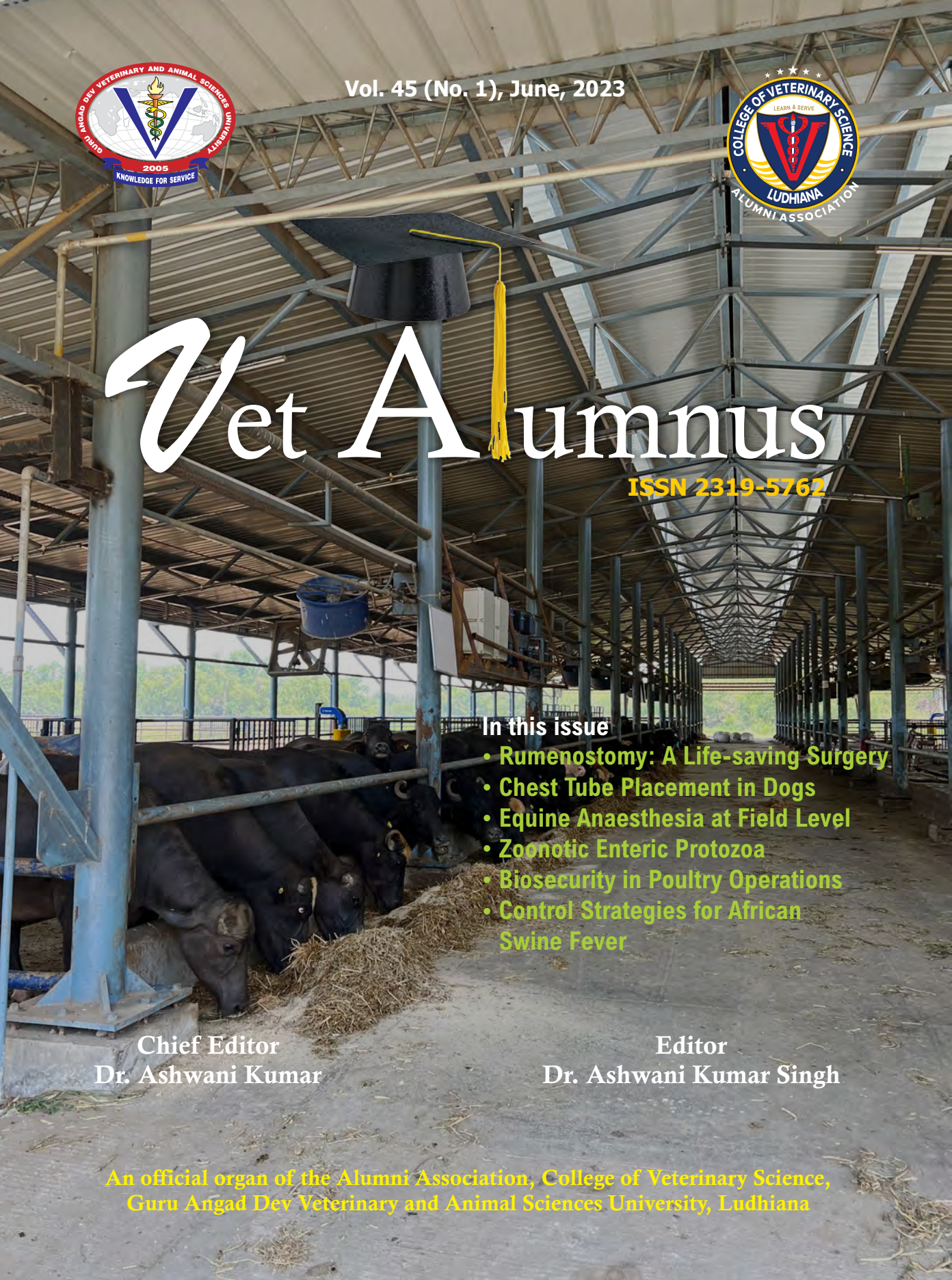
Vol. 45 (No. 1), June, 2023

Vet Alumnus

ISSN 2319-5762



In this issue

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An official organ of the Alumni Association, College of Veterinary Science, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana

Since 2015 all published issues of
the 'Vet Alumnus' are available as OPEN ACCESS (PDF)
on https://www.gadvasu.in/page/vet-alumnus

Printed at Foil Printers, 2051, Gobind Nagar, Civil Lines, Ludhiana-141001, Punjab, India and published on the behalf of the Alumni Association of the College of Veterinary Science, Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana -141004, Punjab, India

VET ALUMNUS

Vol. 45 No. 1 June 2023

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RUMENOSTOMY: A LIFE-SAVING SURGERY

Vandana Sangwan* and Ashwani Kumar

Department of Veterinary Surgery and Radiology, College of Veterinary Science (Ludhiana)
Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India
*Corresponding author email: drvandanasangwan@rediffmail.com

Received: April 2023 Accepted: April 2023

Abstract

Rumensotomy is a technique to make a quick opening in the rumen through left flank. It is a life-saving procedure for the bovine when they are bloated with gas, froth and or fluid in various disease conditions particularly when they become recumbent. This article describes the technique of rumenostomy in bovines which is feasible under field conditions and do not require any specific instruments or equipments.

Keywords*: bloat, bovine, emergency, recumbency, rumen surgery*

Bovine produce 50-60 litres of gas per hour with carbon dioxide and methane as the major components. Eructation is the process which leads to release of these gases into the environment. If due to any problem, which leads to obstruction in the release of gas like; an abscess/lung or liver cyst pressing the reticulo-omasal groove, diaphragmatic hernia involving the reticulo-omasal groove or oesophageal obstruction can cause severe bloat which can be life-threatening. The pressure of the bloat on the diaphragm leads to difficult respiration and if not relieved timely, may lead to death of the bovine (Lozier & Niehaus, 2016). The bovine may become recumbent due to severe bloat or distended rumen. In such condition, it is an emergency to relieve the bovine from bloat or distension and needle might not be sufficient for it.

Rumenotomy is major and time consuming surgery to be performed in a bloated recumbent bovine and if the reason for bloat is not found, may lead to recurrence of bloat once the rumen is closed. However, rumenosotmy is a technique to make an opening in the rumen through the left flank, which can be left open as long as the bovine recovers and may be closed later. Rumenosotmy is a quick and less sophisticated method which require limited instruments and if bovine is serious enough may not even require local anaesthesia **(**Hartnack et al., 2015**)**. It is better than trocharization, if time allows as the leakage of rumen contents in the peritoneum can be avoided. This article describes the technique of rumenostomy in bovine which can be done in filed level.

Indications

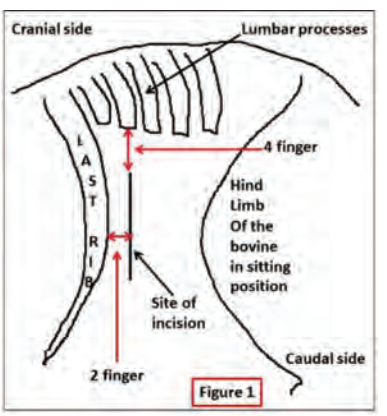
1. Severe bloat
2. Severe rumen distension leading to recumbency

Material Required

1. Local anaesthesia (2% lignocaine hydrochloride, 30-50ml)
2. Syringe 20ml
3. Hypodermic needle 18g
4. Needle holder
5. Cutting needle no. 2 or 3 or 4
6. Silk thread, no. 2
7. BP Blade more than number 20.
8. Fly repellent spray
9. Antibiotics and analgesics

Technique of Rumenosotomy

- Infiltrate 2% lignocaine hydrochloride with an 18 gauze needle at the site of incision, subcutaneously. Do not Infiltrate too deep as due to distension, the muscle thinning occurs and the local anaesthetic may go wasted if infiltered into peritoneal cavity or rumen. Sometimes, in severely bloated bovines, even local anaesthesia is not required.



- The site of incision is the same as for rumenotomy i.e the left flank. The incision is 2 fingers saudal to the last rib and runs parallel to it. The start of incision is almost 4 fingers below the lumbar processes. Make sure, there is no floating rib after the last rib (Figure 1).
- If the severity of the condition allows, the site may be scrubbed 2-3 times with Chlorhexidine (Savlon), otherwise just paint povidone iodine over the skin.
- A bold linear skin incision of about 6 inch is made as per the site described. The incision is extended into the muscles and the parietal peritoneum and one can see the bloated moving rumen inside.
- Start suturing (using silk no. 2) the rumen wall and the skin of one side in a continuous pattern with a knot at one end. The muscles are not included in this suturing. When one side is complete, apply knot at the other end and continue suturing the other side in the similar fashion leaving a small

space in between the two sutures lines in rumen (Figure 2).

- Make sure that the suturing is tight enough and the corners are secured properly so as to avoid leakage of ruminal contents into peritoneal space.
- Incise the rumen wall in between the sutured lines (Figure 2 inset). The gas will come out by itself, while the froth or contents may need to be evacuated manually. Explore the reticulum for any foreign body or abscess and leave the rumen opening as such. Apply fly repellent on the surgical wound (Figure 3). The procedure may be done within 15-20 minutes.
- Move the animal to kaccha floor so that when it tries to stand up, it does not slip.

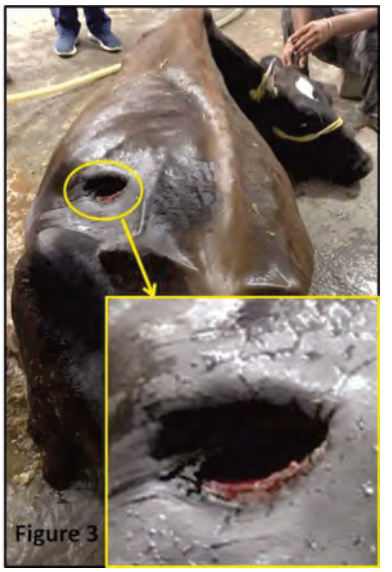


Figure 3

- Inject antibiotics and analgesics and other supportive medication as per the primary condition of the animal. Sutures are to be removed after 14 days.
- Once, everything is normal with the bovine, the rumenostomy wound may be closed. The closure of incision is not an emergency and may be done after 3 days to 3 months as per the condition of the bovine. With time the wound shrinks and the opening become smaller and may close by itself also.

In conclusion, rumenostomy is a simple, quick and lifesaving technique in bloated or distended rumen bovines, which become recumbent. It is feasible under field conditions and should be done early. In delayed cases, the bovine may not stand/recover even after emptying the rumen.

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ANAESTHETIC MANAGEMENT OF HORSES AT FIELD LEVEL

Aswathy Gopinathan and Kiranjeet Singh*

Division of Surgery, ICAR-IVRI, Izatnagar, Bareilly, UP, 243 122

*Corresponding author email: ksuppli@yahoo.co.in

Received: January 2023 Accepted: April 2023

Abstract

Many surgical conditions in horses can be managed at filed level provided there is availability of proper anesthetic agents, expertise and knowhow, facilities and staff support. Here, the anesthetic techniques for producing regional analgesia, sedation and short term surgical procedures are described.

Keywords*: Field anesthesia, horse, nerve blocks, sedation*

Horses respond to anaesthesia with an enormous stress response which increases the morbidity and mortality rate in this species. Yet, anesthetic procedures can be safely conducted to carry out surgical or diagnostic procedures of less than an hour at field level. Still, the risk of anesthesia and surgery on patient morbidity is high when compared to established equine operative care settings. Availability of suitable anesthetics for hypnosis, analgesia and for alleviating stress response to restraint and surgery is to be assured. The type of surgical procedure, site of surgery, age and condition of the patient, availability of supporting personnel for restraint, transport and post operative care are the other important aspects for field level surgery in horses. Preoperative fasting for 12 hour is recommended in short term anesthetic protocols; water is withheld for 6 hours except in nursing foals. Hydration status of the equine should be checked in conditions where possible blood loss is possible like severe lacerations, mandible fracture or in hemorrhage. In such conditions fluid therapy is mandatory since hypovolemic horses can decompensate while in anaesthesia. The environment where the horse is to be restrained for anaesthesia should be free from noise, a clean paddock/pasture or soft bedding should be provided since hard floor can obstruct blood supply to the dependent musculature and pose risk of post anaesthetic myopathy. Always halter head the horse while standing sedation is attempted for better control. Care should be taken to avoid break in the aseptic technique even in minor operative procedures.

Regional Anaesthesia

Lignocaine 2% can be effectively used for local and regional nerve blocks with in recommended dose rates. For epidural analgesia, volume of up-to 5ml can be administered. Because of the low therapeutic index of lidocaine, no more than 10ml of 2% lidocaine is permissible in horses. Local nerve blocks with or without standing

sedation can be employed for ocular examination, removal of parasite from the anterior chamber of eye, castration, perineal laceration repair etc.

1. Nerve block for eye: For routine eye examination and for collection of samples for corneal cytology, auricualopalpebral nerve block (motor nerve) and supraorbital nerve block (frontal nerve, branch of trigeminal n.) can be performed. These blocks will provide immobilization and analgesia for the upper eye lids. 2 ml (2%) lidocaine can be injected into the dorsal rim of the zygomatic arch subcutaneously in a fan shaped manner to block aurculopalpebral nerve. For blocking frontal nerve, 2ml lidocaine can be injected on to the supraorbital foramen palpated over the orbital rim towards the medial canthus of eye. These blocks along with retrobulbar block of the nerves helps to achieve removal of parasite from anterior chamber of eye. For retrobulbar block of eye, upto 10ml of 2% lidocaine can be injected caudal to the dorsal orbital rim and advanced towards the retrobulbar orbital cone and the proper placement of the needle can be assured by the dorsal movement of eye. Slight exophthalmos can be felt when lidocaine is injected slowly into this space and anesthesia ensues by 8-10 minutes and lasts for 1 hr (Labelle & Clark-Price, 2013).
2. Epidural anesthesia: Epidural anesthesia is preferred for standing procedure involving perineum or tail such as tail amputation and caslick operation. Lignocaine 2% solution at the rate of 1ml/100kg body weight is the recommended dose. Lignocaine (0.6 mg/100kg) when combined with xylazine (0.2 mg/kg) is recommended for longer duration of action with lesser chances of recumbancy.
3. Infiltration anaesthesia: Local anesthetic lignocaine 10-15 ml can be directly infiltered into the scrotal tissue before incision and addition 2-3 ml infiltered into the cord before ligation and removal of testicles. Sedation prior to the procedure is required to assure response to visceral traction.

Standing sedation

Alpha 2 agonists can produce sedation for short duration in horses. Xylazine is used at the dose rate of 1 mg / kg body weight IV, it can cause drooping of head within minutes though the animal can kick accurately if prompted to do that. Butorphanol, an opioid in combination with Xylazine at dose rates 0.025mg/kg body weight (IV) increases the quality of sedation. The combination enhances sedation and ataxia which should be taken care of. There are alpha 2 agonists which can produce prolonged sedation like Detomidine 10 microg/kg and Romifidine 50 microg /kg (IV) according to the availability and personal choice (Valverde & Doherty, 2006). The head droop is less with Romifidine than in Xylazine. Addition of Acepromazine at the rate of 0.025 mg/kg IV along with the above said combination of alpha-2 agonist and opioid can produce profound sedation. A

constant rate infusion of Detomidine at the rate of 0.3-0.5 micro/kg/min in saline for 15 minutes followed by 0.15 microg/kg/min for 15-20 minutes can prolong the duration of sedation.

Anaesthesia for 10-20 minutes: A short term anesthesia is useful for procedures like removal of parasite from the anterior chamber of eye; repair of cut/lacerations, pedunculated tumor removal or for a slight detailed examination of organs, or radiographic exposure requiring recumbency. Xylazine @ 1 mg/kg and ketamine @ 2 mg/kg IV can be used to sedate and achieve recumbency for 10-20 min. Inadequate muscle relaxation is a drawback of this combination and may not be suitable to achieve recumbancy in a fractious animal. Addition of Diazepam with ketamine can improve muscle relaxation. Incorporation of Butorphanol (0.2 mg/kg/, IV) can improve analgesia and quality of sedation (Vigani & Garcia-Pereira, 2014). The downtime can be increased to 5- 10 minutes if romifidine (80-100 microg/kg) or detomidine (10-20 microg/kg) is used.

Anaesthesia for 20-30 min: The combination said earlier can be used to achieve recumbency. Xylazine or ketamine top-up with 30-50% of the original dose can achieve recumbancy for an additional 10-15 minutes.

Anaesthesia for 30-60 min: Triple drip technique can provide anaesthesia in field conditions going upto 1 hr. Xylazine-Ketamine combination can be used to induce recumbency. A mixture of Xylazine 500 mg, ketamine 1000 mg in a one litre bag of 5% guaifenesin can be given at the rate of 2.0-2.5 ml/kg/ hr, the infusion rate can be increased if the animal is showing swallowing reflex. Ophisthotonus is a sign of guaifenesin toxicity at which the infusion should be discontinued. Combination of Xylazine and ketamine followed by bolus infusion of thiopentone 0.5-1g can produce adequate muscle relaxation for endotracheal intubation. Thiopentone can be used as maintenance agent at the rate of 0.5 g intermittent boli. Intermingling ketamine boli with thiopentone is also used to achieve general anesthesia in horses. Care should be taken to avoid hypotension.

Sedation in Foals

Young nursing foals should not be fasted prior to anesthesia. Foals older than 4 months can be withheld from food for 4-5 hours. It is not recommended to sedate nursing foals with alpha 2 agonists. Diazepam 0.05-0.1 mg /kg IV along with Butorphanol (0.2 mg/kg/, IV) can produce muscle relaxation to aid recumbancy in foals. Ketamine at the rate of 2-4 mg/kg IV can increase sedation in foals along with Diazepam and Butorphanol.

Monitoring and supportive care

Ensure to alleviate hypovolemia in equines before initiating any anesthetic procedure since most of the anesthetics used are potent hypotensives. Infection should be handled with proper dose of antibiotics. Pain is a major cause of anxiety and violence

in horses, so anti-inflammatory analgesics should be administered in painful conditions well before anesthetic procedures are conducted. Commonly used analgesic/anti-inflammatories are Phenyl butazone (1-2 mg/kg slow IV), Flunixinmeglumine 1mg/kg IV or ketoprofen 2 mg/kg IV. Head and musculature should be well supported by padding to prevent postoperative myopathy or neuropathy. Animal should be anesthetised only when the surgeon is ready for the procedure, which will reduce the duration of surgery and hence anesthesia and reduce complications of longer recumbency. The animal should be well assisted for standing at recovery period since muscle relaxation during the procedure can cause ataxia and excitement. Administration of an alpha 2 agonist at a 30-50% of the recommended dose rate as constant rate infusion during the last 10 minutes of anaesthesia along with fluid therapy can be helpful to reduce excitation during recovery. Even under controlled situations, the anesthesiologist and surgeon should be prepared for adversaries any time during the procedure of equine anesthesia, hence careful patient selection, anesthetic technique appropriate for the procedure and duration, accessories for supportive care and continuous monitoring is essential for field anesthesia of equines.

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COMMON DEVELOPMENTAL ORTHOPEDIC DISEASES IN PUPPIES CAUSING HIND LIMB LAMENESS

Gaurav Chadha* and Arun Anand

Department of Veterinary Surgery and Radiology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab
*Corresponding author email: chadhagaurav96@gmail.com

Received: September 2022 Accepted: April 2023

Abstract

Hind limb lameness is commonly observed in young growing dogs and it may result from congenital abnormalities, developmental orthopaedic diseases, and trauma. Early detection of developmental orthopaedic abnormalities could aid in preventing its progression and their management by conservative or managemental approaches. This article describes diagnosis, clinical signs and management of common developmental musculoskeletal disorders in juvenile dogs.

Keywords*: bone diseases, canine, congenital, joint diseases, juvenile*

Juvenile orthopedic diseases affect the musculoskeletal system of immature animals, and most of these diseases can be traced to pathologic events occurring in this period. Hind limb lameness is common in young dogs and may result from congenital abnormalities, developmental orthopedic diseases, and trauma. In most cases, the lameness can be successfully treated if diagnosed early and appropriate treatment is initiated. Reaching an accurate diagnosis requires consideration of the patient's signalment, good physical examination; complete patient's history, and knowledge of common diseases. The common developmental orthopedic diseases in young, growing dogs causing hindlimb lameness and their management are discussed as under:-

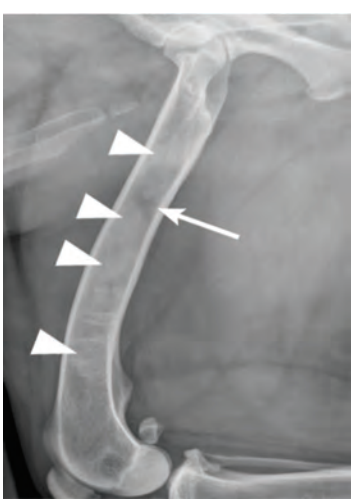

Fig. 1 . Radiograph showing increased opacity in the medullary canal (white arrows)

Panosteitis

Panosteitis is an acquired self-limiting inflammatory condition affecting diaphyseal regions of long bones of dogs from 5 to 18 months of age. German Shephard, Doberman Pinschers, Golden Retrievers, St Bernards, Labrador Retrievers are the commonly affected breeds with panosteitis. The probable etiology is excessively high dietary protein or calcium that causes protein accumulation,

which increases intraosseous pressures and causes pain. An increased osteoblastic and fibroblastic activity affecting the periosteum, resulting in fibrosis of normal medullary cavity (Kieves, 2021).

Diagnosis is based on the presence of hind limb lameness in susceptible breeds and age groups aids in the diagnosis panosteitis, tentatively. Physical examination findings include a shifting leg lameness and pain along the diaphysis on palpation of the long bones. The characteristic radiographic findings include an increased opacity in the medullary canal of the long bones, also described as patchy and mottled radio densities in the medullary cavity (Fig. 1).

Panosteitis is a self-limiting disease. Restricted activity and analgesic medications helps in resolving lameness within 1 to 2 weeks. Intermittent lameness may occur until 18 to 20 months of age.

Legg-Calve-Perthese Disease

This condition is also known avascular or aseptic necrosis of the femoral head. It is characterized as non-inflammatory local ischemia of the femoral head and neck. It is a developmental condition occurring primarily in toy- and miniature-breed dogs in the 4 to 11 month age group (Aguado & Goyenvalle, 2021). Compromised blood supply from epiphyseal vessels may cause ischemic insult. Subsequent synovitis increases intra-articular pressure collapsing fragile veins that further deprives blood flow to the femoral head resulting in necrosis and pathological fracture of the femoral head.

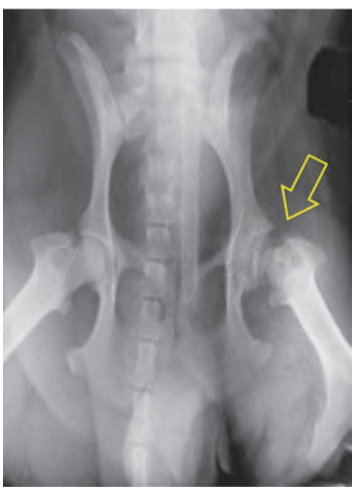

Fig. 2 . Radiograph of Legg Calve Parthes disease (white arrow)

Clinical signs are typically seen with an acute onset of non–weight-bearing lameness. Recent mild trauma acts as inciting cause. Hip pain, crepitus during palpation, and muscle atrophy seen in chronic cases. Radiograph shows irregular bone opacity of the femoral head and neck, collapse, and fragmentation / fracture of the bone (Fig. 2). Chronic cases develop degenerative joint disease.

Conservative therapy consists of rest, limited exercise, appropriate nutrition, and NSAIDs for analgesia. The preferred treatment is femoral head and neck excisional arthroplasty. Surgery alleviates pain and lameness in 84% to 100% of patients regardless of age. After surgery, passive range of-motion exercises and controlled active exercise are encouraged to promote the creation of functional pseudoarthrosis.

Canine Hip Dysplasia

Hip dysplasia is a common skeletal developmental defect in dogs due to genetic predisposition to subluxation of the hip joint. It more commonly affects large-breed dogs but also seen in small-breeds. Joint laxity leads to subluxation and poor congruence between the femoral head and acetabulum. Numerous factors influence the development and progression of HD, including genetics, rapid weight gain in growing animals, a high nutrition level, and pelvic muscle mass (Fries & Remedios, 1995).

Clinical signs vary with the severity of condition and include decreased activity, difficulty in rising, reluctance to run or climb stairs, intermittent lameness, bunny hopping, swaying gait, narrow stance, hip pain, atrophy of the thigh muscles, crepitus, and reduced hip joint motion. Joint laxity (detected clinically by a positive Ortolani sign) is characteristic. The diagnosis is based on physical examination and radiographic findings. Physical examination typically reveals hip pain and a reduced range of motion. Joint subluxation may be observed subjectively on VD hip-extended view of the pelvis (Fig. 3a). The degree of subluxation can be quantified by measuring the Norberg Angle or by calculating the percentage coverage of the femoral head by the acetabulum.

The prognosis for dogs with Hip Dysplasia is variable and it largely depends upon age, body weight and severity of the condition. With proper medical management, many dogs maintain a good quality of life without surgical intervention. In severe cases, femur head and neck excision in indicated. The prognosis after surgical treatment is good (Fig. 3b) with proper patient selection, sound surgical technique, and proper postoperative management. Criteria for selection of various therapeutic regimens for canine hip dysplasia condition have been depicted in Table 1.

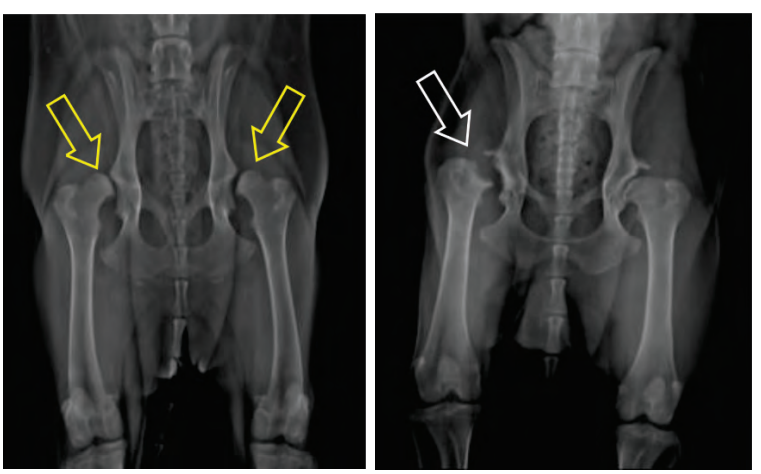

Fig. 3. Radiograph showing bilateral hip dysplasia (yellow arrow) before surgery (a) and after excision arthroplasty surgery (b)

Table 1. Various therapeutic regimens for the management of canine hip dysplasia

Treatment	Patient Age	Criteria	Principles
Medical therapy	Any age and size.	Clinical signs, Response to therapy	Weight loss, physical therapy and exercise control, NSAIDs.
Juvenile Pubic Symphysiodesis	3-4 months of age	Age, No Osteoarthritis, Hip laxity	Reorients the acetabulum to establish congruity between femoral head and acetabulum.
Triple Pelvic Osteotomy	Less than 10 months	No Osteoarthritis, Hip laxity, Clinical signs present	Increases acetabular ventroversion and acetabular angle to improve coverage of femoral head.
Total Hip Arthroplasty	After skeletal maturity (>10-12 months)	Clinical signs, Unresponsive to medical therapy	Implant the prosthetic hip
Femoral Head and Neck Excision	Any age and any size	Clinical signs, Unresponsive to medical therapy	Eliminate hip pain by removing femoral head and neck and initiating development of fibrous pseudoarthrosis

Patellar Luxation

Patellar luxation is usually congenital and developmental condition due to shallow trochlear groove that fails to retain patella while joint motion. It may be caused by abnormal hip conformation leading to displacement of the quadriceps mechanism. Patellar luxations may also occur as a result of traumatic injury to the medial or lateral fascia surrounding the stifle joint. Displacement of the patella from the trochlear groove

Fig. 4 dog with patellar luxation showing skipping gait

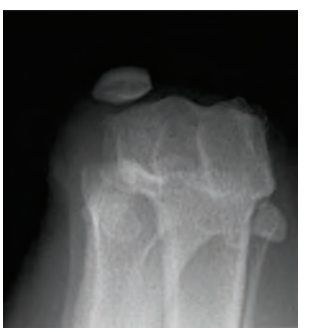

Fig. 5 Skyline radiographic view showing patella located out of the tochlear groove

can cause pain and may predispose the dog to rupture of the Cranial Cruciate Ligament (Harasen, 2006). Medial patellar luxation is a common abnormality in toy, miniature while lateral luxation is more often seen in large-breed dogs. The grading of patellar luxation has been described in Table 2. Initial signs include intermittent lameness and pain. In many cases, the owners describe episodes of "skip" or "hop" for several steps (Fig. 4). Some owners describe feeling a "pop" when they pick the dog up or manipulate the hind legs. In cases of traumatic patellar luxation, the pain and lameness are usually acute and persistent. Diagnosis is made by palpating the stifle joint and evaluation of patellar stability. The displaced patella may also be observed on craniocaudal and Skyline view of the stifle (Fig. 5).

Table 2. Grading of patellar luxation

Grade I	Patella can be manipulated out of its groove but returns to a normal position spontaneously.
Grade II	Patella rides out of the groove occasionally and can be replaced in its groove by manipulation.
Grade III	Patella rides out of its groove most of the time but can be replaced by manipulation.
Grade IV	Patella rides outside the groove all the time and cannot be manipulated into normal position.

Conservative treatment consisting of restricted activity and analgesic medications is often prescribed for dogs with Grade I and II luxation. Surgical treatment such as trochlear recession (deepening the trochlear groove to improve patellar stability), retinacular Imbrication (tightening the fascia surrounding the patella with Lembert sutures or placing sutures from the fabella to the patella) and tibial crest transposition (Realigning/ directing the pull of quadriceps mechanism allowing the patella to remain in the trochlear groove by insertion of the straight patellar tendon) are generally recommended in dogs with more severe patellar luxations (grade III and IV) to prevent recurrence and to help prevent torsional and angular changes in the femur and tibia.

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AN UPDATE ON THE ADVANTAGES OF EARLY NEUTER SURGERY IN DOGS

Harmanpreet Singh Sodhi*, Tarunbir Singh and Ashwani Kumar

Department of Veterinary Surgery and Radiology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India

*Corresponding author email: harmansodhi11@yahoo.com

Received: May 2023 Accepted: May 2023

Abstract

Stray dog over-population is one of the serious problems in India. Lack of awareness on the various advantages of early neuter surgery of pet dogs appears to be a reason for major hindrance in the effective implementation of Animal Birth Control Program. This articles updates the numerous advantages of early neuter surgery in male and female dogs.

Keywords*: Canine, genitalia, mammary tumour, spay surgery*

In order to sterilise male and female dogs as part of an animal birth control plan, neutering is a common procedure. This article will emphasise that neutering prevents them from breeding as well as numerous chronic, incurable diseases. The primary doctor handling cases is a veterinary officer, therefore they are most equipped to advise pet owners to have their dogs neutered as soon as possible for the sake of the animals' long-term health. Some of the benefits of an early elective neutering for pets are covered in the sections below:

Female Dogs

- Mammary cancer – Intact female dogs frequently develop mammary tumours during her life, which account for 25% of all canine tumours. Majority of the mammary tumours are malignant. Early female dog neutering can considerably minimise the incidence of mammary cancer and the risk of cancer significantly rises with each season (Sorenmo et al., 2000). Anaesthesia risk is higher when surgery is performed on an older or sick animal than it is on a young, healthy animal.
- Pyometra - a common and potentially life threatening infection of the uterus requires emergency surgery. It generally occurs in old female dogs and causes them to be very ill associated with severe infection and kidney failure (Hagman, 2022). Spaying eliminates the risk of the female dog developing pyometra.

- Pseudopregnancy: Following heat, many female dogs experience false pregnancies, which can lead to ill health and cause them to become irritable, aggressive, and moody. It is common for a female dog to develop enlargement of the mammary glands that predisposes to mastitis. Besides, there will be behavioural changes such as guarding, aggression and decreased tolerance of other dogs and people. This can be avoided by removing her reproductive organs.
- The neutering of female dogs may have an impact on their behaviour. The animal tends to become friendly, calmer and easier to handle, and interactions with other animals will typically be less unpleasant. In contrast, neutered animals are more predisposed to obesity. The higher propensity for weight gain in neutered dogs can be prevented by dietary management (Vendramini et al*.,* 2020).
- Prevention of 'heat' - Owners of entire female dogs have to be prepared to cope with their pet's heat as well as the issues of mess and hygiene.
- Prevention of unwanted pregnancy - A female dog in heat will be attractive to male dogs. This can cause difficulties when there are male dogs in the house.
- Prevention of genital organ cancer - Spaying removes the ovaries and uterine horns, and therefore prevents ovarian cancer and cysts. The greatest preventative effect occurs when spaying female dog before the first heat. This benefit is less pronounced once the female dog has had two or more heats.

Male Dogs

- Neutering reduces the nuisance of leg-lifting in house on furniture and other items, which is otherwise an act required by wild /stray male dogs done to mark their territory.
- If performed at a young age of 6 months, neutering lowers the animal's dominance and aggression, especially in vicious canine breeds like Rottweilers and Pit bulls.
- By having a male dog neutered, it lower the chance to get attacked by other male dogs while chasing females dogs during breeding season. During this time, dog bites occur most frequently. Another infectious disease that is frequently observed during breeding season is rabies.
- The dog may get infected with transmissible venereal disease, if not neutered, by contacting infected stray dogs.
- The pet becomes more loyal as a result of neutering because its focus to sexual behaviours is less. Additionally, it lessens the male dog's sexual behaviour towards the family members.
- To lower the chance of prostate diseases such tumours, cysts, infections, etc., neutering is crucial. In intact male dogs, enlargement of the prostate is fairly

prevalent from middle to old age. Urinary incontinence, constipation, anal fistulas/wounds, and perineal hernia are additional effects of prostate diseases. Such infections are uncommon in affluent countries where neutering is mandatory. Testicular malignancies are also reduced by neutering.

- The neutering of dogs with retained testis must be done. The non-descent of testicles is known as cryptorchidism. This may be unilateral or bilateral. The retention of these testicles in the abdomen or subcutaneous tissue may cause a variety of hormonal imbalances that result in hair loss, enlargement of the teats, and the formation of a sertoli cell tumour in the retained testicle. It is advised to wait no more than six months for the testicles to descend. Neuter surgery in cryptorchidism may prevent complications associated with this condition.
- A chronic and frustrating disease condition of intact males is anal frunculosis in which multiple fistulas may develop around anus along with inflammation of rectum. Defecation in this condition is excruciatingly uncomfortable. Due to their broad bases and non-lifting tails, German shepherd dogs are especially prone to it. By neutering the dog at the recommended age, the occurrence of this condition can be decreased (Cain, 2019).
- When trying to control the dog population in a nation like India, neutering pet dogs is of the utmost importance as our pet dogs might approach any stray females and mate with them, which would increase the stray dog population. It is responsibility of field veterinary officers to encourage the general public to get their male and female pet dogs neutered.

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COMMON PULMONARY AFFECTIONS IN EQUINES

Asmita Narang* and Swaran Singh Randhawa

Department of Veterinary Medicine, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Ludhiana, Punjab-141004, India

*Corresponding author email: asmi.vet@gmail.com

Received: April 2023 Accepted: April 2023

Abstract

Equine pneumonia is a common respiratory condition which could be life-threatening. High incidence of respiratory diseases in equines can be attributed to climate, close contact between the animals under housed conditions, stressful activities, exposure to dust, irritants, ammonia and respiratory microbes. There are various types of equine pneumonia classified on the basis of etiology. Interstitial pneumonia includes a wide variety of acute and chronic respiratory diseases with infectious, toxic/chemical or allergic causes. Bacterial pneumonia involves both the bronchi and the lung parenchyma. Pleuropneumonia develops secondary to bacterial pneumonia or penetrating thoracic wounds. Rhodococcus equi pneumonia causes pneumonia in foals with 80% morbidity rate in newborn foals. Non-infectious respiratory diseases are commonly due to hypersensitivities to allergens in the stable environment, for example inflammatory airway disease in younger horses and recurrent airway obstruction in older horses. Exercise-induced pulmonary hemorrhage is common in racehorses that require strenuous exercise for short periods of time. Horse's athletic ability and long-term welfare are significantly dependent on minimizing respiratory disease throughout its life.

Keywords*: Equine, interstitial, pneumonia, Rhodococcus*

Respiratory diseases are a serious problem in horses which can lead to high morbidity and mortality. High incidence of respiratory diseases in equines can be attributed to climate, close contact between the animals under housed conditions during winters, stressful activities such as racing and transport, exposure to dust, airborne irritants, bacteria, molds, and ammonia and respiratory viruses (Gerard and Wikins, 2015). Disorders of the upper airways are primarily mechanical in nature while those of the lower airway are often associated with infection. Lower respiratory tract diseases in horses are caused by both infectious and non-infectious agents**.** Most infectious pulmonary diseases are considered to be initiated by viral infections. Non-infectious respiratory diseases are commonly due to hypersensitivities to allergens in the stable environment. Common pulmonary affections in horses are discussed below.

Pneumonia

Equine pneumonia is a common respiratory condition which could be life-threatening, especially in young horses. Viral respiratory infection, general anesthesia, long-distance transportation, and strenuous exercise are common predisposing factors impairing pulmonary defense mechanisms and leading to secondary bacterial pneumonia. Head restraint results in bacterial contamination and multiplication within the lower respiratory tract within 12–24 hr and may be the single most important predisposing factor for development of pneumonia associated with long-distance transport. Equine pneumonia, irrespective of the etiology, requires early and aggressive treatment for a successful outcome.

Interstitial pneumonia

It encompasses a wide variety of acute and chronic respiratory diseases in horses of all ages. Interstitial pneumonia is accompanied by severe respiratory disease and often poor clinical outcome (Wilkins, 2003). The potential causes can be infectious, toxic/chemical or allergic. In humans, the etiology of corresponding interstitial lung disease is immune mediated. Similar antigenic stimulation by various causative agents like bacteria, viruses, fungi, parasites etc. and factors like smoke, silicosis, plant toxins, adverse drug reactions etc. might occur in horses with interstitial pneumonia. Radiographic evaluation of lungs usually shows nodular interstitial pattern in caudal lung lobe in interstitial pneumonia. Transtracheal wash cytology usually reveals moderate neutrophilia with degenerative changes is diagnostic for interstitial pneumonia. Presence of langhans gaint cells in Broncho-alveolar lavage fluid may indicate the chronic interstitial pneumonia in horses. Treatment is often unrewarding in these cases with slight remission of signs.

Bacterial pneumonia

Bacterial infections of the lungs are frequent in adult horses. These involve both the bronchi and the lung parenchyma. Bacterial infections generally occur following the events suppressing the pulmonary immunity. Pulmonary defense mechanisms are altered by stress, poor nutrition, respiratory viral infections, exposure to dust or noxious gases, immunodeficiency conditions, immunosuppressive therapy, and general anesthesia. Aspiration of microorganisms normally inhabiting the nasopharynx or oral cavity in horses usually leads to bacterial pneumonia. The β-Hemolytic streptococci is one of the commonest bacteria isolated from adult horses with pneumonia. *Streptococcus equi* subsp. *zooepidemicus* is an opportunistic pathogen, which may cause mild to severe, suppurative, bronchopneumonia and pleuropneumonia. Other bacteria such as *Staphylococcus spp*., *Klebsiella* and *Escherichia coli* have also been isolated from horses with pneumonia. Common clinical manifestations include dyspnea, hemoptysis, fever and chronic cough. The lungs, especially the cranioventral areas, show diffuse pulmonary consolidation.

Transtracheal wash cytology showing numerous neutrophils with degenerative changes along with apparent pneumonic changes on ultrasonography is strongly indicative of bacterial pneumonia. The prognosis depends on severity, early diagnosis and proper therapeutic measures.

Pleuropneumonia

It develops secondary to bacterial pneumonia or penetrating thoracic wounds in most of the cases. Common predisposing factors impairing pulmonary defense mechanisms include viral respiratory infections, general anesthesia, strenuous exercise, and long-distance transportation. These factors consequently allow secondary bacterial infections. Polymicrobial and mixed anaerobic-aerobic infections are common in horses with pleuropneumonia, with more than one bacteria isolated from transtracheal aspirates (Couetil et al., 2016). The clinical manifestations of bacterial or fungal bronchopneumonia and pleuropneumonia are fever, decreased appetite, weight loss and depression. Blood picture shows leukocytosis with neutrophilia and increased immunoglobulins (Couetil et al., 2016). Specific signs of pleuropneumonia are pleural pain evident as short strides, guarding, and flinching on percussion of chest, shallow respiration and endotoxemia. In anaerobic bacterial infection and necrotic pulmonary tissue, there would be putrid breath or fetid nasal discharge. Laboratory findings are suggestive of bacterial sepsis or toxemia. Confirmatory diagnosis is done by thoracic ultrasonography. Thoracocentesis is performed for diagnostic and therapeutic purposes. Gross examination of pleural fluid includes evaluation of color, odor, volume and turbidity. Gram stain examination and bacterial culture and sensitivity tests are used to select antimicrobial for better clinical response. Medical therapy includes broad-spectrum antibiotics, NSAIDs, analgesics, and supportive care. Intrathoracic fibrinolytic therapy can reduce fibrin deposition and pleural fluid accumulation.

***Rhodococcus equi* Pneumonia**

Rhodococcus equi is one of the most important causes of pneumonia in foals between 1 and 6 months of age. Disease occurs in sporadic form. Morbidity rate reaches 80% in newborn foals (Sangwan et al., 2013). The most common and important route of infection in foals is inhalation of contaminated dust particles (Leclere et al., 2011). The high pathogenicity of the bacteria is due to its ability to survive intracellularly. The disease is manifested as chronic suppurative bronchopneumonia with extensive abscessation and associated suppurative lymphadenitis. Clinical signs include lethargy, fever and tachypnoea. Colonic microabscessation by the bacteria may lead to diarrhea in one-third of foals. Common laboratory findings include neutrophilic leukocytosis and hyperfibrinogenemia. Perihilar alveolization, consolidation and abscessation is apparent on thoracic radiography. The presence of nodular lung lesions and mediastinal lymphadenopathy in foals of 1–4 months age is strongly suggestive of *R. equi* infection.

For confirmatory diagnosis, transtracheal wash cytology and culture isolation of *R. equi* are used. The treatment of choice is erythromycin and rifampin for 3–8 wks. Supportive therapy includes provision of a clean, comfortable environment and highly palatable, dust-free feeds. Judicial intravenous fluid therapy and saline nebulization facilitates expectoration of pulmonary exudates.

Exercise-induced Pulmonary Hemorrhage (EIPH)

The EIPH is common in racehorses that require strenuous exercise for short periods of time. Common signs observed are respiratory distress and an increased rate of swallowing after exercise. There is evidence of epistaxis in a small proportion (1-10 %) of horses with EIPH. Definitive evidence of EIPH is provided by endoscopic (Tracheoscopy) observation of blood in the airways 30–90 min after exercise. Detection of hemosiderin in alveolar macrophages in Broncho Alveolor Lavage (BAL) fluid cytology also aids in diagnosis. Hemorrhages occur in caudo-dorsal lung lobes, which is associated with macrophagic bronchiolitis and fibrosis. Thoracic radiography demonstrates alveolar or mixed alveolar-interstitial opacities in the caudodorsal lung fields. Furosemide and application of nasal dilator bands can reduce the incidence and severity of EIPH in Thoroughbred racehorses.

Inflammatory Airway Disease (IAD)

The IAD includes a group of primarily non-infectious inflammatory conditions of the lower respiratory tract. Racehorses and non-racehorses of all ages and any breed are susceptible. Allergic airway disease, dust inhalation, pollutants, recurrent pulmonary stress and respiratory viral infections (notably EHV-2) are possible etiologies. Chronic cough and mucopurulent nasal discharge are the common clinical signs. Diagnosis of IAD is based on poor race performance and clinical signs. Endoscopic examination shows mucopurulent exudate in the pharynx, trachea, and bronchi. BAL is performed to characterize the type of pulmonary inflammation. Cytologic evaluation of bronchoalveolar fluid shows mixed inflammation with high total nucleated cells.

For the management of IAD, aerosolized bronchodilator therapy before exercise can reduce the chances or severity of exercise or irritant induced bronchoconstriction in affected horses. Aerosol administration of an inhaled corticosteroid preparation (beclomethasone or fluticasone) and systemic corticosteroid therapy can improve clinical signs of respiratory disease.

Chronic Obstructive Pulmonary Disease (COPD) / Heaves

COPD is a common allergic respiratory disease of horses. There is no breed or gender predilection. Clinical signs are manifested when affected horses are stabled, provided with straw bedding, and on feeding hay. Molds and endotoxin associated with bedding and feedstuffs play a role in the etiopathogenesis of COPD. In response to allergen

exposure, there is small-airway inflammation, mucus production, and bronchoconstriction. Clinical manifestations include flared nostrils, tachypnea, cough and a heave line. Hypertrophy of abdominal muscles muscles to assist expiration produces the classic heave line. The diagnosis is based on history and characteristic physical examination findings. Chest radiography shows peribronchial infiltration and overexpanded pulmonary fields. Non septic inflammation with increase in non-degenerative neutrophils in BAL fluid cytology is also indicative of COPD. There is lot of mucus and hyperplastic cells with more of goblet cells. Environmental management to reduce allergen exposure is the most important treatment for horses with COPD. Horses should be maintained at pasture, with fresh grass as the source of roughage, supplemented with pelleted feed. Soaking hay with water before feeding may control clinical signs in mildly affected horses. Straw bedding should be avoided. A combination of bronchodilators and corticosteroids can be used for medical management (Bertin et al., 2011).

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REPRODUCTIVE MANAGEMENT OF GOATS: AN OVERVIEW

Amit Kumar* and Mrigank Honparkhe

Department of Veterinary Gynecology and Obstetrics, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab 141004, India

*Corresponding author email: amitkajal7@gmail.com

Received: March 2023 Accepted: April 2023

Abstract

Though reproductive efficiency is important in dairy goat herds, and selection of highly fertile buck goats is absolutely essential. Still many problems exist that may cause infertility in bucks and goats. Consequently, present article discusses reproductive management strategies to further enhance caprine productivity.

Keywords*: breeding, gestation, small ruminant,*

Caprines have been associated with man since domestication of animals, making them socio-economically very important animal providing products (meat, milk, fibre, hair) and service to man throughout the world, especially in developing countries. In India, as per 20th livestock census goat population is around 148.88 Million (20th Livestock Census, 2019) and globally India stands first in terms of goat population (Basic Animal Husbandry Statistics, 2021-22). Goats play an essential role in the nutrition and economy of small farmers in our country. Furthermore, Punjab has a total of 3.27 lakh goat population, which will increase by 5.93% in the next five years (20th Livestock Census, 2019). The profitability of goat and sheep operations is largely reliant on successful reproduction programs. Therefore, reproductive management should be a vital component of the overall herd management scheme and closely integrated with nutritional and health management.

Normal Reproductive Physiology

Goats are generally seasonal breeders, with the onset resulting from decreased daylight. Most breeding occurs in the late summer through early winter (Sept to Oct, Feb to march and May to June). However, it is possible to alter the breeding season by artificial lighting and ram/buck effect (i.e. sudden introduction of a ram/buck into a herd which have been kept segregated from male animal for 2-3 months). The gestation period is 5 months, and twins are common, though single and triplet births are not rare. Kids may enter into the breeding colony at 5 to 10 months of age, depending on breed. General life span of goat is about 12 -13 years, but in normal husbandry practices, female goats are retained only up to 6-8 years of age, whereas males are retained until 4-6 years to get efficient production. The most viable offspring are produced between 2-5 years of age.

Buckling's are generally fertile by 5 months of age or earlier. In some breeds, puberty is reached at a young age; it is therefore important to separate goat and buck at an early age.

Breeding Management

Buck management: Bucks, because of their size, odor and sometimes temperament, often require special management considerations and experience in handling.

- Bucks used for breeding should have characteristics that will advance the production potential of the herd and should be able to mate successfully to transmit these characteristics.
- Spermatogenesis is susceptible to outside influences such as elevated temperature, season of year and nutrition, and breeding males need to be evaluated for reproductive soundness 3-4 weeks prior to mating season.
- Part of such a 'breeding soundness examination' is an evaluation of the overall condition of the buck and includes his health history, physical soundness, particularly of feet and legs, and examination of the external genitalia for signs of infections and other abnormalities.
- The testis should be of adequate size (20-25 cm scrotal circumference) and tone (firm).
- In case the assistance of a veterinarian or trained personnel is available the breeding soundness examination should also involve the collection and evaluation of an ejaculate for the percentage of motile, normal and live sperm.
- If goats in estrus are available, bucks should be placed with these goats to evaluate libido and mating behavior.
- Bucks deficient in any part of the examination should be considered questionable, and retested after several weeks, a second failed test would indicate reproductive deficiencies and such a buck should not be used for mating.
- The number of goats a buck can breed will depend on a variety of factors, including age of the buck, terrain and pre-breeding management (i.e. synchronization of estrus).
- A mature buck under pasture conditions should be able to breed 30 to 50 goats.
- For yearling bucks this ratio is markedly lower, and dependent on the physical development of the buck.
- Young males can be put in to experienced older does and older bucks to younger does will help in better mating.
- A buck may breed only 5-15 goats if goats are estrus synchronized (hormone treatments that causes all goats to be in estrus at the same time) to ensure adequate

fertility.

- An alternative approach to breeding synchronized goats is the use of 'hand-mating' where access to the goat is restricted to one or two controlled mating's 12 hours apart.
- A useful management tool for breeding is the use of a marking harness, a device which holds a marking crayon on the buck's chest which colors the back of a goat when she is mounted.
- Harness marks will provide an instant confirmation of the breeding activity of a buck, establish mating dates (and subsequently projected kidding dates).
- The color of crayon should be changed after 15-20 days of breeding to identify goats that are rebred, as indicated by marking with the new color.
- A large number of rebred goats would suggest that a buck has fertility problems and should be replaced.
- In multiple sire mating groups alternative color crayons will provide an indication of mating activity of individual bucks.
- The males should be replaced or exchanged once in two years to avoid inbreeding.

Goat Management

- Breeding in indigenous breeds should be at 10 to 12 months of age depending upon their body condition, when they reach 70 per cent of the average adult body weight.
- Breeding too young does result in more weakling and thus results in higher lamb loss.
- Maintain good body condition in goats throughout the breeding season.
- Flushing is feeding of extra concentrate (250 g of concentrate daily or 500 g of good quality legume hay per head per day) to goats normally 3 or 4 weeks before breeding.
- Flushing increases the onset of estrus, ovulation rate and decreases the early embryonic mortality by strengthening the fetal membrane integrity.
- Estrus detection should take place at least twice daily, separated by 12 hours, but should be avoided at feeding or milking, as signs may not be as obvious.
- Heat detection can be achieved using a teaser buck, the herd sire, or by the producer.
- Teasers are particularly attractive, as they can induce heat when introduced to females.

- Signs of a goat in heat include the following: paying attention to or seeking out the buck, decreased appetite, increased restless behavior, frequent urination, increased vocalization, mounting and/or allowing mounting with other females, a swollen vulva with the presence of mucus (clear during early estrus, thicker and opaque later is estrus)
- In order to synchronize them improved hormonal technology may be used or buck may be in a partitioned corral of woven-wire net so that the goats and the buck may have full view of each other. This may be done a week or two before or during the breeding season.
- A 90% conception rate in goats may be ensured if one buck with one goat or more goats (not exceeding 2 to 3) in heat are allowed to remain together for a whole day or whole night provided it is followed over period of 3 cycles.
- For better conception 2 services at an interval of 8 to 12 hours should be done.
- If they have no kidding for complete one year they should be removed from flock.

Management during Gestation and at the Time of Parturition

- Goats which do not return to estrus after 2 cycles are considered as pregnant and should be separated from the dry, non-pregnant flock.
- They should be kept in a group of not more than 15 to 20 goats to avoid infighting.
- Pregnancy diagnosis should be done to ensure the breeding season is not missed, and the use of an ultrasound allows pregnancy diagnosis 32 days post-breeding.
- Embryonic losses early in pregnancy are usually much higher than fetal losses at later stages of gestation, and can be as high a 20-30%, due to the complexity of events associated with fertilization and implantation.
- Embryo mortality is also influenced by extrinsic factors such goat age, and environmental and nutritional stress.
- Undernutrition, vitamin and mineral deficiencies, toxic plants, and certain drugs (i.e levamisole) can contribute to non-infectious abortions.
- Multiple late abortions (‘abortion storm’) usually suggest an infectious cause for the abortion, of which chlamydiosis and toxoplasmosis are the most common source.
- To properly diagnose the cause of an infectious abortion, the fetus, portions of the placenta and a blood sample from the goat should be collected for testing.
- Eight to 12 hours prior to birth, signs including udder development and loosening around the vulva will be observable.

- The goat will also lie down and stand up multiple times. Females should be given a clean, dry place to birth at this time.
- Unless labor is prolonged, females should be largely left alone during parturition.
- Generally, parturition should be completed within 2 hours following the appearance of the water sac.
- If the goat appears to be having difficulty, and if the second stage of labor exceeds two hours call veterinarian for assistance.
- In case of failure of the cervix to dilate properly (ringwomb), cautious manual stretching can be applied.
- Continued failure to dilate may require a cesarean section to deliver the kids.
- Newborn kids should be nursing within a couple hours of birth to ensure that proper amounts of colostrum's are to be consumed.
- The navel of the new born kid should be dipped in iodine.

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CURRENT INSIGHTS IN DIAGNOSIS, CONTROL STRATEGIES AND PREPAREDNESS FOR FUTURE OUTBREAK OF AFRICAN SWINE FEVER

Mohanapriya Thangaraj*, Abhilash Jadhao and Vishal Mahajan

Department of Veterinary Pathology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India

*Corresponding author E-mail Id: priya.vet21@gmail.com

Received: April 2023 Accepted: April 2023

Abstract

African swine fever (ASF) is one of the most contagious, emerging and deadly infectious disease in pigs. ASF virus is an unusual complex, only double stranded DNA virus, it extremely well adapted to replicate in its host in the sylvatic cycle. The disease spread very rapidly due to bite of ticks, improper hygiene and socioeconomic practices of animal handlers and movement of animals into the wild life range. It causes 100% mortality and non-availability of vaccines, which is a greatest concern to the pig industry around the globe. By considering all this facts, this paper is delineated to have a proper approach in effective diagnosis, control and preparedness for the African swine fever in our country.

Key words: *African swine fever, Control, Diagnosis, Emerging disease, Mortality, Pig industry*

African swine fever is a highly contagious, fatal haemorrhagic disease found in domestic and wild pigs. It was first recorded in Kenya in 1921 and it is a world organization of animal health (OIE) notifiable disease. ASF is caused by large, double stranded DNA virus in the Asfarviridae family, genus Asfivirus. This family virus replicates in the cell cytoplasm of macrophages and monocytes, which is the main target cell for ASFV replication. The ASF virus also infects the soft ticks of the genus Ornithodoros spp, which act as natural reservoirs (Jo & Gortazar, 2021). ASFV transmission occurs in multiple pathways like importantly tick to pig, wild boar to pig, fomites to pig and pig to pig mainly by consumption of contaminated food products commonly known as swill feeding, or other fomites such as vehicles, workers, and other equipment. The duration and extent of exposure to the infected host in the field, the interface between the wild boars and domestic pigs will have a major role in determining transmission from wild boars to domestic pigs (Guinat et al., 2016). This disease is characterized by high fever, haemorrhages in skin and internal organs and death within 2-10 days. ASF occurs mostly in wild boar. Mortality rates may be as high as 100% in domestic pigs. ASF will not cause threat to other livestock.

Clinical presentation and lesions

The pig develops high temperature (105ºF), becomes lethargy, listlessness, anorexia, cyanosis, increased respiratory rate, leucopenia and thrombocytosis. The other clinical signs includes vomiting, diarrhea, reddening or darkening of skin, particularly near the ears, snout and ventral abdomen, epiphoretic sticky eyes, labored respiration, cough, unwillingness to stand, still birth, mummification and abortion. The piglets that were born alive to the infected dam had neurological signs, including shivering, trembling, and ataxia, which are signs of congenital tremors. The incubation period is between 5-21 days, when bitten by ticks it can be less than 5 days. The clinical symptoms and gross lesions of ASF look very much similar to those of classical swine fever and it may occur as peracute, acute, sub-acute and chronic forms (Yoo et al., 2020).

1) **Peracute form:** Pigs die without any clinical signs and gross lesions within four days.
2) **Acute form:** Severe skin erythema/ general reddening, vasculitis, pulmonary edema, congested splenomegaly, hemorrhagic lymphadenitis and petechial hemorrhages in the lungs, urinary bladder and kidneys. The death of infected pigs show mortality rate of 90%-100% within the 4-21days.
3) **Sub-acute form:** In general, less remarkable clinical signs with mild vascular changes and edema in all organs. The mortality ranges between 30%-70% with incubation period of 20 days of post infection.
4) **Chronic form:** Absence of vascular lesions and low mortality rate due to low virulence isolates in chronic forms. It showed delayed growth, emaciation, joint swelling, skin ulcers and lesions associated with secondary bacterial infections.

Sampling in the Outbreak Area

For the accurate diagnosis of the ASF infection a thorough investigation of farm and its environment, feed and the utensils used, live and dead pig transport vehicles are sampled in viral transport medium. The whole blood and serum is the appropriate sample in live animal, and tissues like lymph nodes, spleen, liver, lung, heart, kidney, brain, urinary bladder, intestine, diaphragm, bone marrow, synovial membranes and meat-juice (Kosowska et al., 2021).

The sampling should be done as per Gebhardt *et al.* (2022) in different zone wise. **Zone 1** was defined as surfaces with direct contact with suspected source of contamination (feed, live animal, etc.). **Zone 2** was defined as a surface with close proximity (<1 m) or surface with high potential for contamination. **Zone 3** was defined as a surface not in close proximity (>1 m) or with potential to become contaminated through fomite transfer. **Zone 4** was defined as a fixed location in non-production airspace with potential contamination via fomites or non-fixed surfaces with intermittent contact with possibly

contaminated surfaces (brooms, employee footwear, etc.).

Diagnostic Methods

Rapid and reliable diagnosis of the disease is critical for preventing the spread of the disease. ASF infection cannot be detected based on the clinical signs and gross lesions because of the similarities classical swine fever, highly pathogenic porcine productive respiratory syndrome and salmonellosis. The collected samples might be subjected to different efficient laboratory diagnostic techniques. The OIE recommended a gel-based PCR assay and the real-time PCR method for ASF diagnosis based on the VP72 Viral DNA sequence detection, using the primers (forward 5′-CCC AGG RGA TAA AAT GAC TG-3′; reverse 5′-CAC TRG TTC CCT CCA CCG ATA-3′) and (5′-FAM-GGC CAG GA-TAMRA-3′) (Nguyen et al., 2022).

The ELISA or Direct immunofluorescence test and immunoperoxidase monolayer assay is a quick test in which many samples can be investigated at the same time. The virus isolation for the blood or organs, required for further characterization of a viral strain requires specific skills and a highly-equipped laboratory and the cytopathic effect. ASFV has usually hemadsorbing capacities and is attached to erythrocytes inoculated with 2×10^5 hemadsorbing units 50% of ASFV. Finally from the necropsy collected and the formalin-fixed tissue sections stained with routine hematoxylin and eosin stain, microscopically, reveal the swelling and hemorrhage in intestines, mandibular and inguinal lymph nodes and kidney. Further congestion of the lungs, meninges and hydranencephaly was also observed. Further the tissues may be subjected to immunohistochemistry, using a polyclonal antibody against p30 (Pikalo et al., 2021).

Control Strategies and Preparedness for Future Disease Outbreak

First and foremost constraint is, non availability of commercial vaccine due to the sturdiness of the virus or effective treatment available to protect against the disease signifying that the principal tool for disease control is preventive measures. The spread of ASF can be prevented only by early detection and the strict application of classical disease control methods, including surveillance, epidemiological investigation, tracing of pigs, stamping out in infected stocks, quarantine, and man - animal movement control (Yoo *et* al., 2020).

In this current situation, effective bio-security measures are highly required for the proper containment of the diseases. The area covered under 1 km radius is declared as infected zone. An area of 10 km from infected premises is considered as surveillance zone and beyond that is disease free zone, but random sampling has to conduct weekly. The following guidelines are adopted to prevent the introduction of ASF in the pig farm or to create disease free zone (Kim et al., 2021).

1. Isolation of healthy animals from infected animals and contaminated materials.
2. Proper quarantine for 30-45 days of newly introduced animals into the farm
3. Proper double fencing of the pig farm to prevent the entry of wild boars and other wild animals.
4. Disinfection of vehicles, wheels, visitor's shoes/boots with 2% sodium hyochlorite or 3:1000 $KMnO_4$ solutions.
5. Foot dips (2% NaOH, 1% HCHO, 1% bleaching powder or lime/ 1% Calcium hydroxide) at the entrance of the farm and also the exit of individual pig pen/ sheds.
6. Farm workers would properly sanitized with detergent soap and maintain proper hygiene always during the whole process of handling the animals.
7. Farm workers should wear specific dresses and gumboots before entry to till completion of work.
8. Separate the infected and healthy animals immediately when it's showing symptoms or on suspicion.
9. Pigs should be purchased from authenticated / authorized farms.
10. No diseased animals should be sold to people or any traders.
11. Swill feeding (kitchen waste, hotel waste, airport/seaport waste or meat meal) should be prohibited and if practiced, it should be boiled properly (>30 min) before feeding.
12. Movement of animals from one farm to another farm or from one village to another village or any animal fairs should be strictly prohibited.
13. Farm utensils should not be shared between all the animals.
14. Natural breeding should be restricted, instead advised to follow artificial insemination.
15. Regular spraying of acaricides to control the vector (tick) is must.
16. Dead pigs, biological fluids and other tissues should be disposed appropriately and deeply buried upto 6 feet along with lime powder.
17. Proper record should be maintained for entry and exit of visitors and vehicles.
18. Any suspected cases or animals showing clinical symptoms should be reported to Veterinary officers.
19. Complete ban on movement of live and slaughtered pigs in infected zones.
20. Harvesting of bedding materials from the livelihood of wild boars should be prohibited.

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RECENT ADVANCES IN THE DIAGNOSIS OF ZOONOTIC ENTERIC PROTOZOA IN LIVESTOCK

Alveena Ganai*, Nirbhay Kumar, Jyoti and Harkirat Singh

Department of Veterinary Parasitology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India.

*Corresponding author Email: dralveenaganai@gmail.com

Received: April 2023 Accepted: May 2023

Abstract

Zoonotic enteric protozoa infection causes severe morbidity and mortality in various livestock and humans worldwide. Most commonly seen enteric protozoans in the livestock are: Entamoeba histolytica, Giardia spp., Cryptosporidium spp. and Balantidium coli which are of zoonotic significance. These parasites are significant causes of diarrhea, weight loss, low milk production, decrease in body weight gain and nutritional disorders in institutional and community settings, affecting growth and cognitive functions of infected individuals and may cause production losses in livestock. Therefore, these protozoans contribute to the high burden and prevalence of infectious diseases in various livestockmainly due to lack of proper laboratory diagnostic techniques via direct, conventional, immunological and molecular diagnostic techniques which are commended in order to detect them in clinical and environmental specimens.

Keywords: *Balantidium coli, Cryptosporidiosis, Diagnosis, Entamoebiasis, Giardiasis, Zoonotic.*

Gastro intestinal protozoan infections are one of the major constraints for profitable dairy industry in India. They cause considerable global economic losses as a consequence of reduced weight gain, digestive disturbance, lowered production, impaired reproductive performance, condemnation of affected organs and mortality in infected animals. Transmission of these parasitic infections is typically associated with poor faecal-oral hygiene (Gupta et al., 2012). These protozoans are usually included in the WHO Neglected Diseases Initiative because they impair the ability to achieve full potential, development and socio-economic improvements, and they have a common link with poverty (Savioli et al., 2006). This article describes about the most common pathogenic gastrointestinal protozoa like: *Balantidium coli*, *Cryptosporidium* spp., *Entamoeba histolytica* and *Giardia* spp. in the livestock which are of zoonotic significance with latest conventional, immunological and molecular diagnostic techniques.

Entamoeba

It is caused by *Entamoeba histolytica* which is an anaerobic parasite. It is predominantly found in primates (including humans) and occasionally in dogs, cats, cattle and pigs. The parasite has a worldwide distribution mostly prevalent in tropical and subtropical countries.

Transmission and pathogenesis: Transmission is through faecal-oral route via ingestion of food and water contaminated with amoebic cysts. *Entamoeba histolytica* exists in 2 forms: Trophozoite and the cyst form. The motile trophozoites predominate in the faeces of ruminants but rapidly invade outside when excreted in the environment and if accidentally ingested, it would be destroyed by gastric acids. Some trophozoites in the colonic lumen become cysts that are excreted in faeces. *E. histolytica* trophozoites can adhere and kill colonic epithelial cells and polymorphonuclear leukocytes (PMNs) and can cause dysentery with blood and mucus but with few PMNs in faeces. Whereas the mature cyst which is another infective stage predominate in faeces of host and resist destruction in the external environment. They may spread directly from one host to another host or indirectly via food or water (Bruckner et al., 1992). Common symptoms include diarrhea, bloody stools, weight loss, and abdominal pain in the infected host.

Diagnostic techniques: The microscopic diagnosis of the *Entamoeba histolytica* is done in the faeces, body fluid or tissue samples of infected host. Tentative diagnosis is made from the clinical signs that include gastrointestinal discomfort and watery or bloody diarrhea. Microscopic examination of permanently stained smears (trichrome or iron hematoxylin) is an important method. There are a wide number of commercially available antigen detection kits using enzyme-linked immunosorbent assay (ELISA), radioimmunoassay, or immunofluorescence that have varying sensitivities of up to 88% (Saidin et al., 2019).

Giardiasis

It is very unusual, seemingly ancient, eukaryotic single cell organism and shares its characteristics with anaerobic prokaryotes. Most common species of *Giardia* is *G. duodenlalis* (syn. *intestinalis/lamblia*) infecting a wide range of mammals, including humans, livestock, and companion animals with the greatest public and animal health significance. It affects a wide range of livestock like dog, cat, cattle, buffalo, llama, mice, beavers and other domestic animals. Humans are infected by A and B genotypic assemblages of giardiosis (Feng and Xiao, 2011).

Transmission and Pathogenesis: Transmission of giardiasis is typically associated with poor faecal-oral (by contaminated food and water) route. Trophozoites when ingested by the host, it inhibits the small intestine using the ventral adhesive disc to attach to the wall of the small intestine which blocks absorption of nutrients and including fats. The trophozoites usually encyst before they leave the jejunum. Encystation into new cysts (10µm) takes place in the lower part of the small intestine, which again releases four trophozoites from one cyst and multiplies by longitudinal binary fission. Thus, both cyst and trophozoites are released in the environment which is very infectious immediately when passed in faeces.

Clinical manifestations of giardiasis usually appear 1 to 2 weeks after infection and causes acute or chronic diarrhea, abdominal pain, and vomiting, irritable bowel syndrome and weight loss. It causes villous atrophy, diffuse shortening of microvilli, reduce disaccharidase activity, loss of epithelial barrier function, increase permeability and enterocyte apoptosis. Mostly results into disruption of the intestinal barrier function, activation of host lymphocytes,CD8+ lymphocyte-mediated shortening of brush border microvilli with or without coinciding villous atrophy, disaccharidase deficiencies, small intestinal malabsorption, anion hypersecretion, and increased intestinal transit rates. It is also associated with increased mucus secretion and may cause mucin depletion in goblet cells of the small intestine and results into poor cognitive function caused by zinc and iron micronutrient deficiencies.

Diagnostic Techniques: Direct fecal smear via iodine staining method results into 97% sensitivity. Other conventional technique like formol-ether concentration technique and the zinc sulfate flotation methods are also equally sensitive. The immunodiagnostic techniques like: Direct immunofluorescence microscopy of faeces (fluorescentantibody [FAB] coproscopy), using fluorescent antibodies directed against *Giardia* spp. provides greater sensitivity and specificity than flotation methods. Also, on the basis of molecular diagnostic technique, PCR based assays (nested PCR, PCR-RFLP, RT-PCR etc.) have excellent sensitivity and specificity by amplification of β-giardin gene. RT-PCR is one the most powerful method and it has the following advantages: targeting the small specific gene regions of the parasite, rapid cycling time (approximately 1 h), low contamination risk, and ability to measure the DNA amount during the assay without post-PCR analysis (El-Nahas et al., 2013).

Cryptosporidiosis

It is a zoonotic apicomplexan protozoa affecting all terrestrial including wide range of livestock caused by 26 validated species of the genus *Cryptosporidium*. Among them the most important species infecting bovines are *Cryptosporidium parvum, C. andersoni, C. bovis* and *C. ryanae* (formerly known as deer-like genotype).*Cryptosporidium parvum* is considered the most common enteropathogen of neonatal calves. Cryptosporidiosis is recognized worldwide, primarily in neonatal calves but also in lambs, kids, foals, and piglets. Humans are affected by the most common IIa and IId subgenotype of *C. parvum* species of *Cryptosporidium* genus.

Transmission and Pathogensis: It is usually transmitted directly from ingestion of the oocyst from contaminated food or water or either from one host to another or through the environment via faecal oral route. Excystation of oocyst occurs after ingestion by a host, with release of four motile sporozoites which invade and parasitize epithelial cells primarily of gastrointestinal tract. Subsequent developmental stages are intracel1ular but

extra-cytoplasmic, usually found at the microvillar surface of epithelial host cell and finally produce micro and macro gametes. Fertilization of female and male gametes produces sporulated thick and thin walled oocysts which are excreted in the faeces of infected host. Whereas, thin-walled oocysts, burst while in intestine and release sporozoites which give rise to endogenous auto-infection (Soulsby, 1982).

Clinical signs can persist for 4-14 days and shedding of *C. parvum* oocysts takes place as early as 2 days of age and peak occurs at approximately 14 days of age. The disease is manifested by diarrhea (varies from pale yellow with mucus to profuse watery diarrhea), blood clot, mucus in faeces, undigested milk clots, depression, anorexia and abdominal pain. The disease mainly affected young livestock of 0-3 month's age and the first peak of prevalence of infection in infected animals takes place at 2 weeks of age followed by a second peak at 6 months. Due to sloughing of the mucus membrane especially of the jejunum and results into mucus secretion in faeces and invasion and epithelial destruction with the result of mild to moderate villus atrophy and microvillii shortening and destruction. This also leads to impaired nutrient absorption, increased intestinal permeability, chloride secretion, and malabsorption and transport, loss of membrane-bound digestive enzymes and impaired nutrient and electrolyte transport.

Diagnostic Techniques: The identification of the 2-4 μm small and spherical oocysts of *Cryptosporidium* in the faecal sample can be done by various conventional methods such as: direct faecal smear staining, diethyl ether sedimentation staining and sheather's floatation sedimentation staining. The differential staining methods include the safranin-methylene blue stain, modified Ziehl-Neelsen and the DMSO, whereas carbol fuchsin stains pink colour naked sporozoites seen against green colour background because of the malachite green counter stain under the microscope. Immunological-based techniques include polyclonal fluorescent antibody tests, latex agglutination reactions, immunofluorescence with monoclonal antibodies, enzyme linked immunosorbent assays, reverse passive haemagglutination, immunoserology using immunofluorescence detection and solid-phase qualitative immunochromatographic assays. Whereas, PCR based detection of *Cryptosporidium* spp. by amplification of 18S SSU rRNA based nested PCR protocol and amplification of glycoprotein, gp60 of 60 kDa, also called gp40/15 gene locus showed extreme accuracy which generate rapid, highly sensitive and accurate results (Mirhashemi et al., 2015).

Balantidiasis

It is caused by *Balantidium coli*. It is the largest ciliated zoonotic intestinal protozoan with cyst size of 50 to 100μm in length and 40 to 70 μm in width. Balantidiasis effects variety of host species including pigs, camels, ruminants, equines and even human.

Transmission and Pathogenesis: Transmission is generally via the fecal-oral route from the normal host where it is asymptomatic. Water is the vehicle for most of the cases of balantidiasis. The trophozoites and cysts are shed in faeces and if the cysts, in particular, contaminate drinking water or food, the infection can be spread to other host. Also, if the cysts or the trophozoites, being ingested accidentally via contaminated water and food, transmission by coprophagia could occur in most of the animals. Balantidiasis causes thickening of the mucosa, with mucus accumulation and mucous cell hyperplasia and inflammation of the lamina propria by lymphocytes, plasma cells, and eosinophils. *Balantidium* infection is asymptomatic and the infected host shows no clinical signs, suggesting that this ciliate is an opportunistic parasite that could take advantage of the weakened status of the host caused by other infections, lesions, or diseases. In the chronic form of the disease, the symptoms varied from unspecific abdominal disorders (diarrhea, abdominal pain) to cramping rectal pain, nausea and vomiting, whereas in the acute form these symptoms can be accompanied by mucus and blood in feces, and in severe cases, hemorrhages and perforation could occur resulting in parasite dissemination to other tissues or even in the death of the host (Dhawan et al*.,* 2013).

Diagnostic Technique: Trophozoites have fine, visible cilia and a large, kidney-bean-shaped macronucleus that can be easily recognized in both trophozoites and cysts in temporary smears stained with iodine. Other staining methods such as hematoxylin-eosin or trichrome are also generally used in the labs. Cysts can be recovered using common coprological techniques (i.e., centrifugation methods for concentration). The sequences corresponding to the ribosomal genes (the small subunit rDNA and the 5.8s rDNA, and the internal transcribed spacers ITS– 1 and 2), although show some genetic heterogeneity, can be used in order to differentiate morphologically identical cysts (Pomajbíková et al., 2013).

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LABORATORY DETECTION OF ACARICIDE RESISTANCE STATUS AND ITS MANAGEMENT IN TICKS

Jyoti*, Harkirat Singh, Alveena Ganai and Nirbhay Kumar Singh

Department of Veterinary Parasitology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab - 141004, India
*Corresponding author email: jyoti76vet@gmail.com

Received: April 2023 Accepted: April 2023

Abstract

Ticks and tick-borne diseases are a major hinderance to sustainable and profitable dairy industry. Presently, chemical acaricides are the most widely used approach for tick control but its indiscriminate and extended use has led to development of resistance leading to treatment failures and significant economic losses. There is an urgent need for detection and continuous monitoring of acaricide resistance in the field situation through bioassay, biochemical and molecular tools forstrategic application of available acaricides and for maintaining the lifespan of the existing products. The diagnostic protocols are discussed in detail.

Keywords*: Acaricide resistance, detection, ticks*

Ticks are haemtophagous ectoparasites causing direct and indirect losses to animals. Approximately 80 percent of the world's cattle population is exposed to tick infestation. In India the cumulative annual loss due to ticks and tick-borne diseases is 6107.65 Cr INR/US$ 787.63 million (Singh et al., 2022). The control of ticks mainly depends upon chemical acaricides because they arerelatively quick and cost-effective. Long-term indiscriminate use of acaricides leads to selection of acaricide resistance and show limited efficacy in tick control.

Development of acaricide resistance is not universal, and is most widespread and diverse in the one-host cattle tick and is slower to develop in multi-host ticks. Resistance to commonly used acaricides is emerging as one of the important problems because the development of new acaricides is scarce. There are different resistance mechanisms for each class of acaricide including behavioural changes, cutaneous penetration, metabolic detoxification involving enzymes and target site alterations. Detection and characterization of resistance is very important for effective tick control. Resistance is usually first recognized as a failure in effectiveness of an acaricide to eliminate tick burdens from animals. Periodic monitoring of the ticks for development of resistance against commonly used acaricides is very important for sustainable livestock production.

Detection of Acaricide Resistance

Detection of cattle tick acaricide resistance status is essential on a local and global scale. Failure of tick control is not always due to development of acaricide resistance

in tick population but can be caused by other factors like inappropriate preparation or application of acaricide, faulty equipment and expired drug. Hence, when treatment failure is observed, suspected resistance should be confirmed by laboratory tests before selecting a new acaricide. Once resistance is confirmed to certain acaricides or acaricide group, livestock owners need to be advised on using alternative chemical acaricide for controlling the resistant tick populations. For optimum and strategic use of acaricides and to slow down the development of resistance there is a need to monitor acaricide resistance in the field condition. Resistance can be detected using various *in vitro* bioassays along with biochemical and molecular assays to detect metabolic and target site insensitivity-based resistance, respectively.

I. Bioassays

Bioassays are important tools for characterization of resistant tick populations. These are regarded as one of the most suitable methods to detect resistance in arthropods due to simplicity and low cost. Bioassay measure resistance to compounds whose resistance mechanisms are unknown, but do not give idea about the mechanism involved in acaricide resistance. However, when a bioassay is conducted along with more sensitive biochemical and molecular assay, more information and solid evidence come out about the acaricide resistance status and its possible mechanism. Currently, four bioassays are used to detect acaricide resistance, each with their own advantages and disadvantages:

(i) Adult immersion test (AIT): This test was first developed by Drummond et al. (1973). The principle is to treat engorged female ticks with a range of dilutions of an acaricide and to assess the effect of treatment on mortality, egg mass weight and hatching percentage comparing treated and untreated ticks. The AIT, although provides results within 7 days but requires a large number of engorged females.

(ii) Larval packet test (LPT): This test was developed by Stone and Haydock (1962) and was later adopted by FAO. Subsequently, the same test has been standardized in different laboratories using country specific reference tick strain. In this test 14-21 days old unfed larvae are exposed in acaricide treated filter papers. Mortality of larvae is recorded after 24 hours for detection of resistance and it requires 5-6 weeks to complete.

(iii) Larval immersion test (LIT): First developed by Shaw (1966) and it involves the use of 14-21 days old unfed larvae. Here the larvae are immersed in a solution of the acaricide and mortality of larvae is recorded after 24 h and it also requires 5-6 weeks to complete.

(iv) Larval tarsal test (LTT): This test was developed by Lovis et al. (2012). It involves placement of tick eggs in to multi-well plates to allow the evaluation of multiple chemical acaricides. Requires about 2 weeks to obtain data on acaricide resistance.

II. Biochemical Assays

Metabolic resistance mechanism against acaricides is often the most common mechanism and it may confer resistance to acaricides. Ticks use their detoxifying enzyme system to break down or sequester acaricides. These enzymes may also have wide substrate specificity, with the result that they are able to detoxify a broad spectrum of acaricides. Biochemical estimation of enzymes is considered as one of the indicators in addition to bioassays for resistance monitoring. A possible association between higher activity of esterases, monooxygenases and GST (Jamroz et al., 2000) with resistance has been reported. However, these biochemical assays are not complete substitutes for the standard susceptibility tests which are used to measure resistance. Biochemical testing is basically the use of synergists that inhibit enzymes responsible for metabolic detoxification. Currently following biochemical assays are used for monitoring of acaricide resistance:

(i) Qualitative estimation of enzyme: Gel electrophoresis is performed mainly to detect esterase based metabolic resistance mechanism. Further, inhibitors viz. eserine sulfate, copper sulfate, p-p-chloromercuribenzoate, malathion, phenylmethylsulfonyl fluoride and Triphenyl phosphate are used to confirm the role of specific enzyme.

(ii) Quantitative estimation of enzymes: Done by either filter paper/nitrocellulose membrane based assays quantified visually or more commonly used microtitre plate method quantified visually or with a spectrophotometer.

III. Molecular Assays

Molecular assays have the advantage to provide results within a day, require very few ticks and provide confirmatory information about resistance mechanisms. The molecular assay detects quickly at early stages of the spread of resistance genes so that proper control measures can be adopted to slow the spread of these genes which can increase the effectiveness of a particular acaricide. Various molecular techniques have been developed for detecting acaricide resistance in ticks for rapid detection of resistance viz. target gene amplification by Polymerase Chain Reaction (PCR), Allele-specific PCR, PCR-RFLP, Quantitative PCR and Sequencing techniques with DNA.

These molecular based assays are sensitive and rapid alternatives to bioassays for determining the status of acaricide resistance within a population. The success of molecular assays depends on the availability of precise molecular markers, which are identified and validated through different molecular tools in the targeted genes. The study of single nucleotide polymorphism (SNP) potentially associated to the acaricide resistance is one of the most popular approaches in trying to find molecular markers for acaricide resistance monitoring. A resistance management strategy with molecular markers is crucial because it can monitor acaricide resistance when the resistance allele is

recessive and heterozygotes are abundant in the population or the frequency of resistance is low. Molecular diagnosis with molecular markers allows rapid and direct analysis of resistance-associated genes and has the potential to mitigate the arising menace of acaricide resistance before it reaches the tipping point and the effectiveness of acaricides is diminished.

Molecular techniques not only allow early detection of the presence of resistance genes but also allow determination of the genotype of individual ticks. Information on genotype frequency can help define the risk of resistance and can prevent the spread of resistance phenomenon at the regional level.

IV. Acaricide Management of Ticks on Livestock

Intensive tick control: implies frequent applications of acaricides throughout the year to keep animals free of ticks and prevent pathogen transmission. The economic benefits of eradication of ticks through intensive tick control are enormous.

Strategic tick control: aims to reduce tick populations and transmission of pathogens; the timing of acaricide application is based on the seasonal occurrence of ticks with a variable number of applications. Acaricides are applied at strategic times of the year to control seasonal tick abundance peaks AND is mainly aimed at adult ticks. Other strategic tick control includes rotation of acaricidal classes and combining acaricidal classes into the same product. The rotation strategy, which implies different alternating groups of pesticides with no cross-resistance, has not been implemented significantly. Farmers choose between classes of acaricides available on the market rather than follow any predetermined strategy.

Threshold tick control: aims to control ticks when they exceed a predefined, economically damaging number of ticks. There is a significant relationship between the frequent application of acaricides and the development of resistance. Acaricidal applications can be opportunistic, and only those animals with tick burdens considered above the economic threshold could be treated. This should reduce both treatment costs and delay the development of acaricide resistance.

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AN UPDATE ON THE BOVINE TROPICAL THEILERIOSIS

Paramjit Kaur* and Lachhman Das Singla

Department of Veterinary Parasitology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary & Animal Sciences University, Punjab-141004

*Corresponding author email: paramvet53@rediffmail.com

Received: April 2023 Accepted: May 2023

Abstract

Theileriosis, caused by Theileria annulata, is one of the economically important predominant tick borne haemoprotozoan diseases in bovines especially in cattle. The life cycle is complex involves invertebrates and vertebrates animals. The acute cases based on the clinical signs pyrexia, weakness, nasal discharge, conjunctival petechiae, enlarged lymph nodes and anemia is easily diagnosed by routine microscopic examination of stained blood smears; however, latent asymptomatic animals require more sensitive and specific techniques like polymerase chain reaction (PCR). Buparavaquone is the drug of choice for treatment. The integrated methods consist of chemotherapy, vaccination, and vector management is the key approach for the control of Theileria infection.

Keywords*: Bovines, control, tick-borne disease, theileriosis*

Tick-borne diseases (TBDs) are major constraint to livestock sector, affects many domestic animals, especially cattle in the tropical and subtropical parts across the globe. Bovine tropical theileriosis is one of the predominant TBD caused by *Theileria annulata,* an apicomplexan haemoprotozoan parasite, transmitted by *Hyalomma anatolicum* tick (Ntesang et al., 2022). Since the discovery of *Theileria* in 1904, the economic impacts were recognised in the late 1970's after outbreak of theileriosis in crossbreeds and exotic breeds. The disease leads to high mortality, decline in production and draught power of infected animals and hence painstaking a stern challenge to the livestock rearing in developing countries like India. A total of 39 million crossbred cattle population is at risk to the theileriosis (Kolte et al., 2017) that causes annual economic losses up to rupees 8426 crores in India (Narladkar, 2018). Nationally the data on the prevalence of the disease in cattle and buffaloes in different states ranged from 0.28-89.0% (Singh et al., 2017). Variation in prevalence attributed to various factors like management practices, sex, health status, nutrition, breed, herd size, agro-climate (humidity, temperature), tick vector exposure (Ghosh & Nagar, 2014). Considering the impact and epidemiology of theileriosis in bovines, a better understanding about the biology, pathogenesis, and advances in diagnostic and immunological chemotherapeutic is of paramount concern for the veterinarians and livestock owners, for the formulation of better effective control strategies to minimize the economic loss. Intend of this article to acquaint about the etio-pathogensis, diagnosis and effective control measures of the theileriosis in cattle and buffaloes.

Life Cycle

Theileria annulata has a complex life cycle involving the host animal and genus of *Hyalomma* ticks (Fig 1). During tick feeding, the infective sporozoites inoculated through the saliva into the animal's body that are taken up by mononuclear leukocytes where mature into macroschizonts and induce lymphocyte proliferation in the host. Microschizonts fairly thrive into macroschizonts (Koch's blue bodies) and ultimately into merozoites which are released from the lymphocytes. These merozoites invade into red blood cells and develop into piroplasm.

During a next feeding cycle, larval or nymphal vector ticks ingest piroplasms and the released parasites undergo syngamy in the tick gut, forming a zygote, the only diploid stage. The zygote divides into motile kinetes that infect the tick gut epithelial cells and migrate to the haemolymph and then infect the salivary glands. After moulting and beginning of tick feeding multiplication of sporozoites (sporogony) in acini cells of salivary gland before injection into the feeding site by nymphs or adult ticks (Abdela & Bekele, 2016).

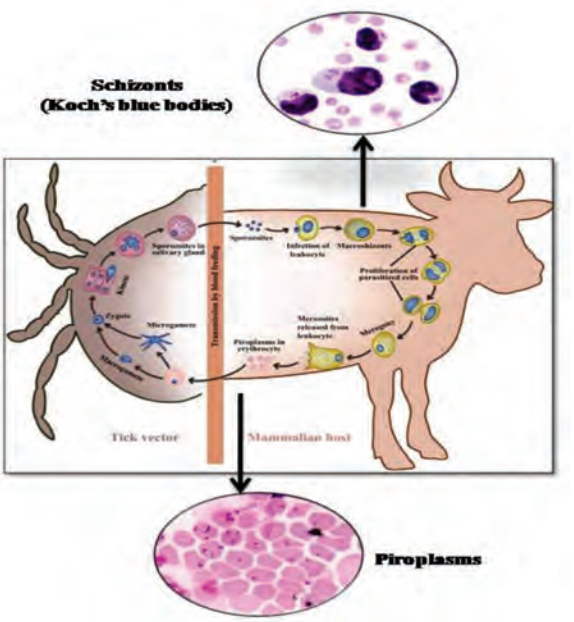

Fig. 1. Life cycle of *Theileria annulata* (modified from Liu et al., 2022)

Pathogenesis and Clinical Signs

The proliferation of the parasites in the lymphocytes in the lymph nodes results into lymphadenopathy. Parasites development in the mammalian host results into consumption of glucose, anorexia may associate with other clinical signs such as hyperthermia, lymph node enlargement, haemolytic anemia, icterus and in rare cases exophthalmia and

haemoglobinuria in cattle also reported. Icterus usually noticed in the early phase and haemoglobinuria in later phase of the tropical theileriosis. *Theileria* Infected cells cause the high level production of inflammatory mediators which are the potent precursor of the typical clinical symptoms of acute tropical theileriosis such as pyrexia, anemia, anorexia, muscle wasting and necrosis. Infected erythrocytes are removed by the macrophages in the organs of the reticulo-endothelial system, erythrophagocytosis and destruction of the erythrocytes results into anaemia. Anaemia causes difficulty in breathing and over functioning of the lung parenchyma leads to the thick copius discharge from the nostrils. The petechial haemorrhages on the sclera of eye are reported and exopthalmia may due to the increase in the intraocular pressure that corresponds to the parasitized lymphocytes that are further related with the high level of TNF-α in the ocular muscles. Lateral recumbency, diarrhea and dysentery are also associated with later stages of the infection.

Diagnosis

Diagnostic techniques employed for the detection of *Theileria* infection are microscopy, serological assays for epidemiological studies and molecular tools for the detection of latent infection and genus/species confirmation of the parasite. Traditional methods in routine use is Romanowasky stained thin blood smears and lymph node aspirates smears for presence of piroplasms in erythrocytes or schizont (Koch's blue bodies) in lymphocytes (Fig. 2) in acute cases, but insensitive in sub-acute and carrier animals as unable to detect the low level of parasitaemia and misidentification of the parasites at genus or species level. For epidemiological surveys a number of immunological assays like complement fixation test, indirect fluorescent antibody test (IFAT), enzyme-linked immunosorbent assays (ELISA) are used. As per OIE recommendation the gold standard test is IFAT, but ELISA is vastly adopted globally for theileriosis, as it is easy to perform, and having high through output. However, the major limitations of sero-assays are cross reactivity, inept to differentiate the present and past infection chiefly in the carrier animals

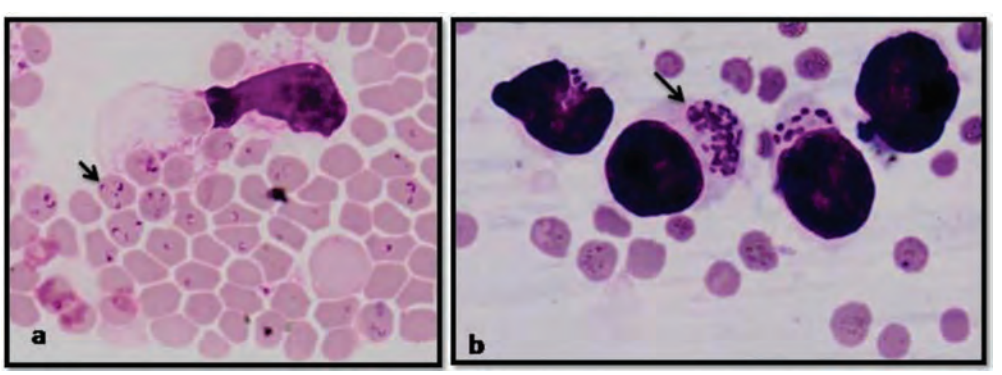

Fig. 2. Piroplasms (a) and Schizonts (Koch's blue bodies) (b) of *Theileria annulata*

that are the source of transmission in non-endemic areas. To overcome the limitation of microscopy and serological assays, nucleic acid based detection molecular techniques like PCR and its variants are proved to highly sensitive and specific, as these are able to detect upto 0.000001% parasitaemia. The prevalence of bovines theileriosis in Punjab by microscopy and nested PCR are recorded as 9.23% and 29.26%, respectively (Tuli et al., 2015).

Chemotherapy

The drug of choice for the effective treatment is Buparvaquone @ 2.5 mg/kg, IM which is found effective both for schizonts and piroplasm stage of the parasites. The Parvaquone @ 20 mg/kg, IM, is only effective against schizonts. Long acting Oxytetracycline @ 20 mg/kg, IM, three doses 4 days apart found effective only at the early phase of the infection.

Control Measures

The integrated approaches targeting the vaccination, tick vector control and managemental strategies to meet the specific requirements of livestock holders in different situations must adopt for the control of theileriosis. Commercial vaccination Rakshavac-T using attenuated schizont-infected cell lines, 3 ml, subcutaneously to the calves of >2 month age group is widely used in India. The most widely used method for tick control is chemical acaricide application. However, multidrug level resistance development is a major concern beside the residual effect of these drugs in tissue and milk and the negative environmental impact. To reduce these impacts, much emphasis has been placed on the search for alternative, environmental eco-friendly tick control strategies with lower chance for the development of resistance. These include the use of phytoacaricides derived from more than 200 plants that possess anti-tick or tick repellent properties. *Azadirachta indica* (Neem), *Lavendula augustifolia* (Lavender), *Pelargonium roseum* (Rose geranium) and *Cymbopogon* spp. (Lemongrass) showed acaricidal and larvicidal effects with 90–100% efficacy, comparable to those of currently used acaricide (Adenubi et al., 2016). Bio-control of ticks by rearing of backyard poultry that eats the ticks off the body, spray of fungus like *Metarhizium anisopliae* and *Beauveria bassiana* , entomopathogenic nematodes of family Steinernematidae and Heterorhabditidae, predatory mites and parasitoids also found beneficial. The management strategies include diet rich in protein; shed with pucca floor, without cracks and crevices the hiding place of ticks and the treatment of the sheds with acaricide if required. The judicious use of the chemical acaricides and on the rotation basis is supportive in the delay of the development of resistance. The indigenous breeds (*Bos indicus,* Sahiwal) are more resistant to tick and tick borne diseases than exotic breeds. Therefore, the development of tick resistant breeds by genetic exploitation and cross breeding programme and vaccination will be an

alternative more sustainable and efficient complementary approach to control theileriosis in future.

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NEW AND ALTERNATIVE APPROACHES TO COMBAT ANTIBIOTIC RESISTANCE

Tania Gupta* and Deepti Narang

Department of Veterinary Microbiology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India
Corresponding author email: dr.tania603@gmail.com

Received: April 2023 Accepted: April 2023

Abstract

Inappropriate, indiscriminate and widespread antibiotic use escalates antibiotic resistant cases and generates public health predicament due to limited synthetic approaches for manufacturing newer antibiotic and limited resource of antibiotic production as most of the environmental bacteria are uncultivable. Newer promising approaches such as a catalytic antibiotic, use of DNAzymes or antisense oligonucleotide for inactivating mRNA that encode for antibiotic resistant protein, inactivation of efflux pump and disruption of the MecR1 regulatory pathway and exploration of newer antibiotic resources via screening of soil bacteria or other methods, may help in combating the existing gigantic antibiotic resistance issue.

Keywords: *Antibiotic Resistance, Catalytic Antibiotic, Antisense Oligonucleotides*

Over the years, antibiotics have saved lives, eased the suffering of millions of people and animals, contributed to the major gains in life expectancy. However, these wonder drugs have started to lose ground rapidly due to the evolution of drug resistance bacteria. Inappropriate, haphazard and widespread antibiotic use establishes a selective pressure, a driving force in the evolution of antibiotic drug resistance such as penicillin-resistant *Streptococcus pneumoniae,* vancomycin-resistant enterococci, methicillin-resistant *Staphylococcus aureus*, vancomycin resistant *S. aureus*, tetracycline resistant Pasteurella spp., extended spectrum beta-lactamase producing *E.coli, Salmonella, Klebsiella* spp., *Pseudomonas aeruginosa,* metallo-beta-lactamase producing *Pseudomonas aeruginosa,* MDR-salmonellae, and MDR-*Mycobacterium tuberculosis, Pseudomonas* and *E. coli*. The spread of drug resistance among bacteria has greatly outpaced the rate of drug development. Escalating number and a variety of drug-resistant pathogens currently emerged as a serious public health problem. This article deals with recent advances on the newer and alternative approaches to combat antibiotic resistance.

Drug resistance in bacteria may be natural or acquired. Bacteria acquire drug resistance via conjugation, transformation and transduction. Bacteria have evolved the various drug resistant mechanisms such as efflux pump, decrease affinity for the target/ alteration in the binding site, development of alternative metabolic pathway, elaboration of a drug inactivating enzyme such as beta-lactamases, decrease drug permeability etc. (Davies et al., 2010). Farm's wastage, nosocomial infection, unused antibiotics flushed

down in the toilet, chemical discharges from pharmaceutical companies etc. are various sources for the evolution of drug-resistant bacteria. The following are the innovative strategies to combat antibiotic resistance

I. Catalytic Antibiotics

Drugs like aminoglycosides are potent bactericidal drug that binds with ribosomes and inhibit protein synthesis. The development of catalytic antibiotic will be able to induce fast and irreversible inactivation of specific target sites via the cleavage of a crucial chemical bond at their binding site in a catalytic manner. This strategy is based on a chemical modification of existing antibiotics in order to make it a catalyst. The possible benefits include activity at lower dosages and subsequent elimination of side effects, activity against drug-resistant bacteria, and reduced potential for generating new resistance.

i) **Aminoglycosides :** These antibiotics inhibit protein synthesis by binding to 30S subunit and 50S subunit of the ribosome as well as their interface and freeze initiation, inhibit translocation and cause misreading of m-RNA code. Bacteria acquire resistance to aminoglycosides by alterations in ribosomal proteins, decreased permeability to the antibiotic or by induction of bacterial drug-inactivating enzymes such as Aminoglycoside nucleotidyl transferases, Aminoglycoside acetyl transferase enzyme, Aminoglycoside phosphotransferase etc. (Azucena et al., 2001). Development of inhibitors of the enzymatic action or of the expression of the modifying enzymes could help to combat aminoglycosides resistance.

ii) **β-lactam antibiotic:** The key catalyst in the synthesis of β-lactam antibiotics like penicillin involve enzyme IPNS (Isopenicillin N Synthase) & DAOCSs (Deacetoxycephalosporin C Synthases) for clavulanic acid is a clavaminate synthetase enzyme. Insight into the machinery of the β-lactam biosynthetic enzymes now offers a unique opportunity for experiments aimed to develop new biosynthetic routes to β-lactamase, cephalosporinase. By combining information on the 3-D structure of active sites of different biosynthetic enzymes, new classes of compound that are active against the resistant bacteria could be developed.

iii) **Vancomycin:** Shi and Griffin (1993) discovered the catalytic activity of vancomycin by chemically altering vancomycin to develop a molecule that will not only bind to the cell-wall precursor and inhibit cell-wall synthesis but destroy the precursor as well. If this is attained, it should increase the potency of vancomycin; the catalytic antibiotic should be able to move to another cell-wall precursor after destroying the first, and so on.

II. Catalytic Activity of L,D-transpeptidase

The β-lactams inhibit D,D-transpeptidase enzyme responsible for cross-linking of peptidoglycan chains and also inhibit carboxypeptidationreaction. L, D- transpeptidase enzyme of *Mycobacterium tuberculosis* catalyzes the formation of 3-3 peptidoglycan cross-links of the cell wall and facilitates resistance against β-lactams (Silva et al., 2014). Carbapenems inactivate these L, D-transpeptidases, and meropenem combined with clavulanic acid is bactericidal against extensively drug-resistant *M. tuberculosis*.

III. Deoxyribozymes

DNAzymes are the catalytic single stranded DNA molecules with enzymatic activity. Many deoxyribozymes catalyze DNA phosphorylation, DNA adenylation, DNA deglycosylation, porphyrin metallation, thymine dimer photoreversion and DNA cleavage. The intracellular introduction of the deoxyribozyme may lead to a decrease or destroy in the levels of the targeted mRNA that encode the proteins responsible for antibiotic resistance and increase the antibiotic sensitivity of treated bacteria. This strategy may be useful for resolving the problem of antibiotic resistance. However, DNAzymes are prone to nucleolytic degradation in body fluids. To prolong the half-life of oligonucleotides for *in-vivo* usage, to enhance biostability, to reduce toxicity and improve target affinity modified nucleotides such as LNA (locked nucleic acid) are usually incorporated (Baum and Silverman, 2008).

IV. Anti-Sense Oligonucleotide

"Antisense" oligonucleotide (ASO) maybe another novel strategy in order to combat drug-resistant bacteria via inactivating mRNA and inhibiting efflux pump that is associated with drug resistance.

V. Disruption of the MecR1 Regulatory Pathway

Methicillin resistance in *Staphylococcus aureus* is mediated by the *mecA* gene which encodes a penicillin-binding protein (PBP2a) possessing low beta-lactam affinity under regulatory signal transduction system MecR1. Disruption of the MecR1 regulatory pathway may inhibit *mecA* expression and restore methicillin-resistant *Staphylococcus aureus* susceptibility to beta-lactams (Hou et al., 2007).

VI. Other Ways to Combat Antibiotic Resistance

Antibiotic resistance is spreading faster than the introduction of new compounds into clinical practice, causing a public health crisis. To discover a newer antibiotic via screening soil microorganism may resolve this problem up to some extent. However, most (approx. 99 %) of the environmental bacteria are uncultivable and limited synthetic approaches for producing a newer antibiotic are helpless. Teixobactin, a newer antibiotic discovered via screening of uncultured bacteria, inhibits cell wall synthesis by binding to

a highly conserved motif of lipid II (precursor of peptidoglycan) and lipid III (precursor of cell wall teichoic acid) (Ling et al., 2015). Teixobactin resistant *Staphylococcus aureus or Mycobacterium tuberculosis* has not been reported yet. The properties of this compound suggest a path towards developing antibiotics that are likely to avoid development of resistance. The bacteria that can be grown in the laboratory are only a small fraction of the total diversity that exists in nature. At all levels of bacterial phylogeny, uncultured clades that do not grow on standard media are playing critical roles in cycling carbon, nitrogen, and other elements, synthesizing novel natural products, and impacting the surrounding organisms and environment. Uncultured organisms have recently been reported to produce interesting compounds with new structures/modes of action, lassomycin, an inhibitor of the essential mycobacterial protease ClpP1P2C1 (Gavrish et al., 2014).

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CANINE IMMUNE MEDIATED HEMOLYTIC ANEMIA

Kanle Shivani Tatyarao*, Kuldip Gupta and Leishangthem Geeta Devi

Department of Veterinary Pathology, College of Veterinary Science (Ludhiana), Guru AngadDev Veterinary and Animal Sciences University, Punjab - 141004, India

***Corresponding author E-mail: shivanikanle@gmail.com

Received: April 2023 Accepted: April 2023

Abstract

Immune-mediated haemolytic anemia (IMHA) is one of the most common cause of anemia in dogs and cats that results from a type II hypersensitivity reaction anda common cause of morbidity and mortality. IMHAcan occur as a result of aprimary (idiopathic or autoimmune) incident or secondary to a variety of infectious or neoplastic illnesses. Middle-aged female dogs, particularly American cocker spaniels, are most susceptible to the disease. It typically results in severe anemia that develops acutely as well as an inflammatory response. It is caused by the development of antibodies that specifically target red blood cells, which either activate the complement cascade, causing intravascular lysis of red blood cells, or opsonize red blood cells, inducing phagocytosis by the monocyte-phagocyte system in the liver and spleen.Unfortunately, despite increasedknowledge of the condition and novel medication treatment strategies, the overall death rate linked with IMHA remains high (about 50%).

Keywords: *Anemia, autoagglutination, coomb's test, IMHA,spherocytosis*

Immune-mediated haemolytic anemia (IMHA) is one of the most typical canine autoimmune disorders characterised by a breakdown in immune-mediated self-tolerance, which results in the rapid premature destruction of RBCs (Griebsch et al., 2009; Manev & Marincheva, 2018).Either extravascular (removal of antibody-coated red blood cells by the macrophages in the liver and spleen) or intravascular (as a result of complement system activation) mechanisms can be used for the destruction of erythrocytes.

IMHA can be categorised into primary (idiopathic) and secondary (caused by an underlying illness) form (Manev & Marincheva, 2018). The primary is more common in canine population (Barker & Elson,1991) and is characterised by the development of antierythrocyte antibodies, which are mostly directed against glycophorins, a sialoglycoprotein on the surface of RBCs that allows them to circulate without sticking to other cells or vascular endothelium (Barker & Elson, 1995). IMHA can develop secondary to avariety of inflammatory, neoplastic or infectious conditions. Feline Leukaemia Virus or hemobartonellosis (mycoplasmosis) in cats, recent immunisation or neoplasia (especially lymphosarcoma) in dogs, are significant causes of secondary IMHA in small animals (Yogeshpriya et al., 2017).Therapeutically, distinction between primary and secondary IMHA is must, since secondary IMHA usually responds poorly to treatment or recur unless the root cause is identified and treated (Balch & Mackin, 2007b).

Signalment

With a females to maleratio of 2:1, female dogs (even spayed) seem to be slightly more prone. According to reports, IMHA can occur between 1 and 13 years old dogs, with a mean of 6 years. A rise in tick-borne infections during the spring and summer may be the cause of a seasonal occurrence (Daisley, 2015).

Etiology of Primary and Secondary IMHA in Dogs has been described by various authors (Balch & Mackin, 2007b; Griebsch et al., 2009; Daisley, 2015)

Etiology of Primary IMHA : Idiopathic

Etiology of Secondary IMHA

Infection
- Ehrlichiosis
- Babesiosis
- Anaplasmaphagocytophilum
- Leptospirosis
- Dirofilariasis
- Abscess
- Pyometra

Neoplasia
- Lymphoma
- Hemangiosarcoma
- Lymphocytic leukemia
- Gastric and lung carcinoma
- Diffuse sarcoma

Drugs
- Penicillins
- Acetaminophen
- Cephalosporins
- Levamisole
- Phenylbutazone
- Dipyrone
- Chlorpromazine
- Vaccination?

Immunological
- Transfusion reactions
- Systemic lupus erythematosus
- Neonatal isoerythrolysis (puppies)
- Anti-lymphocyte globulin (transplantation patients)
- Hypothyroidism

Miscellaneous
- Onion
- Garlic
- Zinc
- Bee-sting envenomation

Pathophysiology of IMHA

IMHA is a type II hypersensitivity reaction due to the development of autoantibodies against the animal's own RBC membrane antigens (Manev & Marincheva, 2018; Balch & Mackin, 2007b). One of the more popular RBC membrane antigens targeted by autoantibodies is a glycophorin, a glycoprotein that bridges the plasma membrane.

Normally, suppressor T cells keep autoantibodies from interacting with host tissues. IMHA-affected animals are believed to have dysregulated suppressor T-cell function or overactive immune systems, which enable autoantibodies to adhere to normal cells and cause RBC destruction. (Balch & Mackin, 2007b).

The synthesis of autoantibodies, predominantly IgM (pentameric) activates the complement system by the classical pathway which leads to generation of membrane-attacking complex and thereafter intravascular hemolysisreleasing free haemoglobin into the plasma and to a lesser extent, C3d-mediated extravascular lysis (Fig. 1) (mostly in the liver). These haemoglobin scavenging systems become rapidly saturated in IMHA patients with intravascular hemolysis, which causes hemoglobinemia and hemoglobinuria (Yogeshpriya et al., 2017). Intravascular hemolysis is more likely to occur with IgM-mediated illness because IgM is more effective than IgG at fixing complement (Balch & Mackin, 2007b). Since their ideal reaction temperature is 4°C, they are to blame for the cold types of autoimmune haemolytic anemia (Barcellini, 2015; Manev & Marincheva, 2018).

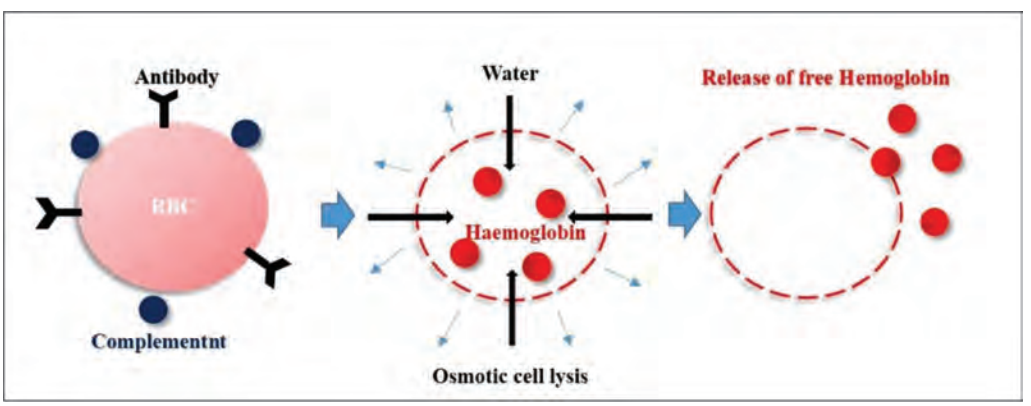

Fig. 1. Mechanism of intravascular hemolysis

The most common autoantibodies to RBCs are IgG, which primarily control extravascular hemolysis via the antibody-dependent cell mediated cytotoxicity in the reticuloendothelial system (spleen and to a lesser extent, liver). When macrophages in the spleen or liver phagocytose and remove the antibody or complement coated RBCs, extravascular hemolysis results. The RBC membrane may also only be partially phagocytized by macrophages, resulting in the formation of spherocytes (Fig. 2) (Yogeshpriya et al., 2017). The Fc portion of the antibody coating the RBC membrane binds to Fc receptors on macrophages in the liver and spleen, causing phagocytosis and death of the RBC. Extravascular hemolysis does not result in hemoglobinemia or hemoglobinuria because RBC haemoglobin enters the bilirubin metabolic pathway

as opposed to leaking into the circulation(Balch &Mackin, 2007b). The IgG subclass determines how much these antibodies limit RBC lifespan. They often respond at 37°C, which accounts for the warm types of autoimmune haemolytic anemia(Barcellini, 2015).

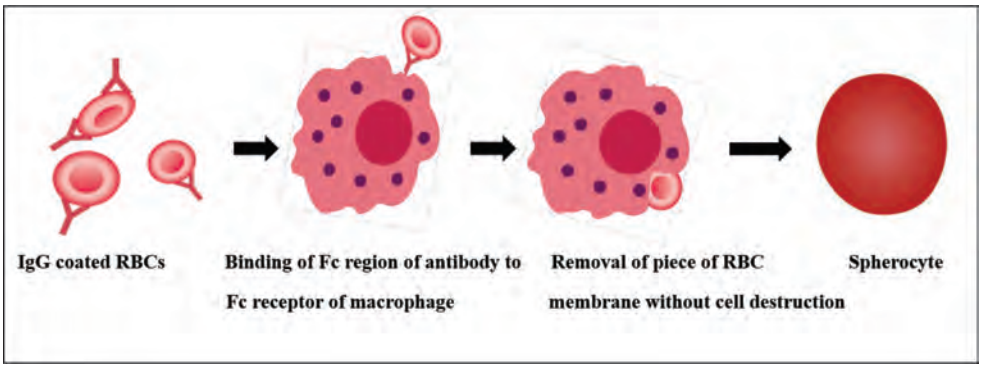

Fig 2. Mechanism of extravascular hemolysis

Clinical Signs

- Anemia and its related symptoms (lethargy, weakness, pale mucous membranes and a hemic heart murmur).
- Tachypnea, tachycardia and bounding pulses - brought on by tissue hypoxia and stimulation of the sympathetic nervous system.
- Fever, anorexia, and less frequently lymphadenopathy - clinical indicators of a persisting inflammatory or immune process.
- Icterus -a significant clinical sign of intravascular hemolysis.
- Petechiae, ecchymoses and melena – due to Evan's syndrome or concurrent thrombocytopenia.
- Extravascular hemolysis caused by subacute or chronic IMHA can partially make up for the absence of erythrocytes in patients, who may still appear impressively bright while having severe anemia.
- Pulmonary thromboembolism - a well-known adverse consequences of IMHA, which is more prevalent in animals with acute severe anemia who are receiving high doses of glucocorticoids. Other causes of dyspnea, such as cardiogenic pulmonary edema or acute bacterial pneumonia, should also be taken into consideration, especially in dogs who are already receiving glucocorticoid and immunosuppressive therapy.

Diagnosis

Complete blood count (CBC): Moderate to severe anemia (mostly regenerative), a high corrected reticulocyte count. However, reticulocyte counts can occasionally be

abnormally low because anemia is peracute (since it takes about 5 days for the marrow to mount a strong regenerative response) or because antibodies are also directed against RBC precursors. In response to both non-specific marrow stimulation and the inflammatory process linked to RBC breakdown, white cell and neutrophil counts are moderately to significantly elevated. Occasionally leukemoid response also will be there. Except in cases of immune-mediated thrombocytopenia or platelet consumption brought on by pulmonary thromboembolism or disseminated intravascular coagulopathy, platelet counts are typically normal in animals (Balch & Mackin, 2007b).

Peripheral blood smear examination: Spherocytosis, polychromasia,macrocytosis, anisocytosis, Heinz bodies, agglutination (Fig. 3) and occasionally elevated nucleated RBC counts. Spherocytes (Fig.4) are microcytic, lack a central pallor and are spherical erythrocytes strongly suggest the presence of either primary or secondary IMHA.About 89 to 95% of dogs with IMHA have large numbers of spherocytes. Mean Corpuscular Volume measurement of RBC stays normal. RBC agglutination (clumping) on a microscopic level can be seen when blood smears are examined.

Serum biochemistry: Hyperbilirubinemia, hyperproteinemia, hemoglobinemia, elevated liver enzymes, decreased haptoglobin concentration and decreased hemopexin concentration (MacNeill et al*.,* 2019). The increase in liver enzymes is thought to be caused by hepatocyte hypoxia (Daisley, 2015).

Slide agglutination test: An IMHA diagnosis is strongly suggested by a positive slide agglutination test, which also indicates that the disease is likely to be acute and severe. To distinguish rouleaux from true autoagglutination, do a saline dilution test (one drop of RBCs to one drop of saline in dogs). Grossly, true agglutination appears as persistent speckles despite saline dilution, and microscopic examination reveals non-linear aggregates of RBCs (Fig. 5). A positive saline agglutination test is seen in 40-80 % of dogs with IMHA.

Immunological testing: A possible diagnosis of IMHA can be supported by specific immunology testing. The direct antiglobulin test (DAT) or Coombs' test, which looks for antibodies and/or complement attached to RBC membranes, is the most often utilised test.According to strict interpretation, a positive DAT confirms the presence of IMHA, whereas a negative test points to the presence of a non-immunological cause of hemolysis.

Bone marrow analysis: All patients suspected of having the non-regenerative forms of IMHA should also undergo bone marrow analysis (aspiration cytology and/or core biopsy histopathology).Pure red cell aplasia is characterised by a relative or total absence of RBC precursors within the marrow, whereas cytological or histopathological evidence of an erythroid "maturation arrest" (predominance of immature precursors, with an absence of more mature RBC precursors), suggests that antibodies are targeted at a later stage

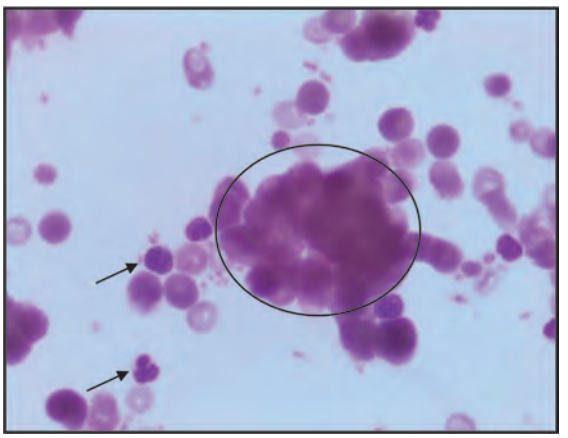

Fig. 3. Peripheral Blood Smear Examination, clumps of red blood cells (circle) indicating IMHA along with infiltration of neutrophils (arrow).

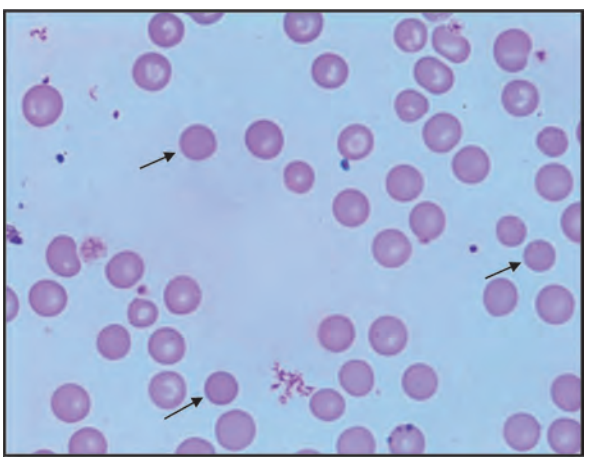

Fig. 4. Peripheral Blood Smear Examination showed numerous spherocytes (arrow).

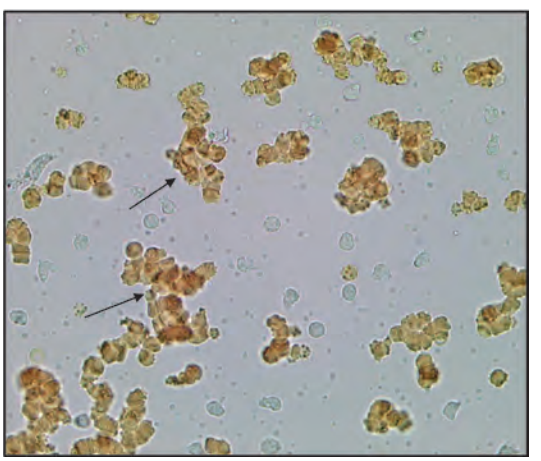

Fig. 5. Slide Agglutination Test, microscopic evidence of agglutination (arrow) in a wet mount. Peripheral blood was diluted in 1:1 isotonic saline (0.9 % NaCl).

of marrow RBC development rather than being directed against very early stem cells. Macrophages phagocytosing erythrocytes or RBC precursors may also be seen by means of marrow cytology and/or histology (Balch&Mackin2007b).

A coagulation profile: The diagnostic panel is completed with a coagulation profile. Prolonged prothrombin time, protracted partial thromboplastin time, low fibrinogen level, and positive D-dimer test are examples of potential changes. They suggest a prothrombotic tendency and impaired hemocoagulation (Manev & Marincheva, 2018).

Prognosis

Unfortunately, the outlook for canines with IMHA is uncertain. Complete therapeutic success can take weeks or months, and some patients may need ongoing therapy for the rest of their lives (Balch & Mackin, 2007a).

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PROFESSIONAL CROSSWORD FOR VETERINARIANS

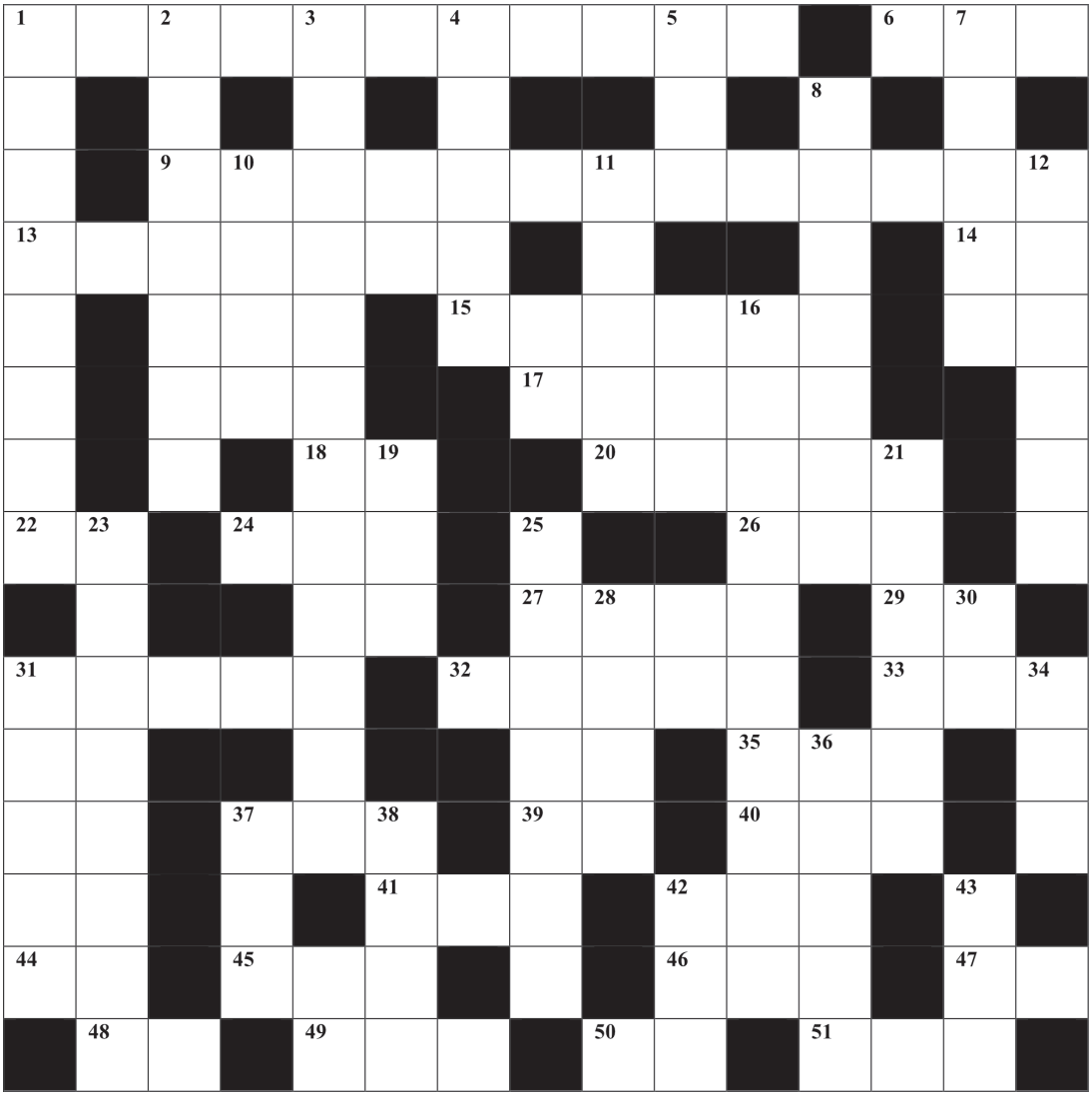
Rakesh Kumar Sharma*

Department of Veterinary and Animal Husbandry Extension Education, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India

*Corresponding author email: rkkural@yahoo.com

Received: December 2022 Accepted: May 2023

(Note: The spellings for this crossword are according to American English)



ACROSS:

1. A hormone produced by adrenal medulla (11)
6. Vasopressin is also known by this acronym (3)
9. Glial cell that forms around damaged nerve cell (9,4)
13. Red blood cells stacked like a pile of coins (7)
14. Prefix meaning an egg (2)
15. Pertaining to a solution that contains salt(s) (6)
17. A condition with elevated body temperature above normal range (5)
18. Chemical symbol for element Krypton (2)
20. Second largest tarsal bone (5)
22. Thirteenth letter of Greek alphabet (2)
24. One of a series of long thin curved bones articulating with thoracic vertebra (3)
26. Suffix used in naming enzymes (3)
27. A filament of keratin consisting of a root and a shaft (4)
29. Abbreviation of nanogram (2)
31. Electromagnetic radiation with shortest wavelength (5)
32. Drugs that inhibit biosynthesis of folic acid (5)
33. Gumboro disease in which Bursa of Fabricius gets affected (3)
35. Another name (Abbreviation) for Dimercaprol (3)
37. The foot or foot like structure (3)
39. A number or value that indicates how much volatile fatty acids can be extracted from a particular fat or oil (2)
40. A rubber bag in which cracked ice is put and is used as cold application (3)
41. An index representing thermal stress among animals (3)
42. A tag containing *this* is put on equipment that is not to be used (3)
44. Prefix for two or twice (in Greek) (2)
45. Symbol of an amino acid that has a single hydrogen atom as its side chain (3)
46. A scan by positron emission tomography (3)
47. Y-shaped protein of immune system that neutralizes pathogens (2)
48. Abbreviation for antibody (2)
49. ____ - down reflex of sucking dams (3)
50. Transferring fertilized egg from donor to recipient animal (2)
51. Female germ cell (3)

DOWN:

2. This hormone helps in the conversion of glucose to glycogen (7)
3. Proteolytic enzyme secreted from the duodenal mucosa (12)
4. The point at which nerves and vessels enter or leave an organ (5)
5. The egg of louse (3)

7. One of the signs of inflammation (5)
8. Jaundice (7)
10. A succulent plant whose gel is used externally for burns/frostbite (4)
11. A passage leading into a cavity (5)
12. A liquid preparation applied on the skin (6)
16. An antineoplastic agent (10)
19. Common abbreviation for an erythrocyte (3)
21. Pertaining to old age (6)
23. Presence of uric acid salts in the urine (8)
25. Cyanogenetic glycoside present in leaves of *Sorghum vulgare* (7)
28. Any double sulfate of a trivalent (Al^{3+}, Fe^{3+}) and a univalent (Na^{+},K^{+}) metal often used as an astringent (4)
30. Small, pear-shaped organ located under the liver that stores bile (Abbreviation) (2)
31. An organ having epithelial cells that are secretory in nature (5)
34. A coat color in horses ranging from grey yellow to light yellow (3)
36. A disease with rapid onset (5)
37. A breed of small dogs with large rounded head, brachycephalic jaw, prominent eyes and tail arched over the back (3)
38. Hordeolum; inflammation of the sebaceous gland of the eyelid (4)
42. A childhood vaccine (combined) against diphtheria, whooping cough and tetanus (3)
43. Common name for *Sus domesticus* (3)

(Crossword answers on page No. 87)

COLOSTRUM - A LIQUID GOLD FOR THE DAIRY FARMERS

Niharika Thakur*, Rajesh Kasrija and Devansh

Department of Veterinary and Animal Husbandry Extension Education, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University Punjab-141004, India

*Corresponding author email: thakurniharika27@gmail.com

Received: September 2022 Accepted: April 2023

Abstract

Colostrum provides high amounts of nutrient as well as non-nutrient factors that promote the immune system and intestinal maturation of the calf. The maturation and function of the neonatal intestine enables the calf to digest and absorb the nutrients provided by colostrum and milk. Feeding of colostrum is thus a crucial step in the calf's development as colostrum intake supports the start of anabolic processes in several tissues, stimulating post natal body growth and organ development.

Keywords: *Anabolic process, colostrum, growth, nutrients, organ maturation*

Usually when we talk about calf management, the first question that farmer asks is when to feed the colostrum to the neonatal calves? Some farmers have perception that the new born calves should be fed colostrum only after the expulsion of placenta (Tiwari et al., 2007) and they think that feeding of colostrum to calves before placenta expulsion will result in poisoning to calves. But, this is a misconception. The neonatal calf should be provided colostrum immediately after birth without waiting for expulsion of placenta. Suckling by calves result into release of oxytocin in the body of parturated dam, which helps in easy expulsion of placenta.

Next query that farmers have in mind is whether to feed the colostrum directly from the teat of the dam or should we have to feed it manually via bottle or via method of pail feeding? When the calf is directly feeding from teat of dam, then the dairy farmer has to first clean the udder and teat of the dam and then allow the suckling of the neonatal calf. Also, the dairy farmer has to ensure that the neonatal calf drinks the colostrum properly in right amounts. If these two conditions are met, then farmer can directly feed its calf via teat. But if a farmer is not sure whether calf will completely drink the colostrum, then he should go for bottle feeding or via method of pail feeding to the calf. It is important for a dairy farmer to understand that calves are born without any immunoglobulins (Ig antibodies); and colostrum contains these immunoglobulins or antibodies (Kertz et al., 2017). Hence, it becomes necessary that calf should get these antibodies via colostrum within first 24 hours after calving for its survival.

It is usually observed that calves are not allowed to consume enough colostrum thus they fail to achieve successful passive transfer of immunity, leading to poor health and less chances of survival. Besides, the efficiency with which a calf can absorb Ig starts declining within one hour after birth and is gone by 24 hours. Thus, the first few hours of birth in calves are critical concerning colostrum feeding. Delay in first feeding of colostrum causes a decline in efficiency of absorption (Kumar et al., 2013). Sometimes it is also seen that farmers do not ensure that calves are drinking colostrum properly when they are left to nurse the dam and as a result calves can sometimes fail to find the udder or teats, and consequently they don't get enough colostrum. They only start drinking that colostrum later, if they were bottle fed or pail fed. The situation gets even worse if the dam has large pendulous udders that are low to the ground. Due to calf's natural tendency (i.e. to nurse up), calf may spend many frustrating hours trying to find the udder instead of nursing colostrum, and may be ingesting bedding or faeces that can contain harmful bacteria. Hence, the calf survival in near future solely depends on what it consumes its first feed i.e. colostrum or faeces.

How to Feed Colostrum via Bottle Feeder to the Calf?

There is a general notion in the mind of the farmers that how can we feed the calf by bottle? Bottle feeding the calf is not a difficult task. First of all the farmer has to pour the colostrum into the bottle by dripping some of the colostrum on his fingers and then let the calf suckle on his fingers. Gradually, calf learns about suckling and then the farmers can immediately shift to the bottle feeding. Always remember that the face of the calf should be raised by the farmer so that there are no chances of drenching pneumonia (Fig. 1). Bottle feeding can facilitate early weaning. The method of bottle feeding provides a natural suckling position of calf which helps in promoting early oesophageal groove closure resulting in early rumen development. This method of feeding also ensures that the adequate amount of colostrum and milk is provided to the calves.

Fig. 1. Bottle feeding method

How can we Feed Colostrum by Method of Pail Feeding?

In this method of feeding, the farmer should first withdraw colostrum from the dam into a clean pail. The farmer should first dip his two fingers (after cleaning) into the colostrum and keep them close to the mouth of the calf. After getting the taste of colostrum, calf will start suckling his fingers. Gradually, the fingers should be lowered to the pail (Fig. 2) and should be dipped into the container containing colostrum. When

the calf slowly starts drinking the colostrums, then remove the fingers. The main advantage of pail feeding are- easier handling, simple and easily cleaning of pail. It can be filled on the spot which can be beneficial in case of large dairy farms. It also facilitates early weaning. The main disadvantage of this method is that this method allows faster consumption which may lead to digestive disorders.

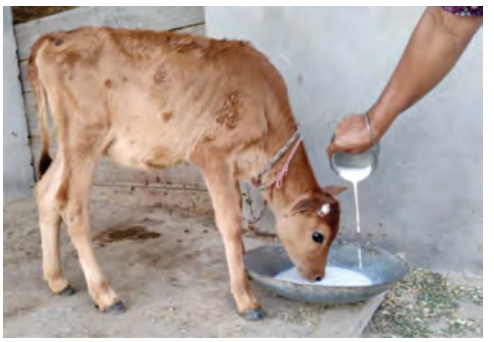


Fig. 2. Pail feeding to calf

How much colostrum to be fed to the calves?

The farmer always has query that how much colostrum is enough for the calf? Usually, colostrum is fed @10% of body weight of the calf which comes to about 2-3 litres of colostrum daily (in two to three divided feedings for 3-4 days). But it is important for the farmer to remember that first 24 hours are more crucial. The amount of scientific colostrum feeding usually depends on various factors such as the body weight of the calf, breed, productivity and the amount of the antibody that is present in the colostrum.

How much Colostrum to be Fed to the Calves?

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What does Colostrum Contain?

Colostrum contains carotenoids due to which it imparts a reddish-yellow colour to the colostrum. The level of carotenoids are high in initial colostrum, in particular the fat fraction but decrease rapidly as the mammary secretions changes to normal colour. Colostrum contains bioactive components with immunity enhancing properties such as immunoglobulins, lactoferrin, lysozyme, lactoperoxidase, alpha lactalbumin, beta-lactoglobulin, fat, protein, energy, vitamins and minerals that are needed for a calf to remain healthy (Table 1).

Table 1: The chemical composition of cow colostrum and buffalo colostrum at calving

Components	Cow Colostrum	Buffalo Colostrum
Total Solids (%)	24.19	26.67
Total Protein (%)	13.45	13.46
Whey Proteins (%)	11.90	11.80
Fat (%)	8.04	9.59

(Source: McGrath et al., Dairy Science and Technology, 2016)

Why Colostrum Feeding is to be done for 3-4 days?

Colostrum is important for the calves as it is the first milk secreted from the mammary glands (for the first 24 hours) after the parturition and provides passive immunity (Mengesha, 2013). The first 24 hours are more crucial as the maximum absorption of antibodies occur within these 24 hours. After twenty four hours the colostrum is called as transition milk. This transition milk is also important for the dairy calf as it helps to keep the calf gut healthy and prevents them from becoming sick. The composition of this transition milk then further changes to that of milk by 72 hours after calving. The comparative composition of colostrum, transition and whole milk is given in Table 2.

Table 2: Difference in composition of colostrum, transition milk and whole milk

Parameter	Colostrum (Within first 24 hours)	Transition milk (48 hours to 72 hours)		Milk (Post 72 hours)
Specific Gravity	1.056	1.040	1.035	1.032
Solids%	23.9	17.9	14.1	12.9
Protein%	14.0	8.4	5.1	3.1
Casein%	4.8	4.3	3.8	2.5
IgG, g/L	48	25	15	0.6
Fat %	6.7	5.4	3.9	3.5
Lactose	2.7	3.9	4.4	5.0

(Source: M J Fahey et al., Journal of Dairy Science, 2020)

Importance of Immunoglobulins in the Colostrum:

Immunoglobulins are antibodies or proteins that help to identify and destroy pathogen in animals. There are mainly three types of immunoglobulins namely IgG

(70-80%), IgM, (10-15%) and IgA (10-15%). These all three immunoglobulins are important to calf to prevent the chances of disease and incidences of death. IgG is present in abundance in colostrum of calf in comparison to IgA and IgM. Additionally, there are further two isotypes of IgG: IgG1 and IgG2. These Ig work together to provide the calf with passive immunity. IgM antibody provides protection against septicemia. IgA antibody provides protects against gastro-intestinal disorders.

What to do if Colostrum is Not Available?

Sometimes, the dam is not able to produce colostrum or milk due to some reason or if she dies while giving birth then the calf can be fed by foster mothers or can be fed on artificial colostrum if there are no foster mothers available.

Preparation of Artificial Colostrum

Artificial colostrum serves as a rich source of nutrients for the newborn calf as it has high amount of energy and protein. To prepare artificial colostrum, the following ingredients are used: an egg, half litre of fresh warm water, half litre whole milk, one teaspoonful of Castor oil and small amount of Cod liver oil. Then mix thoroughly and feed the colostrum to newborne calf for 3-4 days @ of 10% of calf body weight. The egg is a source of protein. Castor oil and Cod liver oil are sources of energy and whole milk acts as a source of lactose and milk protein. The diet of the newborn calf should contain these milk proteins since the enzymes that break down these proteins do not develop in them until seven to ten days after birth.

Colostrum Feeding- Do's and Don'ts:

DO's:

- When allowing the neonatal calf to feed directly from the teat of the animal, always clean the udder before it suckles the teat.
- Try to feed colostrum immediately one hour after the calving and continue to feed it for 3-4 days.
- If calf does not drink colostrum from the teat of the animal try using the bottle feeder or via pail feeding. Ensure that the bottle feeder or pail is clean before and after feeding.
- Raise the head of the calf when feeding via bottle feeders so as to avoid chances of drenching pneumonia.

Don'ts:

- Don't underfeed the colostrum to the calves.
- Do not assume that bottle feeder or pail is clean.

- Do not feed colostrum from the dairy animals that are leaking prior to calving or during calving.
- Do not feed colostrum that may contain blood or may be mastitic.

Colostrum provides high amounts of nutrient as well as non-nutrient factors that promotes the immune system and intestinal maturation of the calf which enables the calf to digest and absorb the nutrients provided by colostrum and milk. Dairy farmers will also see decrease in incidence of calf diseases or diseases in near future when they grow up if they practice scientific management of feeding colostrum which in turn will reduce the cost of treatment of the calf or the future cow or buffalo. Hence, providing boon for the dairy farmers as '***Colostrum is the real Liquid Gold***'.

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CLINICAL DIAGNOSIS AND MANAGEMENT OF PIGMENTARY KERATOPATHY IN DOGS

Kiranjeet Singh* and Aswathy Gopinathan,

Division of Surgery, ICAR-IVRI, Izatnagar, Bareilly, UP, 243 122

*Corresponding author email: ksuppli@yahoo.co.in

Received: January 2023 Accepted: April 2023

Abstract

Pigmentary keratopathy or corneal pigmentation is of multifactorial etiology, occurring as a result of a local biological response to various irritating stimuli. Such stimuli may be mechanical, such as corneal surface abrasion by eyelid skin (entropion) or aberrant hair (distichiasis, trichiasis, or ectopic cilia). It can also occur secondary to exposure of the cornea (lagophthalmos or facial nerve paralysis); or immune mediated reasons (eg, chronic superficial keratitis) environmental stressors like UV radiation, dust and contamination playing a significant role. This article describes risk factors, early diagnosis and management of pigmentary keratitis in dogs.

Keywords*: blindness, canine, corneal pathology, keratitis,*

Pigmentary keratopathy is a type of keratitis where corneal transparency is compromised due to development of brown discoloration in response to various irritating stimuli. Sources of pigmentation on or in the cornea may be either endogenous or exogenous in origin. Endogenous stimuli may be mechanical, such as corneal surface abrasion by eyelid skin (entropion) or aberrant hair (distichiasis, trichiasis, or ectopic cilia); secondary to exposure of the cornea (lagophthalmos or facial nerve paralysis); or immune mediated (eg, chronic superficial keratitis [ie, pannus]). Exogenous irritants like dust, pollen, other allergens, UV rays, therapeutic agents can cause corneal inflammation in response to increased oxidative stress which could lead to pigmentation. Identifying the underlying cause and correction of it is a prerequisite for proper management of this condition.

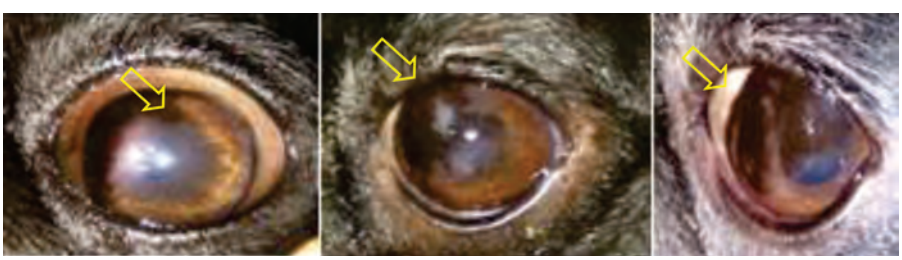

Fig. 1. Representative photographs showing deposition of brown pigment in the cornea (yellow arrow) in dogs.

Risk factors for the Development of Pigmentary Keratopathy in Canine

i) **Brachycephaly:** Brachicephaly is the term used to describe animals that have greatly shortened upper jaws, noses and decreased depth of the orbits (the bony eye sockets). Pekingese, Pug, shitzu, Lhasa apso (Ledbetter et al., 2012) are major breeds predisposed to this condition.

ii) **Congenital abnormalities:** Marked distichia, entropion, euryblepharon, ectopic cilia, nasal fold trichiasis (Labelle et al., 2013) etc. can act as cause of chronic irritation and can trigger inflammatory cascade.

iii) **Environmental factors:** Chronic exposure to UV radiation predisposes to corneal pigmentation. Other ocular surface effects of certain pollens can be generated independent of immune system involvement.

iv) **Drug induced:** Keratoconjunctivitis Sicca (KCS) in dogs has often been associated with use of nonsteroidal anti-inflammatory drug, etodolac as well as with many sulfa derivatives, including trimethoprim sulfamethoxazole, sulfadiazine, and sulfasalazine.

v) **Heavy metals:** Iron, Copper, Gold, Silver, Adrenochromes and Hemochromes can cause pigment deposition in the cornea when these particles get deposited as foreign particle

vi) **Keratoconjunctivitis sicca (KCS):** The KCS, also known as dry eye, is a chronic inflammatory condition of eye, characterized by the deficient production of either aqueous portion of the tear film or due to the inadequate production of lipid layer leading to excessive evaporative loss and consequent pigmentary keratitis.

vii) **Effect of Gender:** The disease is more frequent in female compared to male with more frequent predisposition in breeds like Cocker Spaniels and Brachycephalic breeds in general.

viii) **Chronic superficial keratitis**: Chronic superficial keratitis, also known as pannus or Uberreiter's disease, is an inflammatory condition of the cornea in dogs, particularly seen in the German Shepherd Dog. It is characterized by a chronic progressive inflammatory reaction with formation of granulation tissue and secondary pigmentation in the cornea (pannus).

Irrespective of the causes, presence of persistent inflammation of the corneal surface leads to migration of melanocytes from the limbus towards cornea. It may reach the central cornea, sometimes completely obscuring the entry of light through cornea; inflammatory changes disrupt the anti-oxidant scavenging of corneal surface, further aggravating corneal tissue damage. Release of inflammatory cytokines and subsequent leukocytosis can lead to fibrous metaplasia of corneal surface and corneal stroma. Chronic

keratitis and Keratoconjunctivitis sicca predisposes to pigmentary Keratopathy in dogs.

Diagnosis

Corneal pigmentation is a clinical sign; its presence should prompt thorough ocular examination to identify comorbid conditions. Diagnosis is made on the basis of history from the owner like presence of ulcer, epiphora or redness of eye etc. Reflexes like menace reflex, papillary light reflex, palpebral should be done to identify the status of the eye. Visual function test like maze test, placing test, cotton ball etc. should be carried out to determine the vision status of the animal. Ophthalmic examination with retro-illumination will reveal clear evidence of ulcer, pigment or any other congenital abnormalities.

Lacrimal function test: Schirmer tear testing is done by placing the strip on the ventrolateral aspect of eye for one minute and normal values are 18.9 - 29.9 mm (Gelatt et al., 2021).

Fluorescein dye staining is used to visualize epithelial defects and Rose Bengal stain to identify devitalized cells on the ocular surface. Routine cytology reveals basal and intermediate squamous epithelial cells during inflammation; other cell like neutrophils, leucocytes can also be observed.

Management

Medical management is aimed at abrogating the progression of pigment deposition in cornea. Commercially available corticosteroids are available in topical formulations. Experimental studies indicate that topical prednisolone acetate 1% produces the best anti-inflammatory effect in the intact cornea, followed by dexamethasone 0.1% suspension, prednisolone phosphate 0.1 % solution, and dexamethasone phosphate 0.05% ointment. Despite the advantage of prednisolone acetate suspension, vigorous shaking of the bottle is necessary to deliver the intended dosage, and marked discrepancies can occur between brands of the same product.

Immunosuppressive agent like Cyclosporine is recommended for pigmentary Keratopathy. Cyclosporine acts by interfering with interleukin-2 and T-cell activation, inhibits mitochondrial mediated pathways of apoptosis. It can be used topically in varying concentration from 0.02% to 0.5%. In Indian market 0.5% cyclosporine eye drops are available. Tacrolimus is a similar immuno suppressant which is 10 -100 times more potent than cyclosporine. Picrolimus, a macrolacum derivative and Mitomicin C are also used for the treatment of pigmentary keratitits.

Surgical Treatment

Superficial Keratectomy: Superficial keratectomy is the surgical procedure by which superficial corneal epithelium and a portion of corneal stroma can be removed. Surgery

is usually performed under general anaesthesia. Central positioning of globe and orbital analgesia can be ensured by injecting lignocaine 20% (1:1 dilution with distilled water) into the caudal part of globe. Proparacaine can be instilled on the corneal surface to desensitize superficial corneal nerve supply. The area to be removed can be demarcated using a corneal punch of optimum diameter; the demarcated area can be detached from stroma using a keratome. It provide only temporary improvement by removal since the initial effect short-lived as granulation occurs in healing process again lead to neovascularization which will again bring pigment into the epithelium. Antibacterial, cyclosporine, and NSAIDS postoperatively can be used.

Cryotherapy: Melanocytes are highly cold-sensitive, and hence cryosurgery can be used to treat corneal pigmentation in dogs and conjunctival or scleral melanomas. Melanocytes are more cold-sensitive than other corneal cells because of their high water content; soft cryogens are useful for selective destruction of superficial corneal pigmentation.

The mechanism of action can be explained in 3 stages. The mechanism of action of cryotherapy has an immediate, a delayed, and a late phase. First, freezing causes ice crystal formation. The freezing rate determines where crystals form: with slow freezing, ice crystals are formed extracellularly, and with rapid freezing, they are predominantly intracellular and cause maximal cell death as they expand during a slow thaw. Simultaneously, osmotic alterations causes rupture the cell membrane. The second phase occurs a few hours later as vascular stasis leads to destruction of the microcirculation and tissue ischemia. The late phase consists of an immune reaction Epithelial and stromal cells are destroyed at‑30 °C, but no destructive effect is observed on collagen & fundamental substances (Azoulay, 2013).

Aerosol bottle of liquid cryogen (95% dimethylether, 3% isobutane, and 2% propane) and single-use plastic foam applicator are available which can be applied on to cornea for 20 sec. Liquid nitrogen spray for 15 seconds along with double freezing has been found to be successful in removing pigment epithelium (Holmberg et al., 2008). Cellular repopulation begins with an expansion of adjacent normal cells and pigmentation completely decreased in 2 months. Side effects of cryotherapy included postoperative corneal edema resulting from epithelial injury caused by the cryogen and corneo-conjunctival inflammation. These symptoms resolved by about a month after the procedure. The underlying disease if not kept under control the condition with pigmentation recurs.

Postoperative improvement in the animal's ability to see well enough to ambulate in its environment without colliding with objects, even temporarily, could justify the use of cryotherapy as an adjunctive treatment in the management of severe corneal pigmentation.

Radiotherapy using soft X-rays: The effect of radiotherapy treatment with soft X-rays (15 kV) on canine chronic superficial keratitis along with pigmentation has been evaluated to be long lasting when the underlying cause is also being removed. Those animals demonstrating a poor response to medical therapy alone, all responded well to treatment with soft X-rays.

Constraints of Present Therapy

Therapeutically we are managing immune mediated cells which invade the cornea as a protective mechanism. Surgical management includes the removal of the covering pigment which can restore the vision of the animal to a considerable extent but it does not eliminate the cause of pigment. And if the inciting cause still persists then a vicious cycle of inflammation leads to recurrence of the pigment epithelium and failure of treatment. Currently, the reason for the precipitation of pigment epithelium is least explored and hence there is no standard protocol to prevent and revert the condition.

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OCCURRENCE OF SUPERNUMERARY TEATS IN RUMINANTS: AN ANATOMICAL PERSPECTIVE

Vipul Kumar, Neelam Bansal*, Varinder Uppal and Anuradha Gupta

Department of Veterinary Anatomy, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab- 141004

*Corresponding author email: bansal.neelam@rediffmail.com

Received: March 2023 Accepted: April 2023

Abstract

Supernumerary teat (SNT) is a congenital problem indicating the presence of extra teats in addition to the primary teats. There are different types of SNT based upon the internal anatomical structures i.e. simple and complex type. Depending upon external appearance, three types of SNT are ancillary teats, fish or fork teat or double-orifice teat. SNT also varied according to their location in relation to the normal teat and classified as caudal SNT, intercalary or ramal types. In dairy cows, the caudal SNT are observed more frequently as compared to intercalary or ramal types; whereas the cranial SNT are more common in goats. Among different animals, SNT are more common in goat followed by cattle and least in buffalo. The present article describes applied anatomy, classification and associated complications of the supernumerary teats in different ruminant species that would aid in planning effective diagnostic and therapeutic interventions.

Keywords*: Anatomy, buffalo, cow, goat, sheep, supernumerary teat*

Supernumerary teat (SNT) is a congenital condition in which additional teats are present along with the primary teats. There are four primary teats in cow and two in small ruminants (sheep and goat). Additional teat is known as supernumenary teat or hyperthelia, after teats, abortive teats or extra teats. This condition is due to the failure of secondary ducts to form during embryonic development. The SNTs are considered as a reservoir of bacteria for the whole udder, a risk factor for mastitis, an impediment for suckling, milking or efficient total milk removal and positioning of the milking machine. As all of these are with economic relevance in farms and dairy industry (Gradinaru et al., 2021), therefore, an investigation was done to explore the anatomy of SNT in different ruminants.

Anatomy of Normal Teat

The detailed anatomical and functional anatomy of the teat has been described by the Sarangi and Bansal (2020). Anatomically, the udder is divided into 2 cavities, the right and the left in goat and sheep, and four in cow. The milk is formed from the maternal blood in the alveoli within the mammary lobes, from where this is transported via lactiferous duct to the gland cistern. The latter serves as a temporary reservoir before milk-let-down stimulates milk removal through the teat orifice. The teat is divided into

three parts i.e. base, mid and tip (Fig. 1). The teat skin is thicker than the udder skin. The thickness of teat increased from base to tip region. Mostly teats are hairless, but in sheep and goat fine hairs are present on the teat. The gland sinus and teat sinus are separated by annular folds, which were more prominent in buffalo. The teat sinus was wider at base which narrowed down at tip. The teat sinus continued as teat canal which opened at teat orifice. At the junction of teat sinus and teat canal, small mucosal elevations known as Furstenberg's rosette are present. The number of teat orifices per teat is 1 in ruminants, 2 in mare and sow, 8-14 in bitch. The muscular sphincter is present around the teat canal, which opened during milking. The sphincter contained smooth muscle and collagen fibers in cattle and buffalo, but it is musculo-elastic type in goat. During dry period, the teat canal is closed by the keratin plug which prevents the invasion of pathogens causing intramammary infections. The infection can be lowered down by keeping the animal standing for some time after milking.

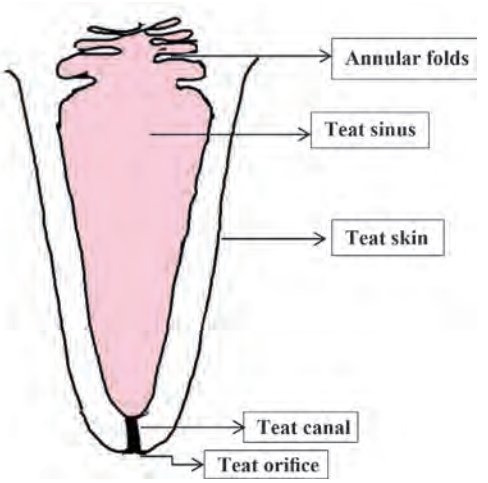

Fig. 1 Schematic diagram showing normal anatomy of bovine teat

Among different animals, SNT are more common in goat followed by cattle and least in buffalo.

Classification of SNT

A. Based upon internal anatomy, SNT are of two types:

1. **Complex SNT (Fig. 2):** Supernumerary teats having either teat canal or teat cistern or both are classified as complex SNT.

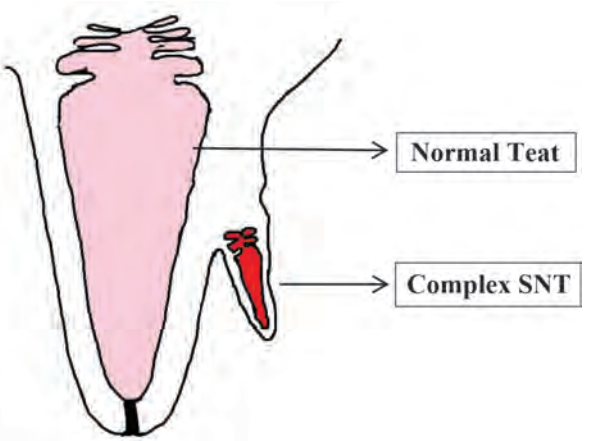

Fig. 2 Schematic diagram of complex supernumerary teat

2. **Simple SNT (Fig. 3):** The SNT without any internal structures as seen in normal teat are simple SNT.

Hardwick, et al. (2019) reported in sheep that anatomically simple SNT (52%) had no apparent connection with the gland cistern proximally or the external environment distally. However, anatomically complex SNT (48%) had internal structure similar to the normal teats i.e. presence of teat canal, teat cistern and gland cistern, but its size may be smaller than normal teat.

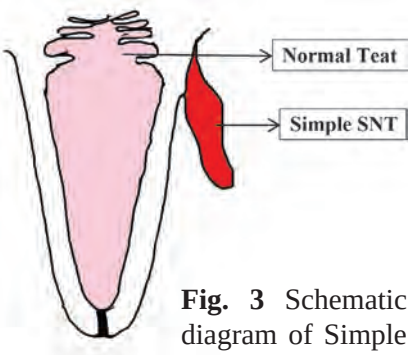

Fig. 3 Schematic diagram of Simple supernumerary teat

B. Based upon external appearance, SNT are of three types

1. **Ancillary teats** are those consisting of more than the normal number of teat in each half of the udder (Fig. 4). In ancillary teat, the primary teat is more conspicuously bigger and easily differentiated from the SNT.
2. **A fish or fork teat** is a single teat that flattens but splits near the end with two separate orifices (Fig. 5). Split teat is a single teat that splits in to two separate parts distally.

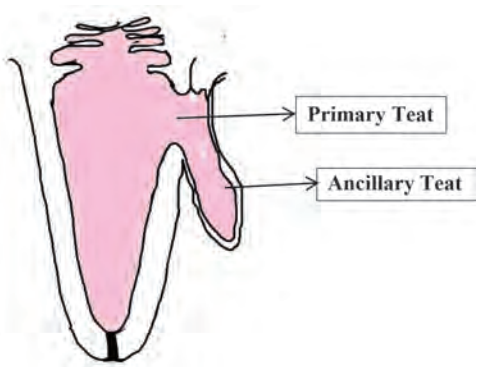

Fig. 4 Schematic diagram of Ancillary supernumerary teat

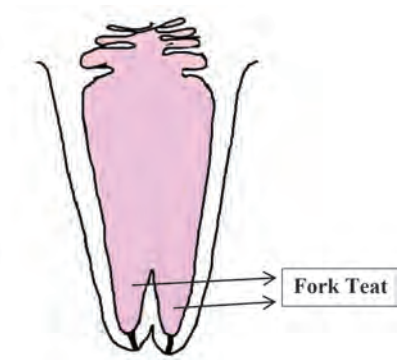

Fig. 5. Schematic diagram of Fork supernumerary teat

3. **Double/triple orifice teat** has more than one orifice per teat. Sometimes, three orifices were also observed in goat teat i.e. one normal and two small orifices were present giving the appearance of smily (Fig. 6 a and b).

Raheem and Leigh (2014) reported various types of SNT in goat and observed that mostly SNT had no duct that connected to the primary mammary gland and was vestigial and non-functional. Some other studies showed that it may be connected to the small or main glands and yielding small amount of milk (Thomas, 2008).

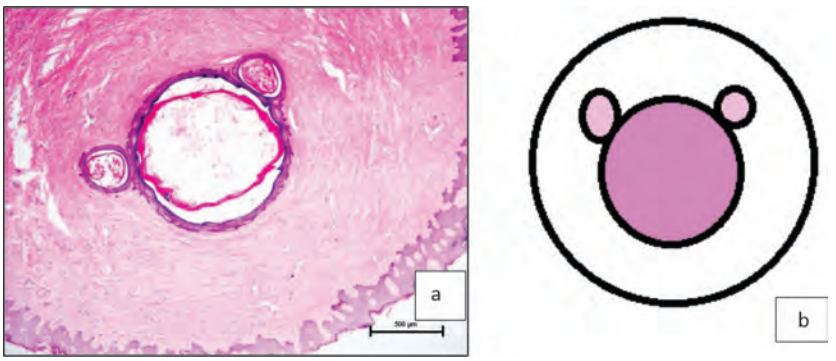

Fig. 6. Photomicrograph (a) and schematic diagram (b) showing triple orifices in goat teat at 40x.

C. According to location, there are three types of supernumerary teats

1. **Caudal SNT:** These are present at the rear of the normal teat (Fig. 7a). In cow, the caudal type may have four supernumeraries, one pair just posterior to the normal teat, and a second pair posterior to the first pair of supernumeraries and placed well up on the rear quarters of the udder, but may be near the median line.
2. **Intercalary SNT:** These are found between the normal teats (Fig. 7b). The intercalary SNT are attached to the floor of the udder and may occur at various points between the normal teats on each half of the udder. They are usually in line with the two normal teats on each half of the udder.
3. **Ramal or the supernumeraries SNT:** These had ramifications and branches of the normal teats. The ramal type appeared at the base of the normal teat or on the side of the teat and may be attached on the inner or outer position i.e. caudal or cranial to the normal teat (Fig. 7c).

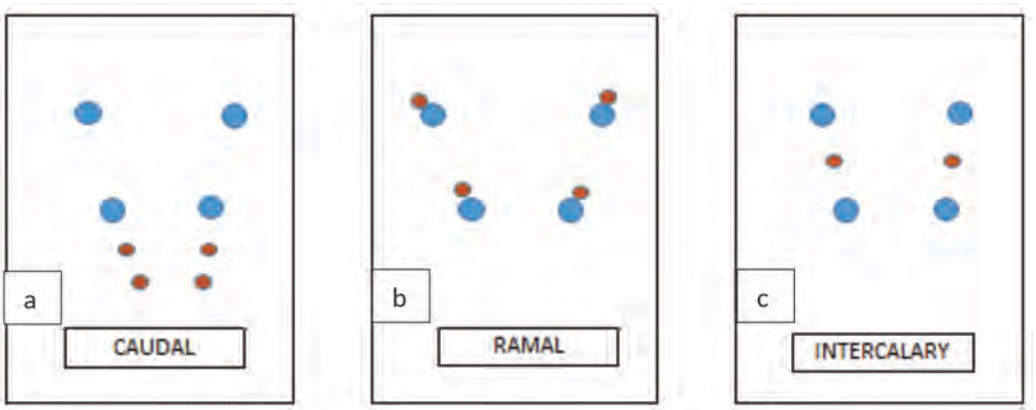

Fig. 7. Classification of Supernumerary teats as per location. Normal teat (Blue colour) and SNT (red colour).

Available literature showed that the caudal SNT are more as compared to intercalary or ramal types in dairy cow, whereas the cranial SNT (>86%) are more common in goat (Famkinde et al., 2019)

Complications of Supernumerary Teats

1. Suckling of non-functional teats may lead to lack of passive immunity in young ones and such animals are more susceptible to diseases which may cause neonatal death.
2. Animals with SNT are more prone to ascending infections through the external orifice of the extra teat which may cause mastitis.
3. Since the occurrence of SNT has been associated with some recessive homozygous genes that can be passed down from one generation to another, so the veterinarians and breeders should be careful while selecting the animals for breeding purposes.

It may be concluded that early diagnosis of SNT is very important for prompt interventions and treatment. Surgical removal of SNT can be done as an early age of the animal or it can be used to replace the damage primary teat by teat grafting.

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BIOSECURITY IN POULTRY OPERATIONS

Garima Chahar and Sidhartha Deshmukh*

Department of Veterinary Pathology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab- 141004.

*Corresponding author email: sid0159@yahoo.com

Received: May 2023 Accepted: May 2023

Abstract

One of the most crucial aspects of the environment for commercial poultry production is safeguarding flocks from microbial invasion. It could have major economic repercussions on production, if a highly contagious, harmful pathogenic organism is introduced into flocks of chickens. The effectiveness of a biosecurity program can be optimized by regional participation. Biosecurity measures include adoption of some essential safety measures that ensure clean poultry production with least possible spread out of infections, besides insuring socio-economic prosperity and growth. Here, this technical article attempts to bring closer some important information including the steps of biosecurity which can easily be implemented to ensure quality production with least possible threats to human and animal health.

Keywords: *Biosecurity, economic, management, poultry.*

Poultry represents an important sector in animal production with backyard flocks representing a huge majority. In order to address the rising population worldwide, demand for animal-sourced foods, high yielding commercial varieties of chicken have risen dramatically. Production of backyard (traditional) poultry, or tiny flocks with some biosecurity precautions, is common in developing nations like India to meet the household food demands and has improved income generation manifold.

High mortality rates are caused by infectious diseases like Newcastle disease, Salmonellosis, Gumboro disease, and fowl typhoid in poultry farms, especially backyard flocks (Conan et al., 2012), where free native unselected breeds of different ages are frequently mixed in the same flock along with subpar measures and inadequate management practices. Low biosecurity standards are implied by backyard production techniques, which can be a source of zoonotic infection.

Biosecurity, which literally means safety of living things in which “Bio” refers to life and “Security”, refers to protection. Biosecurity is an integrated strategy that includes authentic and legislative frameworks for assessing and controlling environmental, animal health and food safety threats (Department of Animal Husbandry, Dairying and fisheries Ministry of Agriculture and farmers’ Welfare Government of India, 2015). According to Siekkinen et al. (2012), biosecurity entails all steps taken to exclude and eradicate infections and effectively control the risks posed by those pathogens.

Aim of Biosecurity

The three main pillars of biosecurity are isolation, traffic control, and sanitation. Biosecurity is, and should be the most primary objective of any poultry farm operations as they make sure to restrict microbial loads, contamination and their dissemination. Thus this basic fabric of farming practices benefits:

- Reduction of microbial contamination of surrounding area.
- Prevention of entry of pathogenic organism into poultry premises.
- Keep way contagious diseases like IBD, ND out of the poultry farm.
- Help to reduce the occurrence of common vertically transmitted disesaes like salmonellosis, MD, mycoplasmosis, chicken infectious anemia (CIA).
- It minimizes or eliminates background immunosuppressive agents such as IBD virus.
- It confers greater food safety security and removes fear of zoonotic spread of diseases.
- It attempts to reduce the risk of continual disease outbreak.

Benefits of Biosecurity

Good biosecurity measure is therefore the vital ingredient for the poultry livestock industry to attain the following benefits:

- Improves overall health of the flock.
- Reduces mortality losses and improves profitability.
- Cut down the costs of disease treatment.
- Improves production performance and less production cost is needed.
- Helps to keep the farm disease free.
- Lower the risk of pathogens being transferred from farm to farm.

Thus, Biosecurity is the set of sanitary and preventive measures involving 3 major steps (Shane, 2005): -

1. Conceptual Biosecurity.
2. Structural Biosecurity.
3. Operational Biosecurity.

1. Conceptual Biosecurity

It is primary level of biosecurity, revolves around the location of animal facilities and their components.The most effective way to limit risk is physical isolation and it should be the primary consideration while planning a new confinement facilities or farms. According to OIE guidelines the following steps should be taken into consideration under

conceptual biosecurity:

- Ideally, the new facilities/ farms should not be located in close proximity to other farms or public roads or next to slaughterhouses, live animal markets, agricultural fairs or animal exhibits.
- There should be a strict isolation method in practice which include limiting the use of common vehicles and facilities; limiting access by personnel not directly involved with the operation and controlling the spread of disease by vermin, wild animals and wind.
- Proper distance among farms, hatcheries, processing units.
- Proper connectivity with roads and proximity of water supply.

2. Structural Biosecurity

It is the secondary level of biosecurity, deals with physical factors such as farm layout, perimeter fencing, drainage, number/location of charging rooms, presence of showers, air filtration systems, enclosed load outs and housing design in general (*Figure 1 and 2*). According to OIE guidelines following steps should be taken into consideration while designing structural biosecurity:

- Fencing of farm to avert trespass.
- Movement restriction in general at farm level and at poultry shed level.
- Secure housing with suitable rodent proofing, concrete floors

Fig. 1. Well ventilated and nicely fenced perimeter allowed free ranging facilities to poultry birds (Photo by Sidhartha Deshmukh)

Fig. 2. Nicely maintained housing (caging) facilities that allowed optimal sun drying provision and day light oppurtinity (Photo by Garima Chahar).

- Correct positioning of exhaust fans to prevent air borne diseases, proper ventilation.
- Ensuring water supplies to farm free of pathogens and chlorinated content [2ppm].
- Proper power supply and water supply to perform operations of decontamination of vehicles entering the farm.
- Installation of bins for pest free storage of bagged feed.
- Installation for disposal of dead birds.

3. Operational Biosecurity

It is the tertiary level of biosecurity, includes proper disinfection and decontamination facilities, personnel hygiene, proper disposal of biomedical waste, feed safety, water quality treatments, vaccination of birds, health related record keeping, quarantine facilities etc. "Biosecurity includes the concept and measures of preventing introduction and spread of new infectious agents into flocks ('*Bioexclusion*') and the potential need to reduce the risk for flocks to spread disease to others ('*Bio containment'*)" (Thieme, 2007). According to OIE guidelines following steps should be taken into consideration while formulating operational biosecurity:

- It includes taking a shower or changing footwear and personal clothes with farm dedicated clothes before entry into the farm, washing hands and disinfecting equipment at point of entry.

- Scheduled and periodic decontamination and disinfection of units.
- Stringent follow up of isolation and quarantine of new birds.
- Strict control for prevention of contact with exotic and backyard poultry.
- Follow up of proper vaccination schedule at all level of poultry keepings.

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FPO- A WAY FORWARD FOR LINKING FARMERS WITH MARKET

Muskaan, Rajesh Kasrija* and Amandeep Singh

Department of Veterinary and Animal Husbandry Extension Education,
College of Veterinary Science (Ludhiana),
Guru Angad Dev Veterinary and Animal Sciences University, Punjab
*Corresponding author email: mjaggagadvasu@gmail.com

Received: April 2023 Accepted: May 2023

Abstract

Farmer Producer Organization (FPO) is one of the major initiatives of Government of India which help the farmers to organize themselves in to a group, provides a business outlook to the farming system, and links farmers with the market so as to improve their socio-economic status. The present article provides an overview of challenges faced by farmers, FPO, FPO promoting and funding institutions, benefits to members of FPO and challenges faced by the FPO. FPO seems to be an excellent model for improving socio economic status of the farmer.

Key words: *Agriculture, Farmer, FPO, Market,*

Agriculture and allied sectors play vital part in Indian economy and major source of livelihood in rural areas. The allied sectors include livestock, fisheries, aquaculture, forestry and logging. These sectors are now growing exponentially and contributing to better farm incomes. The livestock sector grew at a Compound annual growth rate (CAGR) of 7.9 per cent during 2014-15 to 2020- 21, and its contribution to total agriculture GVA has increased from 24.3 per cent in 2014-15 to 30.1 per cent in 2020-21 (Economic survey 2022-2023).

Major population of Indian farmers are small and marginal farmers. Thus, such a small holding make livelihood very difficult. Also, a large chunk of population is directly or indirectly linked with livestock sector. At rural level, almost every household has 2-3 livestock. Livestock products (milk, meat and other value added products) are equally relished by urban and rural people. The livestock sector contributes significantly in spawning employment opportunities and supplementing the income of small and marginal farmers. But, still the farmers are facing many challenges in farming, which lead to less income in comparison to expenditure/investment in farming.

Challenges faced by small farm holders in Agriculture and Livestock sector:

1. Drought, climate change, monsoon failure
2. Crop failure
3. High cost of input (if purchased in small quantities)
4. Perishable nature of livestock produce
5. Forced sale of produce (low in quantity) to middlemen.

6. Lack of knowledge of market and poor marketing skills
7. Inadequate human resource,
8. Lack of resources
9. Less credit available
10. Lack of extension services
11. Low level of technology adoption
12. Low income due to poor infrastructure

In response to the crucial concerns of farmers and agriculturalists, in 2002 the idea of a "Producer Company (PC)" was introduced. An expert panel led by Y.K. Alagh, an eminent economist, had amended the Indian Companies Act, 1956, in 2002-03 to provide for "producer companies".

What is farmer producer organisation?

Farmer Producer Organisation (FPO) is legal entity whose members have come together to improve farm profitability through enhanced marketing, local processing, and production. Members are mainly primary producers such as farmers, milk producers, fishermen, weavers, rural artisans, craftsmen etc. A PO can be a producer company, a cooperative society orany other legal form which provides for sharing of profits/benefits among the members.(Anonymous 2015)

Central Sector Scheme- "Formation and promotion of 10,000 Farmer Producer Organizations (FPOs)"

Sh. Narendra Modi, Hon'ble Prime Minister, launched the new Central Sector Scheme- "Formation and Promotion of Farmer Producer Organizations (FPOs)" for formation of 10,000 new FPOs in the country at a mega event on 29th February, 2020 at Chitrakoot, Uttar Pradesh.Salient features of this scheme are:

1. It will be formed by Cluster Based Business Organizations (CBBOs) and handholding and professional support will be provided for a period of five years to make them economically sustainable.
2. Priority will be given to the Aspirational Districts by way of forming at least one FPO in every block.
3. Efforts will be made to adopt "One District One Product" model.
4. Provision of matching Equity Grant of Rs. 2000/- per farmer subject to the ceiling of Rs.15 lakh/FPO.
5. Provision of Credit Guarantee up to the project loan of Rs.2.00 Crore per FPO.

FPO Promoting institutions

Promoting institutions play vital role in the formation, promotion and functioning of FPOs. In very initial years, promoting institute helps FPOs in documental procedure, provision of technical and financial support in the functioning of the FPO. The performance of FPOs in the state highly depends upon the number of promoting institutions. The funding institutions like NABARD (National Bank for Agriculture and Rural Development) and SFAC (Small Farmers' Agribusiness Consortium) provide fund support to promoting institutions for enabling them to mobilise farmers and to form FPOs (Manaswi et al*.,* 2018).

FPO promoted by GADVASU, Ludhiana

Name of FPO	District	Promoting Institution
Gharyala Dairy Farmer Producer Company Limited	Tarn Taran	Krishi Vigyan Kendra, Tarantaran
Neeli Kranti Aqua Farmer Producer Company Limited	Barnala	Krishi Vigyan Kendra, Barnala
Mushkabad FAM Dairy Producer Company Limited	Ludhiana	Directorate of Extension Education, GADVASU, Ludhiana

Benefits to members of farmer producer organisations:

a) **Better farm income:** An FPO can guarantee a higher farm income for farmers by first evaluating demand and then buying in bulk. Additionally, controlling transportation expenses through bulk shipping lowers the entire cost of production. The FPO may also combine the produce from all of its members and sell it in bulk for a higher price per unit (Rashtrapal et al., 2022)

b) **Information about market:** The FPO can also notify farmers about the market, enabling them to hold onto their produce until the market prices increase. The revenue of primary farmers will rise attributed to all of these interventions.

c) **Access to credit and finance:** FPOs also provide access to credit and finance, which is often a major challenge for small and marginal farmers. Provide loans for crops, livestock, machines, purchase of tractors, pump sets, construction of wells, laying of pipelines, sheds etc.

d) **Market accessibility:** Market access is ensured through the purchase of members' produce, and the purchasing activity also includes quality control and pricing of raw materials. At FPO level, various inputs such as concentrate feed and different medicines can be procured at a cheaper price under bulk order for its members.

e) **Social empowerment and upliftment:** When farmers come together, they socialize, share, and learn from each other leading to building of confidence and social upliftment. Thus, they are socially, economically and managerially more empowered as compared to non-members of FPO.

f) **Networking:** The FPO will facilitate linkages between farmers, processors, traders, and retailers to coordinate supply and demand and to access key business development services such as market information, input supplies, and transport services.

g) **Insurance:** FPO provides various insurances to its member such aslife insurance, crop insurance etc (Khan et al., 2020).

Challenges faced by FPOs

A. **Limited institutional support:** FPOs often struggle to access institutional support from government agencies and other organizations, which can provide them with technical assistance, training, and other resources.

B. **Internal governance and management issues:** FPOs face challenges related to governance, management, and leadership, which can impact their ability to function effectively and achieve their objectives.

C. **Limited access to technology:** FPOs often struggle to access the latest agricultural technologies and equipment that can help them increase productivity and reduce costs.

D. **Limited technical knowledge and skills:** FPOs often lack the technical knowledge and skills needed to improve their agricultural practices, increase productivity, and improve the quality of their produce.

E. **Inadequate market linkages:** FPOs often struggle to find reliable buyers for their produce. This is especially true for FPOs that operate in remote or rural areas where markets are not well developed on other side well established markets are at distance.

F. **Limited access to finance:** FPOs often struggle to access affordable credit, which is essential for scaling up their operations, buying inputs, and investing in infrastructure. Many FPOs are not able to meet the collateral requirements of banks and financial institutions**.**

G. **Lack of infrastructure:** Many FPOs operate in areas with inadequate infrastructure, including roads, power, and water supply, which can make it difficult to transport produce and store it properly. Lack of godowns/storage facility is one of major challenges faced by FPOs.

H. Lack of transportation vehicle: Lack of vehicle greats great hindrance in efficient working and renting the private vehicles leads to more financial expense hence less profitability.

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Answer to the CROSSWORD
(Quiz on Page no. 58)

[1]E	P	[2]I	N	[3]E	P	[4]H	R	I	[5]N	E	■	[6]A	[7]D	H
S	■	N	■	N	■	I	■	■	I	■	[8]I	■	O	■
T	■	[9]S	[10]A	T	E	L	L	[11]I	T	E	C	E	L	[12]L
[13]R	O	U	L	E	A	U	■	N	■	■	T	■	[14]O	O
O	■	L	O	R	■	[15]S	A	L	I	[16]N	E	■	R	T
G	■	I	E	O	■	■	[17]F	E	V	E	R	■	■	I
E	■	N	■	[18]K	[19]R	■	■	[20]T	A	L	U	[21]S	■	O
[22]N	[23]U	■	[24]R	I	B	■	[25]D	■	■	[26]A	S	E	■	N
■	R	■	■	N	C	■	[27]H	[28]A	I	R	■	[29]N	[30]G	■
[31]G	A	M	M	A	■	[32]S	U	L	F	A	■	[33]I	B	[34]D
L	T	■	■	S	■	■	R	U	■	[35]B	[36]A	L	■	U
A	U	■	[37]P	E	[38]S	■	[39]R	M	■	[40]I	C	E	■	N
N	R	■	U	■	[41]T	H	I	■	[42]D	N	U	■	[43]P	■
[44]D	I	■	[45]G	L	Y	■	N	■	[46]P	E	T	■	[47]I	G
■	[48]A	B	■	[49]L	E	T	■	[50]E	T	■	[51]E	G	G	■

TECHNIQUE OF CHEST TUBE PLACEMENT FOR PNEUMOTHORAX IN DOGS

Nikita Gupta, Ashwani Kumar and Vandana Sangwan*

Department of Veterinary Surgery and Radiology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab-141004, India

*Corresponding author email: sangwanvandana1@gmail.com

Received: June 2023 Accepted: June 2023

ABSTRACT

Chest tube or thoracostomy tube placement is necessary for the treatment of certain pleural cavity affections in dogs like pneumothorax, chylothorax, pyothorax, hydrothorax and hemothorax, when these are unmanageable by needle thoracocentesis. On emergency basis, chest tube needs to be placed for pneumothorax due to trauma mostly and a regular suction of air is required through it. This article describes the technique of chest tube placement, along with its potential complications for pneumothorax in dogs.

Keywords: *Chest tube, dogs, pneumothorax, technique*

Chest trauma in dogs might lead to mild to severe pneumothorax due to rib fractures or blunt lung injury. Mild pneumothorax may not exhibit clinical signs of respiratory distress while moderate to severe may lead to emphysema and life threatening distress. Radiographs in lateral and VD/DV view are helpful in assessing the severity of pneumothorax. The moderate to severe pneumothorax need chest tube placement for intermittent suctioning of air from chest as needle suctioning may not be sufficient. If unilateral affection is present, then the placement of chest tube is preferred from the affected side.

Practically, in field conditions also, two types of chest tube may be used for the procedure: baby feeding tubes or the commercially available silicon tubes with or without stellate (Ryle's chest tube) or silicon Foley's catheter. But since these allows passive drainage and no suction cup is attached to them, they are less aseptic. These tube need to be closed with a 2/3 way stop cock or by some other method whichever is applicable when not in use to avoid sucking environment air inside. The size of the tube is chosen to approximate the radiographically estimated diameter of the main stem bronchi (Fossum, 2018), as large size tube might cause pain and discomfort.

Materials Required

1. Sterile new Chest tube/baby feeding tube/Foley's catheter, silicon tubes/Ryle's tube
2. Syringe 20ml and 2ml
3. Sterile Surgical Gloves
4. NSS
5. Local anesthetic/sedation
6. Betadine

TECHNIQUE

Pre-surgical preparation

- The procedure might require sedation in furious dogs but is risky sometimes in recently traumatized dogs.
- Restraint the dog in lateral position with the side where tube is to be placed on dorsal.
- It is highly aseptic procedure. So clip the hair over the dorsal chest (all ribs).
- In-filter local anesthetic inj. Lignocaine hcl 1-2ml on the dorsal aspect i.e. upper one-third of 10^{th} intercostal space (ICS) and 2-3 ICS cranial (7^{th} or 8^{th} ICS) as well from where it is intended to insert the tube into the chest.
- Scrub the site with Savlon or Betadine 2-3 times.
- Open the chest tube aseptically in the pack or an assistant can open it for you during placement. A sterile syringe with 20ml normal saline or sterile distilled water should be kept loaded to distend the bulb of Foley's in-case it is being used.
- Wear sterile gloves.

Chest Tube Placement

- With the help of a scalpel blade, make a small superficial incision on the skin in the dorsal one third of the 10^{th} ICS where local has been given.
- Insert the chest tube from the incision site with the help of an artery forceps/groove director or stylet fixed to its tip and move it subcutaneously 2-3 ICS cranially (7^{th} or 8^{th} ICS), from where the tip of the tube in stabbed into the pleural space with the help of same artery forceps or stylet (Fig.1). The tube insertion into the chest is done cranial to the skin incision to avoid direct exposure of chest to the environment and to prevent the entry of air along the tube, into the pleural space.
- The outside end of the tube should be closed using a 3-way stop cock or tube should be clamped near the chest wall while removing stylet to prevent iatrogenic pneumothorax.

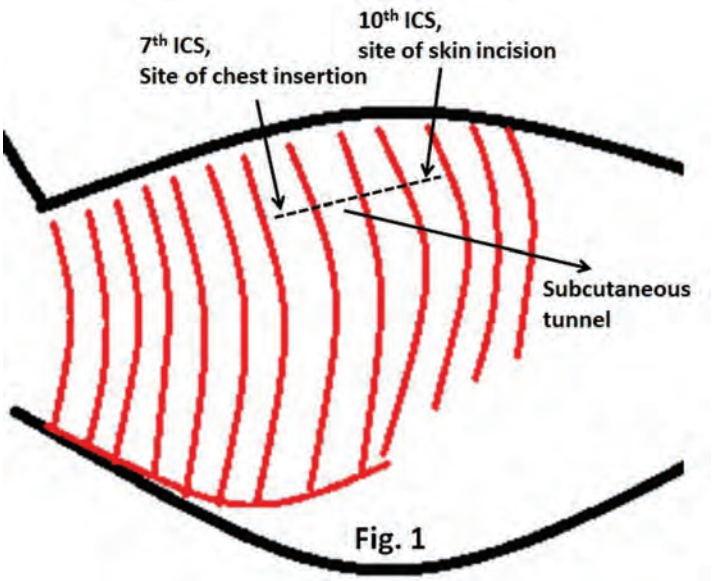

Fig. 1. Schematic diagram showing points of chest tube placement in dog for pneumothorax.

- The tube at insertion site into the chest is blindly secured with a purse string suture and also at the skin incision site with a Chinese finger trap. If using a Foley's catheter, inflate the bulb of the tube along with required normal saline/ distilled water.

Post-operative Care and Suctioning

- The skin incision should be dressed with betadine and a padded bandaging is done over the chest (Bexfield & Lee, 2014).
- The air can be suctioned intermittently using a 3-way stop cock as it is efficient in preventing the entry of atmospheric air and should be drained till negative resistance is felt. A low pressure suction apparatus may be used or if not available a 50ml/20ml syringe may be used through the 3 way stop cock.
- The dog is put in sternal recumbency, once the procedure is complete.
- Post-operative care includes; antibiotics and analgesics as chest tube placement can lead to pain (Day, 2014).
- The residual air after suction can be monitored with X-rays.
- Most of the cases of pneumothorax may require frequent suctioning of air through this tube for the initial few days till the wound in the lungs starts healing and shrinking and air leakage is reduced. The frequency of suction will decrease with healing.

Tube Removal

- The tube can be removed once the requirement for suctioning is reduced to once daily and for a short time only. It is usually 4-5 days for large pneumothorax and 12-24 hours for small ones.
- Check the amount of air using x-rays before removing the tube and the general condition of the dog/respiratory distress.
- Dress the wound from where the tube is removed and may apply one stitch on skin if required.

Potential Complications

- All invasive procedures are associated with complications. Iatrogenic pneumothorax while tube placement, lung laceration, hemorrhage in cases where a blood vessel may be ruptured while placement (Bexfield & Lee, 2014), subcutaneous emphysema, edema or cellulitis due to damage of tube along the tunnel or near the chest wall, may be due to bite of the dog (Kahn, 2007; Day, 2014) are a few potential complications of chest tube placement in dogs.
- Proper asepsis and careful surgical maneuver can avoid many complications.

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ETHNOMEDICINAL APPROACH IN THE MANAGEMENT OF UROLITHIASIS (*ASHMARI)*

Milindmitra K. Lonare*, Rashmi Sagar Bhullar & Santosh S. Mane

Department of Veterinary Pharmacology and Toxicology, College of Veterinary Science (Ludhiana), Guru Angad Dev Veterinary and Animal Sciences University, Punjab

*Corresponding author email: milindmitra@gmail.com

Received: May 2023 Accepted: May 2023

Abstract

The ancient Indian medical system or Ayurveda, is based on a "natural" and holistic approach to physical and mental health. Ethno-veterinary medicine is an alternative treatment option using medicinal plants and herbs locally available in particular. It is a cost-effective, efficacious, and farmer-friendly option to manage common ailments of bovines and other species of animals. However, common problems of the urinary system and kidney are urinary crystals and bladder stones, urinary tract obstruction and failure, and extend to cancer in small and large animals. These diseases give rise to clinical conditions like haematuria, urinary calculi, polyurea, painful micturition, etc. These conditions are usually treated with anti-inflammatory agents, urinary alkalizer, antibiotics, and surgical interventions. Ethno-veterinary medicine would be a good alternative means to treat urinary disease conditions by replacing modern medicine. Thus, proven medicament documented in Ayurveda can be safely explored for the prevention and treatment of animals.

Keywords: *Ayurveda, ethno-veterinary, haematuria, polyurea, urinary calculi*

Urinary tract diseases associated with infectious or non-infectious causes and the most common illnesses in animals. The concept of the urinary system is explained in *Ayurveda* very widely under the section of *Mootravaha Srotas*. They are broadly classified into two conditions i.e. *Mutraghata* (retention of urine) and *Mutrakruccha* (Strangury or dysuria/ passage of urine with difficulty). The clinical signs and symptoms like *Raktamutrata* (haematuria), *Bahumutrata* (polyuria), *Sadah-Mutrata* (burning micturition), *Alpamutrata* (oliguria), etc. Moreover, *Mootravaha Srotas* (urinary system) plays an important role in the manifestation of diseases like *Ashmari* (urinary calculi), *Prameha* (Diabetes mellitus) and *Medoroga* (obesity), etc. These disease conditions can be treated by intervention of traditional system of medicine with holistic approach known as Ayurvedic medicine (Ramdas & Ghotge, 2005). Many herbal medicines have been documented in ancient script related to conditions of urinary tract diseases.

Urolithiasis is a one of the common conditions of urinary tract diseases. This is broadly covered under the term *Ashmari* in Ayurveda. Urolithiasis is also known as urinary calculi. It may cause intense pain, nausea, vomiting, hematuria, and, possibly, chills and fever due to secondary infection. Urinary calculi are caused by renal tubular

acidosis and calcium calculi have primary hyperparathyroidism in dogs and cats. Rare causes of hypercalciuria are sarcoidosis, vitamin D intoxication, hyperthyroidism, multiple myeloma, metastatic cancer, and primary hyperoxaluria in small animals (Brown, 2022). Heavy heat, constipation, diseases of the liver, elevated urinary oxalate levels, excess work may lead to the calculi. Other risk factors include taking high doses of vitamin C, a calcium-restricted diet, and mild hyperuricosuria (Kasote et al., 2017). Clinical findings include excess thirst, bloating, dribbling of urine drop by drop with yellowish or reddish micturition, pain, discomfort, wasting and death. Pain in the flank or kidney area that radiates across the abdomen suggests upper ureteral or renal pelvic obstruction, while along the course of the ureter into the genital region suggests lower ureteral obstruction. Further, pain along with urinary urgency and frequency suggests a distal ureteral, ureterovesical, or bladder calculus (Nirumand et al., 2018).

Management

- Give hot water enema as a usual common treatment for urolithiasis in small animals.
- Orally administer diuretic drugs like potassium nitrate, the root of *Rheum emodi* (*Amlavetasa*, *Rewandchini*) and coconut water, etc. regularly to the small animals.
- The regular administration of root (15-20g) decoction of *Boerhavia diffusa* (*Punarnava*) at dose of 30-40 ml twice or thrice a day is an effective diuretic remedy, it helps to flush out kidney stones, reduces water retention, helps to reduce swelling and pain of kidney. Or another effective way is, the use of *Punarnava* seed powder (1-2 g) or its decoction 30-40 ml taken regularly for 10-12 days helps to flush out the urinary calculi in small animals.
- *Crateva religiosa* (*V*aruna) is effective in the treatment of renal conditions, infection, and enlarged prostate. Prevent formation of oxalate-type kidney stones and helps to reduce inflammation and pain during stone formation. Dry powder of Varuna flower and leaves (3 grams) boil in water, filter, and drink regularly.
- One can prepare a powder with a mixture of 25 g *powder of Varuna* (*Crateva religiosa*), 25 g of *Gokhru* (*Tribulus terrestris* L.), 10 g of *Giloy* (*Tinospora cordifolia*), 10g of *Sarpunkha* (*Tephrosia purpurea*) leaves, 10 g of *Kantakari* (*Solanum anguivi* L.), 10g of *Shatavari* (*Asparagus racemosus*), and 10 g of *Sonth* (*Zingiber officinale*). This mixture can be administered in a dose of 5 g or 1 spoon of powder with water, twice a day regularly to small animals and in a dose of 15gm/100kg body weight for large animals.
- *Cucumis sativus* (Cucumber) is a preventive and curative regimen for the treatment of kidney stones. Take 6-10 cucumber pieces and crush them to obtain juice and add 3-5 g sugar candy and a pinch of cardamom powder. Taking this mixture 2-3 times a day before food regularly will neutralizes toxins and cleanses

Table 1: List of medicinal plants and their recommended dose and parts to be used for urolithiasis

Sr. No.	Botanical name and family	Part used	Oral Dose/mode preparation
1.	Apiumgra veolens L. (Wild Celery. Ajmod, Ajwain-ka-patta)	Root	5–7 g, powder
2.	Anogeissus latifolia Wall. ex Guillem. & Perr (dhau, dhawa, dhawra, or dhaora)	Stem bark	30–50 ml, decoction
3.	Beninca sahispida (Thunb.) Cogn. (Cucurbitaceae)	Fruits	5–10 g, powder
4.	Aerva lanata (L.) (Gorakhdi, Kapurimadhuri)	Whole plant	50–100 ml, decoction
5.	Anisomeles malabarica (L.) (Malabar catmint)	Whole plant	3–5 g, powder
6.	Asparagus officinalis L. (Liliaceae)	Root	3–6 g, powder
7.	Baliospermum solanifolium (Burm.) Suresh (Euphorbiaceae)	Root	1–3 g, powder
8.	Butea monosperma (Lam.) Taub. (Lam.) (Leguminosae)	Seed	0.5–1 g, powder
9.	Cassia fistula L. (Leguminosae)	Stem bark	50–100 ml, decoction
10.	Calamus rotang L. (Arecaceae)	Rhizome	50–100 ml, decoction
			5–10 g, powder
11.	Carica papaya L. (Caricaceae)	Root	2–6 g, powder
12.	Celosia argentea L. (Amaranthaceae)	Seed	5–10 g, powder
13.	Calamus rotang L. (Arecaceae)	Rhizome	50–100 ml, decoction
14.	Carthamus tinctorius L. (Compositae)	Fruit Leaves	2–4 g, powder
16.	Dalbergia sissoo DC. (Leguminosae)	Heart wood	5–10 g, powder
			10–20 g, decoction
17.	Garcinia pedunculata Roxb. ex Buch.-Ham. (Clusiaceae)	Fruit	5–10 ml, juice
18.	Diospyrosm alabarica (Desr.) Kostel. (Ebenaceae)	Fruit	5–10 g

Sr. No.	Botanical name and family	Part used	Oral Dose/mode preparation
19.	Dendrophthoe falcate (L.f.) Ettingsh. (Loranthaceae)	Fruit Leaves Stem Root flowers	10–20 ml, juice
20.	Hygrophila auriculata (Schumach.) Heine (Acanthaceae)	Roots	3–6 g, decoction
21.	Hyoscyamus niger L. (Solanaceae)	Seed	125–500 mg, powder
22.	Moringa oleifera Lam. (Moringaceae)	Root bark	25–50 g, powder
23.	Momordica dioica Roxb. exWilld. (Cucurbitaceae)	Root	3–6 g
24.	Imperata cylindrica (L.) Raeusch. (Poaceae)	Root	10–20 g, decoction
25.	Ocimum tenuiflorum L. (Lamiaceae)	Whole plant	1–3 ml, juice
26.	Phyllanthus acidus (L.) Skeels (Phyllanthaceae)	Fruit	10–20 g
27.	Saccharum spontaneum L. (Poaceae)	Roots	3–6 g, powder
28.	Salvadora persica L. (Salvadoraceae)	Fruits Leaves Root bark	3–6 g
			3–6 g
			10–20 g, decoction
29.	Sesamum indicum L. (Pedaliaceae)	Seeds	5–10, powder
30.	Sesbania bispinosa W. F. Wight (Leguminosae)	Root	3–6 g
31.	Stereospermum chelonoides (L.f.) DC. (Bignoniaceae)	Roots	5–10 g, powder
			25–50 ml, decoction
32.	Tribulus terrestris L. (Zygophyllaceae)	Root Fruit	20–30 g, decoction
			3–6 g, powder
33.	Vallaris solanacea Kuntze (Apocynaceae)	Root	3–6 g, powder
34.	Typha elephantina Roxb. (Typhaceae)	Root	10–20 g, decoction
35.	Vigna unguiculata (L.) Walp. (Leguminaceae)	Seeds	12 g, decoction
36.	Typha australis K. Schum. & Thonner (Typhaceae)	Rhizome root	3–6 g, powder

urinary crystals (Exploration of veterinary practices in Ayurveda-2008; Oburai et al., 2013; Patankar et al., 2010; Rathod et al., 2012; Nirumand et al., 2018; Kieley et al., 2008).

- Additional list of Ayurvedic medicinal plants and their parts documented for the treatment of urinary calculi for small animal and human practice are enlisted in Table 1 (The Ayurveda Pharmacopoeia of India. Part I-VI. 2004, 2009; Kasote et al., 2017).

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