Common congenital surgical affections in large ruminants

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Abstract
The common congenital surgical problems in calves includes atresia ani, atresia ani et recti, atresia ani et recti et coli, atresia ani with rectovaginal fistula, umbilical hernia, contracted tendon, eventration of intestine, pervious urachus, meningocele, patellar luxation, lip and palate defects, maxillo-mandibular and ocular defects. Surgical treatment of such calves is of utmost significance for their survival. The present review deals with common congenital surgical problems in bovine and their surgical management.

Keywords: Common congenital, surgical, atresia ani

Introduction
Congenital Abnormality is defined as defect in structure and function, which are present at birth (Badaway, 2011) [7]. Congenital malformations can result from defective genetics or environmental factors or a combination of both (Shukla et al., 2007) [55] and are mainly by virtue of autosomal recessively inherited genes (Bryan et al., 1993) [12]. Congenital arthrogryposis in calf has also been reported by ingestion of plant genus Lupinus by pregnant cows (Shupe et al., 1967) [56] and infection by viruses of the family Bunyaviridae (Green et al., 2015, Sprake, 2015) [26, 59]. But still the cause of many congenital defects is unknown (Mamta et al., 2014) [37]. Congenital abnormalities occur more in animals during 4–8 weeks of foetal life (Sharma et al., 1986) [53]. Recognized environmental factors include nutritional deficiencies, teratogenic drugs or chemical exposure, some viral infections, toxic plant ingestion, x-rays and per rectal examination during the early stage of organogenesis (Rafid, 2010) [48]. Most of congenital defects need immediate surgical intervention while some congenital defects get importance at the time of production or reproduction. These include affections of teat (Fig. 1a & b) and vagina (persistent hymen). By restricting the breeding of animals with congenital surgical condition that have complete surgical treatment like umbilical hernia, previous urachus etc. can reduce incidence of congenital affections in subsequent progeny. However, inheritability of any condition should be ascertained. Animals with congenital defects should not be bred but their survival can be improved by timely surgical intervention. Some frequently encountered congenital conditions are described briefly as below.

Rectal defects
Defects of rectal or anus are the predominately recorded as congenital gastro-intestinal malformation in ruminants. These mainly includes atresia ani (Imperforate anus), atresia ani et recti, atresia ani et recti et coli, atresia ani with rectovaginal fistula. According to Durmus...
Atresia ani is defined as absence of anal opening since birth. This congenital defect is observed as soon as birth takes place. This condition may occur alone or seen associated with agenesis of other parts like atresia recti, rectovagial fistula, vagino urethral agenesis hypospadias, cleft scrotum. Atresia ani is a congenital abnormality characterized by persistence of the anal membrane resulting in a thin membrane covering the normal anal canal (Noden and Lahunta, 1985) [41]. This condition have been found associated with abnormal chromosomes and interruption of the foetal blood supply to the anus failure of the anal membrane to become perforated, failure of the bowel to become canalized that leads to atresia ani (Singh et al., 2020) [58] but sometimes occur with umbilical hernia (Fig. 2b) also. Atresia ani is more common in male than female (Singh, 1989) [57]. Most of cases of atresia ani are presented within two days of birth. Clinical signs include absence of anal opening (Fig. 2a), suspended defecation, tenesmus, occasional straining, mild to moderate distention of abdomen, bulging at normal anatomical site of anus when abdomen is compressed or during straining to defecate. This ballooning (Fig. 2c) is more evident when rectal wall defect is present at caudal part of rectum. In case of atresia ani et recti, blind end does not bulge out on applying abdominal pressure and the defect is usually located near the pelvic brim (Singh et al. 2020) [58]. Both atresia ani or atresia ani et recti may be hereditary and due to single autosomal recessive gene. Atresia recti and ani can occur with atresia coli. Durmus (2009) [19] observed low survival rate in calves suffering from atresia coli and repaired by laparotomy might be due to peritonitis.

**Fig 2:** (a) Showing atresia ani in cow calf. (b). Showing both umbilical hernia (Arrow) and atresia ani (After surgery) (c) Showing bulging at site of anus in buffalo calf (d) disposable syringe barrel sutured with skin.

Treatment includes surgical intervention under local infiltration analgesia with or without sedation. Sedation may be achieved using xylazine hydrochloride in calves (Durmus, 2009) [19]. Circular incision of 2-3cm (Singh et al., 2020) [58] is made below the base of tail where bulging is most prominent while straining or on pressing the abdomen. Blind end of rectum is identified, isolated and opened to evacuate mucous. Then rectal wall was sutured with the skin to make artificial opening. Major complication is stenosis of anal opening due to cicatrisation during healing. Some times stenosis is also observed if diameter of opening is adequate and to prevent stenosis, disposable syringe barrel (5ml) of 5-6 cm in length, can be put inside artificial opening should be duly sutured by black braided silk with skin (Fig. 2d) after smoothening of cut edges to avoid injury to rectal mucosa.

**Umbilical hernia**

An umbilical hernia is a discontinuity of the abdominal wall at the umbilicus with protrusion of abdominal contents into hernia sac formed by the skin and surrounding connective tissue. Generally intestinal loops are main organ to form the content of hernia. Due to hypoplasia and poor development of abdominal muscles, improper closure of the umbilical opening at birth leads to congenital hernia. Umbilical hernia is more frequently seen in females than males and is primarily hereditary in origin due to dominant gene with low penetrance, autosomal recessive genes or due to environmental factor (Singh et al., 2020) [58]. Most of umbilical hernia is reducible in nature (Fig. 3a). However neglected cases with delayed presentation (Fig. 3b) may become strangulated or incarcerated. Treatment consists of simple elliptic skin incision around the umbilical mass ensuring adequate skin remains to allow skin closure without tension. Freeing of subcutaneous tissue around the umbilical mass until hernial ring and muscles is exposed muscle is exposed. After that either vest over pant sutures or sterile non absorbable monofilament polypropylene mesh (Fig. 2c) can be applied depending on size of hernial ring, after placing herniated content back to abdominal cavity. The male calf presents more challenges in herniorrhaphy because of the preputial orifice being close to the umbilicus. Baird (2008) [9] suggested method to overcome this problem by giving a half-moon or semilunar skin incision centring over the umbilicus with the concave side directed caudally. The semilunar skin incision also allow caudal extension of the skin incision on either side as a paramedian incision. The subcutaneous closure is done longitudinally on the line of the paramedian skin incision. The skin closure may bedone with simple continues or horizontal matters pattern.

**Fig 3:** (a) Showing herniation in cow calf(b) showing delayed presentation of congenital umbilical hernia buffalo calf(c) Use of polypropylene mesh to close hernial ring.

**Eventration**

Congenital eventration is defined as protrusion of visceral organs with its serous sac from faulty closure of the abdominal wall in the prenatal development life. Congenital ventral abdominal defects are very common in calves and these defects in the development of somatopleura leads to various defects in the bodywall especially in the ventral median parts (Veena et al., 2011) [65]. Exposure of the abdominal viscera is very common in schistosomus reflexes which include spinal inversion in bovine fetal monsters (Denis and Meyer, 1965; Denis, 1972) [18, 17]. Congenital eventration of intestine (Fig. 4a) in calves is common and eventration of intestine along with spleen has also been reported in lambs (Gangwar et al., 2014) [24]. To the author’s best of knowledge, there is scarcity of peer-reviewed case report of eventration of under developed liver and gall bladder (fig. 4b, a and c). The abdominal viscera are generally covered with parietal peritoneum at birth but later on get exposed either by licking of dam or self inflicted injury. Reported cases usually involve badly soiled viscera, especially in unassisted parturition that occur at late night. Such calves are prone for peritonitis when content is replaced back to
abdominal cavity. Badly soiled mass has more congestion and are prone to early necrosis.

Treatment includes washing of protruded visceral mass with copious amount of normal saline till soil or faeces are washed out completely. After aseptic preparation of the site, lignocaine 2% was infiltrated around the ring. Sometimes reduction of the contents is impossible through the narrow umbilical opening in such cases opening should be enlarged by cranial caudal incision. The abdominal viscera are replaced back in to the abdominal cavity. If the ballooning of the peritoneum is still present that should be trimmed from the base. The peritoneum, abdominal muscles and skin is sutured in routine manner.

**Pervious urachus**

The umbilicus in newborn calves consists of urachus, a tube that connects the fetal bladder to the placental sac, and the remnants of the umbilical vessels that transported blood between the fetus and its mother. Before birth, urinary bladder communicates with allantois through urachus which becomes atrophied and its lumen gets obliterated after parturition (Laverty and Salisbury, 2002) [35]. Normally, these structures shrink until only tiny remnants remain within the abdomen. If the urachus remains open for longer period it may act as source of ascending infections to bladder (Langan et al., 2001) [34]. There is constant dribbling of urine from the umbilicus and area around it remains wet. Area around the umbilicus shows inflammation, mild oedema and pain if infection is there. Both surgical and conservative treatments are indicated (Singh et al., 2020) [58]. Conservative treatment is successful in initial stage. Cotton swab dipped in 90% phenol is applied inside the urachus at a distance of 4–5 centimetres towards the urinary bladder. Singh et al. (2020) [58] also described surgical treatment when conservative treatment fails or infection is present. In order to ligate urachus an incision is given over umbilicus carnio caudally to ligate umbilical vessels and urachus proximal to infection using absorbable suture material, finally infected mass is dissected out.

**Meningocele**

Meningocele is an anomaly in which fluid-filled meninges protrude through a defect in cranial vault. It is mostly seen in domestic animals, especially in cattle (Ayan et al., 2013) [6] and defect may be due to defective ossification of the skull (Oliver et al., 1987) [42]. When defect occur in the skull called meningoencephalocele and if occur in the spinal cord called as meningoymyelocoele. The defects on the skull have been reported mostly on frontal region but mid frontal, occipital or parietal regions have also been involved (Singh et al., 2020) [58]. In cranial meningocele, fluid filled meninges protrude through a defect in the cranium (Leipold and Davis, 1993) [36]. It is a form of neural tube defect, may be observed at the time of birth, where neural tube is unable to close completely. The neural tube is a hollow, embryonic structure that gradually develops into the central nervous system, comprising of the brain and spinal cord. This in turn creates a gap or hole through which cerebrospinal fluid, brain tissue, and the membrane covering the brain (Meninges) protrude into a sac-like formation (McComb and Chen, 1996) [38]. This defect in the cranium is more common on frontal bone at its symphysis (Kohli and Naddaf, 1998) [32]. Such calves are unable to lift their head and suckle due to weight of the swelling. However, neurological signs in such calves have not been reported. Physical examination of the swelling reveal a fluid filled sac (Fig. 5a) situated over the frontal bone close to the nuchal’s crest. Diagnosis of this condition is done by signalment itself but the nature and degree of involvement of meninges and the magnitude of defect in the cranial vault have to be assessed by diagnostic imaging like radiography, computed tomography and magnetic resonance imaging. Treatment includes excision of the sac by elliptical incision near the base and connecting the hernia cavity to peritoneal cavity through a tube subcutaneously.

![Fig 4: (a) Eventration of intestine (b) Eventration of under developed liver and gall bladder (c) under developed liver and gall bladder after removal of sac](image)

![Fig 5: (a) Meningiocele (b) Prognathism in buffallo calves](image)

**Prognathism**

Prognathism is a congenital deformity in which the lower jaw overlap the upper jaw (Fig. 5b) characterized by shortened nasal bones, an exaggerated upward curvature of the lower jaw, and a broad head generally and also called as “bulldog” head in cattle. The nasal bones are about one third of the ordinary length, but retain almost their normal breadth (Darwin, 1980) [14]. The occurrence of this condition has been reported from abroad in cow (Robetrs, 2004 [53]; Harper et al., 1998) [27] and in buffaloes (Christopher, 2000) [13]. The condition occurs mainly due to autosomal recessive gene (Robetrs, 2004) [51] resulting in chondrodysplasia, which is histogenic abnormality causing disorganization of the cells and other components of a tissue (Spranger et al., 1982) [60]. Chondrodysplasia is a disturbance of endochondral ossification leading bone development disorder (Gentele and Testoni, 2006) [25]. Due to chondrodysplasia not only prognathism but other phenotypic defects like lingual protrusion, a cleft palate (Harper et al., 1998; Agerholm et al., 2004) [27, 3], malformed ribs, omphalocele or even evisceration, micromelia (Harper et al., 1998 [27]; Agerholm et al., 2004) [3] may occur. The shape of the mouth permits these cattle to graze readily on good pasturage, but on short vegetation they could not survive without special care. No surgical treatment is indicated however for such conditions in human are well managed by modern orthodontic techniques.

**Contracted tendon**

Congenital contracted tendon occurs due to shortening of flexor tendons results in knuckling frequently involve fetlock joint (fig. 6a, b) but rarely carpal joint (fig. 6c) also (Singh et
al., 2020; Rashmi et al., 2018) [58, 47]. The aetiological origin of contracted flexor tendons include inherited factors, in utero nutrition, malposition, and overcrowding caused by the size of the foetus relative to the dam (Anderson et al., 2008; Ferguson 1997) [5, 23]. The degree of knuckling varies from slight knuckling of the knee joint to complete flexion of pastern and fetlock joint. Singh et al. (2020) [58] have reported are more involvement of hind limb (Fig. 7 a, b and c) and may be associated with cleft palate and arthrogryposis. Treatment of flexural deformity should be initiated immediately after recognition of the problem because calf get older contracted tissues become less responsive for treatment.

Fig 6: (a) Mild knuckling at fetlock (b)severe knuckling at fetlock (c) knuckling at carpal joint

Animal with mild knuckled fetlock joint stands and is able to walk on its toe, while in advanced cases either animal falls of stumbles when it is made to walk. In severe cases dorsal aspect of pastern or fetlock rest on the ground which leads to excoriation of the skin.

Fig 7: (a) Involvement of hind fetlock joint (b) Involvement of hock joint (c) Involvement of both hind leg pastern joint (dead foetus)

Mild cases generally do not require any treatment and gets self corrected when the calf exercises, frequent manual extension of joints to stretch ligaments, tendons and muscles aids in treatment. Daily improvement is seen and condition gets resolved within few weeks. Temporary splints can be applied. Low toxic dose of oxytetracyline (20 mg/kg IV) daily for three consecutive days also have additional beneficial effect in managing moderate fetlock knuckling (Fazili et al., 2014) [22]. Surgically, partial or complete tenotomy (Fig. 8a) of superficial digital flexor and deep digital flexor tendon is done depending on severity of knuckling. Stabilization of limb using PVC splint and soft bandage from elbow to hoof is recommended (Fig. 8b). Euthanasia is often the best option for some of these calves if the defect is severe.

Fig 8: (a) tenotomy at carpal joint (b)post tenotomy POP bandaging

Hypospadias
Hypospadias is condition in which urethra opens anywhere (Fig. 9a) along its length at one or more locations from the perineum to the tip of penis and often accompanied by hypoplasia of the corpus cavernosum urethra (Alam et al., 2005) [4]. This congenital defect occurs due to incomplete formation of the penile urethra (Boothe, 2003) [11] and imperfect closure of the external male urethra (Radostits et al., 2007) [46] due to disturbance in the process of fusion of the paired urethral folds to form complete urethra after rupture of the urogenital membrane (Kluth et al., 1988) [31] or may be caused by extra/intrauterine factors resulting in abnormalities of androgen metabolism and timing of receptor function during male sexual differentiation at the early period of gestation (Stamper et al., 1999; Kurzrock et al., 2000; Uda et al., 2004) [61, 33, 64]. The condition is classified based on the location of the urethral opening as anal, perineal, scrotal, penile, or glandular, (Ader and Hobson, 1978) [2]. It mostly coexists with other developmental anomalies (Singh et al., 2020) [58] such as cryptorchidism (Shima et al., 1979; Hayes and Wilson, 1986; Rohatgi et al., 1987) [54, 29, 52] or atresia ani (Fig. 9b). Generally surgical correction is not recommended when it co exists with other anomaly (Singh et al., 2020) [58] but contrary to this finding Alam et al. (2005) [4] operated three calves to improve survival. Author also operated a calf for atresia ani and tubecystotomy, as urethral opening was very much constricted resulting in urination in jet fashion and that was after too much straining (Fig. 9 b) but this led to ascending infection to urinary bladder in due course of time.

Fig 9: (a) Abnormal opening of urethra (black arrow) with unilateral cryptorchidism and ventrally incomplete sheath (b) Hypospadias with atresia ani (c) tubecystotomy

Vulvar atresia
Vulvar atresia or agenesis means absence of normal opening of vagina with fused outer labia covering over normal canal (Radostitis et al., 2000) [45]. Complete absence of vulva end in a tubercle like structure at ventral aspect at the site of normal vulva. Sometimes there is complete absence of urethral opening or may have very constricted urethra directed dorsally and caudally (Fig. 10a), through which urine came out after much straining as narrow jet flow. To manage constricted urethra surgically a small incision is to be made at mid of fused vulvar lips to create or widen the opening. The patency is then maintained by temporary fixing a cannula (Surabhi et al., 2018) [63]. But when there is complete absence of urethral opening (Fig. 10b), it is very difficult to manage surgically, tube cystotomy done as salvage procedures to just lengthen the life till weaning otherwise humanely euthanasia is recommended.

Fig 10: (a) Vulvar atresia (b) tubercle like structure

Ocular dermoid
Ocular dermoids are rare in cattle, with the prevalence between 0.002% and 0.4 percent (Kiliç et al., 2012) [30]. It is an uncommon congenital and developmental defect characterized by the islands of skin that are histologically normal but misplaced to an abnormal location (Bekele et al., 2012; 2018). Surgical removal is recommended (Fig. 8b) along its length at one or more locations from the perineum to the tip of penis and often accompanied by hypoplasia of the corpus cavernosum urethra (Alam et al., 2005) [4]. This congenital defect occurs due to incomplete formation of the penile urethra (Boothe, 2003) [11] and imperfect closure of the external male urethra (Radostits et al., 2007) [46] due to disturbance in the process of fusion of the paired urethral folds to form complete urethra after rupture of the urogenital membrane (Kluth et al., 1988) [31] or may be caused by extra/intrauterine factors resulting in abnormalities of androgen metabolism and timing of receptor function during male sexual differentiation at the early period of gestation (Stamper et al., 1999; Kurzrock et al., 2000; Uda et al., 2004) [61, 33, 64]. The condition is classified based on the location of the urethral opening as anal, perineal, scrotal, penile, or glandular, (Ader and Hobson, 1978) [2]. It mostly coexists with other developmental anomalies (Singh et al., 2020) [58] such as cryptorchidism (Shima et al., 1979; Hayes and Wilson, 1986; Rohatgi et al., 1987) [54, 29, 52] or atresia ani (Fig. 9b). Generally surgical correction is not recommended when it co exists with other anomaly (Singh et al., 2020) [58] but contrary to this finding Alam et al. (2005) [4] operated three calves to improve survival. Author also operated a calf for atresia ani and tubecystotomy, as urethral opening was very much constricted resulting in urination in jet fashion and that was after too much straining (Fig. 9 b) but this led to ascending infection to urinary bladder in due course of time.
2014) [10] and are often noticeable soon after birth (Abou-Rayyah et al., 2002) [1]. According to Perry and Tuthill (2003) [44], dermoid cysts are benign tumours that represent the simplest form of teratoma. Defective epidermal closure along embryonic fissures is found to be the etiology behind dermoid cyst (Nagar et al., 2015) [40]. The cyst usually contains hair, keratin, and sebum, and these materials may produce progressive enlargement of the structure so that it becomes clinically apparent (Edwards, 2002) [20]. Dermoids are most commonly seen over lateral canthus, medial canthus, limbus, conjunctiva, nictitans, sclera, cornea (fig.11.a, b) and eyelid. It may be found associated with other ocular manifestation like congenital corneal opacity, which have been described in Holsteins as recessive condition by Deas in 1959 [16] or with other malformations like nasal hyperplastic tissue which was reported by Hashim et al. (2016) [28] in a calf. It can be unilateral or bilateral but most commonly it is reported in one eye (Williams and Gelattk, 1981) [67]. Bilateral ocular dermoids have been reported in cattle by Barkyoub and Leipold (1984) [9] and Yeruham et al. (2002) [68] whereas Moore et al. (1999) [39] reported bilateral dermoid in camel. Bilateral ocular dermoids are genetically transmitted defects in Hereford cattle (Barkyoub and Leipold, 1984). [9] Hair from the lesions is mostly responsible for the associated irritation resulting in chronic inflammation of the conjunctiva and cornea, mild blepharospasm and epiphora (fig.11.a) and may cause visual impairment (Pandey et al., 2011) [43]. It can easily be diagnosed by history and clinical signs. Superficial lamellar keratectomy is the only option for treatment of ocular dermoid in calves and it is performed under local anaesthesia by blocking auriculopalpebral nerve and Peterson nerves to desensitise the eyelid and eye ball respectively. Ocular dermoid should be dissected carefully from the underlying cornea to avoid penetration of anterior chambers of the eye (Roberts and Lipton, 1975) [50]. Once it is removed completely, chances of reoccurrence are very rare.

**Fig 10:** (a) Vulvar atresia (b)Vulvo urethral agenesis in female calf

**Fig 11:** (a, b) bilateral ocular dermoid cyst in a heifer showing epiphora due to dermoid hair irritation, (c) unilateral dermoid cyst in a buffalo

**Bifid Tongue**

Bifid tongue or glossochisis is a rare congenital abnormality (Rifai et al., 2006) [49]. It has been reported in humans, mules, dogs and bovines (Surej, 2010; Rifai et al., 2006; Villagomez and Alonso, 1998) [63, 48, 66] along with other congenital malformations like mandibular cleft and palate agenesis or cleft palate (de Paula Lopes et al., 2019) [13]. The etiology of fetal malformations is still unclear. Tongue formation in bovines occurs with the fusion of three structures at the end of the fourth week of gestation. These structures fuse to form the free part of the tongue, which is capable of moving inside the oral cavity. Disturbance in organogenesis of tongue may lead to incomplete fusion of the lateral tongue buds, thus resulting in a deep groove in the midline of the tongue (Emmanouli and Kerameos, 1992) [21]. Correction surgery involves glossoplasty under general anaesthesia where an incision is given over medial rim of tongue and ventral and dorsal part of tongue is then sutured with non absorbable suture material starting from root to apex of tongue (de Paula Lopes et al., 2019) [15]. The bifid tongue prevents the calf from naturally suckling its mother’s milk which ultimately affects general body condition. Therefore, it becomes necessary to initiate artificial feeding to keep the animal alive and with a good body score until reduction glossoplasty is performed. The affected animal should not be used for reproduction because there is a risk of disseminating the genes of the fetal malformation among the herd (de Paula Lopes et al., 2019) [15].

**Fig 12:** Bifid tongue with abnormal mandible in a buffalo calf.

**Conclusions**

The best control of genetic diseases is to avoid breeding of animals that carries defective genes. Bulls or semen should be purchased from reputable breeders, produced by parents who are not known to carry undesirable genes. Prompt surgical intervention should be advocated to save and improve quality life of affected new born animals.

**References**


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