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SURGICAL MANAGEMENT OF ATRESIA ANI IN A CAMEL CALF

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ABSTRACT

A camel calf was presented to the clinic with a history of inability to pass faeces. Clinical examination revealed absence of anal opening and protrusion was noticed in the perineal region of the calf during straining for defaecation. Clinical parameters of the calf were within normal range. Surgical reconstruction of anus using rectal pull through technique was achieved after analgesia by epidural injection and local infiltration with 2% lignocaine hydrochloride.

Key words: Atresia, perineum, reconstruction, rectal pull through technique

Intestinal atresia has been reported as a congenital defect in all species of domestic animals (Van Der Gass and Tibboel, 1980). Any segment of the intestine from duodenum to anus may be atretic (Dreyfuss and Tulleners, 1989).

Atresia ani is the lack of opening of anal sphinctor resulting in blockage of intestinal transit and accumulation of meconium and gas in the gastrointestinal tract. The neonate becomes progressively bloated and depressed and may die if anal patency is not restored.

Case history and clinical examination

An eight hours old male camel calf was presented to the clinic with a history of straining for defaecation leading to protrusion in perineal region but calf had no anal opening. The calf was sucking mother's milk and passing urine normally. The clinical parameters were within the normal range. A bulged out area in perineal region during straining for defaecation indicated the presence of the blind end of rectum very near to the perineal skin.

Surgical management

The camel calf was positioned in sternal recumbency. Perineal region was clipped and prepared for aseptic surgery. The xylazine hydrochloride was given intravenously @ 0.1mg per kg body weight for sedation and local analgesia was achieved by epidural injection at sacro-coccygeal space (0.1ml/kg) of 2% lignocaine hydrochloride. Circular incision of 2.5 cm diameter was made through the skin in perineal region at the site of protrusion. The blind end of bulged rectum was identified after blunt dissection through the subcutaneous tissue.

The rectal submucosa was sutured to perineal subcutaneous tissue using a series of simple interrupted sutures of 2-0 polygalactin-910. The initial sutures were placed at 180° and then at 90° intervals to allow accurate circumferential alignment. Additional interposing sutures were placed to complete the marsupialisation.

A circular portion of rectal blind end was excised using scalpel central to first row of sutures. Skin and full thickness rectum were opposed with simple interrupted pattern of 1-0 silk. A copious volume of meconium was passed after rectum was opened. Remaining sticky and tenacious meconium was removed by gentle digital manipulation followed by soap-water enema.

The camel calf was administered injection cefataxime 1gm intramuscularly for five days, gentamycin 200mg intramuscularly for 5 days and meloxicam 25mg intramuscularly for 3 days. The wound was dressed with povidone iodine and fly-repellent spray till healing. Skin sutures were removed after 10 days. The wound healed uneventfully.

Discussion

Intestinal atresia is thought to be associated with interruption of the blood supply to a localised segment of embryonic intestine resulting in atrophy, disappearance of the affected segment or failure of

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the affected segment to undergo further development (Jubb, 1990). Stenosis and atresia of the lumen are the most common congenital malformations of the gastrointestinal tract in most species (Noden and De Lahunta, 1985). Atresia ani was reported to be the most common congenital malformation of the intestinal tract in domestic ruminants (Singh, 1989). However, the most common lethal congenital abnormalities that affect the camelid neonate are choanal atresia, atresia ani or coli and heart defect (Tibary *et al,* 2008). In atresia ani the rectum is complete but not patent. The blind end of the rectum in this case was lying just below the skin. Several other congenital abnormalities like involving the urinary tract (renal agenesis, horse shoe kidney, polycystic kidney), genital tract (recto-vaginal fistula, cryptorchidism), central nervous system (spinal dysraphia and lack of cauda equina) and skeletal system (coccygeal or sacral vertebral agenesis) may accompany atresia ani but were not in present case as evidenced by normal physiology and external examination of the animal.

The pathogenesis of intestinal atresia is unknown. Any reason leading to disruption of the mesenteric vasculature *in utero* may lead to intestinal atresia. It has been experimentally induced by ligation of mesenteric vessels in foetal pups, lambs (Clark *et al,* 1978), rabbits (Tsujomoto *et al,* 1972) and chikens (Tibboel *et al,* 1979). In cattle, the palpation of amniotic vesicle at the time of pregnancy examination during period of principal organogenesis (between 36-42 day of pregnancy) has been implicated as a cause of intestinal atresia (Ness *et al,* 1982). However, in pigs and calves it has been reported to be a heritable defect by Norrish and Ronnie (1968) and Jubb and Kennedy (1970). In the present case there was no history of rectal palpation for pregnancy diagnosis.

The success of surgery depends on the extent of rectal development (Hay, 1991), early recognition and successful establishment of a patent intestinal tract. Moribund, recumbent animals have a lower survival rate than those that are alert and able to stand. In the present case, the recognition of the condition was very early and animal was quite alert. Postoperative complications can include anal stricture, wound dehiscence, faecal incontinence and

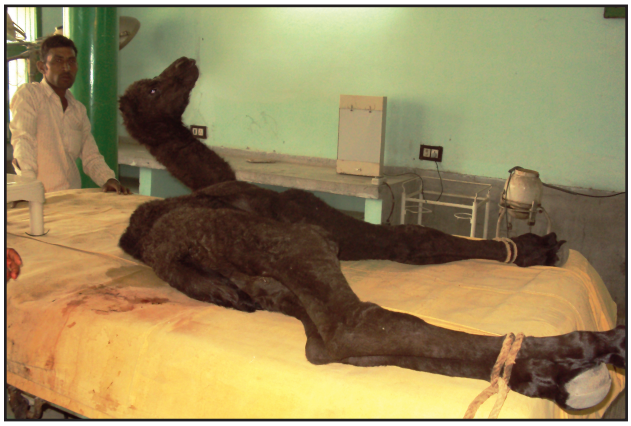

Fig 1. Camel calf presented for treatment.

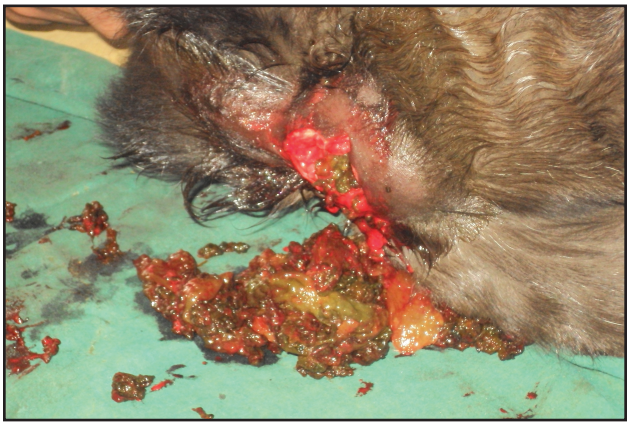

Fig 3. Faeces passed after opening rectum.

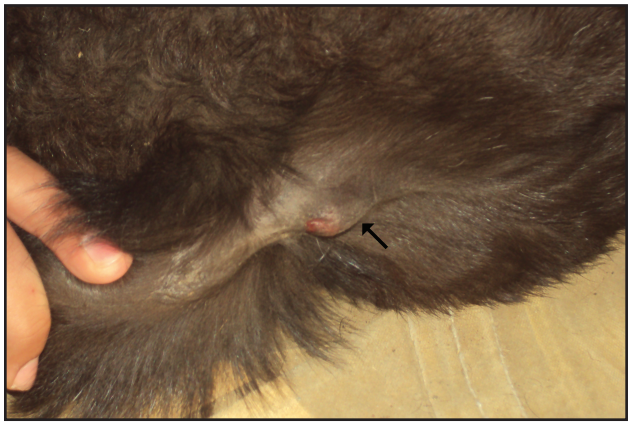

Fig 2. Absence of anal opening (arrow).

Fig 4. Reconstructed anal opening.

constipation. However, the circular excision and rectal pull through technique was used in this case which reduces the chances of complications than cruciate incision technique (Dreyfuss and Tulleners, 1989; Suess *et al*, 1992).

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