Surgical Repair of Atresia Ani (imperforate anus) in Newborn Kids and Lambs

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ABSTRACT

Atresia ani is a congenital defect describes absence of a normal anal opening. It is fatal unless surgical correction is carried out to provide anal opening. In female, the rectum may break through vagina forming a rectovaginal fistula permitting defecation via the vulva. Surgical treatment of atresia ani is indicated to save the animal life and to improve body weight gain. Ten kids and eight lambs suffering from atresia ani referred to the veterinary health center at the Jordan University of Science and Technology in the period of 2004-2009 suffering from atresia ani. Clinical examination and ultrasonography confirmed atresia ani without any other intestinal congenital deformities. Affected animals were subjected to surgical anal reconstruction following animal casting, site preparation and analgesia. A circular perineal skin tissue was excised to explore the pelvic cavity associated with blunt dissection of the perineal region exposing the blind cull- de- sac of the rectum. The blind rectum segment was opened and the meconium was evacuated. Full thickness rectum and was secured using simple interrupted stiches. Anal reconstruction proved successfully for all treated kids and lambs. The new stoma provides a permanent patent orifice for normal passage of feces until the age of slaughter. This surgery was legitimate, considering the high incidence of atresia ani, simple, economic, and lifesaving surgical technique. Anal reconstruction in affected animal is an obligatory surgery rather than alternative euthanasia. Anal reconstruction increases body weight gain and decreases the economic loss in the small ruminant herd industry due to atresia ani.

Keywords: Kids, Lambs, Atresia Ani, Conginital, Surgery, Reconstruction.

INTRODUCTION

The rectum is formed when a mesenchyme partition (urorectal septum) divides the cloaca into dorsal and ventral chambers. The dorsal chamber, which is continuous with the hindgut, becomes the rectum and most of the anal canal. The ventral chamber (the urogenital sinus) is continuous with the allantois. The urorectal septum grow caudally and divides the cloacal membrane into an anal membrane dorsally and a urogenital membrane ventrally. The membranes subsequently disintegrate in normal development. The cranial part of the anal canal (most of the canal) is formed with the rectum; this part of the anal canal is lined by a mucosal epithelium derived from endoderm. The caudal part of the anal canal (caudal to the adult anocutaneous line) is lined by stratified squamous epithelium. It forms tissue surrounding the anal membrane grows caudally creating a depression called the proctodeum; when the anal membrane degenerates, the proctodeum becomes incorporated into the anal canal (McGeady *et al.*, 2006).

By entering the pelvis, the descending colon becomes the rectum, which passes caudally as the most dorsal organ of the

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pelvic viscera. Most of the rectum is suspended by the mesorectum, but the terminal part is totally retroperitoneal. The retroperitoneal space is filled with soft tissue rich in fat covers the anus. The short anal canal is the terminal part of the alimentary canal, which opens to the outside with the anus. The external and internal anal sphincters control the anus. External sphincter is striated muscle arising from the caudal vertebrae while the internal sphincter consists of smooth muscle and is a modification of the circular layer of the muscle coat of the rectum. At the anus mucosa, the columnar intestinal epithelium is replaced by the stratified epithelium of the skin (Konig and Liebich, 2006).

The major blood supply to the rectum is cranial rectal artery, a branch of the caudal mesenteric artery, which runs within the mesentery and over the dorsal rectal wall. The anal region is supplied by branches from the caudal rectal artery, an indirect branch of the vaginal artery of the female or prostatic artery of males. The cranial rectal veins lead to the portal circulation through the mesenteric trunk, while those from the anal region drain into internal pudendal veins.

One or more caudal rectal nerves (S4-5) run between the rectum and the internal face of the pelvic diaphragm. They are composed of somatic motor fibers destined for the pelvic diaphragm and voluntary anal muscles (Dyce *et al.*, 1987).

Congenital defects, abnormalities of structure or function present at birth, may be caused by genetic or environmental factors, or a combination of both; in many cases, the causes are unknown (Servet *et al.*, 2009).

The term atresia describes congenital occlusion of the lumen of the digestive tract. Failure of the anal membrane to break down during the development gives rise to the condition termed imperforated anus and some times termed as atresia ani (McGeady *et al.*, 2006).

There are four major types of intestinal atresia. Type I atresia is a mucosal blockage within the intestinal lumen. In animals with type II atresia, the proximal segment of intestine terminates in a blind end and the distal segment beings similarly with two ends being joined by a fibrous cord devoid of lumen. Type IIIa atresia is similar to type II except that the proximal and the distal intestinal segments blind ends are completely separated and there is a mesenteric defect corresponding to the missing segment of intestine. Animals with type IIIb atresia have a coiled distal segment of intestine. Type IV atresia involves multiple sites of atresia (Kilic and Sarierler 2004; Rahal *et al.* 2007).

Atresia ani is fatal affection to the male unless surgical correction is carried out to provide anal opening, in female rectum frequently break through to vagina forming a rectovaginal fistula and thus permit defecation via the vulva (Norrish and Rennie, 1968).

Atresia ani was reported as a possible genetic defect in Swedish Highland Cattle, Holsteins, and other breeds. Atresia ani is the most common intestinal defect in sheep and is believed to be due to an autosomal recessive gene. In a series of 64 cases of atresia ani in sheep, 42 (62%) were associated with defects of other body systems, especially the urogenital and musculoskeletal systems (Newman *et al.*1999; Ghanem *et al.*, 2004; Kiliç and Sarierler 2004; Loynachan *et al.* 2006; Rahman *et al.* 2006; Magda and Youssef 2007; Bademkıran 2009).

Affected animals may survive for up to 10 days and can be identified by their depression, anorexia, colic, marked gradual abdominal distension and lack of feces (Radostitis *et al.*, 2000).

Atresia ani should be treated by surgical operation to solve the problem, improve body weight gain, and reduce economic loss caused by this defect (Servet *et al.*, 2009).

Anal reconstruction in small ruminant is not routinely attempted in veterinary practice, this study aimed to determine the efficacy of anal reconstruction in improving survival rate, health and bodyweight gain of affected small ruminants.

Materials and Methods

Animals

Ten mixed breed newborn kids (7 males and 3 females) and eight newborn lambs (6 male and 2 female) weighing, 3-5 kg (mean, 4 kg) aged 3-8 days , none of them were twins, from north Jordan flocks. They were referred to veterinary health center in Jordan University of Science and Technology in the period of 2004 to 2009. These animals were physically examined by a standard examination procedure. Diagnosis was obvious when the anus was absent. In these animals, there was a pronounced protrusion of the perineal region by deep palpation of abdomen. Diagnosis was confirmed using ultrasonography.

Surgical procedure

Anal reconstruction was the recommended treatment. Animals were sedated using 2% Xylazine (Xyla-Ject, 50 ml, 2%, the Egyptian Co. for chemicals and pharmaceuticals (ADWIA), Egypt) at doses 0.05 mg/kg, IM (Magda and Youssef 2007). On surgical table, animals were casted in sternal recumbency and both hind limbs were secured. Perineal area was shaved; cleaned, scrubbed and standard surgical site preparation was performed. Local analgesia using Lidocaine HCl 2% (Lidocaine injection, 50 ml, 2%, Rotexmedica, Trittau, Germany) was infiltred around the anal scar (Fig: 1).

Anal reconstruction:

Following the procedure of Frank (1964), atresia ani was treated by excision of a circular piece of skin around anal scar. The blind ended rectum was exteriorized after blunt dissection of the perineal canal (Fig: 2). Then, the blind end of the rectum was retracted caudally, and fixed to the perineal skin. Four stay sutures were placed dorsally, ventrally and bilaterally using 2-0 absorbable suture material (MONOCRYL, violet, size 2-0, monofilament absorbable suture, rounded 1/2 circle, ETHICON, Johnson Company, USA). These sutures were performed in simple interrupted pattern holding sero-mascular layer of rectum to the perineal skin. Numerous sutures were placed all around the rectum using 2-0 absorbable suture material in simple interrupted pattern holding full thickness layer of rectum with full thickness of perineal skin (Fig: 3). This was to ensure good and fully contact between the rectum and the perineal skin. Then, the blind end of the rectum was incised to evacuate the content of the lumen. Meconium was evacuated to allow good exposure of surgical site (Fig: 4).

Figure 1: Pre-operative photograph of lamb perineal area showing where the anus would normally be located and the local infiltration of lidocaine HCL.
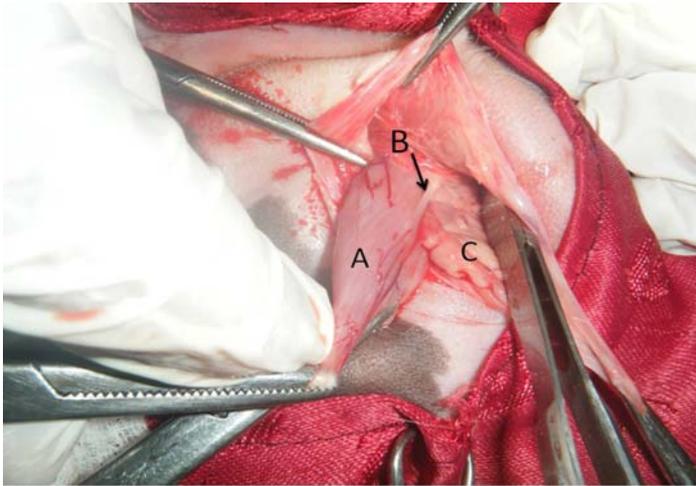
Figure 2: Intra-operative photograph of lamb perineal area showing bluntly dissected pelvic canal, the rectum retracted in caudal position. (A) The rectum, (B) the mesorectum, (C) perineal fat.

Figure 3: Intra-operative photograph of lamb perineal area showing the retracted rectum during rectopexy to the pelvic diaphragm.

Figure 4: Intra-operative photograph of lamb perineal area showing the incised blind rectum to evacuate meconium producing a patent anal stoma.

Figure 5: Pre-operative sonograph of lamb abdomen showing the meconium impacted gastrointestinal tract.

Postoperative care

A thorough follow up physical examination was performed daily for fourteen days to evaluate wound healing and improvement of animal health. Animals were monitored for six months (age of slaughter) to evaluate body weight gain.

The following antibiotic and anti-inflammatory regime was given for all animals.

- Penicillin and streptomycin (pen-strep, 100 ml, Norbrook Laboratories Limited Newry, Co): 10,000-30,000 IU/kg, IM, Bid for 5 days.

- Gentamycin 5% (Alfamycin, 100 ml, AlfaMedic): 3-6 mg/kg, IV, Bid for 5 days.

- Flunixine meglumine (Finadyne, 50 ml, 50 mg/ml, Schering Plough Animal Health): 1.1 mg/kg, IV daily for 10 days.

Results

In addition to clinical examination, ultrasound of the perineum (rectal and vaginal areas) was useful to confirm atresia ani diagnosis. Ultrasonography revealed the meconium filled intestine loops (Fig: 5). It also determined the distance between a meconium-filled distal rectum and the perineum. Ultrasound could determine any anomalies of the urinary tract or the spinal cord.

Anal reconstruction as a treatment of atresia ani by excision of a circular piece of skin at bulging perineal area facilitated dissection of the blind end of the rectum. It fixed the rectum by stay sutures (rectopexy) at the site where the anus is normally located providing a permanent functional anal stoma. Blunt dissection saved all adjucent anal muscels and strucures. This gave a clear surgical field and good access to fix the rectum to perineal skin. Body weight increased by 3 kg per month untill the age of slaughter (six months).

In all animals, anal reconstruction was performed satisfactory. After postoperative period, all animal's wounds fully healed without any significant complications. All animals were able to nurse their dams. No clinical side effects were observed after surgery except mild sign of digestive discomfort in three treated animals without any need for further treatment.

Defecation was normal soon after surgery. Fecal passage was achieved without any need of spescific care or interference which improved animal health and body weight gain. Full function anal stoma was achieved after wound healing at about one month post operative. The diameter of new artificial opening enlarged by time. It

was suffcient to by pass the feces without any compliction or straining despite the abcence of external and internal anal sphincter functions.

Discussion

Atresia ani has been reported to be a heritable condition in pigs and calves (Kilic *et al.* 2004). Based on this all treated animals were not considered for breeding; they were slaughtered at six months of age. A genetic basis has been documented for some cases of atresia ani, but the specific cause in sporadic cases in domestic species and humans is not always known (Johnson *et al.*1980; Newman *et al.*1999).

Some authors Johnson *et al.* (1983), Leipold (1986) and Noh *et al.* (2003) reported that the most congenital anomalies of digestive system observed in calves were atresia ani and atresia recti. Besides, the anomalies of urinary system such as renal agenesis, polycystic kidney and skeleton system such as cocygeal or sacral vertebral agenesis were observed at the same time in calves. However, in this study animals were suffering from atresia ani only and were treated by anal reconstruction.

Atresia ani is a fatal affection to the male unless surgical intervention occurs to provide new anal stoma. In some females, fecal pressure result in rectum break through vagina forming a rectovaginal fistula and thus permit defecation via vulva, therefore affected female does not require a further care or surgical correction, and may not be identified (Norrish and Rennie, 1968). In present study, treated females did not develop rectovaginal fistula, this was because of early diagnosis

and treatment of atresia ani after birth. Some affected females grow normally and may contribute in herd reproduction, keeping the high incidence of atresia ani. In this study, all animals were not considered for breeding and were slaughtered at six months of age.

Four major types (I, II, IIIa, IIIb and IV) of intestinal atresia have been described involving different intestinal segments (Kilic and Sarierler 2004; Rahal *et al.* 2007). The animals presented in this study suffered from atresia ani only without any other intestinal anomaly.

Anal reconstruction was relatively painless using sedation and local anesthesia and was not invasive surgery. Mild signs of digestive discomfort were detected in three treated animals, as postoperative complications can occur, including fecal incontinence and colonic atony secondary to prolonged preoperative distension (Maria and Karen, 2005). In the present study during suckling period the feces was soft and normally excreted, once the newborn animal changed diet a normal pellet like feces was excreted and no postoperative complications were found. This finding showed that the new anal orifice increased in size with age and was able to excrete feces in spite of absence of anal sphincters.

It could be concluded that anal reconstruction is the only treatment for atresia ani in newborn animals. It is recommended in ruminants as it saves life of animal, improves body weight gain and reduces herd economical loss. The operation proved to be painless and economic. In addition, the future breeding of surgically treated animals should be discouraged.

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