Abdominal Hernia in a 3 year old Suffolk Ewe

Introduction

Abdominal hernias may occur in sheep as a result of increased pressure during pregnancy. The pathophysiology is not well described, but may be related to pre-pubic tendon rupture, gradual weakening of muscle layers by continuous stress, or acute rupture of abdominal muscles by trauma. The pre-pubic tendon in small ruminants is strongly attached to the pelvis by the symphyseal tendon and is composed of the rectus abdominis muscle, internal and external abdominal oblique muscles and the pectineus muscle (Figure 1). Rupture of the pre-pubic tendon is rare in ruminants, but has been reported occasionally in cattle, sheep and goats. Overall, abdominal herniation in small ruminants is rarely discussed in the literature. The incidence of pre-pubic tendon rupture causing abdominal herniation in ruminants is not reported, and it is possible that herniation may be secondary to abdominal muscle weakening, without prepubic tendon involvement, as small ruminant abdominal wall is relatively thin. A study from Saudi Arabia indicates that 30% of sheep presented to the veterinary hospital for hernias had abdominal hernias, all of which were acquired, but involvement of the prepubic tendon was not described[1]. Genetics may be a factor in herniation, as it has been reported more frequently in sheep breeds with higher birth weights, greater fecundity and increased milk production. Higher incidence is also reported in females. In pregnant ewes, herniation typically occurs on the right side, possibly due to displacement of the gravid uterus by the rumen, resulting in increased pressure and weakening of abdominal muscles. Abdominal muscle weakness may occur with age or multiple parities.

History for abdominal hernia includes known trauma, late gestation, multiple fetuses or advanced parity. Patients present with an acute or chronic swelling/distention in the lateral or ventral abdomen, which may fluctuate in size depending on position and chronicity. The animal is typically otherwise healthy, unless there is strangulation or incarceration within the hernial cavity. If bowel is strangulated, the animal may

present with signs of depression, bloat or shock. Differential diagnoses for an abdominal hernia include abscess, hematoma, neoplasia, or udder edema. Thorough palpation of the site may confirm an abdominal hernia diagnosis. Uncomplicated hernias are non-painful to palpation, while strangulating hernias may illicit pain. If the hernia is acute or traumatic, marked inflammation can make palpation difficult initially. A distinct hernia ring is often palpable with reducible hernias, where a defect in the musculature has occurred. Non-reducible hernias occur when there are sufficient adhesions to the hernia sac or ring. Ultrasound examination is useful in determining the contents of the swelling. With abdominal herniation, any abdominal organs may be visualized, and a decrease in muscle wall thickness should be identified overlying the site.

Treatment, Management and Prognosis

Depending on the defect size and purpose of the animal, management for abdominal herniation may include surgical repair, or benign neglect. If the animal is to be culled, monitoring for signs of incarceration and ensuring safety from further trauma is a viable option. This may include housing the animal separately if fighting occurs. Definitive treatment requires surgery. One study reports 87% positive surgical outcomes with no post-operative complications for abdominal hernias ranging from approximately 3-20cm in diameter[1]. Herniorrhaphy or hernioplasty are options for surgical correction. Herniorrhaphy involves closure of the hernia ring by apposing the edges. Hernioplasty is the incorporation of surgical mesh if tension does not allow primary closure, or if the muscle is too weak to provide support. Post-operative complications include incisional dehiscence or infection, hernia recurrence, and abdominal adhesions. Analgesics, such as non-steroidal anti-inflammatories, are required for acute and post-operative pain, and peri-operative anti-microbials should be given. Post-operatively, feed should be re-introduced slowly so rumen fill is gradual. The abdomen may be supported with a belly-band for 5-7 days to alleviate ventral pressure. Generally, prognosis is excellent for recovery. In a breeding animal, prognosis for return to function is poor to good, depending on the size and location of the defect.

Case History and Presentation

A 3 year old Suffolk ewe presented for acute, marked swelling of the ventro-lateral abdomen, cranial to the udder. The ewe was reportedly pregnant, but stage of gestation was unknown. She continued to eat and drink, but appeared depressed and had separated herself from the flock of 14 other ewes. She was unvaccinated, offered free choice hay and approximately 2lbs of oats per day. She lambed with twins uneventfully the year prior. On presentation, the ewe had a heart rate of 148 beats per minute (normal: 60-120), respiratory rate of 76 breaths per minute (normal: 12-72) and temperature of 39.1˚C (normal: 39.0˚C – 40.0˚C) . Body condition score was 4/9. Cardiothoracic auscultation was within normal limits. Rumen contractions were of normal rate (3 per 2 minutes) and rhythm. Capillary refill time was normal (< 2 seconds), and mucous membranes were pink and moist. There was a large (approximately 45cm in length) swelling cranial to and incorporating the udder (Figures 1 and 2). On palpation, the swelling had pitting edema and poorly defined borders.

Figure 1. 3 year old Suffolk ewe on presentation, with large ventral swelling.

Figure 2. 3 year old Suffolk ewe on presentation, with large ventral swelling.

The edema was too diffuse to appreciate anything else by palpation, and the abdominal wall was not discernible. Ultrasound evaluation of the abdomen revealed a minimum of two fetuses, with an approximate gestational age > 110 days, based on fetal head length (11.8cm). Diffuse edema was visualized overlying the swelling, and the layers of the abdominal wall could not be identified (Figure 3). Blood beta-hydroxy butyrate (BHB) reading by handheld ketometer measured 3.1mmol/L (normal < 1.2 mmol/L).

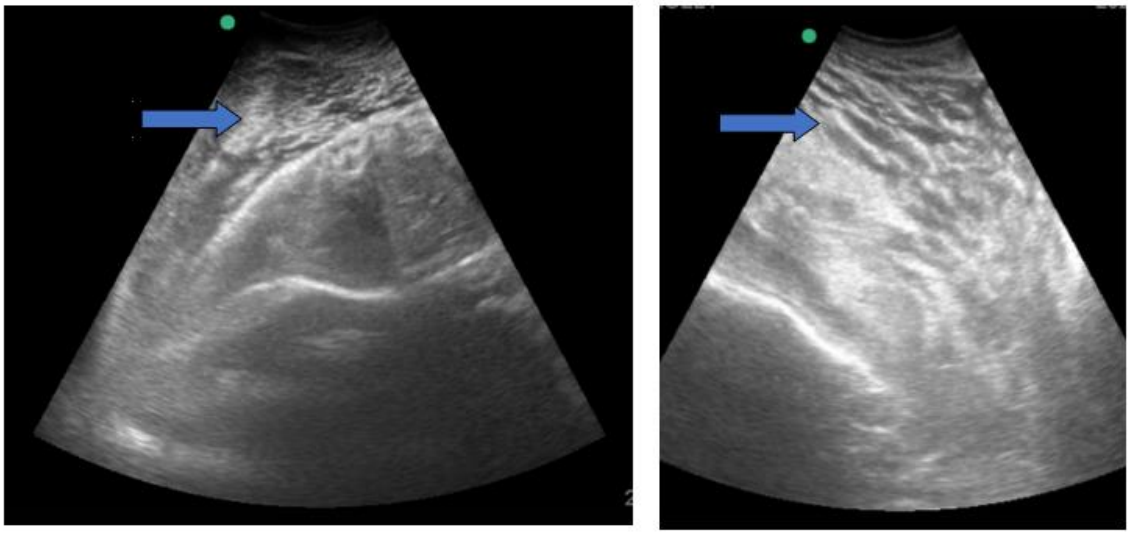

Figure 3. Ultrasound images of ventral swelling using 5-2MHz convex probe. Blue arrow indicates edema. Green dot indicates cranial.

Identified problems were tachycardia, tachypnea, hyperketonemia and acute, marked ventral edema. The ewe was diagnosed with pregnancy toxemia and suspect prepubic tendon rupture or abdominal wall herniation, based on history and appearance of the swelling. Abscess, hematoma, and neoplasia were ruled out based on the acute history, ultrasonographic appearance, and palpation of pitting edema. Udder edema was not completely ruled out, but was less likely as the caudal aspect of the udder seemed relatively unaffected. Rectal and vaginal examination were performed to rule out uterine torsion causing increased abdominal pressure, and no evidence of torsion was found.

Case Management and Outcome

Blood was collected for serum chemistry and complete blood count (CBC) to assess any concurrent illness or underlying cause of clinical signs. Serum chemistry revealed mildly decreased calcium, albumin and total protein, which may be related to 3rd space loss (edema), and increased creatine kinase indicating muscle damage (Table 1). The CBC revealed mild lymphopenia, likely related to stress (Table 2).

Table 1. Serum chemistry on day of presentation.

Test	Laboratory Result	Reference Interval	Units
Serum Chemistry			
Sodium	150	137-152	mmol/L
Potassium	4.5	3.9-5.7	mmol/L
Chloride	111	97-111	mmol/L
Bicarbonate	23	17-29	mmol/L
Anion Gap	21	17-33	mmol/L
Calcium	2.15	2.43-3.23	mmol/L
Phosphorus	1.45	1.06-2.62	mmol/L
Magnesium	0.86	0.77-1.17	mmol/L
Urea	6.4	4.5-12.1	mmol/L
Creatinine	84	55-107	µmol/L
Glucose	3.2	1.9-8.5	mmol/L
Total bilirubin	5.7	1.0-11.0	µmol/L
Direct bilirubin	2.5	0.0-3.0	µmol/L
Indirect bilirubin	3.2	n/a	µmol/L
GGT	37	9-61	U/L
GLDH	4	n/a	U/L
AST	183	62-260	U/L
CK	1258	31-347	U/L
Total protein	50	61-81	g/L
Albumin	27	33-39	g/L
Globulin	23	n/a	g/L
A :G Ratio	1.17	0.70-1.38	

Table 2. Complete blood count on day of presentation.

Test			
Leukocytes	**Laboratory Value**		**Ref. Int. x10^9/L**
WBC	7.8 x10^9/L		4.0 – 12.0
NRBC/100 WBCs	0		rare
Differentials	**Rel%**	**Abs. x10^9/L**	**Ref. Int. x10^9/L**
Segs	76	5.928	0.700-6.000
Bands	0	0	0.000-0.100
Metamyelocytes	0	0	0.000
Myelocytes	0	0	0.000
Toxic change	None	n/a	n/a
Eosinophils	0	0	0.000-1.200
Basophils	0	0	0.000-0.300
Lymphocytes	20	1.560	2.000-9.000
Monocytes	4	0.312	0.000-0.750
Atypical	none		
Erythrocytes	**Laboratory Value**	**Reference Interval**	**Units**
RBC	8.22	8.00-16.00	X10/^{12}L
HGB	103	80-160	g/L
HCT	0.280	0.240-0.500	L/L
MCV	34.1	23.0-48.0	fL
MCH	12.6	9.0-12.0	pg
MCHC	369	310-380	g/L
RDW	18.5	n/a	%
Plasma Protein by Refractometry	**Laboratory Value**	**Reference Interval**	**Units**
Total protein	55	60-79	g/L
Fibrinogen	5	1-6	g/L
Total Protein :Fib ratio	11 :1		

The ewe was treated with 60ml each of propylene glycol[a] and mineral solution[b] orally, 1mg/kg meloxicam[c] subcutaneously, and 60 ml calcium borogluconate[d] subcutaneously. The owner was advised that pregnancy toxemia requires fastidious supportive therapy, and that the ewe will require assistance with lambing. The owner was amenable, and a plan was made to treat the ewe with 60ml oral propylene glycol and mineral solution 3 times daily for 5 days, while monitoring for appetite, demeanor and labor. The ewe would be re-assessed if any decline in appetite or demeanor were noted. Cesarean section was discussed, but declined as the gestational day was undetermined.

The ewe lambed with triplets (2 alive, 1 dead) 10 days later, with assistance from the owner. She was re-checked 48 hours later. At that time, her vital parameters were within normal limits, and her blood BHB was 1.8 mmol/L. She was producing very little milk, and had mild edema of her cranial udder. The abdominal protrusion was decreased in size, but still prominent (Figure 4). On palpation, the contents appeared to be abdominal viscera; they were soft and easily reducible. There was no pain on palpation, and no distinct borders or rings were appreciable. Ultrasound examination revealed a gradual decrease in body wall thickness from the caudal aspect to the center of the mass, measuring 7.5mm caudally, 3mm centrally and 6mm cranially (Figure 5). Lymph nodes, uterus, loops of intestine and rumen were identified beneath the mass, and there were no indications of incarceration.

Figure 4. The abdominal mass and udder 48 hours after lambing. Left is caudal, right is cranial

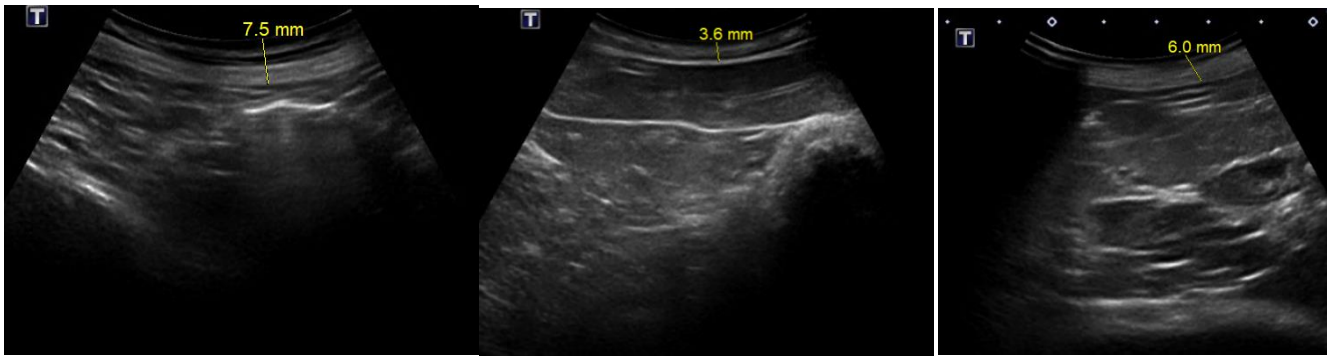

Figure 5. Ultrasound images of the abdominal protrusion using a 3.5MHz convex probe, showing gradual decrease in body wall thickness. Left image is caudal mass, middle image is central mass and right image is cranial mass.

A large abdominal hernia was diagnosed. Given the size of the defect, prognosis for return to breeding was poor. The owner opted for surgical correction, in order to keep the ewe as a pet. The lambs were removed for bottle feeding, and the ewe was dried off. Surgery was scheduled in 1 month, to allow for mammary gland reduction and complete uterine involution. However, the ewe was only presented 7 months later due to the owner having a medical emergency. At this time the hernia size was further enlarged (Figure 6).

Figure 6. Abdominal hernia, on the day of surgery. 7 months after initial presentation.

With the guidance of a board-certified large animal surgeon, a hernioplasty was performed using prolene mesh. Pre-operatively, she was given 1mg/kg of meloxicam and 6.6 mg/kg of ceftiofur[e] subcutaneously. The ewe was maintained under general anesthesia, and the defect was first investigated laparoscopically. A thick muscle band was seen delineating the caudo-medial margin, identified as the rectus abdominis muscle. Moving laterally, the muscle fibers were markedly thinned, and appeared to be frayed/ruptured in areas. The anatomy was abnormal and difficult to interpret, but it appeared that only the transversus muscle, and at some points the internal abdominal oblique muscle were intact. The defect was outlined on the skin, and a 40 cm curved incision was made at the ventro-medial aspect. The mesh was sutured to the medial aspect of the defective abdominal wall circumferentially. The ewe recovered uneventfully. Upon standing, the mass effect was gone, and bandaging was placed around the abdomen for incisional support (Figures 7 and 8).

Figure 7. Post-operative appearance of hernia site.

Figure 8. Belly bandage applied post-operatively to reduce pressure on incision site.

Feed was re-introduced at a rate of 25% of daily intake, split into 4 feedings, over 5 days. The ewe was maintained on antimicrobials; 6.6mg/kg ceftiofur subcutaneously, once to be given in 3 days, and 21,000 IU/kg penicillin[f] intramuscular, once daily for 5 days. The bandaging remained for 2 weeks, until skin sutures were removed, and was changed if soiled or wet. The ewe was discharged from hospital 2 days

after surgery, and rechecked for suture removal 2 weeks later. At recheck, she was bright and alert, had normal physical examination and the incision was healing well. No post-operative complications were encountered, and the ewe was maintained successfully as a pet.

Endnotes

a. Propylene glycol USP 100%, manufactured by Radix Laboratories Inc., WI
b. Ketamalt 50, Bimeda-MTC Animal Health Inc., ON Canada
c. Meloxicam 20mg/ml solution for injection, Metacam, manufactured by Boehringer Ingelheim Animal Health, ON, Canada
d. Calcium Borogluconate 23%, manufactured by Vetoquinol, QC Canada
e. Ceftiofur crystalline free acid 200mg/ml, Excede 200, manufactured by Zoetis, NJ
f. Benzylpenicillin procaine solution 300,000 IU/ml, Depocillin, manufactured by Merck Animal Health, QC Canada

References

1. Al-Sobayil FA, Ahmed AF. Surgical treatment for different forms of hernias in sheep and goats. *J Vet Sci* 2007; 8: 185-191.
2. Al-Ani FK, Khamas W. Ruptures of prepubic tendon in Shami (Damascus) breed pregnant goats. *Transl Biomed* 2016; 7: 74.
3. Amare E, Haben F. Hernias in farm animals and its management technique – a review. *IJCMC* 2020; 4(4): 001.