

URINARY BLADDER RUPTURE SECONDARY TO OBSTRUCTIVE UROLITHIASIS IN A JAMNAPARI GOAT

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SUMMARY

This report describes the complications of obstructive urolithiasis in the lower urinary tract causing urinary bladder rupture in a Jamnapari buck. A 3-year-old Jamnapari buck was presented with the complaint of stranguria, subsequent anuria and a progressively distended abdomen for the past three days. Upon physical examination, body temperature, pulse rate and respiration rate were increased. Uroliths could be felt within the urethra in the ventral abdomen region. A urolith was removed via amputation of the urethral process, but the patency of the urethra could not be established. Transabdominal ultrasound revealed anechoic areas around the bladder, and the bladder was relatively small for a urinary obstructed goat. The bladder wall was thickened and shadow of sludge was observed within the bladder. Abdominocentesis was done and fluid analysis revealed that it was a haemorrhagic effusion. Blood results revealed renal disease, liver disease, muscle injury and haemoconcentration. Retrograde cystourethrogram revealed no uroliths within the urethra but there was leakage of the contrast agent from the bladder into the peritoneal cavity. The final diagnosis was complete blockage of the lower urinary tract leading to bladder rupture. Exploratory laparotomy was done and emergency cystorrhaphy was planned. Due to the poor condition of the urinary bladder with presence of septic peritonitis, the goat was euthanised.

Keywords: Obstructive urolithiasis, bladder rupture, goat, cystourethrogram

INTRODUCTION

Obstructive urolithiasis within the urinary tract is a condition where salt precipitates and insoluble minerals obstruct the urine flow anywhere from the renal pelvis where urine is formed, to the distal urethra where urine is excreted from the body. In goats and sheep, the common site for uroliths to lodge is at the sigmoid flexure and urethral process (Ewoldt *et al.*, 2008, Khairuddin *et al.*, 2016). Obstructive urolithiasis is the most common urinary tract disease in breeding rams and goats (Haven *et al.*, 1993). The irritation at the lodged site causes inflammation and swelling leading to urethral occlusion. A partial obstruction will show signs of dribbling blood-tinged urine after stranguria, tenesmus, tail twitching, colic signs, bloat, and rectal prolapse may also be seen. On the other hand, a complete obstruction may give signs similar to partial obstruction with the absence of dribbling urine after stranguria. The severe sequelae following a complete obstruction is urinary bladder rupture causing uroperitoneum. Uroperitoneum is accumulation of urine in the peritoneal cavity caused by leakage of urine from any part of the urinary tract such as kidneys, ureters, urinary bladder, urethra, or from a rupture persistent urachus (Braun *et al.*, 2015). Surgical intervention such as cystorrhaphy is required to repair the ruptured bladder. The purpose of this report is to describe a case of lower urinary tract obstructive disease causing urinary bladder rupture in a Jamnapari goat. The diagnosis for this case was based on contrast urethrography and exploratory laparotomy performed.

CASE REPORT

History

A 3-year-old intact Jamnapari buck was presented to the University Veterinary Hospital (UVH), Faculty of Veterinary Medicine, Universiti Putra Malaysia with the complaint of stranguria, subsequent anuria and progressive distention of the abdomen for the past three days. The goat was a pet that was managed intensively and fed with concentrates throughout its life.

Clinical and Diagnostic Findings

Upon physical examination, the animal was dull and depressed with a body condition score of 3 out of 5 and weighing 56 kg. The goat had a normal body temperature (38.5°C) but was tachycardic (140 bpm), and tachypneic (48 bpm). Mucous membranes were congested with a capillary refill time of 3 sec and a dehydration status of less than 5%. A bilaterally distended abdomen with fluid wave was felt upon ballottement. Gritty texture could be felt within the urethra at the ventral abdominal region. A urolith was removed via urethral process amputation, but the patency of the urethra could not be established. Based on the history and clinical signs, the tentative diagnosis was obstructive urolithiasis and a possible ruptured bladder.

Haemogram and serum biochemistry results revealed hyponatraemia, hypochloreaemia, extremely elevated urea and creatinine at 5-6 folds increased from normal value, slight elevated muscle enzymes, elevated GGT, and hyperproteinaemia. All these findings

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Figure 1. Ultrasonography of the bladder revealed thickened wall and sediment within the bladder - It was considered relatively small urinary bladder for a goat with history of four days urinary obstruction



Figure 2. Cystourethrogram revealed leakage of contrast agent from the apex of bladder

combined with clinical findings are supportive of possible uroperitoneum and urethral obstruction. Transabdominal ultrasonography revealed thickened urinary bladder wall and hyperechoic sediments within the bladder. The size of the bladder was relatively small for a buck that has been obstructed for four days. The discontinuity of the bladder wall was not observed but anechoic areas surrounding the bladder were suggestive of a ruptured urinary bladder (Figure 1).

Abdominocentesis was conducted using a 20 ml syringe and a 21G butterfly catheter inserted at the ventral midline abdomen at the level cranial to the distal urethra. One litre of serosanguinous fluid was aspirated to relieve the abdominal distension temporary. The fluid analysis resultsshowed a pH of 9.0, specific gravity of 1.016, numerous erythrocytes (4+), moderate turbidity (2+), proteinaceous (4+), moderate glucose content (2+) and moderate leukocyte count (2+). Based on the specific gravity, protein level, and cellularity, the fluid was classified as modified transudate.

Urethral catheterisation was performed using a 5 French feeding catheter (Kruuse, UK). The catheter was advanced up to 12 cm from the urethral opening. Contrast material (Omnipaque, IohexolGE Healthcare, 0.5 ml/kg,

diluted with 100 ml of normal saline) was injected into the urethra for a cystourethrogram. A total of 50 ml Iohexol and 100 ml of normal saline was infused and the urinary bladder was outlined with a small amount of contrast agent observed to leak out from the apex of bladder together with some small and numerous radiopaque structures (Figure 2). Based on this evidence, the final diagnosis for this buck was bladder rupture. The goat was immediately scheduled for a cystorrhaphy.

Surgical Procedure

The buck was pre-medicated with tramadol 3 mg/kg (Analab, Biolab Co. Ltd.), induced with ketamine 4 mg/kg (Ilium Ketamil, Australia) and diazepam 0.2 mg/kg (Diapine Injection, Atlantic Laboratories, Thailand) and maintained with isoflurane (Isoflurane Pennsylvania, USA) at 3%. Normal saline at the rate of 50 ml/min for was administered intravenously throughout the surgery.

The goat was positioned on dorsal recumbency with both forelimbs and hindlimbs extended. The whole ventral abdomen and inguinal region were clipped and surgically prepared. A ventral midline abdominal incision of approximately 10 cm was made at the caudal abdomen with a size #20 scalpel blade. The subcutaneous tissues were undermined using Mayo scissors. The incised skin and subcutaneous tissue were retracted laterally and the lineaalba and the external fascia of the rectus abdominis muscle were identified. The lineaalba was incised with a size #15 scalpel blade. Immediately after incision of the lineaalba, the tip of a suction pump was inserted through the incision into the peritoneal cavity to aspirate the peritoneal effusion. The incision was extended and the urinary bladder was identified.

The peritoneal wall was severely inflamed (Figure 3) and the abdominal organs were adhered with fibrin, which indicated acute severe peritonitis. The serosal layer of the bladder was hyperaemic and severely inflamed. A point of rupture was identified at the dorsal aspect of the bladder body, approximately 1.5 cm in diameter (Figure 3). Urine constantly leaked out from the rupture site. Stay sutures were placed on the serosal wall of the bladder. It was unable to hold the bladder in place as the sutures were detached easily due to the fragile necrotised bladder wall. The bladder was then isolated manually from the abdominal organs with hands and placed on moistened laparotomy pads for examination. The bladder mucosawas severely inflamed and necrotised with an irregular surface (Figure 4). Stones or sand were not found within the bladder, hence it was suspected that the stones were now within the peritoneal cavity. Five liters of serosanguineous peritoneal fluid mixed with large amounts of brick red fibrin were removed from the abdominal cavity.

Due to the poor prognosis from the severe cystitis and septic peritonitis, the owner elected to euthanase the animal. Post-mortem reveals severe inflammation of the bladder mucosa, with an ulcerated and irregular, hemorrhagic and hyperemic surface (Figure 5).

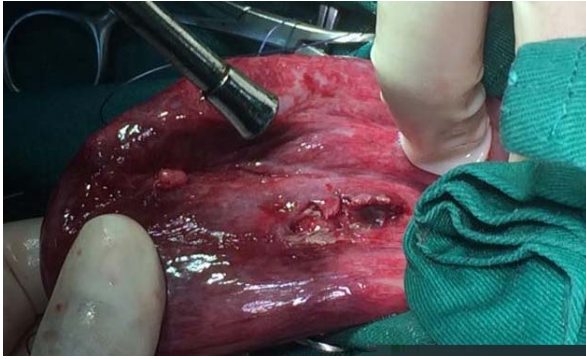


Figure 3. Bladder wall point of rupture was identified at the dorsal aspect of the bladder body

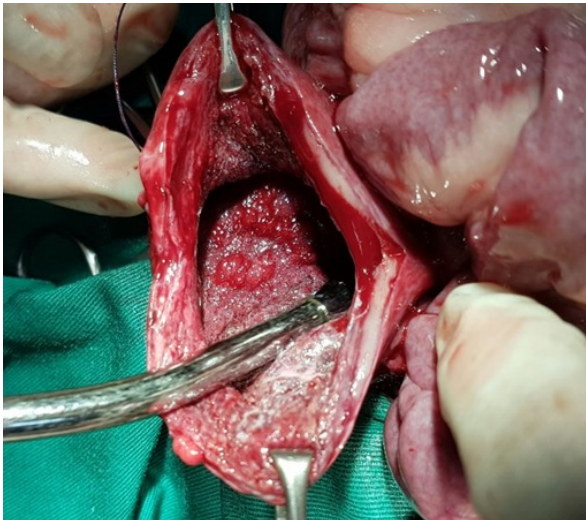


Figure 4. The bladder mucosa was severely inflamed and necrotized with irregular surfaces



Figure 5. Post mortem section of the bladder mucosa showed severely inflamed and ulcerated with an irregular, haemorrhagic and hyperaemic surface

DISCUSSION

In the case of prolonged complete urinary blockage, urinary bladder rupture is expected (Baxter *et al.*, 1992). Upon rupture, the animal will have relief from the abdominal pain, feel better, and have an improved demeanour and resumes eating. Over time however, the demeanour and appetite will gradually deteriorate due to septic peritonitis. The abdomen will be progressively distended bilaterally with a fluid filled consistency, giving the appearance of a pear-shaped abdomen. Upon succession of the abdomen, sloshing sounds can be heard. The animal may have tachycardia, ruminal atony as well as dehydration with enophthalmus and reduced skin turgor (Braun *et al.*, 2006).

Physical examination finding of uroperitoneum may not be enough to confidently diagnose a case of urinary bladder rupture. Further tests need to be done to confirm this diagnosis of a ruptured bladder. Upon rupture, large amounts of liquid accumulate in the abdomen. This liquid will proceed to equilibrate its electrolytes, small molecules, and osmolarity with extracellular fluid (Schechter *et al.*, 1933). Blood serum analysis will show hyponatremia and hypochloraemia due to the low concentration of sodium and chloride in the urine. Thus, these ions will move from the interstitial compartment into the peritoneal cavity through the peritoneal wall. However, one study on 5 calves showed that serum sodium and chloride did not change significantly over a 40 hours (Wilson *et al.*, 1998). Serum will also show high plasma protein indicating haemoconcentration as water is drawn out from the interstitial compartment through the peritoneum into the peritoneal cavity due to the high osmolarity of the peritoneal effusion, causing further dehydration. Similarly, urea and creatinine from the urine in the peritoneal cavity will move from a high concentration area back into the interstitial space and back into the intravascular compartments causing an increase in blood urea and creatinine. However, creatinine which is a larger molecule than urea will take a longer time to diffuse back into the intravascular compartments (Donecker *et al.*, 1982).

Peritoneal fluid can be analysed to diagnose a case of uroperitoneum. By comparing the peritoneal to serum creatinine ratio, results of 2:1 or greater are diagnostic of uroperitoneum (Radostits *et al.*, 2007). On the other hand, the colour of fluid and classification of effusion can vary depending on the chronicity and septic status and therefore is not very useful in diagnosing uroperitoneum (Mayhew *et al.*, 2004).

Contrast radiograph is helpful to outline the urinary bladder wall and the leakage of the contrast will be apparent in cases of bladder rupture. To appreciate the condition and thickness of the bladder wall, a double contrast can be performed to outline the wall thickness and the condition of the mucosa (Palmer *et al.*, 1998). Abdominal ultrasonography is another useful modality to show the outline of the urinary bladder and rule out any other causes of peritoneal effusion. In cases of uroperitoneum, abdominal organs can be seen separated from each other and from the peritoneum, with large

volumes of anechoic to echoic fluid present between the organs and appear suspended in the fluid. The bladder could be collapsed, flaccid or contain varying amounts of urine depending on the severity of the rupture (Braun *et al.*, 2015). Cystoscopy is another useful technique for the detection of bladder rupture, whereby a flexible video endoscope with an optimum diameter of about 9 mm and working length of about 100 cm is used to show the location and extent of the rupture in the bladder wall. In this case, the leakage of the contrast agent from the urinary bladder in the abdominal radiograph is sufficient to diagnose it as a urinary bladder rupture.

For this case, the urinary bladder ruptured as a sequela of prolonged obstructive urolithiasis of the lower urinary tract of the buck. Feedlot animals or small ruminants which are fed a high concentrate diet are commonly affected with obstructive urolithiasis caused by magnesium ammonium phosphate uroliths. Phosphate uroliths generally have multiple small, soft, amorphous stones or sludge in the urinary tract. One of the main factors for development of urinary stones is the high urinary phosphorus level. Prolonged high levels of phosphorus in the urine are more likely to increase the chances to develop uroliths.

Naturally, excessive phosphorus level in the circulation will be excreted in the ruminant's saliva during chewing, and swallowed back to enter the intestinal tract, either to be reabsorbed or lost in the faeces. In a dietary regime with inadequate roughage, less chewing and less excretion of phosphorus in the saliva will result in an excess of phosphorus in the circulation that will then be excreted via the kidney and urinary system. Prolonged high concentrations of phosphorus excreted in the urine contribute to the formation of the consolidated form of phosphate uroliths (Hay, 1990). In order to increase salivation, diets high in roughages are recommended to increase chewing to stimulate salivary flow and rumination. Loose forms of concentrate also increase salivary flow compared to pelleted forms.

During the early growth stage of goats, dietary phosphorus levels should not exceed 0.6% of the ration, while later in life the level is lowered down to 0.4%. Calcium supplementation will ensure that the balance of calcium:phosphorus ratio is greater than 2:1, which will lead to a decrease in phosphorus absorption from the intestinal tract, thus leading to decreased urinary excretion. Similar with other type of uroliths, concentrated and small amounts of urine will increase the precipitation of phosphate crystals, predisposing to the formation of uroliths. Ad libitum clean fresh water and feeding of roughage instead of concentrate will increase fluid intake to increase the urinary volume. Gradual inclusion of sodium chloride into the diet will also encourage water intake. Urinary pH also plays an important role in formation of phosphatic uroliths. Roughage diets will generally increase the pH of urine up to between pH 7.8 and 8.5 which is favourable for the precipitation of magnesium ammonium phosphate uroliths. However, an acidic urine would favour the formation of an apatite form of urolith. Urine acidifiers of dietary chloride ions such as ammonium chloride or calcium chloride can be added into the diet to reduce the

pH and degree of saturation of urinary phosphate ions (Hay, 1990).

It is very important to owners and veterinarians to understand that complete urinary blockage in small ruminant are a life threatening emergency. As the condition progresses to a full urinary blockage and the animal is unable to pass any urine, it will lead to bladder rupture and kidney failure if left untreated for more than 48-72 hours. As in this case, the goat was brought for veterinary attention only after clinical signs of stranguria occurred for three days which ultimately resulted in a ruptured bladder.

The prognosis of urinary bladder rupture mostly depends on the causative factor. A traumatic bladder rupture has an excellent prognosis due to the short progression of the disease and the condition of the bladder wall is still good. On the other hand, a bladder rupture secondary to obstructive urolithiasis has a guarded prognosis which also depends on the extent of necrosis of the bladder wall (MacPhail *et al.*, 2013). Another study found that, serum sodium and serum phosphorus concentrations are useful prognostic indicators in steers with ruptured bladder. The lower the serum sodium (123.6 ± 10.6 mmol/L) and the higher the serum phosphorus (4.1 ± 0.8 mmol/L) would indicate a poorer patient's prognosis (Donecker *et al.*, 1982).

In conclusion, obstructive urolithiasis remains a problem for goats that are reared intensively and fed with a high concentrate diet and low roughage. Surgical intervention is often necessary to relieve urinary obstruction. However, economic feasibility and the value of the animal dictates the treatment options available to the patient especially if it is a production animal.

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CONFLICT OF INTEREST

None of the authors have any potential conflicts or interest to declare.

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