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Urolithiasis in Boer Bucks

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ABSTRACT

This paper describes three cases of urolithiasis in adult Boer bucks. The affected bucks were among the 50 breeders kept under intensive system given cut and carry Napier grass at the rate of 2 kg/animal/day. In addition, the animals were also supplemented with commercial goat pellets at the rate of 300 g/animal/day, 200-300 g of palm kernel expeller (PKE) and mineral block. The affected animals showed clinical signs of stranguria, anorexia, prolonged urination, dribbling urine, tail flagging and abdominal pain. Prior to death, they appeared to be depressed, recumbent, and showed abdominal distension. Supportive treatments in the form of anti-inflammatory drugs and oral drench of ammonium chloride (1%) were attempted. All the animals in this study died within 2 weeks following the onset of depression. Post-mortem examinations revealed swollen testis and severe haemorrhages in the urethra with blackish sandy material deposited within the lumen. There were ascites and swollen kidneys, while cloudy and thick urine filled the bladder. Histological examinations revealed the presence of purplish sandy material, either within the lumen or closely associated with the wall of the urethra. The urethral areas where the calculi were in-contact with the wall showed extensive necrosis with destruction of the epithelial layer and haemorrhages. Urinary calculi have a complex aetiology, but management, nutritional, and anatomical considerations can be helpful.

Keywords: Urolithiasis, Boer bucks

INTRODUCTION

Urolithiasis is a condition of the urinary tract, in which insoluble mineral and salt aggregate around a nidus of proteinaceous material within the bladder or urethra (Belknap & Pugh, 2002). These stones consist of combinations of various minerals and come in many shapes and sizes. Once the stones become too numerous or too large, they cause obstruction of the urethra at the vermiform appendage, the ischial arch or the neck of the bladder (Pinsent & Cottom, 1987). Urolithiasis can rapidly progress to bladder or urethral rupture, uremic crisis, and death (Baxendell, 1984). Males are more likely to be affected as females generally have a shorter, wider urethra (Matthews, 1999). Uroliths can occur in all species but they are a common problem in domestic ruminants (Matthews, 1999; Belknap & Pugh, 2002).

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In the past, urolithiasis had rarely been reported in Malaysia. However, with the increase in the import of and goat rearing, especially the Boer goat, urolithiasis has been promoted in the recent years. This paper describes the occurrence and pathological changes in Boer goats with urolithiasis.

CASE DESCRIPTION

Case History

This report describes three cases of urolithiasis involving imported adult Boer bucks aged >2 years old. The affected bucks were among the 50 breeder males kept under the intensive system. They were fed cut and carry Napier grasses at the rate of 2 kg/animal/day and supplemented with commercial goat pellets at the rate of 300 g/animal/day, palm kernel expeller (PKE) and mineral block. The affected animals showed clinical signs of stranguria, anorexia, prolonged urination, dribbling urine, tail flagging, and abdominal pain. Prior to death, they appeared to be depressed, recumbent and showed abdominal distension. Supportive treatments in the form of anti-inflammatory drugs and oral drench of ammonium chloride (1%) were attempted. Nonetheless, all the affected animals died within 2 weeks following the onset of depression.

CASE FINDINGS

In all cases, the post-mortem examination revealed swollen testis due the accumulation of fluid in the scrotum (*Fig. 1*). The prepuce was stained while the abdomen was distended, containing fluid (*Fig. 2*). Upon opening the urogenital system, there were severe haemorrhages and necrosis along the urethra with blackish sandy material within the lumen (*Fig. 3*). The urinary bladder contained cloudy and thick urine, while the kidneys were swollen with hydronephrosis (*Fig. 4*).

Histopathological examination revealed the presence of purplish sandy material, either within the lumen or closely associated with the wall of the urethra (*Fig. 5*). The urethral areas, where the calculi were in-contact with the wall, showed extensive necrosis with destruction of the epithelial layer and haemorrhages with numerous inflammatory cells (*Fig. 6*).



Fig. 1: Swollen testis with subcutaneous oedema and soiled prepuce



Fig. 2: Fluid-filled the abdominal cavity

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Fig. 3: Urethra of affected goat showing necrosis and accumulation of blackish, sandy materials



Fig. 4: Swollen kidneys with evidence of hydronephrosis



Fig. 5: A photomicrograph of an affected urethra showing the presence of urolith and surrounding necrosis (HE x40)

DISCUSSION

Goats of various breeds and purposes have been documented with urolithic stone problems in captivity (Bellenger *et al.*, 1981; Smith & Sherman, 1994; Gutierrez *et al.*, 2000). These stones often occur when concentrated or supplemented feed with high phosphorus content is presented to the goats (Blood *et al.*, 1989; George *et al.*, 2007). Therefore, diet and



Fig. 6: A photomicrograph showing the urolith, haemorrhages and inflammatory cells (HEx400)

animal management are considered as important risk factors in the formation of uroliths in ruminants (Blood *et al.*, 1989; Kahn *et al.*, 2005; George *et al.*, 2007). Although the relationship between diet and urolith formation in goats has not been clearly established, previous studies have suggested that particular diets contribute to the formation of struvite calculi (Blood *et al.*, 1989; Aitken, 2007). High-concentrated grain diets with approximately equal proportions of calcium and phosphorus and diets high in magnesium, potassium, and phosphorus are thought to predispose animals to calculi (Huang *et al.*, 1999).

Therefore, obstructive urolithiasis remains an often deadly problem for male goats, kept intensive. The solution involves understanding and implementing a proper balance of minerals and nutrients in their diets, while mitigating environmental factors to minimize the risk of this disease.

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