



Unilateral renal torsion in a lamb

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ABSTRACT

In ruminant, the kidneys are a major excretory organ that eliminate metabolic byproducts and regulate water-electrolyte homeostasis and maintain acid-base balance in the body. Therefore, kidney abnormalities cause a life-threatening effect on ruminants' health. Renal torsion has not been reported in any animal species. In the present report, a case of a two-month-old male lamb that has unilateral renal torsion has been described. In necropsy, the twisted left kidney was severely congested and was enlarged in size. Histopathological studies revealed renal tubular and glomeruli necrosis. Vascular changes consisted of congestion and thrombi in the blood vessels. Abnormalities in the embryonic developmental period and increased mobility of the renal structure due to ligaments associated with a twisted kidney are suspected causes of the occurrence of renal torsion.

Keywords

Kidney, torsion, ruminant, urinary tract

Abbreviations

H&E: Hematoxylin and eosin:

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Introduction

The kidneys are located in pairs in the sub-lumbar and retroperitoneal regions. They regulate water-electrolyte homeostasis and maintain acid-base balance in the body [1, 2, 3]. Each kidney has a convex lateral border and a concave medial border [1, 2]. In the medial border of the kidney, there is a fossa called the hilus, in which the nerves, arteries, and ureters of the kidney are located. Around the kidneys, depending on the animal species, a mass of adipose tissue called perirenal fat is seen. These fat masses are involved in maintaining the kidneys in their position in the abdominal cavity [1, 2].

The rotation of the kidney around its vascular pedicle is a very rare condition. To our knowledge renal torsion has not been described in animals. In human beings, renal torsion is an unusual surgical complication and causes occlusion of vascular structure and parenchymal infarction [4,5,6]. Renal torsion was reported in humans after kidney-pancreas transplant surgery usually in infants which have a prune-belly syndrome or any condition that allows the organ to move freely within the pelvis [4, 5, 6]. Probable causes of renal torsion in humans include the excess vascular pedicle or ureteral length, and lack of adhesion, which allows for abnormal mobility of the kidney and a higher probability of twisting [4, 5]. Renal torsion causes severe abdominal pain and decreased urine output and may occlude the ureter, resulting in hydronephrosis or renal pelvic wall thickening [4, 5]. Therefore, any damage and dysfunction of the kidneys can lead to serious complications. Renal torsion in a lamb which has led to kidney damage has been reported for the first time.

Case Presentation

In the winter of 2021, a dead two-month-old lamb was submitted to the veterinary clinic of Urmia University for necropsy. According to the owner, the animal died suddenly and before that, it only showed a decrease in appetite and activity. Because of sudden

death, it was not possible to obtain blood samples and laboratory tests. Post mortem examination of the lamb revealed the presence of unilateral renal torsion of the left kidney in the lamb (Figure 1). The kidney was dark brown and enlarged in size (renal length: 9.5 cm, width: 6.2 cm). Dimensions of the normal kidney were 6.3 × 3.8 cm. Appropriate tissue samples were taken from the kidney and were fixed in 10% neutral buffered formalin for 24 hr. The sections were stained by the hematoxylin and eosin (H&E) method. Histopathological studies revealed renal tubular necrosis characterized by pyknotic nuclei that sloughed into the lumen of the tubules (Figure 2). The glomeruli tufts were atrophied. Severe vascular changes comprised congestion and thrombi in the blood vessels. The interstitial tissue was diffusely expanded by edema and hemorrhage (Figure 2).

Discussion

Renal torsion in lamb has not been reported in the literature. The compression on the vessels due to the torsion is such that arterial blood still gets into the organ because of their thick muscular wall but the pressure on the thinner-walled veins, restricts the blood outflow and venous blood accumulation, resulting in edema, congestion, hemorrhage and eventually necrosis [9, 10]. Ureteral obstruction is associated with urinary retention and destruction of the renal parenchyma, leading to enlarged kidneys [9, 10]. Unilateral ureteral obstruction can quickly cause the infiltration of inflammatory cells into the interstitial tissue of the kidney [9, 10]. Oxidative stress appears to play a key role in the onset and continuation of inflammation after obstruction, resulting in renal tubular damage and interstitial tissue fibrosis [11, 12]. The histopathological findings of this case show that renal torsion caused necrosis in renal tubules and glomeruli. According to the human medical literature, renal torsion can be an early or late complication and may be incomplete and intermittent [7, 8]. In the best of circumstances, renal torsion can be repaired; in the worst cases, the kidney is lost [7, 8].

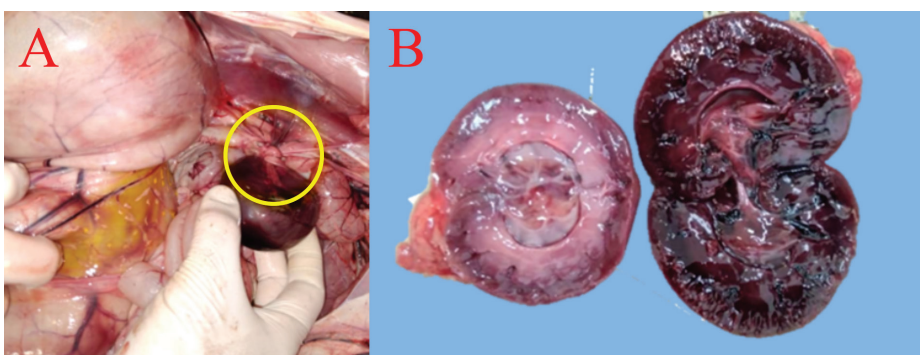


Figure 1.

The necropsy of the two-month-old lamb after sudden death. A) The axis of renal torsion around the vascular pedicle (circle). B) The sagittal sections of the normal and abnormal kidneys. Severe congestion and enlarged size are obvious.

Since this abnormal condition of kidney torsion has occurred at a very young age, it is likely to be due to a disorder in the fetal developmental stage. However, no increase in length was observed in the examination

of ligaments related to the twisted kidney, therefore the possibility of torsion due to increased mobility of the renal structure seems unlikely, although it can be one of the causes.

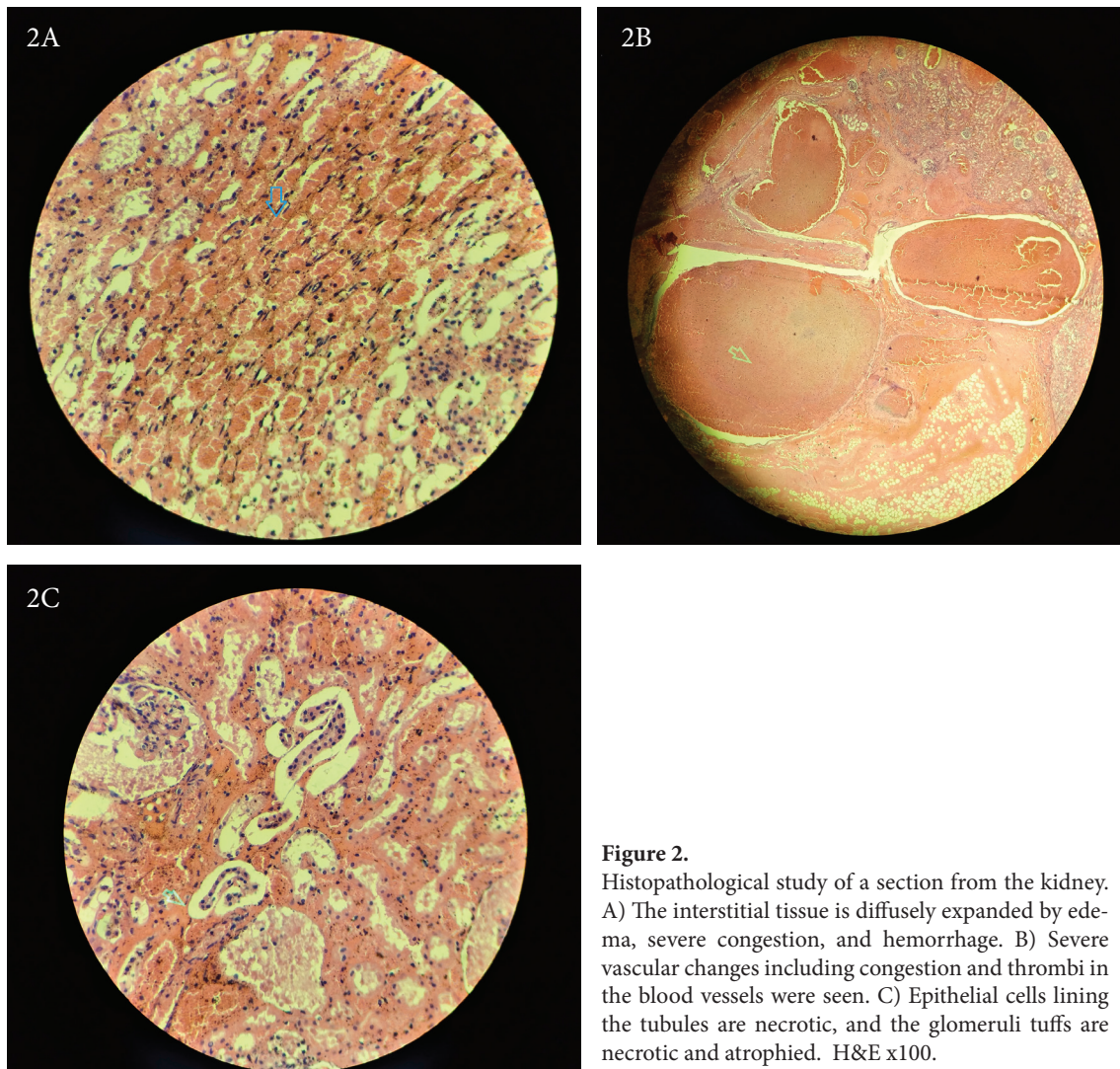


Figure 2. Histopathological study of a section from the kidney. A) The interstitial tissue is diffusely expanded by edema, severe congestion, and hemorrhage. B) Severe vascular changes including congestion and thrombi in the blood vessels were seen. C) Epithelial cells lining the tubules are necrotic, and the glomeruli tufts are necrotic and atrophied. H&E x100.

Authors' Contributions

Y.N., G.J., A.T., S.R., and A.M. contributed to sample preparation. A.B. took the lead in writing the manuscript. Y.N., G.J., A.T., S.R., and A.M. contributed to the interpretation of the results. G.J. took the lead in writing the manuscript.

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Competing Interests

The authors declare no competing financial interest.

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