

A CASE OF FATAL NEPHROLITHIASIS IN A PUG DOG: NECROPSY BASED STUDY

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SUMMARY

A two years old pug bitch was presented in post-mortem hall of Department of Veterinary Pathology, LUVAS, Hisar for the necropsy examination. Bitch had history of vomition and hematuria since eight days. Detailed necropsy of bitch was conducted and tissue samples were collected in 10% neutral buffered formalin for histopathological examination. Necropsy examination revealed brown discoloration of kidneys with petechial to ecchymotic haemorrhages on surface, firm consistency and tight adherence of renal capsule. Cut surface of kidney showed presence of white large calculi in renal pelvis. Urinary bladder was engorged with brown urine and mucosal surface showed presence of small calculi with marked echymotic haemorrhages. Histopathological examination revealed severe chronic nephrolithiasis and nephritis along with hemorrhagic cystitis.

Keywords: Cystitis, Jaundice, Nephritis, Nephroliths, Pug

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Urolithiasis is the formation of calculus, which can affect kidneys, ureters, bladder or the urethra. When these are present in the kidneys, are called nephroliths. Less than 5% of canine uroliths are found in the kidney and ureter (Osborne *et al.*, 2009). Uroliths has the potential to cause physiological changes in the urinary tract, since the development of inflammatory process at the place of affection until complete obstructions, that can cause signs of post-renal uremia and effects on other visceral organs (Lulich *et al.*, 2011). Among the factors that contribute to formation of the uroliths, we can cite the urine pH level, low water consumption and the type of diet that is consumed, beyond the race. There are few reports from surgical point of view on urolithiasis in dog (Uma *et al.*, 2018; Rick *et al.*, 2018).

Few case reports on necropsy based investigation on urolithiasis were found in other breeds of canine (Muhammad *et al.*, 2016; Rick *et al.*, 2018). However, there are no reports on necropsy based pathomorphological investigation on nephroliths and cystoliths along with other vital organs in pug breed. Therefore, the present report describes the necropsy based pathological alterations in case of nephrolithiasis in pug breed of dog.

A carcass of female pug dog was presented for the post mortem examination in the Department of veterinary pathology, LUVAS, Hisar immediately after its death with a history of hematuria and vomition. Post mortem was performed within 24 hr and samples from the affected organs were collected. Tissues were fixed in 10% neutral buffered formalin, processed routinely, stained with Hematoxylin and Eosin (Luna, 1968). DeGallantha staining for crystal demonstration in tissue was also

performed (Luna, 1968). At necropsy, body condition of carcass was emaciated (Fig. 1 A). Abdomino-pelvic cavity was filled with blood tinged red coloured fluid. Both the kidneys were enlarged, markedly hard in consistency, dark brownish in colour with profuse haemorrhages (Fig. 1 B). Cut surface of kidneys showed presence of large whitish uroliths in renal pelvic area (Fig. 1 C). Urinary bladder was filled with blood tinged urine. Mucosa of urinary bladder was thickened, corrugated and hemorrhagic. Small uroliths were attached and partially embedded in mucosa of urinary bladder causing marked erosion (Fig 1D). Liver was yellowish in colour and having multifocal congested areas (Fig. 1 E). Spleen was slightly enlarged (Fig. 1 F). Gastric mucosa was thickened and showed presence of small ulcers (2-4mm in diameter) (Fig. 1 G). Stomach was filled with blackish indigesta. (Fig. 1 H). Microscopic examination revealed crystal formations in lumen of proximal tubule with interstitial nephritis in both kidneys. Extensive haemorrhages (Fig. 2 A) were also observed in cortical area of kidney. Marked proliferation of fibrous tissue in interstitial space compressing the tubules causing tubular atrophy indicated chronic interstitial nephritis (Fig. 2 B). It was further accompanied by formation of hyaline cast in tubular lumen and infiltration of mononuclear cells (Fig. 2 C). DeGallantha staining confirmed the presence of black coloured crystals in renal parenchyma (Fig. 2 D). Urinary bladder also revealed pathological changes such as necrosis of epithelium, marked haemorrhages and mononuclear cell infiltration in mucosa and submucosa (Fig. 2 E). Liver showed presence of yellowish bilirubin pigment in hepatocytes and spleen revealed marked hemosiderosis (Fig. 2 F). The incidence of formation of renal calculus in dogs is extremely slow compared to human species (Bartges *et al.*, 2015). The diet of the patient

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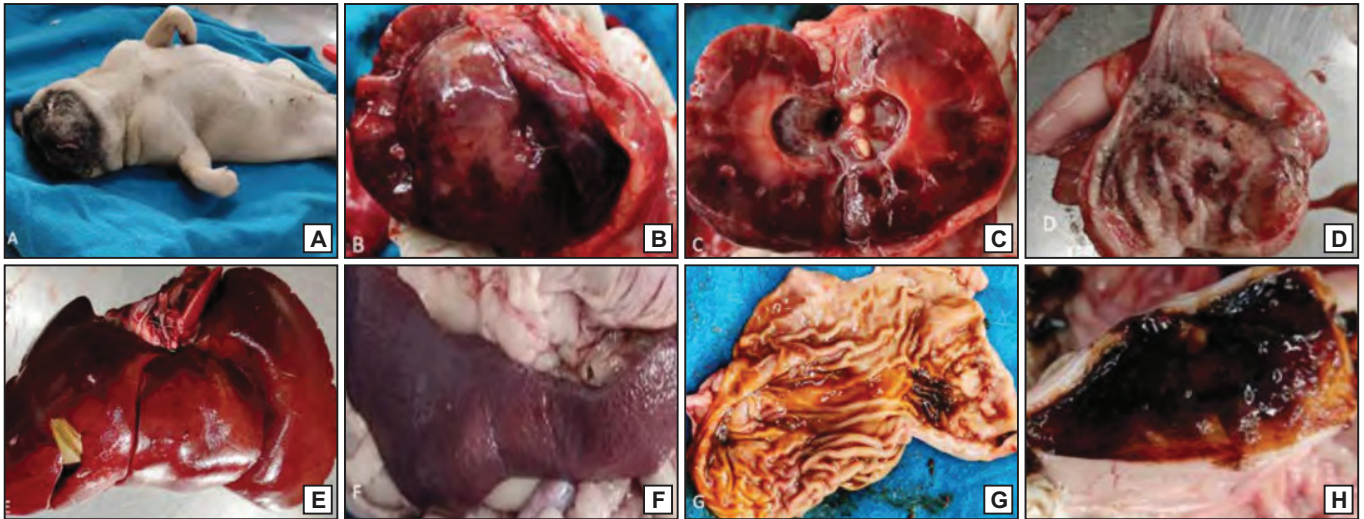


Fig. 1. Gross lesions in kidney and other visceral organs in pug A : Emaciated condition of pug; B : Kidney with fragile consistency, diffuse petechial to ecchymotichemorrhages and adherence of capsule; C : Incised kidney studded whitish large triangular nephroliths in renal pelvis; D : Urinary bladder with thickened, corrugated hemorrhagic mucosa with cystoliths; E : Liver showing moderate yellowish discoloration of surface; F : Spleen showing slight enlargement; G : Stomach showing severe corrugation and reddish discoloration of gastric mucosa; H : Blackish content of stomach indicating gastritis and bleeding from ulcers on mucosa

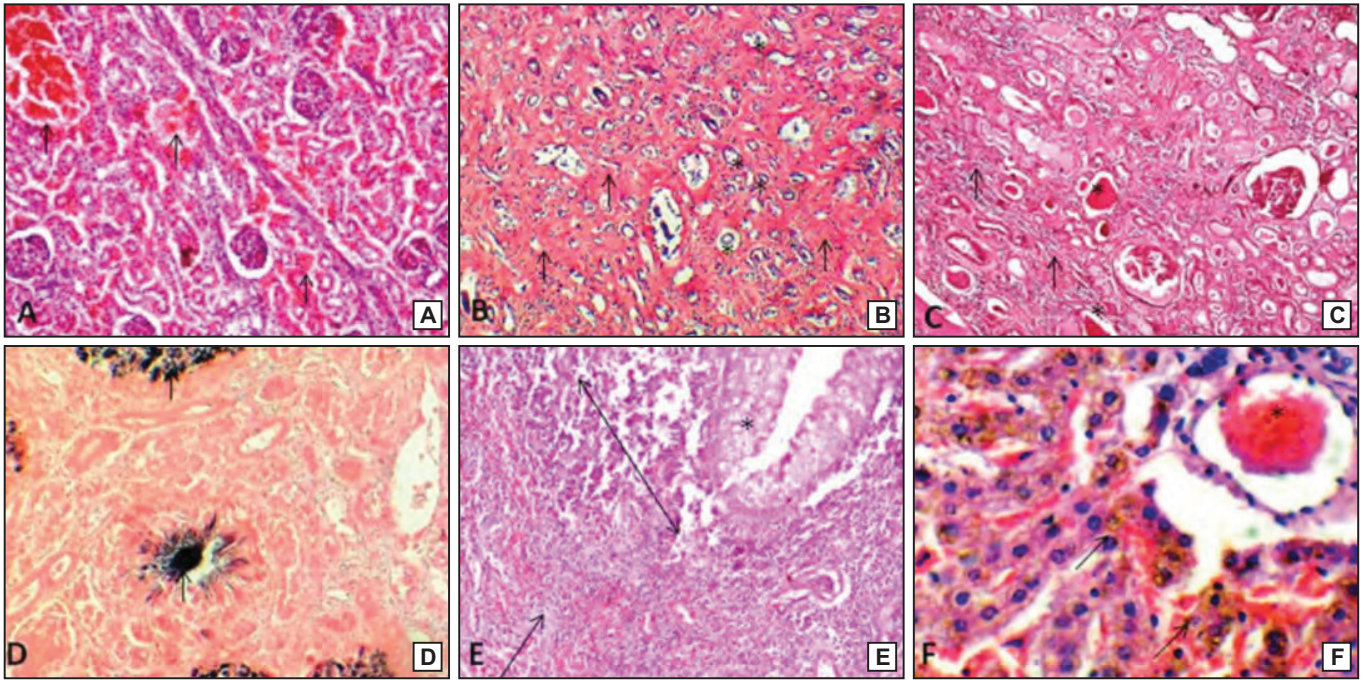


Fig. 2. Microscopic lesions in kidney and other visceral organs in pug A : diffuse severe haemorrhages (arrow) in intertubular area of renal parenchyma H& E stain x100; B : Proliferation of fibrous tissue (arrow) in interstitial space with tubular atrophy (asterisk) H& E stain x100; C : Presence of hyaline casts in tubules (asterisk), mononuclear cell infiltration (arrow) and glomerular atrophy H& E stain x100; D : Blackish coloured crystals in renal parenchyma, Degallantha stain x100; E : Urinary bladder showing necrosis of epithelium (asterisk) edematous appearance of cells, heavy infiltration of leucocytes (double arrow) and haemorrhages H& E stain x 100; F : Liver showing presence of yellowish coloured bilirubin pigment (arrow) in hepatocytes in centrilobular region, H & E stain x 100

can be a predisposing factor to the occurrence of calculus along with the less daily intake of water (Grauer *et al.*, 2009). Diet composed of partially with dog ration and homemade food, can increase the chances of uroliths formation. In the present case, dog was on mixed diet which could have acted one of the important contributory factor in formation of uroliths (Hunprasad *et al.*, 2019). This fact can be explained, by the reasoning that the

sodium in low concentrations in the diet, would not induce a low voluntary consumption of water and consequently would decrease the urinary volume. Hematuria/oliguria and anemia observed before death resulted due to chronic injury by stones and process of inflammation (Rovaris *et al.*, 2020; Sonu *et al.*, 2020). Severe chronic interstitial nephritis observed in present case is due to accumulation of toxic wastes in kidney over time causing severe necrosis

of tubules, hyaline cast formation and fibrous tissue proliferation (Lobetti *et al.*, 2001; Gupta *et al.*, 2021). Necrohemorrhagic cystitis observed in present case showed characteristic lesions such as thickening and corrugated mucosa and cystoliths in lumen and attached to mucosa (Rovaris *et al.*, 2020). Due to obstruction of urinary tract, uremia was resulted in present dog as evidenced by significant pathological changes in other visceral organs such as yellowish discoloration of liver, splenic enlargement, ulcers and thickening of gastric mucosa (Mshelbwala *et al.*, 2016). These alterations indicate that a circulatory disturbance occurred in a retrograde manner because of the increase in hydrostatic pressure caused by the blockage of urinary flow. Early detection, diagnosis and proper diet composition can prevent occurrence of urolithiasis in canine.

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