
VISCERAL GOUT IN A ROCK DOVE

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ABSTRACT

A rock dove was presented to the Department of Veterinary Pathology at DUVASU, Mathura, for post-mortem examination with a history of reduced water and feed intake, greenish diarrhoea and anorexia for four to five days. On external examination, the carcass showed pale visible mucous membranes. White chalky material was deposited in visceral organs, especially on the surface of the heart, kidneys and peritoneum. Histopathological examination revealed congestion in the liver and kidneys. The De Galantha stain of the kidney and heart tissue showed black uric acid crystals.

Keywords: Visceral gout, De Galantha, Urate Crystals

INTRODUCTION

Gout is a metabolic disorder commonly observed in birds that leads to the abnormal accumulation of urates. It is

encountered in infectious and non-infectious conditions. Factors such as high levels of calcium and vitamin D₃ coupled with low phosphorus in the diet, excessive intake of sodium bicarbonate, dehydration, vitamin A deficiency, exposure to mycotoxins and certain antibiotics like sulphonamides and aminoglycosides can initiate gout (Schmidt *et al.*, 2003). Moreover, various infectious agents including nephropathogenic infectious bronchitis virus (IBV), avian nephritis virus (ANV) and chicken astrovirus (CAstV) have the potential to induce gout in poultry (Bulbule *et al.*, 2013). When kidney gets damaged, the excretion of uric acid is impaired, hyperuricaemia ensues and the urate crystals get precipitated in various visceral organs (Wu *et al.*, 2020). Gout manifests in two forms namely, articular gout and visceral gout, the latter being more common. Incidence of both visceral and articular gout have been observed in various bird species, such as pheasants, Japanese quail, ducks, pigeons

and chickens (Rao *et al.*, 1993). This paper discusses a case of visceral gout in a rock dove.

MATERIALS AND METHODS

A piebald rock dove carcass was presented to the Department of Veterinary Pathology at DUVASU, Mathura, for post-mortem examination with a history of reduced water and feed intake, dullness, dehydration, diarrhoea and anorexia for four to five days (Fig. 1A).

The tissue samples from heart,

lungs, kidneys and liver were collected in 10 % formalin for routine H&E staining. The formalin-fixed tissues were washed overnight under running tap water and then dehydrated in ascending grades of alcohol (70%, 80%, 90%, 95% and two changes of 100% ethanol). The tissues were cleared in two changes of xylene and impregnated with two changes of paraffin at 58°C for 2 hours. The paraffin blocks were prepared. The sections were cut at 4.5 to 5 µm thickness with a semi-automatic rotary microtome and the slides were stained with routine H&E stain.

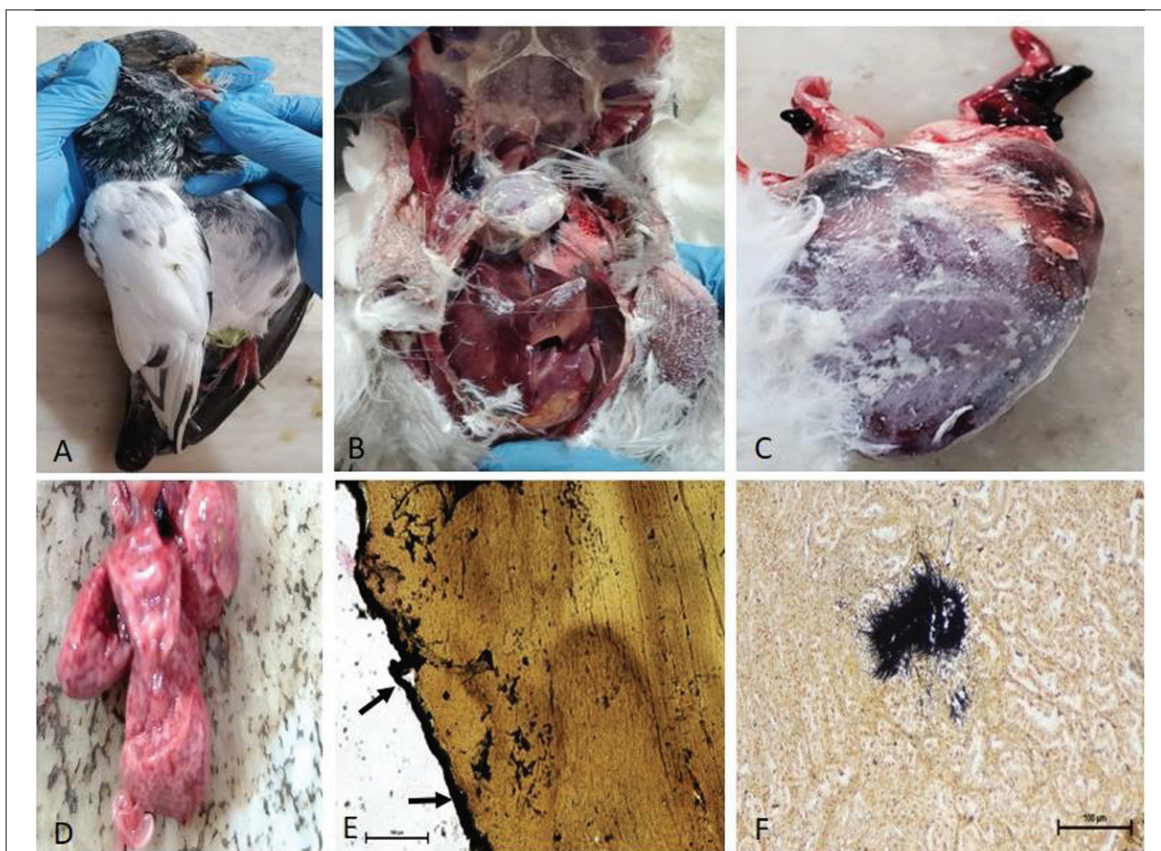


Fig. 1 A. A rock dove carcass presented with the history of greenish diarrhea. B. Chalky white deposits of urate crystals over the pericardium and other viscera. C. Heart showing white frosty material on the surface. D. Kidneys are grossly enlarged. E. Pericardium showing black urate deposits (arrows) De Galantha's stain 200x F. Renal parenchyma showing black needle shaped urate crystals. De Galantha's stain 200x.

For De Galantha's stain the tissue samples were collected in absolute alcohol and fixed for 48 hours. Further, the tissue samples were transferred in xylene paraffin solution (1:1) for 2 hours and then in paraffin for 1 hour (58°C). The tissues were embedded in paraffin and the paraffin blocks were prepared. The tissue sections were cut at 8 µm thickness on clean glass slides. The tissue sections were deparaffinized and dehydrated using two changes of xylene and absolute alcohol each. The tissue sections were transferred to silver nitrate solution and exposed to the sunlight for three hours. Sunlight exposure enhances the staining process, resulting in the bright rose colour of urates, a key indicator of successful staining. Freshly prepared developing solution was applied over the slides until urates turned black and connective tissues yellow. The slides were quickly rinsed in hot water (58°C) to remove excess reagents and halt the staining reaction. Dehydration was done by immersing the slides in absolute alcohol and clearing in xylene using two changes of each solvent (De Galantha, 1935; Luna, 1968).

RESULTS AND DISCUSSION

Gross examination of the carcass revealed deposition of white chalky material on the surface of various visceral organs (Fig. 1 B). Diffuse chalky white material

was seen on the pericardium (Fig. 1C), visible congestion in the lungs, liver and swelling of kidneys (Fig. 1 D) were also seen as reported in earlier studies (Srivastav *et al.*, 2024). On microscopical examination, distension of hepatic sinusoids with blood and degeneration of hepatocytes were noted. Kidney sections showed congestion in renal parenchyma. The De Galantha stain of heart showed diffuse deposition of uric acid crystals in the pericardium (Fig. 1E). The De Galantha staining of kidney showed black needle shaped uric acid crystals in the renal parenchyma deposited focally (Fig. 1F).

Visceral gout is a common metabolic disorder occurring in birds as a result of multifactorial etiology like nutritional and infectious causes, mycotoxins and water deprivation. In renal dysfunction, the normal uric acid excretion is interfered resulting in hyperuricaemia and deposition of urate crystals in various visceral organs like pericardium, liver and spleen (Lierz, 2003). Histologically the uric acid can be found within the lamina propria of the proventriculus, ventriculus, intestine or within the renal parenchyma, or in any tissue (Echols, 2006).

CONCLUSION

Uric acid crystals can be demonstrated in the histological sections

by De Galantha stain which imparts black colour to the crystals. Although uric acid itself is not toxic, precipitated crystals can be highly irritating and can result in severe damage to tissues, including the kidneys, heart, lungs, intestines (leading to visceral gout) and joints (causing articular gout). Gout is a disorder that causes huge economic losses to the poultry industry. Due to its multifactorial aetiology, the underlying mechanism of gout must be understood for better management of the condition.

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