Metastatic Calcification on Digestive System of Sumatran Rhino "Torgamba" with Polycystic Kidneys Disease

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Introduction

Torgamba was one of the first wild-born Sumatran rhinos that rescued for study and breeding purposes in captivity. After living for 11 years in Port Lympne Wild Animal Park, England he was transferred back to Indonesia to newly-opened Sumatran Rhino Sanctuary (SRS) in Way Kambas National Park, Lampung Province. He had a normally behavior and good browser with natural diet. This elderly male Sumatran rhino (estimated to be 32 years old) was dead, following a long illness of chronic kidney disease. The latest laboratory result showed ureum 98 mg/dL, creatinine 7.2 mg/dL, and imbalance ratio of calcium-phosphor.

Materials and methods

A cadaver of Torgamba was brought to our Pathology Division of the Faculty of Veterinary Medicine, Bogor Agricultural University (IPB). Necropsy was performed on the carcass within 13 hours of death at the same day, and all organs were collected for routine histopathological examination with Hematoxylin-Eosin and Von Kossa stainings.

Results

Gross findings :

The surface of esophagus showed some erosion and the stomach was full of compact and fermented food due to gastric dysfunction. The first part of the colon near the caecum was ruptured of about 20 cm wide, with the blood clot was accumulating into the caecum (Figure 1). There are numerous white granular foci (barely visible up to 0.5 cm in diameter) scattered along the mesenterium, lgl. mesenterica, and white plaques observed in the wall of the stomach (Figure 2, 3).

Histopathological findings :

There are erosion, hydrophic degeneration of epithelial mucosa, multiple thrombus and vasculitis in lamina propria of the tongue. The lingual salivary glands are necrotic and mineralized. There is severe hydropic degeneration and necrosis of the parotid glands. The esophagus showed hiperkeratinisation and erosion of keratin layer, with foci of *Candida* spp. and non spesific bacterial infection.

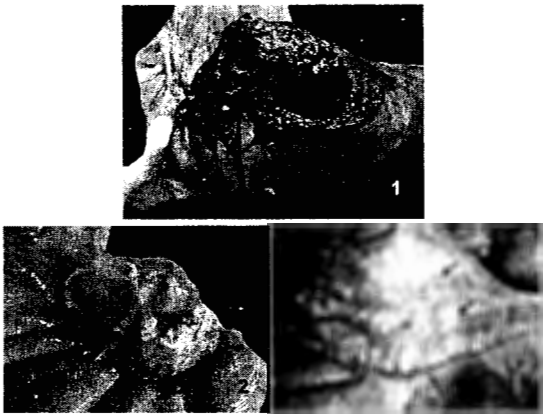

Figure 1. Ruptured colon of about 20 cm long.
Figure 2, 3. Numerous white granular foci of mineralization scattered along lgl. mesenterica and mesenterium.

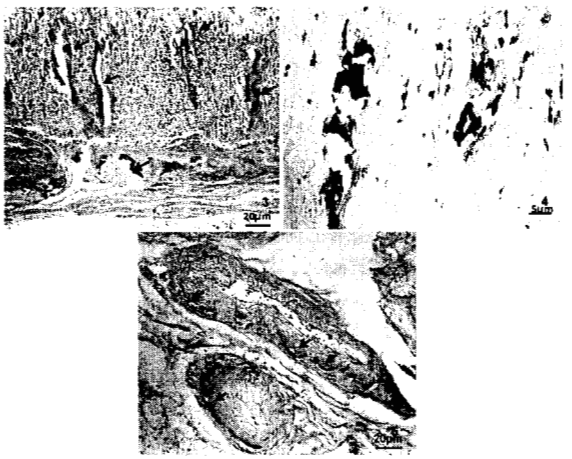

Figure 4. Stomach, severe mineralization and fibrosis observed within mucosa, submucosa and muscularis mucosa. HE staining, bar=20 μm.
Figure 5. Stomach, severe mineralization of gastric pits and glands. Von Kossa. Bar = 5 μm.
Figure 6. Note the characteristic purplish color of calcium deposits in the blood vessel walls, bar = 20 μm.

Multifocal moderate to severe mineralization were observed within the stomach mucosa, submucosa and muscularis mucosa (Figure 4). The gastric pit are dilatated and mineralized with gastric glands degeneration, necrosis and also mineralized. Note the calcium deposits visualized as black staining upon treatment with silver nitrate (Figure 5). Edema, inflammation, hemorrhage and fibrosis are observed in the gastric mucosa. The gastric muscular layers have severe hyalin degeneration. The underlying vasculopathy in the stomach vessels were degeneration, necrosis and mineralization (Figure 6). The degenerated vessel wall at the site of mineralization exhibited a thickened basement membrane with fibrosis. All of gastric lesions diagnosed as uremic gastropathy.

Discussion

Torgamba had a long illness of chronic kidney disease. Based on gross and histopathological findings, he was diagnosed as polycystic kidneys disease (PKD) and categorized as end stage kidney. This disease is found in several domestic and laboratory animal species as well as in humans, but little is known about polycystic kidneys disease in wildlife and zoo animals (1). The severity of kidneys lesions considered to produce renal failure with resulting uremia.

The extra-renal lesion of the uremic end stage kidneys showed systemic soft tissue mineralization or metastatic calcification. Metastatic calcification is defined as the deposition of calcium salts in previously normal tissue due to abnormal biochemistry with disturbances in the calcium or phosphorus metabolism (2). In Torgamba, the mineralization were found on lingual salivary glands, parotid glands, mesenterical lymphnodes and scattered along the wall of the mesenterium, stomach and colon. The white plaque observed macroscopically on the wall of the stomach is the result of mineralization. Mineralization of lingual salivary glands and parotid glands due to high concentration of ammonia considered to hyposalivation. All this lesion were the underlying mastication problem of Torgamba.

Uremic gastropathy is characterized microscopically by mineralization of the mucosa and submucosal blood vessel, edema, vasculopathy and glandular atrophy. The uremic syndrome leads to the increase of parietal cell mass with the result hyperacidity and considered as local acidosis (3). Mineralization of gastric mucosa probably develops through the combined effects of ischemia, local acidosis and increased plasma concentrations of ammonia, calcium and phosphorus. Mineralization and glandular atrophy decreased secretory activity of the gastric glands. Digestive tract vasculopathy, glandular atrophy and muscular degeneration with resultant of gastric dysfunction, clinically explained the indigestive condition of Torgamba.

The irritating ammonia on the vascular system was able to cause thrombosis, vasculitis and loss of elasticity (4). As an outcome of capillary injury there will be fragility of gastric and colon wall. Constipation is a relatively common complication in chronic kidney disease (5). Vasculopathy of the colonic blood vessels near to the rupture region were also present in addition to the subserosal hemorrhages. All the above factors were considered as the causative rupture of the colonic wall.

Chronic kidney disease have an increased risk of developing infections due to their decreased immune system response. When the primary barrier and the humoral defense fail, pathogens as from normal flora can colonize and initiate local disease. *Candida* spp. infection was suggested as secondary to the immunosuppressive condition.

Conclusion

Chronic kidney pathology such as polycystic kidneys disease (PKD) in this wild captive sumatran rhinoceros is a very rare cases, if not the first to be exposed in the paper. The PKD and its metabolic consequences were considered as the underlying factors to the hemorrhagic rupture on the colonic wall.

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