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Hemato-biochemical and pathological alterations of chronic nephritis in a dog

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Abstract

The present study was carried out to investigate the haemato-biochemical, gross and histopathological changes of chronic nephritis in a dog. Decreased value of Hb, PCV, TEC and platelet counts were observed in haematological evaluation. Increase value of BUN and creatinine was found in biochemical estimation. On necropsy, the kidneys were reduced in size and after removal of the capsule cortex showed an uneven and pitted appearance with multifocal petechial haemorrhages. Histopathological examination revealed different stages of tubular degeneration and necrosis, widening of the Bowman's space, decrease glomerular cellularity, presence of infiltrating cells predominantly lymphocytes and plasma cells. Other histopathological findings include focal area of fibrosis, dystrophic calcification, vascular congestion and focal haemorrhages in the interstitial spaces.

Keywords: hemato-biochemical, histopathology, chronic nephritis, dog

Introduction

The kidneys are paired organs that function in excretion, metabolism, secretion, and hormonal or electrolyte regulation (including the hormones rennin and erythropoietin), and are susceptible to diseases of the glomeruli, tubules, interstitium, and vasculature [1]. Chronic kidney disease (CKD) is defined by presence of functional or structural changes in 1 or both kidneys for more than 3 months [2]. It is the most common kidney disease in small animals. Chronic kidney disease affects dogs of all ages, but more commonly older dogs. Increase level of blood urea nitrogen (BUN) and creatinine is observed in chronic renal diseases of canines [3]. Haematological parameters such as haemoglobin (Hb), packed cell volume (PCV), total erythrocyte count (TEC) and platelet count (THR) are significantly decreases in renal failure [3]. In chronic renal diseases reduction of the kidneys size, tough consistency, and nodular capsular surface with pale cortex is common gross findings. Histopathological studies of such kidneys shows, marked loss of functional nephrons, focal to diffuse interstitial fibrosis, tubular dilatation with focal mineralisation of tubular epithelium and basement membranes [2, 4, 5]. Apart from these, renal glomerular changes also observed in chronic nephritis which includes decrease size of glomeruli with segmental fibrotic areas and dilated Bowman's spaces [4, 5]. The present study reports hemato-biochemical and pathological alterations of chronic nephritis in dog.

Materials and Methods

For haematological study, 3 ml of blood was aseptically collected in EDTA vacutainer. Another 3 ml of blood was collected in a clot activator for collection of serum. Hematological parameters such as Hb, PCV, TEC, TLC and platelet counts were estimated with the help of automated haematological cell counter (Melet Schloesing Lab., MS4e model). Serum samples were utilized for the estimation of biochemical parameters like BUN and creatinine by a semi automatic biochemical analyser using kits. Kidney tissues were collected at the time of necropsy in 10 percent formal saline solution for histopathological study. After proper fixing tissue samples were processed, sectioned and stained with routine H & E stain.

Results and Discussion

Hemato-biochemical study

The haemoglobin value of the animal with chronic nephritis was recorded lower than the normal standard range (12-17.8 g/dl). The recorded value of haemoglobin was 2.54 g/dl. The PCV was recorded as 16 percent which was found to be considerably lower than the normal

range (40-55%). The TEC value was $2.54 \times 10^6/\mu\text{l}$, which is much lower than the normal standard range ($6.4-8 \times 10^6/\mu\text{l}$). The platelets count also decreased than the standard range ($120-600 \times 10^3/\mu\text{l}$).

The estimated value of platelet count was $32 \times 10^3/\mu\text{l}$. The TLC value was found to be within the lower side of the normal range ($6-20 \times 10^3/\mu\text{l}$) which was $6.80 \times 10^3/\mu\text{l}$. These findings suggest that affected dogs were suffering from anaemia which is possibly due to deficiency of erythropoietin production by the diseased kidneys as reported by Silverberg *et al.* (2002) [6] or bone marrow fibrosis as suggested by Cowgill and Francey (2004) [7] also, loss of blood in gastrointestinal tract as melena and haematemesis as mentioned by Castaldi *et al.* (1966) [8] also, anaemia due to chronic disease as described by Giger (2005) [9], also, shortened survival period and haemolysis of red blood cells due to uremia reported by Ly *et al.* (2004) [10]. Similar findings are reported by Polzin *et al.* (2005) and Pradhan and Roy (2012) [11, 12].

The BUN and creatinine level recorded was 112.6mg/dl and 4.1mg/dl respectively which were much higher than the normal standard range. The BUN level is elevated when approximately 70 percent of the nephrons are non-functional [13]. Suggestive reason of increased creatinine level in chronic renal disease might be due to marked reduction in glomerular filtration rate (GFR), diminished renal excretion, enhanced tubular absorption of urea and impaired ability of kidneys to excrete proteinaceous catabolites. Creatinine is a resultant of muscle metabolism and its elevated level in blood indicated kidney disease. Similarly findings of increased value of BUN and creatinine in dogs with chronic renal disease have been recorded by other workers (Pradhan and Roy, 2012 and Sumit *et al.* 2018) [3, 12].

Gross and histopathology

Grossly, the kidneys were reduced in size and having a granular appearance. The capsule was removed with difficulty and cortex showed uneven and pitted appearance with multifocal petechial haemorrhages (Fig.1). These observations correlates with the findings of previous workers [2]. The cut section of the kidney revealed finely granular, pale cortex which is not remarkably reduced in size and congestion at the cortico-medullary junction as well as medulla (Fig.2). Similar findings were observed by Slausok and Lewis in 1979 [14].

In histopathological examination, diffuse tubular necrosis was observed. The tubular epithelium undergoes different stages of degeneration. Desquamated epithelium and cellular debris are present at the centre of the tubules. The glomeruli shows decrease cellularity with increase bowman's space (Fig.3). Focal area of lymphocytes and plasma cell infiltration (Fig.4) particularly at the periglomerular and perivascular areas [2]. Earlier workers also reported similar findings [2]. Blood vessels showed marked congestion and focal area of haemorrhages observed in the interstitial spaces (Fig.5). There was thickening of the vascular wall with narrowing of the lumen.

Proliferation of fibrous connective tissue was noticed in some places. Accumulation of proteinaceous substances as a homogenous pinkish appearance was observed in the tubular lumen. Dystrophic calcification was found in some places (Fig.6). Similar findings were recorded by Chetan *et al.* 2019 [2].

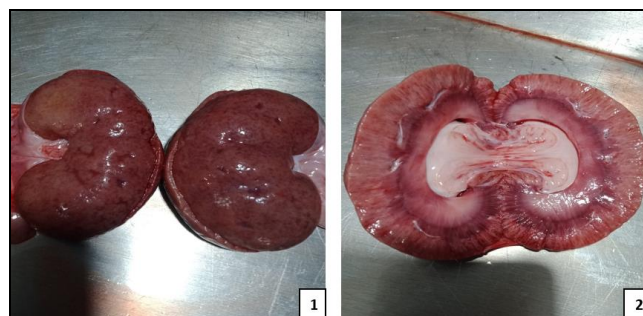


Fig 1-2: 1. Photographs showing (1) Uneven and pitted appearance of the cortex with multifocal petechial haemorrhages, (2) Finely granular cortex and congestion at the cortico-medullary junction and medulla

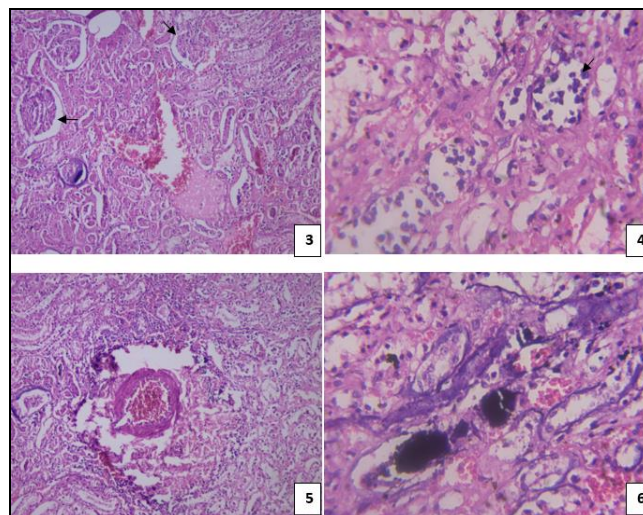


Fig 3-6: Photomicrographs showing (3) Decrease glomerular cellularity with increase bowman's space (arrows), H&Ex100; (4) Focal area of lymphocytes and plasma cell infiltration (arrow), H&Ex400 (5) Congestion and focal area of haemorrhages in the interstitial spaces, H&Ex400 and (6) Dystrophic calcification, H&Ex400

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