CASE REPORT

Juvenile Nephropathy in a Shih Tzu Dog - A Case Report

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Juvenile nephropathy (JN) in dogs refers to any non-inflammatory, degenerative, or developmental chronic kidney disease in young animals (approx. 2 years of age or less), characterized by progressive renal failure, which cannot be associated with primary renal inflammation (Maxie, 1993). This condition may be congenital (present at birth), inherited (genetically derived), familial (a trait present in a group of related dogs) or acquired with onset reported from few weeks to several years, but in often 4 to 18 months of age (Maxie, 1993). Clinical signs might not become apparent until later in life (Cianciolo *et al*., 2018) and will be presented as renal failure with azotaemia, inability to appropriately concentrate urine and possibly signs of uraemia. This case report communicates the findings of Juvenile Nephropathy in a Shihtzu dog.

Case History and Observations

A six months old male Shihtzu dog was presented to small animal medicine ward, Department of Veterinary Clinical Complex at the College of Veterinary Science, Proddatur (Andhra Pradesh, India) with signs of vomiting, inappetence, dullness and weight loss since one week. History revealed frequent episodes of vomiting since three months with continuous elevation of blood urea nitrogen and creatinine which was treated symptomatically by local veterinarian. Clinical examination revealed normal vital parameters, conjunctival mucous membranes with moderate dehydration (STT- 4 secs).

Haematological evaluation revealed mild anaemia (Hb 10.2 g/dL) while serum biochemical profile showed azotemia (BUN 236 mg/dL, creatinine 7.5 mg/dL), hyperphospatemia (8.1 mg/dL), hypoproteinemia (4.10 g/dL) and hypoalbuminemia (1.96 g/dL). Abdominal ultrasonography of both the kidneys revealed hyperechoic cortex with loss of architecture and indistinct corticomedullary junction (CMJ) (Fig. 1, 2). Renal flow Doppler of interlobular artery was done which recorded increased resistive index (LK: 0.86, RK: 0.76) and pulsatility index of 1.8 indicating increased renal vascular resistance (Fig. 3, 4). Echocardiography revealed hyperdynamic left ventricle with concentric left ventricular hypertrophy (LVPWs: 10.9 mm, LVPWd: 9.3 mm) and elevated Ejection Fraction and Fractional Shortening (Fig. 5). Taking into consideration the above findings and correlating with the age of onset, the case was diagnosed as Juvenile nephropathy.

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Treatment and Discussion

Treatment for juvenile nephropathy relies mainly on managing the signs of renal failure. Therapy was initiated with Inj. Ringers lactate @ 10 mL/kg b.wt. IV, BID, tab. Benazepril @ 0.25 mg/kg b.wt. PO, BID, tab. Sevelamer ½ tablet with food twice a day, Inj. Ondansetron @ 0.2 mg/kg IV, BID, Inj. Pantoprazole @ 1 mg/kg IV, OID and prebiotic and probiotic renal capsules at a total dose of three capsules daily orally, which were given as two capsules in the morning and one capsule in the evening. Apart from the above, the pet parent was advised to give renal diet in the recommended quantity. The pet showed pronounced improvement in clinical signs by fifth day of therapy with reduction in vomiting episodes and restoration of appetite. However, there was relapse of the vomiting episode after one month and the patient was succumbed despite the treatment. As the pet parent declined necropsy, a post-mortem examination could not be performed.

The Juvenile Nephropathies include a variety of morphological disease entities such as agenesis, hypoplasia, dysplasia, primary cystic diseases, glomerulopathies, tubulointerstitial nephropathies and tubular transport dysfunction (Peeters *et al.*, 2000). Clinical signs predominantly arise due to progressive renal failure, which include vomiting, lethargy and weight loss (Lavoué *et al*., 2010). In the present case, major haemato-biocemical alterations noticed, *viz.*, anaemia, azotemia, hyperphosphataemia, hypoproteinemia and hypoalbuminemia were comparable to those described

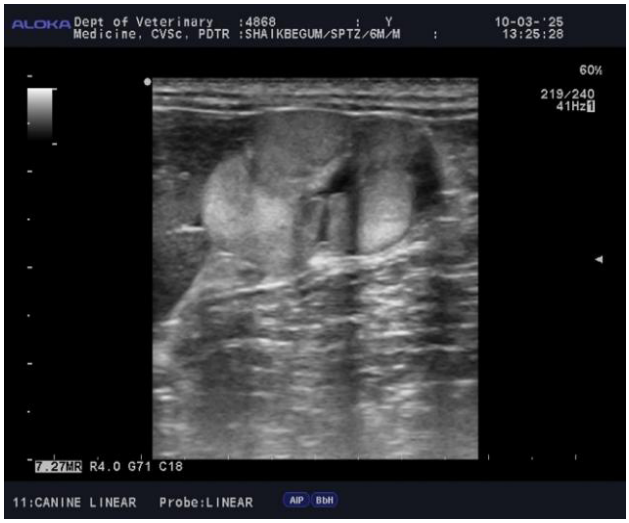

Fig.1: Right kidney with indistinct CMJ

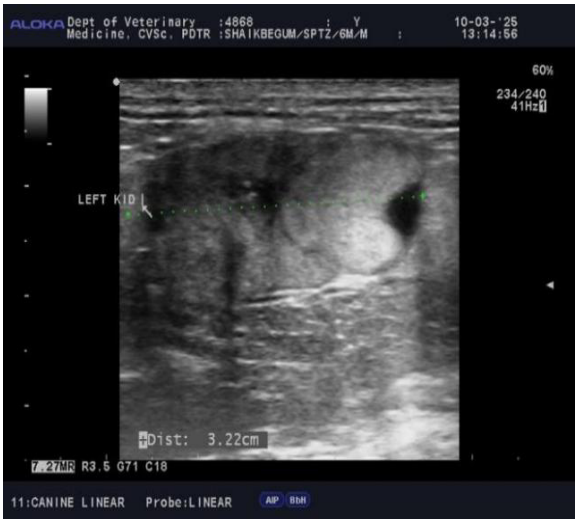

Fig. 2: Left kidney with indistinct CMJ

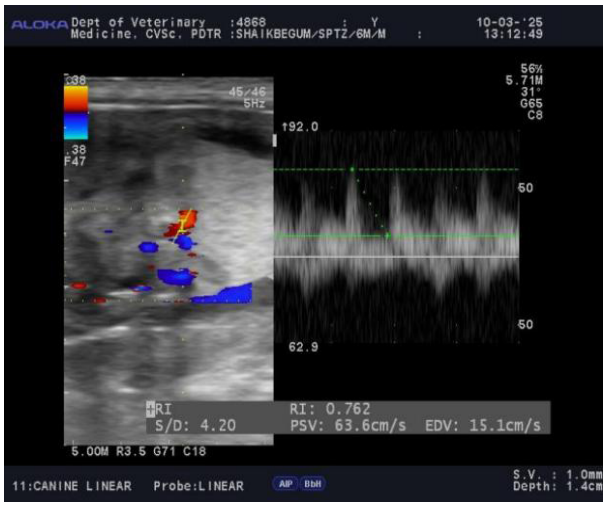

Fig. 3: Color flow Doppler of right kidney

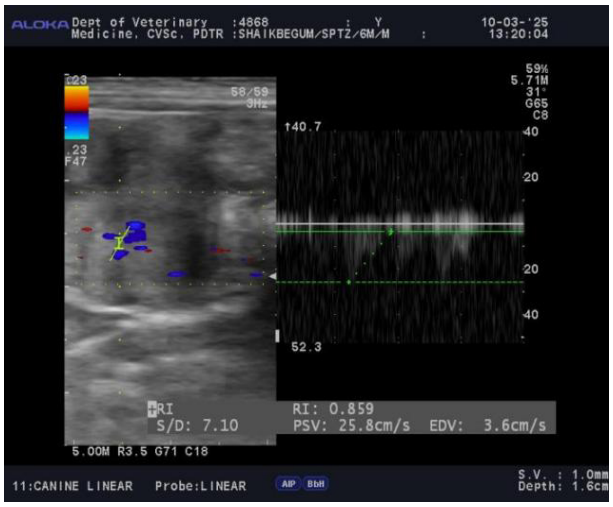

Fig. 4: Color flow Doppler of left kidney

earlier (McKay *et al*., 2004; Chandler *et al*., 2007; Lavoué *et al*., 2010; Basile *et al.,* 2011). The abdominal ultrasonographic findings were consistent with those previously reported in similar cases (Chandler *et al*., 2007; Lavoué *et al*., 2010). Histopathology of renal tissue obtained either from FNAC or biopsy plays a vital role in etiological diagnosis (Gleadhill, 1997). However, renal biopsy was not performed in the present case due to reluctance of the pet parents for the procedure. Upon diagnosis, therapy is focused on fluid therapy, ACE inhibitors, phosphate binders, anti-emetics, enteric dialysis and renal diet which is aimed in managing the signs associated with chronic kidney disease. In an isolated case like this, it is often difficult to identify whether it is acquired or inherited (McKay *et al*., 2004). Considering possible breed specific predispositions and genetic background, preliminary study should be encouraged for the timely diagnosis and to guide appropriate treatment strategies to improve the quality of life for pets.

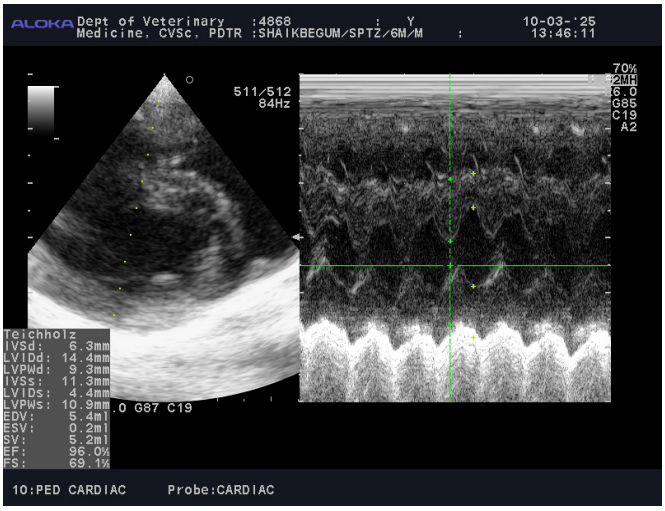

Fig. 5: Right parasternal short axis view: M-mode measurement of Left ventricle

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