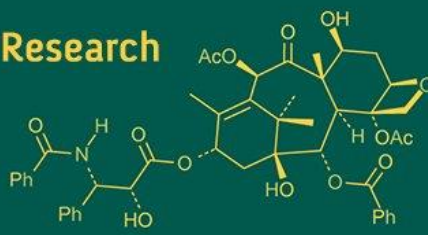


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Therapeutic management of hypertension induced epistaxis in chronic kidney disease stage-4 in canines: A case series

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Abstract

Introduction: Chronic Kidney Disease (CKD) involves a loss of functional renal tissue due to a prolonged (>2 months), usually progressive process.

Objectives: This study wanted to explore the role of antihypertensive therapy in the management of hypertension induced epistaxis in CKD-Stage 4.

Methods: Three different breed dogs were presented to clinics with the history of anorexia, lethargy and gastroenteritis from the past 5 days. On USG, cortico-medullary junction differentiation was lost, renal pelvis was dilated. On CBC, hemoglobin was low; platelets were within the normal range. On KFT, BUN and creatinine values were elevated. Serum electrolytes concentrations were within the normal reference range; however, phosphorus level was elevated. Supportive treatment with IV fluids, phosphate-binders, proton pump inhibitor, probiotics, herbal nephroprotective agents was started. After one week the owner revisited clinics with the complaint of epistaxis. Blood pressure was monitored and found to be on the higher side confirmed substage AP3. Antihypertensive therapy with Enalapril (Envas^R), Benazepril (Aceptor^R), Amlodipine (Amlkind^R) and Furosemide/Spironolactone (Lasilactone^R) was started.

Results: After antihypertensive drug therapy, the complaint of epistaxis got resolved.

Conclusions: Hypertension induced Epistaxis is therapeutically manageable with oral Enalapril, Amlodipine & Furosemide medications.

Keywords: Amlodipine, Enalapril, furosemide, phosphate-binders

Introduction

Chronic kidney disease (CKD) is characterized by an irreversible and progressive impairment in kidney function, resulting from the reduction in the number of functioning nephrons [1]. Paradoxically, the body's compensatory responses to nephron loss, such as glomerular hypertension and hyper filtration, contribute to the progression of CKD. In fact, these compensatory mechanisms may play a more significant role in the disease's advancement than the initial injury that triggered the condition [2]. Glomerular filtration rate (GFR) has traditionally been regarded as the gold standard for assessing kidney function [3]. However, directly measuring GFR is a complex and time-intensive process, making it impractical for routine clinical use [4]. As a result, indirect markers are often employed as substitutes to evaluate kidney function. These include plasma creatinine (CREA), serum symmetric dimethylarginine (SDMA), and Cystatin C (CysC) levels [5]. Chronic kidney disease (CKD) is typically categorized into several stages based on laboratory test results and clinical signs, as outlined in the Table1:

Stage 1: The kidneys are being damaged, but azotaemia and clinical signs have not yet developed. Unfortunately, renal disease is rarely detected at this early stage.

Stage 2: The disease has progressed, with glomerular filtration rate (GFR) falling to less than 25% of normal. Azotaemia is present, but clinical signs are not yet visible. However, this stage may be associated with impaired urine-concentrating ability and increased urine volume.

Stage 3: GFR has declined further, azotaemia is present, and clinical signs often become apparent.

Stage 4: The disease has progressed even more, resulting in severe azotaemia and the presence of clinical signs. Kidney disease is the most prevalent cause of systemic hypertension (SH) in both dogs and cats. It has been proposed that systemic hypertension may lead to epistaxis in small animals [6]. The causes of epistaxis in canines are characterized as local (e.g., nasal neoplasia, trauma, idiopathic rhinitis, or periapical abscess) or systemic (e.g., severe thrombocytopenia, thrombocytopathies, coagulopathy, hypertension, or vasculitis) [7]. Epistaxis is uncommon with platelet counts over 50,000/ μ L but systemic hypertension has been implicated as the cause of epistaxis [6]. Systemic hypertension leads to increased capillary fragility thereby resulting in epistaxis and it is mostly bilateral [7]. Treatment for chronic kidney disease and ensuing hypertension consists of fluid therapy with crystalloid solutions (ringer's lactate, normal saline), antihypertensive drugs (Enalapril, Benazepril, Amlodipine, Furosemide, Spironolactone), antacids like proton pump inhibitors (Pantoprazole) and H₂-blockers (Ranitidine), gastro-protectants (sucralfate), antiemetics (ondansetron, metoclopramide), phosphate binders (Sevelamer, aluminium hydroxide), probiotic powders, haematinics and erythropoietic hormonal preparations etc [2].

Place of Study

The study was conducted at VTCC-IVRI on client owned dogs breeds viz. The study was conducted for a period of 6 months starting from October 2023 to April 2024. During the study period, a total of 12 cases suffering from CKD were presented to the clinics.

Case Presentation

Case 1

An eight-year-old male pug was presented with a history of anorexia, haemorrhagic enteritis, recurrent vomiting, polyuria, emaciation, lethargy, and uraemic breath. On clinical examination, conjunctival mucous membrane was slightly pale, heart rate was recorded as 146 beats per min, respiration rate was normal and rectal temperature was recorded as 101.2°F.

Case 2

A twelve-year-old female Dalmatian was presented to the clinics with the history of anorexia, recurrent vomiting, emaciation, recumbency, bloody dysentery and lethargy. On clinical examination, temperature recorded was 99.6°F, conjunctival mucous membrane was pale, heart rate was recorded as 160 bpm.

Case 3

An eleven-year-old female Belgian Sheepdog was presented to the clinics with the history of anorexia, recurrent vomiting, lethargy, melena, hyperpigmentation in inguinal region and slightly enlarged abdomen. On clinical examination, rectal temperature recorded was 100.8°F, pink mucous membrane, heart rate of 110 bpm and normal respiration rate.

Laboratory Examination

Haematological examination was carried out using auto-hemoanalyser and serum biochemistry was performed using

standard methods (Table 1). The most consistent haematological findings included anaemia, elevated phosphorus levels, elevated creatinine and blood urea levels. However, in all the dogs the platelet counts were in the normal reference ranges. Dogs were negative for all the blood protozoan parasites on PCR. Ultrasonography was performed using curvilinear probe at 3.5-4.0 MHz frequency. The ultrasonographic findings included increased echogenicity of kidneys in comparison to spleen, loss of cortico-medullary junction differentiation, increase in cortical thickness and decrease in the size of kidneys (Figure 1, 2, 3).

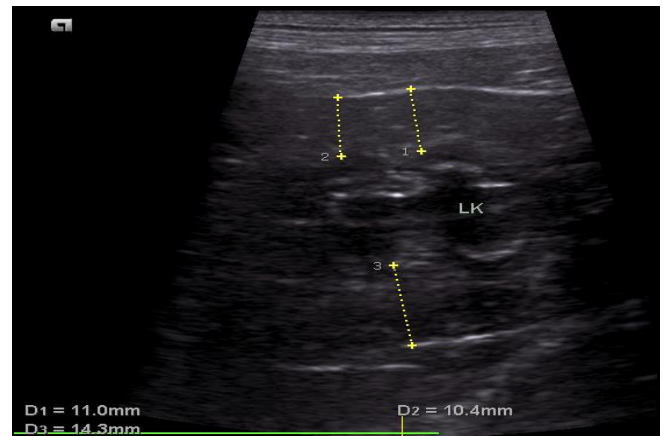


Fig 1: Increased echogenicity, increased cortical thickness and loss of cortico-medullary junction differentiation (Dalmation)

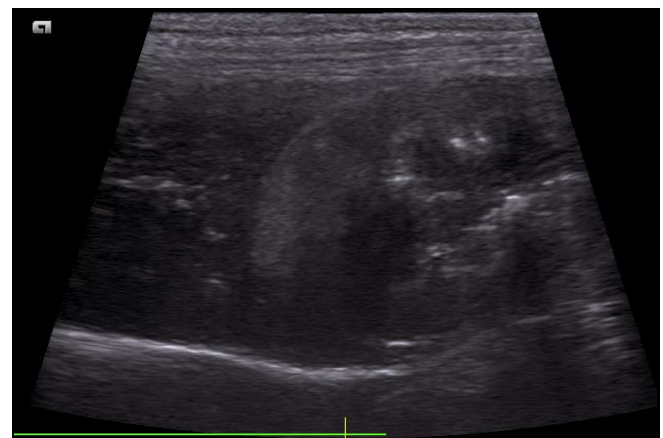


Fig 2: Hyperechoic kidney with loss of cortico-medullary junction differentiation and decrease in size of kidney (Pug)



Fig 3: Loss of cortico-medullary junction differentiation and decrease in size of kidney

Treatment (Initial)

Treatment was started with fluid therapy with intravenous isotonic crystalloids (ringer's lactate, normal saline) at the dose 20ml/kg BW BID, pantoprazole at the dose 0.75mg/kg BW IV OD, ondansetron at the dose 0.5mg/kg IV BID, ethamsylate at the dose 25mg/kg BW IM OD, sevelamer (Tab. Phoslore^R) at the dose 40mg/kg PO OD, aluminium hydroxide (Syrup. Gelusil^R) at the dose 30mg/kg BW PO BID, amoxicillin-sulbactam at the dose 12.5mg/kg BW IV BID, probiotic preparations viz. Sporolac^R and Enterogermina^R at the dose 1 teaspoon full PO OD, Sucralfate at the dose 500mg per dog PO BID, haematinic preparations aRBC-Pet^R at the dose 1ml/4kg BW PO BID, and herbal and homeopathic preparations Neeri-KFT^R, NL2^R at the dose 1-2 teaspoon full PO BID.

Follow up

After continuing the therapy for one week, dog owner's

noticed episodes of epistaxis from 4-5 days of initial therapy. Haematology examination was conducted again that showed normal platelet counts in all the three cases. Blood pressure was monitored with non-invasive blood pressure multi-parameter machine. Blood pressure recorded was on the higher side in all the three cases (Table 2). Antihypertensive therapy was started with enalapril (Tab. Envas^R) in one out of the three dogs (Pug) and benazepril (Tab. Aceptor^R) in two out of the three dogs, both at the dose of 0.25-0.5mg/kg BW PO BID, diuretics furosemide and spironolactone (Tab. Lasilactone^R) at the dose 2mg/kg BW PO BID and calcium channel blocker amlodipine (Tab. Amlkind^R) at the dose 0.2-0.3 mg/kg BW PO BID. Within 2 days after treatment, the complaint of epistaxis got resolved and dogs were re-examined for blood pressure and blood pressure recorded was within normal reference ranges (Table 2).

Table 1: IRIS Staging for CKD

Parameter	Category/Stage	Dog Criteria
Serum Creatinine (mg/dL)	Stage 1	< 1.4
	Stage 2	1.4-2.0
	Stage 3	2.1-5.0
	Stage 4	> 5.0
Proteinuria Substaging (UPC Ratio)	Non-Proteinuric (NP)	< 0.2
	Borderline Proteinuric (BP)	0.2-0.5
	Proteinuric (P)	> 0.5
Blood Pressure Substaging	AP Stage 0	Systolic <150 mmHg; Diastolic <95 mmHg
	AP Stage 1	Systolic 150-160 mmHg; Diastolic 95-99 mmHg
	AP Stage 2	Systolic 160-179 mmHg; Diastolic 100-119 mmHg
	AP Stage 3	Systolic >180 mmHg; Diastolic >120 mmHg

Table 2: Blood pressure values before and after antihypertensive therapy in three dogs with hypertension-associated epistaxis, showing normalization of blood pressure following treatment

Case No.	Breed	Initial Systolic (mmHg)	Initial Diastolic (mmHg)	Initial Mean (mmHg)	Post-Therapy Systolic (mmHg)	Post-Therapy Diastolic (mmHg)	Post-Therapy Mean (mmHg)
1	Pug	182	112	147	132	81	97
2	Dalmatian	176	100	117	149	76	104
3	Belgian Sheepdog	196	117	157	143	89	109

Table 3: Hematological and serum biochemical parameters of the three dogs with chronic kidney disease.

Parameters	Case-1	Case-2	Case-3
Hb (g/dl)	10	9.1	13.2
PCV (%)	30.1	27.3	38.4
TEC ($\times 10^{16}/\mu\text{L}$)	4.02	4	5.52
TLC ($\times 10^{13}/\mu\text{L}$)	18.2	10.4	14.9
Neutrophils (%)	80	75	70
Lymphocytes (%)	13	15	26
Monocytes (%)	07	10	03
Eosinophil (%)	00	00	01
Basophil (%)	00	00	00
Platelets ($\times 10^{13}/\mu\text{L}$)	368	551	210
Serum Creatinine (mg/dL)	12.28	14.66	5.63
Blood Urea (mg/dL)	237	180	240.6
Total Protein (g/dL)	5.6	5.47	6.1
Albumin (g/dL)	2.0	2.50	2.8
Sodium (mEq/L)	130.5	134.6	155.9
Potassium (mEq/L)	5.7	4.84	4.79
Chloride (mEq/L)	93.1	99.3	118.8
Phosphorus (mg/dL)	13.6	10.6	13.3
Serum Cholesterol (mg/dL)	220	257	268.1

Discussion

Chronic kidney disease is a progressive disease involving the loss of functional renal tissue over a prolonged period of time (>2 months). Dogs with serum creatinine level of >5 mg/dL, systolic blood pressure of >180mm Hg and diastolic blood pressure of >120mm Hg are grouped into stage-4 (AP substage-3) CKD. Epistaxis is rare with platelet counts over 50,000/ μL , but systemic hypertension has been implicated as the cause of epistaxis. In dogs, epistaxis can occur as a sequel to renal failure and is typically attributed to indirect mechanisms like hypertension (Bisset *et al.*, 2007; Mylonakis *et al.*, 2008; Cianciolo & Mohr, 2016), and less frequently, increased capillary fragility, disseminated intravascular coagulation, and other coagulation abnormalities (Cianciolo and Mohr, 2016). Systemic hypertension and increased capillary fragility result in epistaxis. The epistaxis seen with all the three cases was bilateral which in turn supports the cause to be of systemic origin. All the three cases were negative for haemo-protozoans and bacterial diseases on PCR. Despite having normal platelet counts, the presence of epistaxis gave a hint about hypertension might be the cause of epistaxis. Blood pressure was measured using non-invasive blood pressure

multi-parameter machine. Three readings were taken in all the cases and average calculated in each case was taken as the final reading. Antihypertensive therapy led to the resolution of epistaxis in all the cases on day 2 of therapy in pug and dalmatian, on day 3 in case of Belgian shepherd.

Conclusion

Epistaxis induced by hypertension associated with CKD is manageable with antihypertensive drugs in dogs. Chronic kidney disease can be a potential cause of hypertension in small animals.

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