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# HEMODIALYSIS-THERAPEUTIC MANAGEMENT IN A DOG WITH VANCOMYCIN-INDUCED ACUTE KIDNEY INJURY: CASE REPORT

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**Abstract** A 3 year old, 23 kg, neutered male Alaskan husky, diagnosed at another clinic with osteomyelitis, presented at the clinic with the following symptoms: lethargy, appetite loss, hypersalivation, moderate weight loss, dehydration (10% with considerable loss of skin turgor) and hypertensive. After long-term Vancomycin and NSAID's administration, the blood work revealed mild anemia and significant increase in BUN 144 (RR: 7-25 mg/dL), CREA 15.3 (RR: 0.4-1.2 mg/dL), PHOS 13.7 (RR: 2.9-6.6 mg/dL), CA 19.8 (RR: 8.6-11.8) mg/dL) and K 6.4 (RR: 3.4-5.6 mmol/L). Urinalysis was performed with UPC 0.2-0.5 (borderline proteinuric), pH 5.0, specific gravity 1.030 and microalbumin >25 mg/L. Vancomycin is an effective highly hydrophilic glycopeptide antibiotic. Vancomycin is the gold standard for treating methicillin-resistant Staphylococcus aureus, but its long-term clinical use is associated with adverse effects of oxidative stress and kidney injury. Hemodialysis was decided as an extracorporeal replacement therapy for sustaining renal function. Hemodialysis was performed for two times in a period of 10 days. With a protocol of intensive care, fluid therapy and hemodialysis, the values were reduced in BUN from 144 mg/dL to 25 mg/dL, CRE from 15.3 mg/dL to 5.9 mg/dL and PHOS from 13.7 mg/dL to 5.5 mg/dL.
**Keywords:** hemodialysis, dog, vancomycin, kidney, BUN.

## • Introduction

Antibiotics are one of the most common causes of drug-induced nephrotoxicity. Mechanisms of antibiotic-induced nephrotoxicity include glomerular injury, tubular injury or dysfunction, distal tubular obstruction from casts, and acute interstitial nephritis (AIN) mediated by a type IV (delayed-type) hypersensitivity response (2). Vancomycin is an effective highly hydrophilic glycopeptide antibiotic. It is also considered to be the gold standard for treating methicillin-resistant Staphylococcus aureus, but its long-term clinical use is associated with adverse effects of oxidative stress and kidney injury. Hemodialysis is a therapeutic procedure that uses the extracorporeal circulation of a patient's blood in order to improve azotemia, fluid overload, electrolyte, and acid-base abnormalities due to uremic syndrome (15, 16).

## • Material and method

A 3 year old, 23 kg body weight, neutered male Alaskan Husky, was referred for a nephrology consult after being diagnosed in another practice with osteomyelitis (antibiogram showed the presence of Staphylococcus spp.) after three orthopedic interventions in two months due to tibial fracture and acute kidney injury (AKI). The main goal of the therapy was to stabilize the patient 24 hours prior to hemodialysis. Hemodialysis was decided in order to improve and support renal function. A central venous catheter (Haemocat® Signo 12Fr 20 cm temporary double-lumen catheter) was placed under light sedation with alfaxalone (dosage: 3.5 mg/kg intramuscularly) and butorphanol (dosage: 0.2 mg/kg intravenously). The urea reduction ratio was calculated at 60% for the first session and the duration of therapy was 6 hours.

## • Results and discussions

At presentation (29th of February 2024), blood biochemistry showed severely increased CREA 15.3 (RR: 0.4-1.2 mg/dl), BUN 144 (RR: 7-21 mg/dl), PHOS 13.7 (RR: 2.9-6.6 mg/dl) and mild hyperkalemia K+ 6.4 (RR: 3.4-5.6 mmol/l). Moderate anemia was revealed on the complete blood cell count (CBC) with RBC 5.46 (RR: 5.5-8.5 10^12/l), HGB 11 (RR: 12-18 g/dl), HCT 35.89%, and MCHC 30.6 (RR: 31-29 g/dl). Urinalysis showed UPC ratio of 0.2-0.5 (borderline proteinuric), microalbumin >25 mg/l, and creatinine >56.4 mmol/l. Based on IRIS AKI Grading criteria, the patient is diagnosed with grade V acute kidney injury, border proteinuric, and hypertensive. Abdominal ultrasound revealed slightly irregular enlarged kidneys, loss of corticomedullary junction with increased renal cortex echogenicity (Fig. 1, Fig. 2).

Prior to intermittent hemodialysis (IHD), the patient was submitted for 24 hours of intravenous fluid therapy, electrolyte rebalancing, and partial parenteral nutrition based on levo-amino acids. After 24 hours of intravenous therapy, the blood biochemistry showed: BUN 139 (RR: 7-21 mg/dl), CREA 15.2 (RR: 0.4-1.2 mg/dl), PHOS 10.6 (RR: 2.9-6.6 mg/dl) and CA 10.2 (RR: 8.6-11.8 mg/dl) with CA*P of 108.12 and CA/P ratio a of 0.96. CBC showed decreased RBC 5.23 (RR: 5.5-8.5 10^12/l), HGB 10.6 (RR: 12-18 g/dl), HCT 34.33%. IHD was decided as an extracorporeal renal replacement therapy on the 1st of March. On 2nd of March 2024, the blood tests were improved with a great decrease as follows: BUN to 45 (RR: 7-21 mg/dl), CREA to 8.0 (RR: 0.4-1.2 mg/dl), PHOS to 6.0 (RR: 2.9-6.6 mg/dl) and CA to 8.9 (RR: 8.6-11.8 mg/dl), with CA*P of 53.4 and CA/P ratio of 1.48. It was decided to pause hemodialysis for 24 hours and give the patient intensive and specific intravenous therapy. On the 3rd of March 2024, blood work showed: BUN 54 (RR: 7-21 mg/dl), CREA 10 (RR: 0.4-1.2 mg/dl), PHOS 6.9 (RR: 2.9-6.6 mg/dl) and CA 9.5 (RR: 8.6-11.8 mg/dl), with CA*P of 65.55 and CA/P ratio of 1.37 and hemodialysis was decided. On the 4th of March 2024, blood work showed: BUN 21 (RR: 7-21 mg/dl), CREA 6.4 (RR: 0.4-1.2 mg/dl), PHOS 5.2 (RR: 2.9-6.6 mg/dl) and CA 8.9 (RR: 8.6-11.8 mg/dl), with CA*P of 46.28 and CA/P ratio of 1.71.

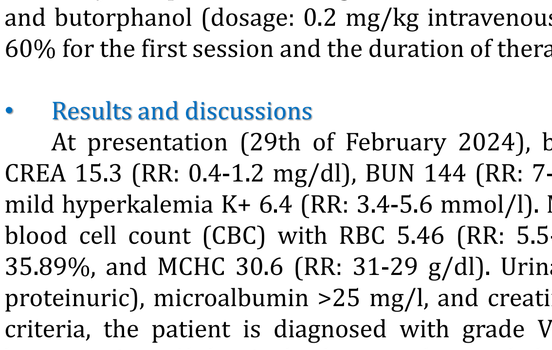


**Fig. 1. Ultrasound aspect of the left kidney (original)**

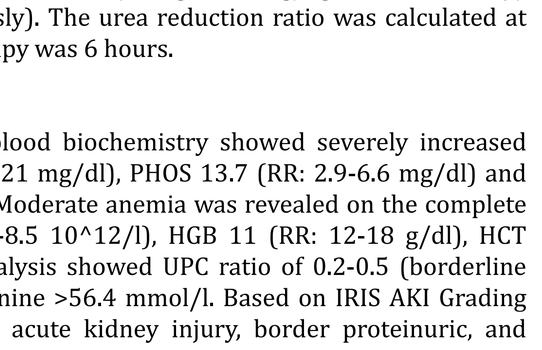


**Fig. 2. Ultrasound aspect of the right kidney (original)**

The patient continued with the fluid therapy and partial parenteral nutrition among the adjuvant therapy for supporting kidney functions until he was discharged. On the 9th of March 2024, when the patient was discharged, the blood biochemistry was: BUN 25 (RR: 7-21 mg/dl), CREA 5.9 (RR: 0.4-1.2 mg/dl), PHOS from 5.5 (RR: 2.9-6.6 mg/dl) and CA from 10.1 (RR: 8.6-11.8 mg/dl) with CA*P of 55.5 and CA/P ratio of 1.83.

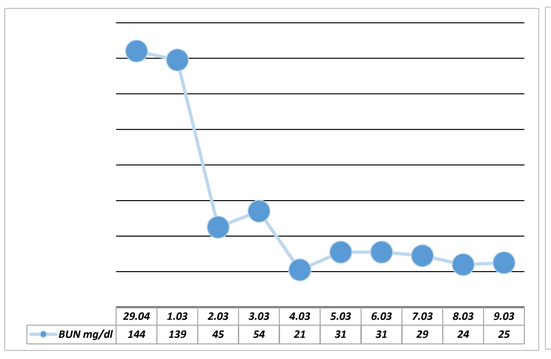


**Fig. 3. Evolution of BUN during hospitalization**

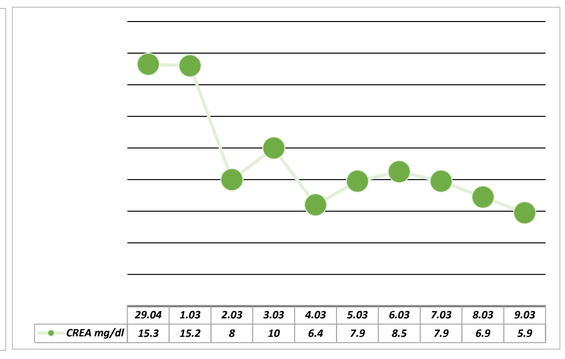


**Fig. 4. Evolution of CREA during hospitalization**

44 days after the patient was discharged with all the recommendations for sustained complex therapy at home, it was observed a great decrease of CREA to 1.12 (RR: 0.4-1.2 mg/dl), BUN to 19.9 (RR: 7-21 mg/dl), PHOS to 4.7 (RR: 2.9-6.6 mg/dl) and CA to 9.7 (RR: 8.6-11.8 mg/dl) with CA*P of 45.59 and CA/P ratio of 2.06.
As demonstrated below, from the first day of admission (29th of February), until the last clinical recheck on 21st of April 2024, the CA*P and CA/P ratio were improved (Fig. 7, Fig. 8).

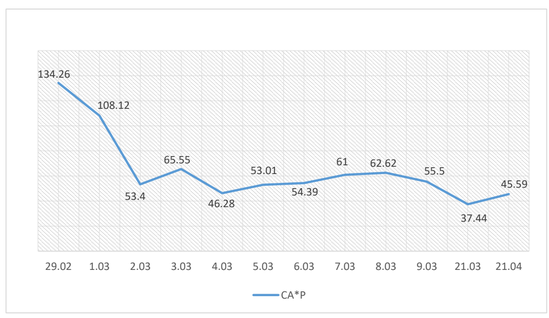


**Fig. 5. Evolution of CA*P**

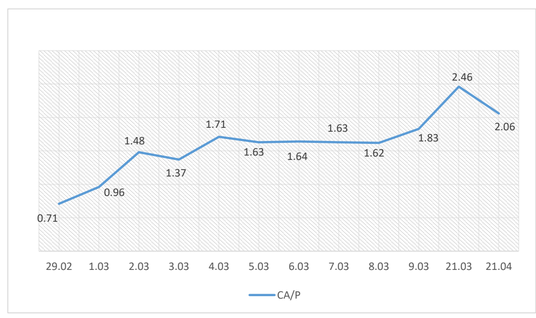


**Fig. 6. Evolution of CA/P ratio**

## • Conclusions

In critically ill patients diagnosed with Vancomycin-induced renal toxicity, intensive care therapy should be initiated as soon as possible and blood urea nitrogen and phosphorus levels should be decreased quickly. Vancomycin is the gold standard for treating methicillin-resistant Staphylococcus aureus, but its long-term clinical use is associated with adverse effects of oxidative stress and kidney injury.

The decision to pursue hemodialysis in patients with AKI should be made as quickly as possible in order to improve the likelihood of a successful outcome. IHD is a useful and suitable modality to improve outcomes in dogs with Vancomycin-induced kidney injury (VIKI) complicated with acute tubular necrosis. If it is used properly, hemodialysis is a life-saving procedure.

Elevated levels of creatinine and blood urea nitrogen, hyperphosphatemia, hypercalcemia, or metabolic acidosis can be solved using hemodialysis and also intensive adjuvant treatment.

For this patient, a key treatment for VIKI was represented by intensive care, fluid therapy, and hemodialysis which stands for a good prognosis and maintaining a positive evolution for this patient.

In conclusion, based on the data presented, the reduction of elevated blood urea nitrogen, creatinine, phosphorus, and calcium levels, along with the improvement of CA*P and CA/P ratios in patients with Vancomycin-induced kidney injury, required 54 days of adjuvant therapy and two hemodialysis sessions starting from the first day of hospitalization.

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