

Salbutamol toxicity in a Golden Retriever dog- A case report

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Abstract

Salbutamol is a beta2 adrenergic agonist commonly used in human and veterinary practice. Toxicity due to salbutamol is uncommon and if happened its therapeutic management is important to prevent lethal consequences. Three year old Golden retriever was presented to the hospital with accidental consumption of salbutamol tablets. On clinical examination dog was dull, tachycardic and having tachypnoea. Serum biochemical examination revealed severe hypokalemia and sinus tachycardia on electrocardiography. Fluid therapy was initiated along with intravenous potassium and oral beta blocker propranolol. Significant clinical recovery was noticed by 24 hours of therapy.

Keywords: Salbutamol, Toxicity, Golden retriever

Salbutamol, a selective beta-2 adrenergic receptor agonist is a bronchodilator commonly used for treating asthma, bronchitis, COPD and related respiratory problems. Toxicity due to salbutamol is rarely reported in veterinary practice. The present article reports the clinical signs, laboratory findings and treatment of salbutamol toxicity in a golden retriever.

Case history and observations

Three year old Golden retriever was presented to the hospital with the history of consuming few salbutamol

tablets (approx.15 no's). Upon clinical examination, the dog was found to be dull with tachycardia (228 bpm) and tachypnoea (36/min) (Fig.1). Other clinical parameters were found to be within the normal range. Haematology revealed no significant changes, while serum biochemistry revealed potassium (2.1 mmol/L), sodium (134mmol/L) and chloride (93 mmol/L) levels. Sinus tachycardia was noticed on electrocardiographic examination.



Fig.1 Dog affected with salbutamol toxicity being monitored using vital signs monitor



Fig.2 Heart rate 117 bpm recorded after 12 hrs of therapy

Treatment and Discussion

Therapy was initiated with Ringers lactate (@ 10ml/kg IV, slow) and propranolol (@ 0.2 mg/kg orally BID). Continuous rate infusion of potassium was

administered at a dose rate of 0.5 mEq/kg/hour diluted in normal saline. Continuous monitoring was done to check cardiac electrical activity using vital signs monitor. At the time of initiation of therapy the heart rate was 228 and

gradual decline of heart rate was noticed which reached to 117 bpm by 12 hours of therapy (Fig.2). Electrolyte levels reached normal range (serum potassium improved to 4.2 mmol/L, serum sodium 146mmol/L and serum chloride 106mmol/L). The general activity of the pet was improved, all vitals were stable within 24 hours and was discharged from the hospital. Salbutamol is a β_2 agonist that triggers the movement of potassium into cells by stimulating endogenous insulin and also by activation of membrane-bound Na/K-ATPase pumps outside the cell membrane, without requiring insulin (Ogrodny *et al.*, 2022). Reduced potassium in the blood suppresses potassium conductance which results in reduction of conduction reserves. Hypokalemia also results in increased intracellular Na^+ and Ca^{2+} through inhibition of $\text{Na}^+ - \text{K}^+$ ATPase activity. This positive feedback promotes early after depolarisation arrhythmias (EAD), delayed after depolarisation arrhythmias (DAD) and automaticity (Weiss *et al.*, 2017). Dogs affected with salbutamol toxicity commonly exhibit tachycardia, tachypnea, depression and vomiting as reported by Crouchley and Bates (2022). Oral thermal injury due to salbutamol inhaler was also reported in dog which is a rare complication (Mackenzie *et al.*, 2012). In cases

affected with salbutamol toxicity, timely correction of the electrolyte abnormality is crucial as delayed cases with tachycardia and arrhythmias can lead to untoward outcome. Propranolol, a non-selective beta antagonist was orally administered to counteract the beta agonistic action of salbutamol and reducing the tachycardia.

References

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