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Successful treatment of hypermagnesemia with atropine sulfate in sheep

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ABSTRACT

A hypermagnesemic adult sheep during an experimental study showed gastraointestinal and respiratory changes along with cardiovascular alterations included of obvious bradycardia and heart block. In order to save the animal from fatal bradycardia, atropine sulfate was administrated instatntly at one dose 0.3 mg/kg. The animal was survived and rhythm returned to sinus *rhythm from marked bradycardia and sinus arrest aft*er administration of atropine. *The present report describes signs of severe* hypermagnesemia *due to an* overdose of *magnesium sulfate during an experimental study and its successful therapeutic outcome.*

Key words: hypermagnesemia, atropine sulfate, sheep.

INTRODUCTION

Hypermagnesemia is an uncommon condition in large animals but may be seen with overdose administration of Epsom salts (MgSo4), either orally as a drench by means of nasogastric intubation or as an enema for the treatment of digestive disorders. Hypermagnesemia can depress the conduction system of the heart. Symptoms and signs of hypermagnesemia are usually predictable. Bradycardia and hypotension are the earliest manifestations [1].

In the absence of reversible causes, atropine remains the first-line drug for acute symptomatic bradycardia. One randomized clinical trial in adults and additional lower-level studies. IV atropine improved heart rate and signs and symptoms associated with bradycardia. An initial dose of 0.5 mg, repeated as needed to a total of 1.5 mg, was effective in both in-hospital and out-of-hospital treatment of symptomatic bradycardia [2-3].

Case History

During of a hypermagnesemic experimental study dult female native Iranian sheep aged 1 yrs showed symptoms of depression, rumen atony, urine retention, muscular tremors and tachypnea. The affected sheep fell into lateral recumbency with mydriasis. Sheep received 600 mg/kg bw magnesium sulfate 40 % (Nasr Pharmaceutical Company) IV in 3 divided doses. Five minutes after the last, sheep showed marked symptoms of poisoning. The clinical signs were characterized by neurological dysfunction including of varying degrees of depression and flaccid paralysis. Eelectrocardiogram (ECG) showed life-threatening bradycardia, atrial tachyarrhythmia and sinus arrest at around 37 beats per minutes. Serum biochemical analysis revealed hypermagnesemia (6.1 mg/dl, reference range: 2.2 to 2.8 mg/dl) at this stage.

Atropine sulfate 0.3 mg/kg was administered intravenously immediately for treatment of bradycardia secondary to increased vagal activity. Administration of atropine 0.3 mg/kg IV increased the heart rate to 98 beats per minute.

After the treatment, cardiac rhythm returned to sinus rhythm from bradycardia and sinus arrest and signs improved after administration of atropine.

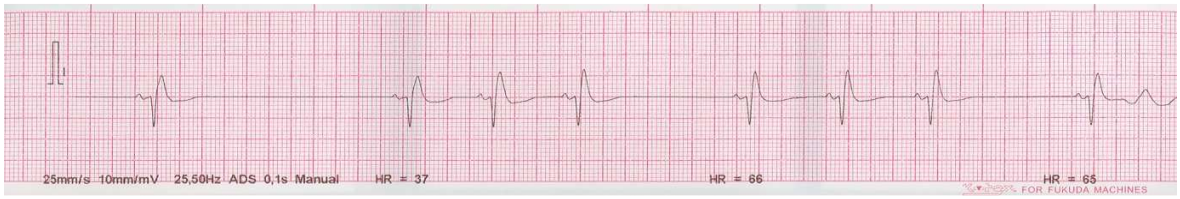

Fig 1- Lead I electrocardiogram showing bradycardia and sinus arrest in sheep. The heart rate is 37 bpm, paper speed 25mm/s, 1 cm=1 mv

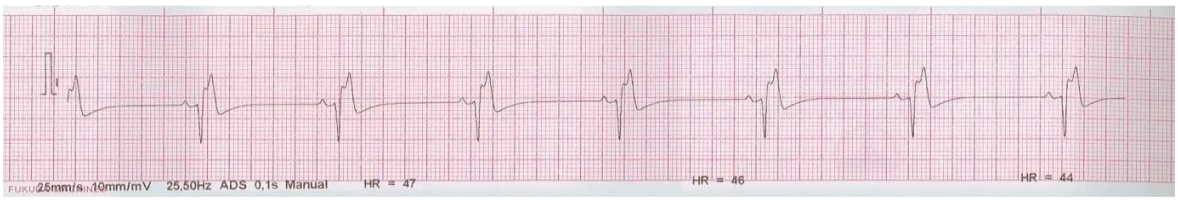

Fig 2- Lead I electrocardiogram showing bradycardia in sheep. The heart rate is 44 bpm, paper speed 25mm/s, 1 cm=1 mv

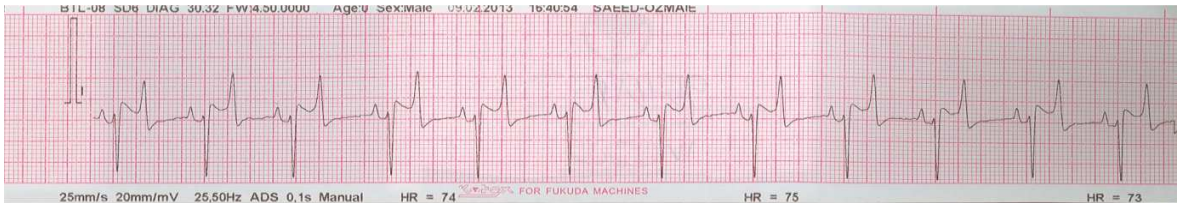

Fig 3- Lead I electrocardiogram showing normal sinus rhythm in sheep after the administration of atropin. The heart rate is 75 bpm, paper speed 25mm/s, 1 cm=1 mv

RESULTS AND DISCUSSION

Symptoms observed in the present case include depression, rumen atony, urine retention, muscular tremors, tachypnea, fell into lateral recumbency and mydriasis due to Magnesium toxicosis has also been reported earlier by other researchers [4]. The affected sheep recovered from recumbancy and mydriasis. Mild hypermagnesemia has been observed in patients receiving lithium therapy, or those with underlying hyperparathyroidism, hypothyroidism, diabetic ketoacidosis, viral hepatitis, in certain neoplasms with skeletal involvement, in pituitary dwarfism and the milk-alkali syndrome, or with adrenocortical insufficiency. The most common cause is iatrogenically encouraged excessive intake of magnesium containing laxatives, cathartics, or supplements, particularly in the presence of a perforated viscus, constipation, small-bowel hypomotility disorder, ureteral irrigation, parenteral replacement magnesium, excessive magnesium in dialysate solutions, or concomitant use of anticholinergic and narcotic agents and multiple doses of magnesium-containing cathartic therapy given in conjunction with activated charcoal [5].

Combined hypermagnesemia and hypercalcemia has been reported in a bitch associated by an iatrogenic parenteral overdose. The clinical picture in this case resembled that of hypermagnesaemia in human beings. In human patients with iatrogenic magnesium overdose, the major life-threatening clinical manifestations are delays in cardiac conduction, asystole, apnoea and coma [6]. Hypermagnesemia occured in cats with renal failure that were receiving IV fluid therapy [7]. In two cases, hypermagnesemia has been reported following administration of excessive oral doses of magnesium sulfate as a cathartic for treatment of large intestine impactions [8]. Hypermagnesemia can depress the conduction system of the heart. Symptoms and signs of hypermagnesemia are usually predictable. Bradycardia and hypotension are the earliest manifestations. Excess magnesium is known to have direct and indirect cardiovascular effects. Magnesium has been described as “nature's physiologic calcium blocker, and cardiovascular effects seen in hypermagnesemia may be caused by disruption of calcium action. Electrocardiographic observations in humans and animals have shown an increase in the P-R interval at concentrations of 6 to 12 mg/dL (2.5–5

mmol/L), which may progress to heart block and asystole at levels greater than 18 mg/dL (7.5 mmol/L). Mechanisms include decreased vascular smooth muscle contraction and peripheral sympathetic blockade. Bradycardia may, in part, be a result of sympathetic blockade. [1, 9, 10, 11]. Cardiovascular changes observed in this case is consistent with other research conducted [10,12]. Treatment of hypermagnesemia consists of discontinuing the source of magnesium, respiratory and hemodynamic support, calcium supplementation and treatment with diuretics and hemodialysis [13]. Atropine, a potent cardiac parasympatholytic blocking agent, increases the heart rate and is widely used to treat bradycardia. There are three distinct phases of atropine action on the heart, which include an initial vagotonic effect, a transient period of vagal imbalance at different levels of the conduction system, and a final prolonged period of parasympathetic blockage [14]. In a report of acute hypermagnesemia in a 10-month–old girl, two doses of atropine used for cardiopulmonary resuscitation and bradycardia 0.02 mg/kg (20 mcg/kg) by IV push [13]. Although there are currently no established guidelines for the treatment of bradycardia, it is widely believed that a heart rate below 60 beats per minute after the induction of SA requires treatment. Approximately one-half of patients who received atropine in the prehospital setting for compromising rhythms had either a partial or complete response to therapy [3].

CONCLUSION

We report a case of severe hypermagnesemia and normal renal function following overdose of magnesium sulfate during an experimental study that resulted in gastrointestinal and respiratory changes along with cardiovascular detection. As in this case, administration of atropine can be used for successful therapeutic management of magnesium toxicosis in ruminants.

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