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Case report
Κλινικό περιστατικό

Iatrogenic magnesium overdose in a Golden retriever - case study

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ABSTRACT: This case study describes the clinical presentation, diagnosis, evolution and treatment of a 2-year-old golden retriever dog who accidentally ingested 100 tablets of magnesium-based dietary supplement. 48 hours after ingestion, he showed clinical signs associated with digestive disorders and cardio-vascular impairment such as vomiting, hypotension, decreased heart rate and the appearance of cardiac compensatory phenomena that are prior to cardiac arrest. The report highlights the therapeutic approach and clinical management strategies, as well as the results of the treatment applied. Hypermagnesemia has been reported rarely, so the protocol applied was focused on stabilization and careful cardiovascular, renal and endocrine monitoring.

Keyword: Hypermagnesemia; Dog; Bradycardia; Atropine

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CASE HISTORY

The present study describe the case of an, 2 years old female, Golden Retriver from an authorized kennel, with no history of medical problems, which voluntarily ingested over 100 tablets of magnesium based food supplement (composition: magnesium bisglycinate, microcrystalline cellulose, magnesium stearate, hypromellosecapsule). The ingestion took place in the absence of the owner supervision and was suspected when the owner found the tube on the floor completely empty. The ingested product is a dietary supplement for humans that has a composition with magnesium in a concentration of 100 mg/tablet, so we deduced that the dog ingested approximately 10.000 mg of magnesium in a single administration.

The female dog was brought to the University Emergency Hospital "Prof univ Dr. Alin Birtoiu" 48 hours after the time of ingestion because the owner started noticing some behavioural and general changes like disorientation, vomiting, periods of apathy which alternates with normal behaviour, difficult and fearful walk and the preference for lateral decubitus for the most of the day.

The dog underwent general clinical examination, cardiological examination and paraclinical laboratory test (electrolyte dosage, blood chemistry and blood count).

Clinical examination: At the time of presentation to the hospital, the dog was in a general impaired condition showing exercise intolerance and an inability to maintain a quadrupedal stance. The mental status was depressed, with no abnormal movements observed and a reduced menace response. The oral mucosa was pink, with a slightly delayed capillary refill time and hypersalivation. Abdominal palpation revealed no signs of pain or sensitivity.

Auscultation showed no abnormal lung sounds, but heart rate was below 45 bpm with low pulse intensity on extremities. Laboratory tests were performed throughout the hospitalization period, with daily monitoring of electrolytes and general parameters, particularly renal function. At presentation—approximately two days after ingestion—biochemical parameters were in between normal limits. Electrolyte monitoring revealed significant fluctuations in magnesium levels over the 5-day hospitalization. 14 days after discharge, the patient returned for follow-up tests.

Based on the values presented in Table 1, we can observe the evolution of the values for magnesium, creatinine and urea during the 14 days, which suggest important information regarding metabolism and kidney function. Serum magnesium levels showed a gradual decrease over the 14-day monitoring period, starting above the normal range and progressively reaching physiological limits. Initial values indicated mild hypermagnesemia, with levels slowly declining during hospitalization. By Day 14, magnesium had normalized, suggesting restoration of metabolic balance.

Creatinine and urea were within normal limits throughout the period, suggesting that kidney function was not significantly affected and allowed an effective elimination mechanism through glomerular filtration.

Cardiological examination was performed every day for 5 days and it included echocardiography, blood pressure monitoring and electrocardiography. It was correlated with heart rate monitoring, so it was decided to install a permanent emergency monitor in the first 48 hours of hospitalization, and then hourly for 72 hours.

From an echocardiographic point of view, the patient showed no evidence of congenital malformations or structural heart disease that could explain the bradycardia or predispose to arrhythmias. Blood

Table 1.

	IONIZED MAGNESIUM VALUE (0.3-0.6 mmol/L)	CREATININE VALUE (0.5 -1.8 mg/dL)	UREA VALUE (7.0-27.0 mg/dL)
DAY I	0.80 mmol/L	1.2 mg/dL	19.2 mg/dL
DAY II	0.86 mmol/L	0.6 mg/dL	11.6 mg/dL
DAY III	0.82 mmol/L	0.7 mg/dL	11.8 mg/dL
DAY IV	0.74 mmol/L	0.7 mg/dL	12.8 mg/dL
DAY V	0.66 mmol/L	0.6 mg/dL	13.4 mg/dL
DAYS XIV	0.42 mmol/L	0.7 mg/dL	15.4 mg/dL

Table 2.

Day	SYS (mmHg)	DIA (mmHg)	IOP (mmHg)
Day I	82 mmHg	55 mmHg	13 mmHg
Day II	84 mmHg	58 mmHg	14 mmHg
Day III	88 mmHg	64 mmHg	13 mmHg
Day IV	108 mmHg	71 mmHg	13 mmHg
Day V	122 mmHg	77 mmHg	14 mmHg
Day XIV	142 mmHg	80 mmHg	14 mmHg

pressure monitoring, showed important variations right from the moment of presentation at the hospital and it was negatively correlated with the variation of intraocular pressure which remain relatively constant throughout monitoring period. To verify the accuracy of blood pressure measurements, both the oscillometric and Doppler methods were used (Tabel 2). Maximum blood pressure limits are commonly cited as: 160 mmHg for systolic and 95 mmHg for diastolic blood pressure (Ynaraja Ramírez, E., 2022).

The table shows the evolution of systolic (SYS) and diastolic (DIA) blood pressure (Oscillometric method) over the 14 days, indicating an increase in both systolic and diastolic values.

From electrocardiographic point of view, during the 5 days of hospitalization, the most important change was bradycardia. The heart rate in the first 4 hours after admission varied between 40-60 bpm with a median change of 54 bpm. Based on low frequencies of cardiac rhythm it was decided to perform "Atropine response test" which showed an adequate response, the frequency of the complexes increased >150 bpm (Fig 2). The increase in heart rate was associated with the appearance of premature ventricular complexes because it led to the appearance of the "R on T phenomenon", thus the fusion at the level of the T wave indicating the need to exclude primary cardiac pathologies (already excluded by echocar-

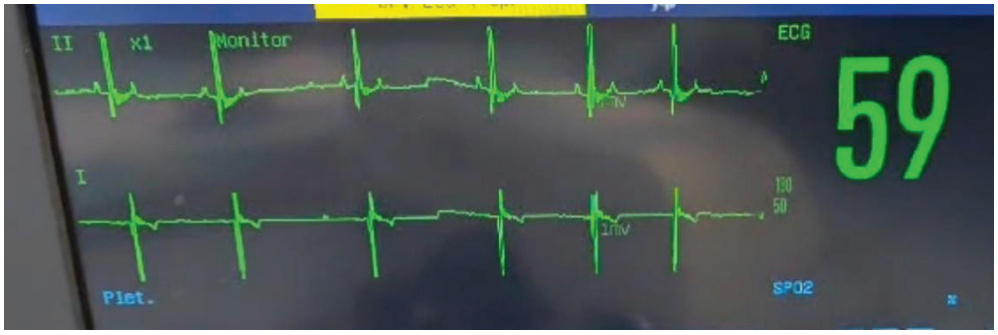

Figure 1. Bradycardia (HR 59 bpm) with a sinus pause.

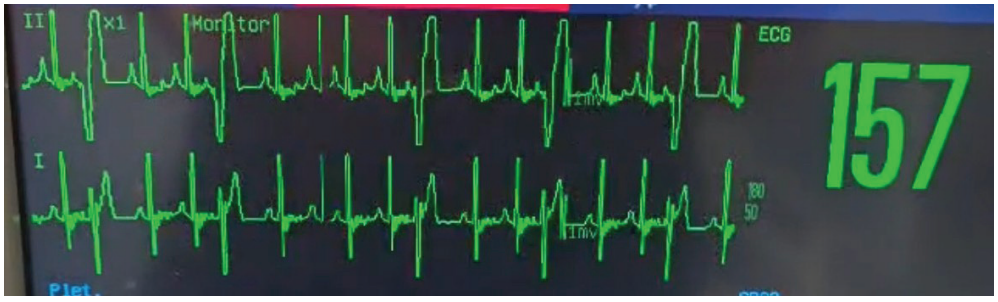

Figure 2. Atropine response test result. Ventricular premature beat with a compensatory pause. Heart rate 157 bpm.

diographic examination) and systemic pathologies (diseases excluded by blood tests and thorough clinical examinations) and supporting the diagnosis of electrolyte imbalances (hypermagnesemia).

Heart rate was difficult to stabilize**.** Bradycardia in dogs is associated with a decrease in heart rate, below 60 bpm in adults and below 70 bpm in puppies. The Golden retriever breed is known for a lower heart rate compared to other breeds, but in the presented case the heart rate from the first day showed important fluctuations (Fig. 3).

Over the course of the 14 days, a gradual increase in heart rate (HR) was observed, from low values in the first days (bradycardia below 60 bpm) to almost normal values on Day V (88-95 bpm), followed by a significant increase on Day XIV (155 bpm in the morning, 145 bpm in the afternoon and 152 bpm in the evening), suggesting a normal heart rate for a dog under stress.

The primary treatment approach focused on enhancing diuresis through intravenous saline fluid therapy. To increase the heart rate, atropine was administered at a dosage of 0.02–0.04 mg/kg, initially at 4 hours, followed by doses at 6-hour and 12-hour intervals during the first 48 hours of hospitalization. In the first 4 hours after the increase in heart rate and the appearance of arrhythmias, we decided to administrate calcium gluconate (100 mg/kg) twice over a period of 20 minutes to counter the toxic effects upon the heart. The patient did not require peritoneal dialysis or hemodialysis, renal function being deemed adequate to sustain effective diuresis during the course of treatment.

To counter digestive phenomena (vomiting and hypersalivation) we used maropitant 1 mg/kg/day in the first 48 hours.

This therapeutic protocol allowed the restoration of metabolic balance and physiological functions, preventing severe complications associated with the excess of magnesium in the blood, such as cardiac arrhythmias, respiratory depression and kidney impairment.

DISCUSSIONS

Hypermagnesemia is generally associated with renal dysfunction and hypoadrenocorticism, with reported effects including cardiac toxicity (myocardial ischemia, hypercoagulable states) and digestive symptomatology (Humphrey et al., 2015; VMD, C. et all., 2004; Mochizuki M. et all, 1998.).

The physiological mechanism underlying cardiovascular impairment involves the action potential of cardiac myocytes, which is generated through coordinated ionic fluxes and comprises five distinct phases: phase 0 (rapid depolarization via inward Na^+ flow), phase 1 (initial repolarization), phase 2 (plateau phase), phase 3 (repolarization), and phase 4 (resting potential). During phase 4, cells are reactivated at membrane potentials below −40 to −45 mV, with a typical resting membrane potential around −65 mV. Cellular excitability—the capacity of a myocyte to generate an action potential in response to a stimulus—depends on both the stimulus intensity and the resting membrane potential reaching the depolarization threshold. Variations in cell excitability influence heart rate, conduction velocity, and the risk of arrhythmias.

Magnesium plays a critical regulatory role in the

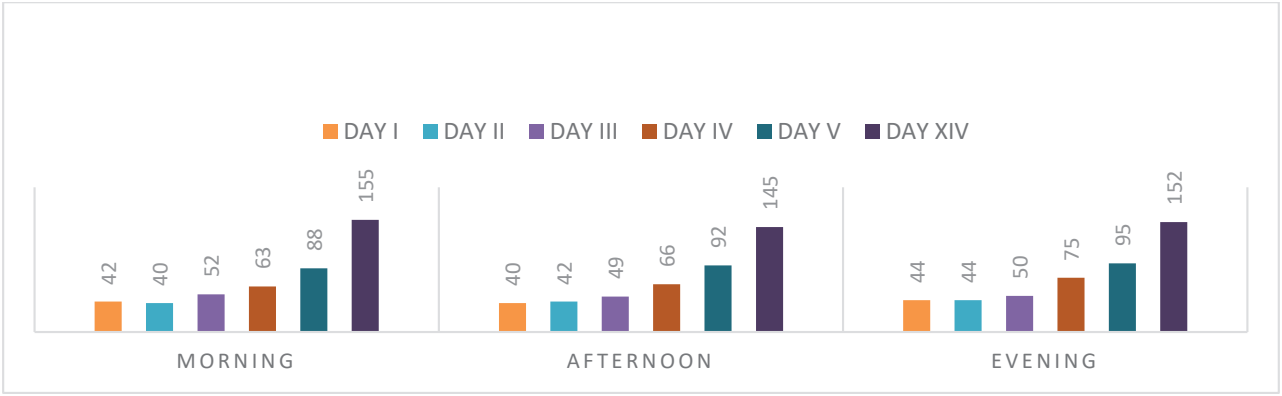

Figure 3. Heart rate evolution during the monitoring period of the female dog.

electrophysiological behaviour of cardiac myocytes. It modulates the activity of ion channels, particularly calcium and potassium channels, thereby affecting the duration and stability of the action potential (de Baaij et al., 2015). By influencing calcium influx through L-type calcium channels and supporting the function of the Na^+/K^+-ATPase pump, magnesium contributes to maintaining ionic gradients across the cell membrane and stabilizing the resting membrane potential (Romani, 2013). This directly impacts the threshold required for depolarization and helps regulate the timing and strength of myocyte contractions.

Additionally, magnesium's stabilizing effect on membrane excitability counteracts excessive cellular activation, reducing the likelihood of arrhythmogenic events (Touyz, 2004). In phase 4, for example, magnesium helps balance K^+ and Ca^{2+} currents, mitigating the risk of early or delayed afterdepolarizations. Moreover, by ensuring proper calcium handling in both cardiac and smooth muscle cells, magnesium supports adequate myocardial contractility and overall cardiovascular function.

The Atropine response test is used to assess the functioning of the cardiac conduction system in cases where dysfunction is suspected, while determining the extent to which it is influenced by the vagal system. Sinus tachycardia with a heart rate greater than 135 beats per minute suggests normal sinus node function in the dog (Bonagura et all., 2018; Kittleson et all., 1998). Atropine is an anticholinergic agent that inhibits the activity of the parasympathetic nervous system.

This test is performed after recording the initial ECG at rest and establishing the heart rhythm and rate. It consists in the injection of 0.04 mg/kg of atropine subcutaneously or intramuscularly and repeating the electrocardiogram 20 minutes after administration. The answer can be adequate, partial or negative (Willis et al., 2018).

Depending on how bradycardia occurs (sudden or chronic), side effects can vary. In dogs where bradycardia sets in suddenly, as in our case, decreased cardiac output can cause syncope and weakness. Hypotension has been associated with decreased vascular tone (Haase et al., 2013). On the other hand, in the case of chronic bradycardia, compensatory mechanisms, such as increasing blood volume by activating the renin-angiotensin-aldosterone system and increasing diastolic time, lead to an increase in ejection volume (Riegger et al., 1984; Merck Veterinary Manual, n.d.). These compensatory mechanisms explain why patients with conduction dysfunctions associated with low heart rates (usually below 50 bpm) can live normal lives (Willis et al., 2018; Vos et

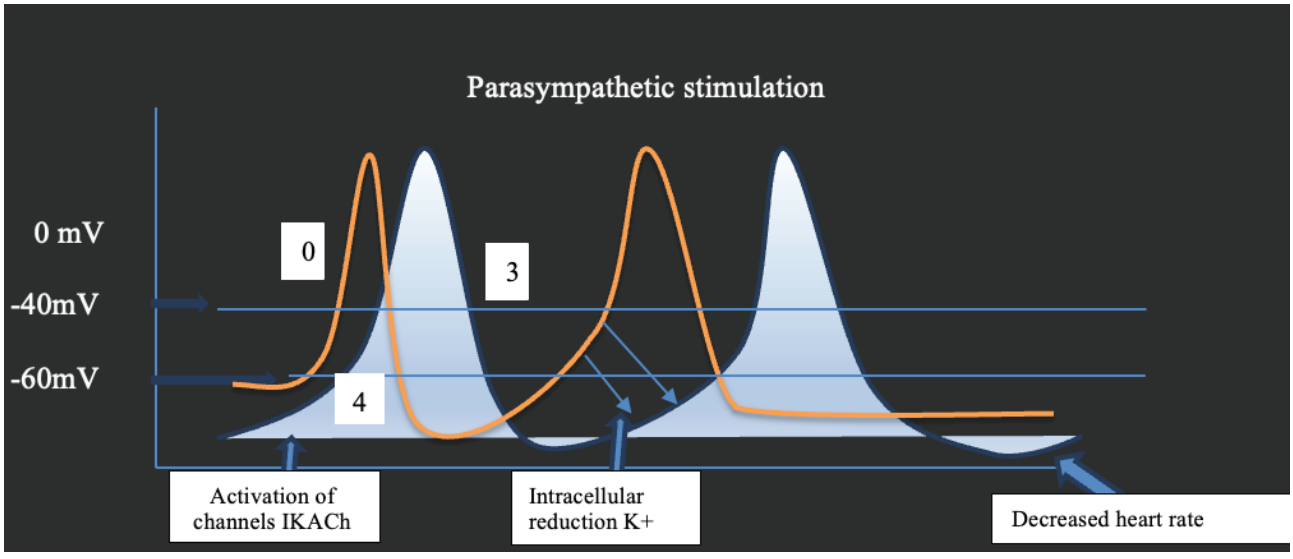

Figure 4. Parasympathetic stimulation –Promotes an increase in outward K^+ currents during phase 4 through activation of acetylcholine-sensitive potassium channels (IKACh). The resulting intracellular K^+ loss leads to membrane hyperpolarization, lowering the resting potential. This makes it more difficult to reach the activation threshold, thereby reducing heart rate. Orange line = normal electrical activity; Blue area = parasympathetic influence (reduced pacing rate).

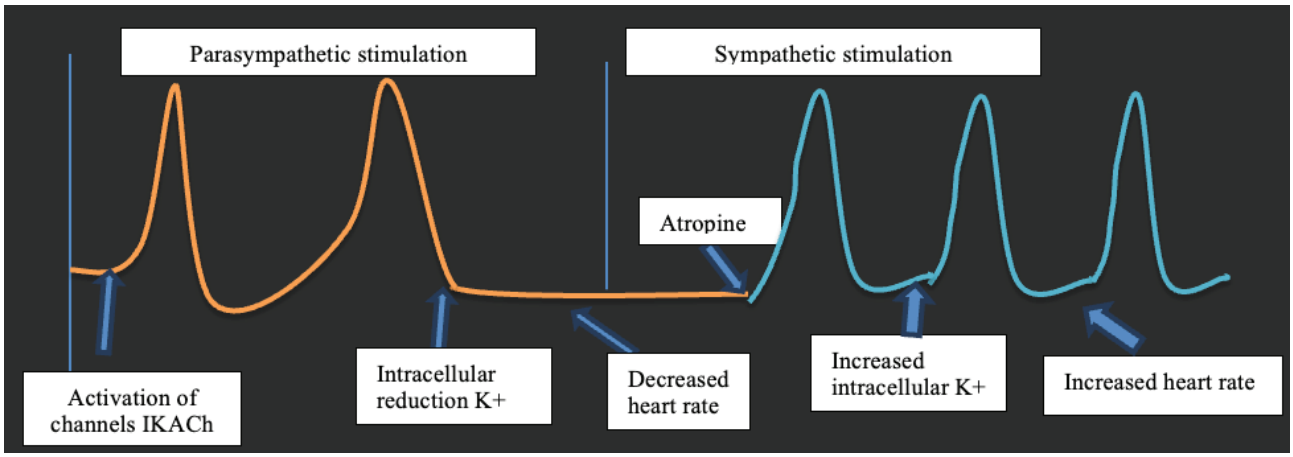

Figure 5. Parasympathetic stimulation and sympathetic stimulation. Parasympathetic stimulation (Orange line) - releases acetylcholine, which activates IKACh channels, increasing K^+ efflux. This causes hyperpolarization, making depolarization harder and thus reducing heart rate. Sympathetic Stimulation (Blue line) - Sympathetic stimulation increases the If current via cAMP, enhancing the opening of If and ICa-L channels. This accelerates phase 4 depolarization, leading to an increased heart rate.

al., 1998). The increase of heart rate was associated with the appearance of ventricular premature complexes. Sinus bradycardia is a type of sinus rhythm where the sinus node discharge rate is low (<50 bpm in an awake dog), it is physiological during sleep and pathological during physical activity, being correlated with pathologies of the sinus nodule. Premature or Escape Beats as well as ventricular premature complexes can occur prematurely and form the so-called ''R on T phenomenon'', a phenomenon in which the superimposition of the ventricular depolarization (R wave) to the T wave of the preceding beat (Cortes, Y. et all., 2007; Oyama, M. A. et all., 2011). The T wave is associated with a period of ventricular vulnerability influenced by primary heart disease or systemic disease or electrolyte imbalances (Tilley et all., 2015; Ware, 2016).

From a laboratory standpoint, a decrease in serum magnesium concentration was associated with a corresponding reduction in the severity of bradycardia and compensatory cardiac phenomena. To exclude potential endocrine disorders that could alter magnesium homeostasis—particularly those affecting glomerular filtration or cortisol secretion through direct or indirect mechanisms—basal cortisol levels were assessed (Mouton A. et all., 2025; Goggs R. et all., 2017).

Following patient stabilization and discharge, ongoing biochemical monitoring was implemented to evaluate possible delayed toxic effects. From a therapeutic perspective, the stabilization strategy primarily targeted the correction of bradycardia and mitigation of compensatory cardiac mechanisms (Silverstein, D.C., 2022).

CONCLUSIONS

Although from the electrolyte point of view the values obtained did not exceeded the physiological limits, the impact on the cardiovascular system was major and led to cardiac impairment. We believe that the blood magnesium assessment was helpful in setting the diagnosis, but we consider that the indicated value did not represented the total metabolized magnesium or the real value of magnesium in the body.

From a cardiac perspective, the decrease in heart rate to bradycardic levels was a key indicator of magnesium's toxic effects on the heart, given its significant role in calcium channel activation and in maintaining the function of the Na^+/K^+-ATPase pump.

Therapy was aimed at counteracting the cardiotoxic effects by increasing heart rate and promoting renal excretion. From a therapeutic standpoint, the treatment was considered successful, as renal function remained uncompromised.

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