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ORIGINAL ARTICLE

Effects of Magnesium Sulfate on Some Cardiac Arrhythmias in Cattle

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ABSTRACT
Milk fever (paresis puerperalis), the clinical manifestation of parturient hypocalcaemia, is a disease of considerable importance for dairy cow welfare and economy. Three main preventive principles against milk fever were evaluated in this literature review, and the efficacy of each principle was estimated from the results of controlled investigations. Oral calcium drenching around calving apparently has a mean efficacy of 50%–60% in terms of milk fever prevention as well as prevention of milk fever relapse after intravenous treatment with calcium solutions. After potassium, the naturally ubiquitous magnesium is the second most abundant intracellular cation in the human organism. Magnesium catalyzes or activates more than 300 enzymatic reactions as a metallocoenzyme. Cardiac arrhythmias are the most side effects of calcium IV treatment in cattle and may cause death in them. A way to control of these side effects is Magnesium sulfate administration. The aim of this study is effects of Magnesium sulfate administration on induced cardiac arrhythmias with Calcium borogluconate. Eight healthy dairy cows (weight 350-450 kg and 3-4 years old) were referred for electrophysiologic testing to evaluate known or suspected cardiac arrhythmia and after 250 ml Calcium borogluconate IV injection, there was some arrhythmias in their ECG, and after IV injection of 10 ml Magnesium sulfate, the all of arrhythmias eliminated in about 5 minute. So we suggest Magnesium sulfate is the safe and sound way to control the arrhythmias after Calcium borogluconate injection.
Keywords: *dairy cows, , Calcium borogluconate , Magnesium Sulfate*

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INTRODUCTION

Milk fever (paresis puerperalis), the clinical manifestation of parturient hypocalcaemia, is a disease of considerable importance for dairy cow welfare and economy. Although treatment with intravenous infusion of calcium salt solutions cure most clinical cases of hypocalcaemia, such cows are later more susceptible to other metabolic and infectious diseases [1, 2].After potassium, the naturally ubiquitous magnesium (Mg^{2+})is the second most abundant intracellular cation in the human organism. Mg^{2+} catalyzes or activates more than 300 enzymatic reactions as a metallocoenzyme. A human body weighing70 kg contains about 24 g Mg^{2+}, 99% of which is located intracellularly. Changes in the low intracellular concentration can lead to major effects on the functioning of the ion channels and the second messenger systems of cardiac cells. Therefore, Mg^{2+} is regarded as an important regulator or modulator of the heart cell function. Intravenous magnesium has been used to treat ventricular arrhythmias in patients with and without hypomagnesemia, with and without digitalis-induced arrhythmias, with monomorphic ventricular tachycardia or torsade depointes, and with acute myocardial infarction. Previousreports describe the therapeutic efficacy of intravenous magnesium in patients with ventricular arrhythmia.However, in most cases, magnesium therapy was added to conventional antiarrhythmic drugs that had failed. There are few or no controlled data on the antiarrhythmic efficacyof magnesium as monotherapy in patients without theseassociated conditions. The electrocardiographic and electrophysiologic effects of intravenous magnesium have been studied in patientswithout clinical evidence of significant cardiac disease and in patients in whom sustained atrial or ventricular tachycardia could not be initiated by electrophysiologic evaluation. However, a systematic evaluation of the electrophysiologic and antiarrhythmic properties of intravenous magnesium in patients with clinical

ventricular arrhythmia and inducible ventricular tachyarrhythmia hasnever been reported [3, 4, 5]. In many countries prevention of parturient hypocalcaemia is therefore given a high priority.It has been proposed that a specific control program is relevant when the incidence of milk fever increases to above 10% among high-risk cows, i.e. cows entering third or later lactations. The per parturient or transition period of 4 weeks before and 4 weeks after calving is characterized by a greatly increased risk of disease. Hypocalcaemia around calving is a risk factor for many of these diseases and is an indirect risk factor for increased culling. The incidence of clinical hypocalcaemia (milk fever) in the field generally ranges from 0-10%, but may exceed 25% of cows calving. In research trials conducted on milk fever the incidence has approached 80% of cows calving. Homeostasis of calcium (Ca) is regulated by calcitonin, parathyroid hormone and 1,25(OH)(2) vitamin D(3). Age increases the risk of milk fever by approximately 9% per lactation. Control of milk fever has revolved around stimulation of homeostatic mechanisms through feeding a pre-calving diet low in Ca. More recently, the role of the dietary cation anion difference (DCAD) in the prevention of Ca disorders has been examined, both by field research and meta-analysis. The most appropriate form of the DCAD equation has been contentious, but recent meta-analyses have shown that the equation (Na(+)+K(+))-(Cl(-)+S(2-)) is most effective for predicting milk fever risk. Decreased risk of milk fever is linear with DCAD, whereas the effect of DCAD on urinary pH is curvilinear. A pivotal role of providing dietary magnesium (Mg) before calving has been confirmed by meta-analysis, and a quadratic effect of Ca on milk fever risk was found with a peak occurring with dietary levels of 1.1-1.3% of dry matter. Risks of milk fever increase with increased dietary phosphorus (P) fed pre-calving and with increasing days of exposure to a pre-calving diet. Meta-analysis has revealed that the important roles of dietary Ca, Mg and P, as well as the duration of exposure to the pre-calving diet in milk fever control strategies are independent of DCAD. Studies on the effect of exposure to well-designed pre-calving diets have shown that substantial improvements in production, reproduction and animal health can be made but further examination of the influence of the period of exposure to different diets is warranted [6-8].

MATERIAL AND METHOD

Eight healthy dairy cows (weight 350-450 kg and 3-4 years old) were referred for electrophysiologic testing to evaluate known or suspected cardiacarrhythmia.

After confirmation of no evidence of any cardiac arrhythmias, we noted the heart rate and inject the Calcium borogluconate 40% from Nasr fariman company (Iran) that injected fast IV in jogular vein in all of group.

In time of infusion the calcium borogluconate, heart rate noted and electrocardiogram take every 5 minutes. After the heart rate changes or any cardiac arrhythmia seen, we finished the calcium borogluconate infusion and then start the Magnesium sulfate 40% infusion. In all time, every 5 minute, ECG was taken. The time of arrhythmias elimination and the volume of drugs were noted.

RESULTS AND DISCUSSION

The Mean heart rate of all cattles before Calcium injection was 63 beats per minute that increases after 250 ml IV injection to 76 beats per minute. In ECGs, there was some arrhythmias. The Cardiac rrhythmias were Sinus arrhythmia, Ventricular Tachycardia, Grade 1 and 2 atrioventricular block. These arrhythmias have seen in all of ECGs. After 10 ml Magnesium sulfate IV injection, in about 5 minute of injection, all the arrhythmias gone and cardiac rhythm became regular.

Based on the results of serial electrophysiological testing, intravenous magnesium has antiarrhythmic effects in patients with cardiac arrhythmias.

Magnesium is essential in the activation of the sodium potassium denosine tri-phosphatase (ATPase) pump and thus plays a critical role in the maintenance of the cellular transmembrane potential.Hypomagnesemia is associated with intracellular potassium deficiency which can increase cellular excitability and automaticity. Also, there are epidemiologicdata linking low magnesium levels to ventricular arrhythmiaand sudden cardiac death in patients receiving long-termdiuretic therapy and in patients with coronary artery disease [4,3]. Thus, hypomagnesemia is associated with arrhythmia, andmagnesium therapy in such cases appears justifiable.Hypomagnesemia when combined with hypocalcemia can result in QT-interval prolongationand predispose to ventricular arrhythmias, particularly torsadede pointes. Magnesium administration in such circumstancesshortens the QT interval and probably is the treatment of choice for polymorphic ventricular tachycardia including torsade de pointes and other arrhythmias associated with QT prolongations Magnesium therapy is also effective in the treatment ofdigitalis-associated arrhythmia.Hypomagnesemia is afrequent finding in patients with digitalis toxicity lo and is more common than hypokalemia in this condition. Magnesium counteracts the inhibitory effects of digitalis onsodium potassium ATPase therapy, reducing cell excitability and digitalis-associated arrhythmia.Thus, magnesium therapy has an important role in the therapy of arrhythmias

associated with hypomagnesemia,QT prolongation (especially torsade de pointes), and digitalisexcess [4,3].

CONCLUSION

In the present study we exclude patients with milk fever conditions that had some pathologic and non-Pathologic arrhythmias, could treat with intravenous magnesium sulfate.

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