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ORIGINAL ARTICLE

The effect of subacute intoxication of monensin on minerals and trace elements in goats

Mahdi Deljou · Mehrdad Mohri · Mohammad Reza Aslani · Mohammad Heidarpour · Ahmad Reza Movassaghi

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Abstract The aim of this study was to evaluate the status of minerals and trace element concentrations in serum samples from goats with subacute intoxication of monensin. For this purpose, monensin with the dose of 13 mg/kg boy weight was orally administered to seven goats for 5 consecutive days. Elements including sodium (Na), potassium (K), Calcium (Ca), magnesium (Mg), phosphorous (P), iron (Fe), copper (Cu), zinc (Zn), and selenium (Se) were determined in baseline and daily serum samples for 10 days. Subacute monensin intoxication significantly decreased serum concentration of Ca, Mg, Na, and Zn in most of samples. Serum concentration of P significantly increased at days 2 and 7–10. There was no significant change in serum concentration of Fe, Se, and Cu during the experiment. These finding indicate that subacute monensin intoxication alters serum concentrations of minerals and certain trace elements in goats.

Keywords Monensin · Goat · Minerals · Trace elements

M. Deljou · M. Mohri · M. R. Aslani · M. Heidarpour
Department of Clinical Sciences, School of Veterinary Medicine,
Ferdowsi University of Mashhad,
Mashhad, Iran

M. R. Aslani (✉)
Department of Clinical Sciences, Faculty of Veterinary Medicine,
Shahrekord University,
Shahrekord, Iran
e-mail: mraslani@um.ac.ir

A. R. Movassaghi
Department of Pathobiology, School of Veterinary Medicine,
Ferdowsi University of Mashhad,
Mashhad, Iran

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Introduction

Monensin, a monovalent carboxylic ionophore produced by *Streptomyces cinnamonensis*, is the first antibiotic that has been made and sold exclusively for use as an anticoccidial in chickens (Chapman et al. 2010). However, in recent years, it has widely been used as a feed additive to improve performance in livestock production systems (Duffield et al. 2012). Monensin has also minor additional uses in the treatment of ketosis, lactic acidosis, bloat, and acute pulmonary edema and emphysema (Radostits et al. 2007). However, the marginal safety of monensin is not wide, and its careless use has been associated with major losses. Accidental monensin intoxication, usually associated to overdosage, misuse, or mixing errors in feed preparation, has been reported in cattle (Basaraba et al. 1999; Gonzalez et al. 2005), water buffaloes (Rozza et al. 2006), sheep (Jones 2002; Mendes et al. 2003), horses (Peek et al. 2004), swine (Miskimins and Neiger 1996), chickens (Zavala et al. 2011), ostriches (Dedoussi et al. 2007), deer (Glover and Wobeser 1983), and dogs (Wilson 1980; Condon and McKenzie 2002). Acute cases of monensin intoxication have also been described in humans (Kouyoudjian et al. 2001; Caldeira et al. 2001).

The exact mechanism of monensin toxicity is not known. However, reasonable hypotheses have been generated based on its inherent ionophoric activity. Carboxylic ionophores form cationic complexes that enhance their transport across biomolecular lipid membranes. Monensin, classified as sodium-selective ionophore that bind to sodium outside the cell, carries it into the cell and produces higher intracellular concentration of sodium (Novilla 2007). This, in turn, enhances the influx of extracellular calcium ions and the release of bound, intracellular calcium ions from sites such as sarcoplasmic reticulum. Accumulation of calcium ions