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Role of magnesuim in ICU

Essay

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LIST OF ABBREVIATIONS

APACHE II	Acute physiology, and chronic health evaluation score
score	for critical ill patients
ATN	acute tubular necrosis
ATP	adenosine-5`-triphosphate
CBF	Cerebral blood flow
СРР	Cerebral perfusion pressure
DCI	delayed cerebral ischaemia
Enos	Endothelial nitric oxide synthase
ET-1	Endothelin -1
FeMg	fractional excretion of magnesium
FEV1	forced expiratory volume during the first second of expiration
GFR	glomerular filteration rate
Hb	haemoglobin
ICP	Intracranial pressure
ICP	Intracranial pressure
IgE	immunoglobulin E
iNOS	inducible nitric oxide synthase
MgSO4	Magnesium sulfate
MLCK	myosin light chain kinase
MPT	mitochondrial permeability transition

MVP	Mitral valve prolapse
NMDA	N-methyl-Daspartate
nNOS	neuronal nitric oxide synthase
No	Nitric oxide
oxyHb	oxyhemoglobine
PEFR	peak expiratory flow ratio
Pg	prostaglandin
PTH	parathormone
ROMK	channel transporter and the potassium-rectifying channel
SAH	Sub arachnoid hemoorhage
TAL	Thick ascending limb
TRPM	transcellular transporter transient receptor potential channel melastatin member
Tx	Thromboxane
VOCC	voltage-operated calcium channels

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Introduction



Introduction

There is an increased interest in the role of magnesium ions in clinical medicine, nutrition and physiology. Magnesium affects many cellular functions, including transport of potassium and calcium ions, and modulates signal transduction, energy metabolism and cell proliferation. Magnesium deficiency is not uncommon among the general population: its intake has decreased over the years especially in the western world. The magnesium supplementation or intravenous infusion may be beneficial in various diseased states. Of special interest is the magnesium status in alcoholism, eclampsia, hypertension, atherosclerosis, cardiac diseases, diabetes, and asthma (*Shils et al.*, 1994).

The Mg ion, the second most abundant intracellular cation after potassium, plays essential roles in the structure and function of the human body; it is an essential cofactor in awide variety in physiological processes, including protein synsthesis and stability, neuromuscular excitability and the conduction of neural impulses, stimulus-contraction coupling and muscular contraction, magnesium is an indispensable part of the activated Mg ATP complex, and it is required for adenosine triphosphate (ATP) synthesis in the mitochondria. magnesium is anecessary cofactor in over 300 enzymatic reactions; it is required for the activity of all rate limiting glycolytic enzymes, protein kinases and more generally, all ATP and phosphate trans- associated enzymes. magnesium may also bind the enzymes directly (i.e RNA and DNA polymerases) and alter their structure, therefore, the availability of an adequate quantity Mg may be considered acritical factor for normal body and cellular homeostasis and function (*Barbagallo and Dominguez*, 2007).

Magnesium is a critical physiological ion, and magnesium deficiency might contribute to the development of pre-eclampsia, to impair neonatal metabolic problems extending into and to Pharmacologically, magnesium is a calcium antagonist with substantial vasodilator properties but without myocardial depression. Cardiac output usually increases following magnesium administration, compensating vasodilatation and minimising hypotension. Neurologically, the inhibition of calcium channels and antagonism of the N-methyl-D-aspartic acid (NMDA) receptor raises the possibility of neuronal protection, and magnesium administration to women with premature labour may decrease the incidence of cerebral palsy. It is the first-line anticonvulsant for the management of preeclampsia and eclampsia, and it should be administered to all patients with severe preeclampsia or eclampsia. Magnesium is a moderate tocolytic but the evidence for its effectiveness remains disputed. The side effects of magnesium therapy are generally mild but the major hazard of magnesium therapy is neuromuscular weakness (Saris et al., 2000).

Some studies have shown that the magnesium intake by patients with diabetes is often below recommended levels. Additionally, there is evidence that the magnesium status of patients with diabetes tends to alter, and that low body concentrations of this mineral may influence the evolution of the disease and generate further complications (*Paula et al.*, 2001).

The use of intravenous magnesium as a bronchodilating agent in the treatment of acute asthma has been recently reported in several articles. The authors report their experience with magnesium in acute severe asthma, defined by clinical examination and by a peak expiratory volume (PEV) of less than 40% predicted. The authors found that infusion of magnesium caused a significant improvement in PEV and improvement in clinical signs and symptoms. They recommend its use as an adjunct to beta-agonist therapy in this setting (Noppen M., et al. 1990).

Magnesium has a lot of physiological and clinical values in human body as itis has therapeutic importance in treatment of acute bronchial ashma, arrhythmia, diabetes, pre eclamsia, resistant hypokalemia and postoperative pain (Shils ME et al., 1994).

Aim of the work

The goal of this essay is to focus on pathophysiology of magnesium in the human body, causes of magnesium derangement in the form of hyper and hypomagnesemia and their clinical symptoms and management of each and the increasing role of magnesium in critical ill patients.

Physiology of Magnesium in human body

