Magnesium Deficiency in Ulcerative Colitis.

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A 32 year old white woman presented with ulcerative colitis of approximately two years duration. The physical signs, symptoms and findings on chemical examination of serum were compatible with magnesium deficiency. Remarkable improvement with treatment of magnesium sulfate was obtained in a short period of time. Additional treatment, high protein diet and multiple vitamins with minerals resulted in an uneventful recovery.

Key words: magnesium, ulcerative colitis

A lowered magnesium concentration occurs in various disease states. A case is reported in which the syndrome of magnesium deficiency was associated with ulcerative colitis.

REPORT OF CASE

A 32 year old white married woman was admitted to another hospital in March 1964, following onset of abdominal pain, bloody diarrhea, fever, and anemia. After a diagnosis of ulcerative colitis was established, she improved and was discharged following treatment with Depo-Medrol, enemas, blood replacement and diet.

In May 1965, she had an exacerbation associated with marked weakness and was admitted to Orange Memorial Hospital. On physical examination, the temperature was 102.8 F., pulse rate 100 per minute, respiration 20, and blood pressure 110/70 mm. Hg. Icterus, generalized abdominal tenderness, enlargement of the liver and marked pallor were present. The hematocrit value was 15, the white blood cell count 5,400, serum bilirubin 3.2 mg. and potassium 3.5 mEq.

She received whole blood transfusions, intravenous ACTH drip, Terramycin and potassium supplements, but after initial improvement severe anemia and clinical and electrocardiographic evidence of potassium depletion developed. After vigorous blood and potassium replacement, a proctocolectomy with permanent ileostomy was performed on July 13, 1965.

The postoperative period was complicated by wound dehiscence and a need to continue intravenous feeding because of gastric suction. Fifteen days postoperatively, she became delirious, disoriented, hallucinatory and aggressive and manifested tremor and twitching. The electrocardiogram showed ST-T abnormality. Reflexes were hyperactive (table 1).

Four grams of magnesium sulfate was given intravenously in 250 ml. of 5% dextrose in water over a two hour period. Shortly after she received the magnesium sulfate, the sensorium cleared and evidence of neuromuscular irritability subsided. She received additional magnesium sulfate over the next four days.

After a prolonged convalescence she was discharged on September 16.

DISCUSSION

The adult body contains 21 to 28 Gm. of magnesium or about 43 mg. per kilogram of fatfree tissue. About half of the total is present in bone. The ash containing 0.5 to 0.7% magnesium is similar to potassium in its distribution being relatively concentrated in the intracellular space. The liver and striated muscles have the highest concentration of about 20 me_2. The brain and kidney contain about 17 and 13 me_2 and the red cells concentration contains about 6 me_2. Magnesium is a major intracellular cation. Though its precise mode of action is not known, the majority of the reactions involving adenosine triphosphate are activated by magnesium ion. In their absence activity is either much reduced or absent. The individual systems in which the magnesium as metal participates are too numerous to be detailed here. This ion usually participates in group

TABLE 1.

me*=me_2

Date	NA me*	CL me*	K me*	Mg mg%	Ca mg%	Total protein
7-13-65	140	105	4.2		7.7	
7-14-65	146	108	4.6			4.5 Gm.
7-15-65	141	102	3.3		8.3	
7-28-65	148	105	4.	1.4	9.2	4.7 Gm.
7-30-65			3.9	1.7	7.3	
8- 3-65	140	104	4.7	1.8	9.3	5.9 Gm.
8- 9-65				1.8		
8-18-65				1.8	8.8	
8-24-65				2.1		
8-31-65				2.4		
9- 7-65				1.9	9.2	
9-16-65	139	104	4.3	2.0	8.9	7.3 Gm.

transfer reactions. The transfer of phosphate, pyrophosphate, sulfate, methyl, formyl, acetyl, alkyl and glycol aldehyde groups utilize magnesium for activation. Thus it is involved in virtually all important metabolic processes such as oxidative phosphorylation of protein, fat, carbohydrate and nucleic acid.

The Cause of Magnesium Deficiency: Malnutrition has been a factor in all patients afflicted, and reduced dietary intake of the element occurs in most of them. In each instance that the syndrome has been observed it was brought about or intensified by some factor which either prevented the absorption or increased the excretion of magnesium.

A. Conditioned Deficiency

1. Failure to absorb a metabolite, inability to synthesize it into biologically active intermediate, and excessive excretion are the simplest examples of conditioning factors.

a. Severe debilitating disease.
b. Prolonged acute infections.
c. Severe alcoholism accompained by malnutrition.
d. Prolonged intestinal malabsorption, or drainage of gastrointestinal contents.
e. Continued parenteral treatment with magnesium-free fluids provides the setting in which this syndrome may be observed. Conclusive proof that the syndrome of magnesium deficiency is due to an alteration of magnesium metabolism is afforded by the correlation of symptoms and signs with chemical changes in the serum. Hypomagnesenemia may accompany other conditions. A lowered magnesium concentration has been observed in idiopathic epilepsy, cirrhosis, congestive heart failure, crhonic nephritis, eclampsia, diabetic acidosis, pancreatitis, hyperparathyroidism and hyperaldosteroidism. At present it is clinically important to recognise the possybility of magnesium deficiency as a causative factor in disturbance of neuromuscular and central nervous system activity particularly in a patient with malnutrition due to any cause. Sudden onsed of delirium, hallucinations, delusions, and wild combative behavior, was seen in our patient.

Clinical and experimental work on magnesium and its relation to the cardiovacular system has been reported (2, 3).

2. Studies in man have shown that as age increases so does the content of calcium and magnesium in the human aorta. Studies on a limited number of patients with uncomplicated hypertension have shown that the serum magnesium levels were significantly lower and serum sodium levels were higher than were those of the controls. It was suggested that this finding might reflect increased adrenal cortical activity similar to that noted in aldosteronism in which a low level of magnesium is seen. Magnesium depletion induced by various diuretics has been reported by Smith and his coworkers (4).
3. The authors stress the inherent danger of magnesium want in edematous patients receiving long term therapy with certain diuretic agents, and they also point out that an even greater risk of magnesium depletion accompanies various diuretics therapy in alcoholic pa-

tients whose supplies of magnesium are often already deficient.

Magnesium depletion was reported in postsurgical patients by Gerst and his associates (1).

4. Where magnesium deficiency was found in one patient 108 days after the patient was a victim of abdominal trauma and underwent partial gastrectomy, in a second case the patient had repeated intestinal perforation due to regional enteritis.

LITERATURA

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Sažetak

NEDOSTATAK MAGNEZIJA U ULCERATIVNOM KOLITISU

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Prikazan je slučaj 32-godišnje pacijentice s dvogodišnjim ulcerativnim kolitisom. Fizički znaci, simptomi i laboratorijski nalazi seruma upućivali su na nedostatak magnezija. Liječenjem magnezij sulfatom postignut je značajan napredak u kratko vrijeme. Dodatni tretman, dijeta bogata proteinima i multimineralovitaminska supstitucija rezultirali su mirnim oporavkom.

Ključne riječi: magnezij, ulcerativni kolitis