



Pharmacological Treatment may Impair Mineral Status in Blood

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Abstract

Some medicines may interact with mineral, with the result that changes occur in the concentration of minerals and other blood parameters that are associated with mineral status. Interpretation of the results of morphological and biochemical blood analyses therefore needs to take into account the drugs used by patients.

Keywords

Minerals; Medicines; Interaction

Hematological and biochemical blood parameters are rather stable in healthy subjects. However, there are many factors that can influence mineral status in blood, including diseases, diet, and drugs. It has been found that several medicines can alter both mineral concentrations and other mineral-status-related parameters in blood.

In experimental and human studies, it has been shown that hypotension therapy disturbs zinc status in the body [1]. Some of our results suggest that indapamide and amlodipine treatment can decrease zinc concentration in serum and erythrocytes. Moreover, the decrease in zinc is accompanied by an increase in glucose serum levels [2]. In our experimental study, we observed that treatment with indapamide and amlodipine was associated with increased red blood cell counts and hematocrit values in the serum of spontaneously hypertensive rats [3]. In patients, high hematocrit could reflect the increase in red blood cell mass resulting from the reduction in plasma volume due to treatment with thiazide-type diuretics.

Long-term therapy with captopril and enalapril may lead to zinc depletion in erythrocytes and monocytes. Preliminary data suggest that indapamide can cause a slight elevation in blood calcium levels. Several results indicate that loop diuretics, especially furosemide, reduce serum calcium and magnesium levels. It has also been observed that beta-blockers may induce hypocalcemia and hypomagnesemia [4].

Antiepileptic therapy with valproic acid may cause abnormalities in serum copper concentration. Some authors have found that therapy with valproic acid can diminish copper levels in erythrocytes and serum in epileptic patients [5,6].

Nonsteroidal anti-inflammatory drugs, when taken regularly, may cause bleeding from the gastrointestinal tract and iron loss with the blood. The use of aspirin and other medicines belonging to this group of drugs may therefore increase the risk of iron deficiency,

which can be identified as a low hemoglobin and serum ferritin concentration [4,7].

In patients with digoxin therapy, a decrease in intracellular magnesium levels and a relatively low level of magnesium in serum were observed [8]. The long-term use of corticosteroids, cisplatin, cyclosporine is also associated with a depletion of magnesium in the serum and an increase in the risk of hypomagnesemia [4,9].

It is known that dietary restriction may impair mineral status; however, pharmacological treatment of obesity may also alter the concentration of minerals in the blood. In our clinical study, we found that 12 weeks of sibutramine therapy decreases zinc and increases magnesium concentrations in the serum, leading to mineral imbalances in obese women [10].

Oral contraceptives (OCs) are among the most commonly used drugs in developed countries. One of the side effects of this group of drugs is their influence on the mineral status in women. In several studies, it was observed that the use of oral contraceptives is associated with decreased zinc, magnesium, and selenium serum concentrations, and with increased copper levels in serum. It has also been found that oral contraceptives can increase ferritin and iron levels, TIBC, and mean corpuscular hemoglobin in serum, while decreasing red blood cells and hematocrit in whole blood in women. The results of human studies suggest that the effect of OCs on mineral status can vary with the form, dose, concomitant agents, and patient characteristics [4,11].

The mechanisms behind the observed interactions between drugs and minerals cannot always be explained. Some may be associated with changes in the excretion of minerals in the urine. Drugs may also influence the absorption and/or distribution of minerals in the body [4].

In conclusion, many drugs influence mineral status in the blood. Pharmacological treatment may require the monitoring of mineral concentrations in blood. Moreover, the interpretation of the results of morphological and biochemical analyses in blood requires a consideration of the drugs being used by the patient.

References

1. Braun LA, Rosenfeldt F (2013) Pharmaco-nutrient interactions - a systematic review of zinc and antihypertensive therapy. *Int J Clin Pract* 67: 717-725.
2. Suliburska J, Bogdanski P2, Jakubowski H3 (2014) The influence of selected antihypertensive drugs on zinc, copper, and iron status in spontaneously hypertensive rats. *Eur J Pharmacol* 738: 326-331.

3. Suliburska J, Bogdański P2, Krejpcio Z1 (2014) Evaluation of nutritional and biochemical parameters in spontaneously hypertensive rats following antihypertensive treatment. *Acta Sci Pol Technol Aliment* 13: 103-110.
4. Stargrove MB, Treasure J, McKee DL (2008) Herb, nutrient, and drug interactions. Clinical implications and therapeutic strategies. St. Louis MO, Mosby Elsevier.
5. Doneray H, Kara IS, Karakoc A, Tan H, Orbak Z (2012) Serum thyroid hormone profile and trace elements in children receiving valproic acid therapy: a longitudinal and controlled study. *J Trace Elem Med Biol* 26: 243-247.
6. Lampón N, Tutor JC (2010) Effect of valproic acid treatment on copper availability in adult epileptic patients. *Clin Biochem* 43: 1074-1078.
7. Wang X, Uzu T, Isshiki K, Kanasaki M, Hirata K, et al. (2007) Iron status and the use of non-steroidal anti-inflammatory drugs in hemodialysis patients. *Ther Apher Dial* 11: 215-219.
8. Iezhitsa IN, Spasov AA, Zhuravleva NV, Sinolitskii MK, Voronin SP (2004) Comparative study of the efficacy of potassium magnesium L-, D- and DL-aspartate stereoisomers in overcoming digoxin- and furosemide-induced potassium and magnesium depletions. *Magnes Res* 17: 276-292.
9. Hodgkinson E, Neville-Webbe HL, Coleman RE (2006) Magnesium depletion in patients receiving cisplatin-based chemotherapy. *Clin Oncol (R Coll Radiol)* 18: 710-718.
10. Suliburska J, Bogdański P, Szulińska M, Pupek-Musialik D (2012) Short-term effects of sibutramine on mineral status and selected biochemical parameters in obese women. *Biol Trace Elem Res* 149: 163-170.
11. Palmery M, Saraceno A, Vaiarelli A, Carlomagno G (2013) Oral contraceptives and changes in nutritional requirements. *Eur Rev Med Pharmacol Sci* 17: 1804-1813.