Zinc balance normalization: an important mechanism of angiotensin-converting enzyme inhibitors and other drugs decreasing the activity of the rennin-angiotensin-aldosterone system.

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BACKGROUND: Imbalances of zinc (Zn) metabolism in arterial hypertension are related to increased urinary Zn excretion, Zn transfer between extracellular and intracellular spaces, and redistribution of this element inside the cells. The changes include an increase of the absorption of Zn in the gastrointestinal tract and decreases of its concentration in lymphocytes, bone, and arterial walls. The Zn content of erythrocytes, cardiac muscle, and kidneys also increases. The condition eventually leads to Zn deficiency (1-5).

DISCUSSION: Zinc plays many roles in biological systems. It is a component of over 300 enzymes, performing catalytic, cocatalytic, and/or structural functions. Among others, it conditions the activities of carbonic anhydrase (CA) and the angiotensin-I converting (ACE) and endothelin-converting (EC) enzymes. Zn is essential for forming the quaternary structure of numerous regulatory proteins and hormone receptors that conditions binding with DNA, such as zinc-fingers, zinc-twists, or zinc-clusters. It is a structural element of the nucleic acids and takes part in its metabolism. Zn stabilizes and regulates cell membrane functions. Cellular growth and division depends on the content of Zn inside the cell and on its transport inside the cell’s compartments (6-11).

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