ARCHIVES OF HELLENIC MEDICINE: ISSN 11-05-3992

CONTINUING MEDICAL EDUCATION
ΣΥΝΕΧΙΖΟΜΕΝΗ ΙΑΤΡΙΚΗ ΕΚΠΑΙΔΕΥΣΗ

ARCHIVES OF HELLENIC MEDICINE 2018, 35(4):571
ΑΡΧΕΙΑ ΕΛΛΗΝΙΚΗΣ ΙΑΤΡΙΚΗΣ 2018, 35(4):571

Acid-Base Balance-Electrolyte Quiz – Case 66

A. Liontos,
T. Panagiotopoulou,
D. Sfairopoulos,
M. Elisaf

Department of Internal Medicine, Medical School, University of Ioannina, Ioannina, Greece

Which is the main mechanism of thiazide-induced kaliuria and hypokalemia?

(a) Increased hypovolemia-induced-aldosterone levels

(b) Decreased effective circulating volume depletion

(c) Increased diuretic-induced sodium and water delivery to the distal nephron

(d) Direct stimulatory effect on renin release

(e) a+c

(f) b+d

Comment

Thiazides inhibit the electroneutral NaCl cotransporter (NCC) in the early distal convoluted tubules. Thus, an increased distal delivery of sodium and water is observed, which is associated with increased sodium reabsorption (through the amiloride sensitive epithelial Na^+ channel [ENaC]) which leads to an increase in the electrical gradient across the luminal membrane (increased electronegativity of the lumen) favoring potassium secretion. Diuretics can also stimulate renin-angiotensin-aldosterone cascade through contraction of the extracellular volume. It should be mentioned that only loop diuretics can directly stimulate renin release since they can inhibit Na^+-K^+-$2Cl^-$ cotransporter in the macula densa. Aldosterone plays a paramount role in potassium homeostasis since stimulates potassium secretion through: (a) Increased sodium reabsorption in the distal nephron which favors potassium secretion as previously mentioned, (b) increased luminal potassium permeability, and (c) stimulation of the K^+-Na^+-ATPase in the basolateral membrane resulting in increased intracellular potassium concentration.

Corresponding author:

M. Elisaf, Department of Internal Medicine, Medical School, University of Ioannina, 451 10 Ioannina, Greece
e-mail: melisaf54@gmail.com

Answer: α+c