

One more option to void

NEW(S)

A possible new target for diuretic therapy in patients with fluid overload, the inhibition of the chloride-absorbing transporter pendrin, and the NaCl co-transporter (NCC) may provide an effective diuretic regimen, research suggests.^[1]

(RE)VIEWS

Pendrin is a protein involved in chloride/iodide transport in many cell types – nephrons of the kidney, follicular cells of thyroid and inner ear. Mutations of the gene responsible for pendrin may lead to diseases such as “Pendred syndrome” characterized by deafness, hypothyroidism, and vestibular weakness.^[2]

Pendrin is expressed on the cells of distal convoluted tubule and the collecting duct. It is involved in $\text{Cl}^-/\text{HCO}_3^-$ exchange (mediates HCO_3^- secretion and Cl^- absorption) and has been demonstrated to play an important role in compensatory salt absorption in response to thiazide and loop diuretics. It has been proposed that pharmacological inhibition of pendrin and thiazide-sensitive NaCl co-transporter can provide a novel and strong diuretic regimen in fluid overload conditions such as congestive heart failure, nephrotic syndrome, etc.^[3]

Pendrin production and function are upregulated in conditions with increased circulating aldosterone or angiotensin II (cardiac failure, hypertension). Aldosterone and angiotensin II are thought to modulate the renal regulation of blood pressure, in part, by regulating pendrin-mediated Cl^- absorption and epithelial Na^+ channel (ENaC)-mediated Na^+ absorption.^[4]

Inhibition of pendrin seems yet another option to mobilize edema fluid apart from the existing diuretics.

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