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Revisiting salt and water retention: new diuretics, aquaretics, and natriuretics.

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Abstract

Diuretics continue to be a mainstay in patients with CHF. Conventional diuretic therapy is associated, however, with potentially deleterious neurohumoral activation and renal impairment. It is not known to what extent these neurohumoral effects are offset by concurrent therapy with ACE-I, beta-blockers, and other agents. In the past, there was no alternative to conventional diuretic therapy, so their potential for adverse outcome in the long term could not be assessed. Enhancement of the natriuretic peptide system could provide us with a better strategy to treat sodium and water retention. In a unique way, the natriuretic peptides combine several of the beneficial actions of the other diuretics, but without the associated cost. Natriuretic peptides, like conventional diuretics, are natriuretic and diuretic. There are important differences, however. First, unlike conventional diuretics, NPs do not activate RAAS. Activation of this system is associated with progression of CHF. Second, NPs inhibit the sympathetic nervous system, the activation of which is associated with heart failure progression, myocyte necrosis and apoptosis, and arrhythmias. Third, unlike conventional diuretics that lead to a decrease in GFR by reflex mechanisms. NPs maintain or even improve GFR. We now appreciate that some "old" drugs may be beneficial to CHF patients in a new way, as is the case with spironolactone. The survival benefit of this aldosterone antagonist is clear: its usefulness, however, may be more a result of both its antifibrotic actions in addition to its tradional role as a potassium-sparing and natriuretic agent. It is hoped that the SARAs will provide the same survival benefit, but with fewer of the sex-steroid side effects. In addition, AVP-receptor antagonists may become useful tools in the treatment of patients with hyponatremia. Likewise, the A1 AR antagonists may find a role in the CHF armamentarium by providing good diuresis and natriuresis while at the same time maintaining GFR through inhibition of TGF. Many questions remain unanswered, and studies are needed to demonstrate that the positive results seen in basic research translate into improved morbidity and mortality.

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MeSH Terms:

Aldosterone/metabolism
Aldosterone Antagonists/pharmacology
Aldosterone Antagonists/therapeutic use
Atrial Natriuretic Factor/antagonists & inhibitors
Atrial Natriuretic Factor/metabolism
Diuresis/drug effects*
Diuretics/therapeutic use*
Heart Failure/drug therapy*
Heart Failure/metabolism*
Heart Failure/physiopathology
Humans
Kidney/metabolism*
Kidney/physiopathology
Natriuresis/drug effects*
Natriuretic Peptide, Brain/antagonists & inhibitors
Natriuretic Peptide, Brain/metabolism
Neprilysin/antagonists & inhibitors
Receptors, Purinergic P1/antagonists & inhibitors

Receptors, Vasopressin/antagonists & inhibitors
Sodium Chloride/metabolism*
Water-Electrolyte Imbalance/metabolism*

Substances:

Aldosterone Antagonists
Diuretics
Receptors, Purinergic P1
Receptors, Vasopressin
Natriuretic Peptide, Brain
Aldosterone
Sodium Chloride
Atrial Natriuretic Factor
Neprilysin

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HL36634/HL/NHLBI NIH HHS/United States

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