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The superiority of new algorithm to approach hyponatremia: High prevalence of renal salt wasting without cerebral disease in general medical wards

Hyponatremia is the most common electrolyte abnormality that is undergoing dramatic changes in terms of its diagnostic approach and clinical outcomes. The approach for more than 50 years has been dominated mainly by the assessment of the volume status of the patient and determinations of urine sodium concentrations (UNa) that exceed 30 to 40mEq/L. This approach has been hampered by a universally agreed inability to accurately assess the volume status of patients by usual clinical criteria and perceived rarity of cerebral/renal salt wasting (C/RSW) among internists. Differentiating SIADH from C/RSW has been difficult because of identical clinical parameters that characterize both syndromes. We have accumulated enough data to identify the various causes of hyponatremia by determining fractional excretion of urate (FEurate) to propose an algorithm to approach hyponatremic conditions. In this algorithm, conditions with low FEurate <4% include extrarenal salt losses with normal kidney function, Addison's disease, and edematous states such as heart failure, cirrhosis, and nephrosis. Normal FEurate of 4-11% include psychogenic polydipsia and reset osmostat (RO), and increased FEurate > 11% SIADH/hydrochlorothiazide and C/RSW. SIADH can be differentiated from C/RSW by correcting the hyponatremia by any possible means and observing normalization of FEurate in SIADH as compared to being persistently increased in C/RSW. This algorithm greatly simplifies the diagnosis of patients with Addison's disease and RO. Our report of C/RSW occurring in patients without cerebral disease has led to our proposal to change cerebral to renal salt wasting (RSW). We utilized this algorithm and differences in physiologic response to isotonic saline infusions between SIADH and RSW to evaluate hyponatremic patients from the general medical wards of the hospital. Isotonic saline does not induce excretion of dilute urines or correct the hyponatremia in SIADH as compared to inducing excretion of dilute urines and correction of the hyponatremia in RSW. In 62 hyponatremic patients, 17 (27%) had SIADH, 19 (31%) had RO, 24 (38%) had RSW, 1 due to HCTZ and 1 Addison's disease. Interestingly, 21 of 24 with RSW had no evidence of cerebral disease and 10 of 24 with RSW had UNa <20mEq/L. We conclude that 1. RSW is much more common than is perceived, 2. The term cerebral salt wasting should be changed to RSW 3. RO should be considered a separate entity from SIADH and 4. SIADH needs to be redefined. This new approach proves the ineffectiveness of the volume approach and the limited value of determining UNa, plasma renin, aldosterone or BNP. Lastly, we discuss the evolving identity of a natriuretic factor in RSW and its therapeutic applications.

Biography

John K Maesaka was born in Hawaii, received degrees from Harvard College and Boston University School of Medicine, did his medical residencies at Barnes-Jewish Hospital at Washington University in St. Louis and Mount Sinai Hospital in New York and renal fellowship at Mount Sinai Hospital. His interest-driven decision to spend 5 years exclusively in the renal physiology laboratory at Mount Sinai Hospital as a renal fellow and member of the faculty proved to be the best investment he made to pursue an academic career in medicine. He was involved in developing colorimetric methods for the determination of uric acid and phosphorus in blood and urine that were applied to studying the transport characteristics of both electrolytes by renal micropuncture techniques in rat kidney. He developed several bioassays to demonstrate the presence of a natriuretic factor in the blood of patients with renal salt wasting and Alzheimer's disease and more recently identified the elusive natriuretic factor after more than a 25-year pursuit.

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