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Complexity of differentiating cerebral/renal salt wasting from SIADH: Emerging importance of determining fractional urate excretion

The evolving recommendation to treat virtually all patients with hyponatremia creates an urgency to differentiate SIADH from cerebral/renal salt wasting, (RSW) because of divergent therapeutic goals for both syndromes, to water-restrict in SIADH and administer salt and water in RSW. Contrary to common opinion among internists, RSW is much more common than SIADH based on data that will be presented, especially in neurosurgical patients. Derivation of this perception of the rarity of RSW can be explained in part by overlapping clinical findings and associations between both syndromes and an ineffective approach to evaluating nonedematous hyponatremia, especially our failure to assess accurately the state of extracellular volume. It is generally agreed that one cannot accurately determine whether a patient is euvolemic vs. hypovolemic and this approach, therefore, appears to have little practical value. This lecture will comment on the technical and practical failure of initiating our approach to hyponatremia by determining whether the patient is euvolemic, hypovolemic or hypervolemic. Moreover, the diagnostic and therapeutic dilemma has been further complicated by reports of renal salt wasting occurring in patients without cerebral disease.

There will be a detailed discussion of how the determination of fractional excretion (FE) of urate can be used to differentiate SIADH from RSW when increased and identify patients with reset osmostat (RO) when normal. This lecture will review the complexity of differentiating SIADH from RSW, provide supportive data to change cerebral salt wasting to RSW, eliminate RO as a subtype of SIADH because of normal FEurate and predictable ADH response to hypo-osmolality, and present a new algorithm to evaluate hyponatremia and RSW where determination of FEurate supersedes the value of assessing or determining extracellular volume, urine sodium concentration, or plasma renin, aldosterone and BNP levels.

Biography

Dr. John Maesaka is a graduate of Harvard College and Boston University School of Medicine with medical residencies in internal medicine at Barnes Hospital in St. Louis, MO and Mt. Sinai Medical Center in New York City, and nephrology fellowship at Mt. Sinai Medical Center. He spent 5 years in the micropuncture laboratory at Mt. Sinai, studying uric acid and phosphate transport. He was Chief of the Renal section at the E. Orange VA Medical Center in New Jersey, Chief of Nephrology and Hypertension at the Long Island Jewish Medical Center and presently at Winthrop-University Hospital, which is the clinical center of SUNY Stony Brook Medical School where he is Professor of Medicine. His publications presently focus on urate metabolism, most importantly the determination of fractional urate excretion rates in hyponatremic conditions. He just completed a chapter on renal salt wasting for two books. His description of renal salt wasting in patients without cerebral disease is an impetus to change cerebral salt wasting to renal salt wasting and his report on reset osmostat provides valid data to justify eliminating reset osmostat as a subtype of SIADH. Based on a large body of evidence, Dr. Maesaka will utilize the determination of fractional urate excretion as the pivotal determination in a new algorithm that he will present in the evaluation of nonedematous hyponatremia.

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