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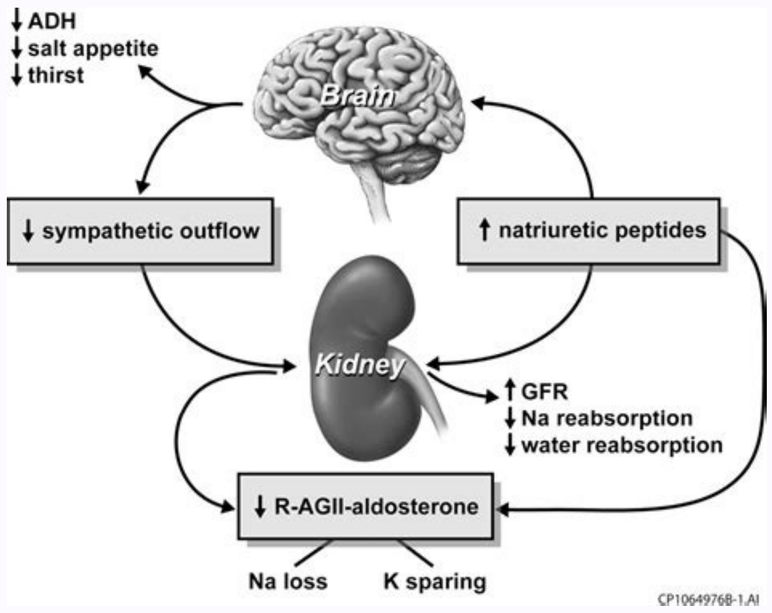
DIAGNOSIS

- Hyponatremia (less than 135 meq/L) with a low plasma osmolality
- An inappropriately elevated urine osmolality (above 100 mosmol/kg and usually above 300 mosmol/kg)
- A urine sodium concentration usually above 40 meq/L
- A low serum uric acid concentration due to urate wasting in the urine

Clinical evidence of hypovolemia is crucial since all of these laboratory findings are also seen in SIADH.



Cerebral Salt Wasting: Pathophysiology, Diagnosis, and Treatment

Alan H. Yee, DO[a,*], Joseph D. Burns, MD[b], Eelco F.M. Wijdicks, MD, PhD[a]

KEYWORDS
• Natriuresis • Natriuretic factors • Hyponatremia • SIADH

Hyponatremia can be a vexing problem for those who care for critically ill neurologic patients. Although seemingly simple at first glance, the accurate diagnosis and effective treatment of hyponatremia can be complex. The chief difficulty in this setting often lies in determining what is driving the fall in serum sodium concentration. Cerebral salt wasting (CSW) is a disorder of sodium and water handling that occurs as a result of cerebral disease in the setting of normal kidney function. It is characterized by hyponatremia in association with hypovolemia and, as the name implies, is caused by natriuresis. In routine clinical practice, distinguishing this condition from the more familiar syndrome of inappropriate secretion of antidiuretic hormone (SIADH) can be quite difficult. Nonetheless, this task is crucial because treatments for the two conditions are fundamentally different. Accordingly, it is important for physicians caring for critically ill neurologic patients to have a thorough understanding of this disorder. This article reviews the pathophysiology of CSW. Building on these basic concepts, a rational approach to its diagnosis and treatment is outlined.

HISTORICAL ASPECTS

Early studies of hyponatremia in patients with cerebral disease published in the 1950s described the presence of polyuria, elevated urinary sodium levels, and dehydration despite the presence of a low serum sodium concentration and adequate fluid intake. This syndrome was termed "cerebral salt wasting." At the time, CSW was suspected to be the major cause of hyponatremia in patients with central nervous system (CNS) injury. Shortly after its original description, however, a syndrome of euvolemic hyponatremia associated with normal urine output and inappropriately high levels of antidiuretic hormone (ADH) was described in a patient with bronchogenic carcinoma.[1] This was later termed as the "syndrome of inappropriate antidiuretic hormone release." Following this discovery and over the subsequent 30 years, hyponatremia that developed in patients with neurologic diseases, such as subarachnoid hemorrhage (SAH), was generally attributed to SIADH.[2–6] Beginning in the 1980s, several key studies[7–9] challenged this concept by demonstrating in patients with aneurysmal SAH a syndrome of low blood volume, natriuresis with a net negative sodium balance, and high urinary output, which was consistent with CSW and not SIADH. These publications led to the modern acceptance of CSW as an important cause of hyponatremia in patients with brain injury and to important research that followed investigating the pathophysiologic disturbances of salt and water homeostasis in patients with neurologic disease.

CLINICAL RELEVANCE

Hyponatremia is frequently encountered in patients with neurologic disease. A recent analysis

[a] Department of Neurology, Mayo Clinic, 200 1st Street SW, Rochester, MN 55905, USA
[b] Department of Neurology, Boston University, School of Medicine, 72 East Concord Street, Neurology C-3, Boston, MA 02118, USA
* Corresponding author.
E-mail address: Yee.Alan@mayo.edu

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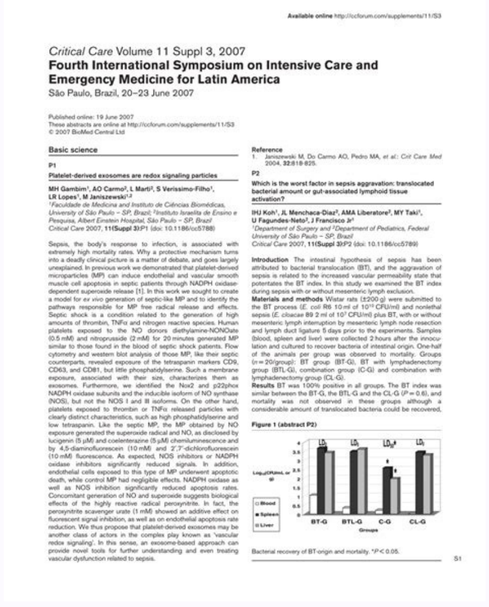
Table 2

	CSW	SIADH
Plasma volume	⇓	⇑ or normal
Salt balance	Negative	Variable
Water balance	Negative	⇑ or normal
Signs and symptoms of dehydration	Present	Absent
Central venous pressure	⇓	⇑ or normal
Serum Osmolality	⇓	⇓
Hematocrit[a]	⇑ or normal	Unchanged
Plasma BUN/creatinine	⇑ or normal	⇓
Urine sodium	⇑ ⇑	⇑
Urine volume	⇑ ⇑	⇓ or normal
Treatment	Normal saline Hypertonic saline Fludrocortisone	Fluid restriction Hypertonic saline Democycline Furosemide

[a] Hematocrit does not differentiate post-operatively.



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Basic science

P1

Platelet-derived exosomes are redox signaling particles

MH Gambim[1], AO Carmo[2], L Marti[2], S Verissimo-Filho[1], LR Lopes[1], M Janiszewski[1,2]

[1]*Faculdade de Medicina and Instituto de Ciências Biomédicas, University of São Paulo – SP, Brazil;* [2]*Instituto Israelita de Ensino e Pesquisa, Albert Einstein Hospital, São Paulo – SP, Brazil*

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Sepsis, the body's response to infection, is associated with extremely high mortality rates. Why a protective mechanism turns into a deadly clinical picture is a matter of debate, and goes largely unexplained. In previous work we demonstrated that platelet-derived microparticles (MP) can induce endothelial and vascular smooth muscle cell apoptosis in septic patients through NADPH oxidase-dependent superoxide release [1]. In this work we sought to create a model for *ex vivo* generation of septic-like MP and to identify the pathways responsible for MP free radical release and effects. Septic shock is a condition related to the generation of high amounts of thrombin, TNFα and nitrogen reactive species. Human platelets exposed to the NO donors diethylamine-NONOate (0.5 mM) and nitroprusside (2 mM) for 20 minutes generated MP similar to those found in the blood of septic shock patients. Flow cytometry and western blot analysis of those MP, like their septic counterparts, revealed exposure of the tetraspanin markers CD9, CD63, and CD81, but little phosphatidylserine. Such a membrane exposure, associated with their size, characterizes them as exosomes. Furthermore, we identified the Nox2 and p22phox NADPH oxidase subunits and the inducible isoform of NO synthase (NOS), but not the NOS I and III isoforms. On the other hand, platelets exposed to thrombin or TNFα released particles with clearly distinct characteristics, such as high phosphatidylserine and low tetraspanin. Like the septic MP, the MP obtained by NO exposure generated the superoxide radical and NO, as disclosed by lucigenin (5 μM) and coelenterazine (5 μM) chemiluminescence and by 4,5-diaminofluorescein (10 mM) and 2',7'-dichlorofluorescein (10 mM) fluorescence. As expected, NOS inhibitors or NADPH oxidase inhibitors significantly reduced signals. In addition, endothelial cells exposed to this type of MP underwent apoptotic death, while control MP had negligible effects. NADPH oxidase as well as NOS inhibition significantly reduced apoptosis rates. Concomitant generation of NO and superoxide suggests biological effects of the highly reactive radical peroxynitrite. In fact, the peroxynitrite scavenger urate (1 mM) showed an additive effect on fluorescent signal inhibition, as well as on endothelial apoptosis rate reduction. We thus propose that platelet-derived exosomes may be another class of actors in the complex play known as 'vascular redox signaling'. In this sense, an exosome-based approach can provide novel tools for further understanding and even treating vascular dysfunction related to sepsis.

Reference

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P2

Which is the worst factor in sepsis aggravation: translocated bacterial amount or gut-associated lymphoid tissue activation?

IHU Koh[1], JL Menchaca-Diaz[2], AMA Liberatore[2], MY Taki[1], U Fagundes-Neto[2], J Francisco Jr[1]

[1]*Department of Surgery and* [2]*Department of Pediatrics, Federal University of São Paulo – SP, Brazil*

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Introduction The intestinal hypothesis of sepsis has been attributed to bacterial translocation (BT), and the aggravation of sepsis is related to the increased vascular permeability state that potentiates the BT index. In this study we examined the BT index during sepsis with or without mesenteric lymph exclusion.

Materials and methods Wistar rats (±200 g) were submitted to the BT process (*E. coli* R6 10-ml of 10^{11} CFU/ml) and nonlethal sepsis (*E. cloacae* B9 2 ml of 10^7 CFU/ml) plus BT, with or without mesenteric lymph interruption by mesenteric lymph node resection and lymph duct ligature 5 days prior to the experiments. Samples (blood, spleen and liver) were collected 2 hours after the inoculation and cultured to recover bacteria of intestinal origin. One-half of the animals per group was observed to mortality. Groups (*n* = 20/group): BT group (BT-G), BT with lymphadenectomy group (BTL-G), combination group (C-G) and combination with lymphadenectomy group (CL-G).

Results BT was 100% positive in all groups. The BT index was similar between the BT-G, the BTL-G and the CL-G ($P = 0.6$), and mortality was not observed in these groups although a considerable amount of translocated bacteria could be recovered.

Figure 1 (abstract P2)

Bacterial recovery of BT-origin and mortality. *P < 0.05.

Cerebral salt wasting syndrome vs siadh. Cerebral salt wasting syndrome symptoms. Cerebral salt wasting syndrome treatment. Cerebral salt wasting physiology. Cerebral salt wasting syndrome diagnosis.

Most of the sodium we consume is in the form of salt, and the large majority of the sodium we consume is in processed and restaurant foods. Your body needs a small amount of sodium to work correctly, but too sodium is bad for your health. Excess sodium can increase its arterial pressure and its risk of cardigal disease and stroke. Together, heart disease and stroke kill American every year. processed food? The sodium is already in processed and restoration foods when buying them, which makes it difficult to reduce the daily sodium intake on its own. Although it is prudent to limit its use of added table salt while cooking and at the table, only a small amount of the sodium that we consume each day comes from the salt whisper. Do you know that sodium and potassium affect arterial pressure? In general, people who reduce sodium, who increase potassium, or who benefit from having less arterial pressure and reduce their risk of other serious health problems. Eating enough potassium can help balance some of the harmful effects that sodium intake can have in the arterial pressure. But lowering sodium intake is key to this balance. The CDC works nationally, state and local to help reduce sodium in the food supply. The CDC approach for sodium reduction includes supporting and evaluating ongo Educate the Pãºblico. Get more information about CDC sodium reduction efforts. In October 2021, the administration Food and medicines of the United States (FDA) announced the orientation to establish voluntary reduction objectives of mesurable mesurable external icon for processed foods, packaged and prepared. FDA orientation establishes a short term sodium dettimda saw ehS .otnelurup odiuqÀl nu ojudorp euq ,rabmul nªÃicnup anu y ,lamron are euq ,laenarc aÃfargomot anu oid el es y olleuc le ne zedigir renet oiv el es ednod ,aicnegreme ed otnematraped la ªÃitimer el es euq ol rop ,aicneicnoc ed levin us ed nªÃiccuder anu ,etnaleda s¡Ãm ,y erbeif ,sotimªÃv ,azebac ed rolod ed oda±Ãapmoca abatse y osnetni s¡Ãm otleuv aÃbah es rolod le ,nªÃisimda al ed setna sa-Ãd sonU .sem nu etnarud odreiuqzi odÃo le ne nªÃicarupus y rolod ªÃteserp etneicap lE .nªÃisimda al ed s©Ãupsed sesem 3 odreiuqzi odÃo le ne avitidua adidr©Ãp noc ,sitito ed socinªÃrc soidem sol a odibed aitsalponapmopit noc adarrec aÃmotcediotsam al arap adarepo so±Ãa 46 ed rejum anu are etneicap lE .setneiugisbus airuilop y aimeartanopih noc otnematraped ortseun ne oditimda ococomuen rop sitigninem noc etneicap nu ed osac le somatneserP .3871â5771:)91(531;7102 .nªÃicalucriC .onretxe onoci sacif¡Ãrgoeg senoiger 3 ed sesnedinuodatse sotluda ne oidos ed setneuF .la te ,MC airoL ,C eipselliG ,DC rendraG ,MJ ynakihS ,EM llewsgoC ,JL kcanraH).3(06;1102 .peR tatS latiV taN .9002 rof atad lanif :shtaeD .CH gnuK ,MA o±ÃiniM ,LS yhpruM ,QJ uX ,DK kenahcoK .s¡Ãm y ,oidos ed omusnoc le ricuder arap recah edeup ©Ãuq ,etnatropmi se oidos ed atsegni al ricuder ©Ãuq rop ,las y oidos ertne aicnerefid al erbos adnerpA .oidos erbos setneucerf satnugerp a satseupser aeL Q muidoS .nªÃisnetrepih e larberec emarred ,acaÃdrac dademrefne ed ogseir ronem nu a ragul ¡Ãrad y)s¡Ãm y so±Ãa 41 solleuqa arap(aÃd la gm 003.2 ed oidos ed etimÃl sadadnemocer sesnedinuodatse sol arap sacit©Ãteid secirtceriD sal raznacla ed acrec s¡Ãm ocilbºÃp la avell aÃug atsE .so±Ãa 5,2 somixªÃrp sol etnarud ,aÃd la gm 000.3 a)gm(somargilim 004.3 ed oidos ed aidem atsegni al riunimsid otejbo rop neneit oidos led nªÃiccuder ed sovitejbo soL .sotnemila ed saÃrogetac 061 ed rodederla arap ,soiratnemila soicivres ed sotneimicelbatse y setnaruatser ,sotnemila ed setnacirbaf sol arap sovitejbo the intensive care unit with a diagnosis of acute bacterial meningitis with a likely focus in the ear. ear. The treatment began with cefotaxima, vancomycin and corticosteroids. A few hours after admission, it required mechanical ventilation due to a decreased level of consciousness and an increase in respiratory work. Streptococcus pneumoniae was an isolated in the cultivation of the libochide and, after identifying the antibiogram (intermediate sensitivity to the cefotaxima), the dose of cefotaxima was increased and rifampicin was added. It was satisfactorily extubated after 2 days, although it still presented a confused state. Three days after, he began with polyuria (> 5l/dãa) and progressive hyponatremia of up to 130 meq/l, high natriuresis (148meq/l) with high urinary osmolality also high (277mosm/kg), while maintaining a good function renal. Then, the suspicion fell on a cerebral salt disposal syndrome (CSW), with additional hypouricism evidence (1.2 mg/dl), high levels of pycid pycid Tica in a normal range (4.2ã®â¼g // l). The proper restoration of the volume with saline solution in the light of urinary sodium and the water pages allowed its condition to be controlled, and the nature, the diuresis and the neurological state returned to normal. Hydration is one of the most common electrolytic disorders, both in the usual closing practice and associated with the diseases of the central nervous system (CNS) .1,2 The causes of hyponatremia are numerous, but most of them They are symptoms of a reduction in plasmatic osmolality (SIADH), with water restriction that form the basis for treatment. However, especially in cases of intracranial pathology, hyponatremia may also be due to a different mechanism, with a sodium pédid Water intake is contraindicated. of inappropriate natriuresis for levels of ,etnemacipÃT ,etnemacipÃT 3.nemulov y oidos ed etnalucric The beginning of hyponatremia due to CSW occurs in the first 10 days after the neurological or neurosuruãºral event. Therefore, in the context of a disease in the CNS, the CSW is diagnosed in a patient with clinical evidence of hypovolemia and with the following findings4: the clinical evidence of hypovolemia is essential since these same findings can be found in SIADH4 (Table 1). Copyright author's information and DeclaimarCapyright license information It is associated with significant morbidity and mortality, especially if the underlying cause is incorrectly diagnosed and is not properly treated. Often, the hospitalist faces a clinical dilemma when a patient has hyponatremia of an unclear etiology with an uncertain volume state. The inappropriate antidiuride hormone syndrome (SIADH) is frequently diagnosed in this clinical context, but the waste of brain salt (CSW) is an important diagnostic to consider. A better understanding of differential diagnosis for hyponatremia. Conclusion: CSW is â € â € ‹A process of exhausting extracellular volume due to a tubular defect in sodium transport. Two postulated mechanisms for CSW are the excess secretion of natural nature and the synthetic stimulation ptidos to the ri ± ín. Making the distinction between CSW and Siadh is important because the treatment for the two conditions is very different. A man, 43 years old, is evaluated for hyponatremia. He was admitted to the hospital six days before for intracranial hemorrhage that required emergency evacuation. The patient was extubated later when his neurological state better. His medical history includes poorly controlled hypertension and rheumatoid arthritis. to be 121 mEq/L. He has been receiving 0.9% normal saline since admission, and his sodium level has been gradually decreasing from 141mEq/L, the level at admission. A hyponatremia workup reveals a low serum osmolarity of 256 mOsm/kg, a normal serum creatinine level of 0.6 mg/dL, a urine sodium level of 89 mmol/L, and an inappropriately high urine osmolarity of 588 mOsm/kg. His urinalysis reveals a specific gravity of 1.030, with no blood or protein. His levels of serum thyroid-stimulating hormone and random cortisol are within normal limits. His brain natriuretic peptide (BNP) and fractional excretion of uric acid are elevated, at 686 pg/mL and 83.8%, respectively.The patient appears euvolemic, with a blood pressure of 115/70 mm Hg and pulse of 90 beats per minute. His intake and outtake documentation reveals that his urine output has been 2 to 4 L/d despite a fluid restriction of

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