Cerebral salt wasting

Cerebral salt wasting (CSW): renal loss of sodium as a result of intracranial disease, producing hyponatremia and a decrease in extracellular fluid volume.

CAUTION: CSW after aneurysmal SAH may mimic SIADH; however, there is usually also hypovolemia in CSW. In this setting, a fluid restriction may exacerbate vasospasm induced ischemia.

The mechanism whereby the kidneys fail to conserve sodium in CSW is not known and may be either a result of a natriuretic factor or direct neural control mechanisms.

Laboratory tests (serum and urinary electrolytes and osmolalities) may be identical with SIADH and CSW.

Furthermore, hypovolemia in CSW may stimulate ADH release. To differentiate: CVP, PCWP, and plasma volume (a nuclear medicine study) are low in hypovolemia (i.e., CSW).

An elevated serum [K+] with hyponatremia is incompatible with the diagnosis of SIADH.

Cerebral salt wasting syndrome (CSW-cerebral salt wasting) was first described in 1950 by Peters.

Renal loss of sodium as a result of intracranial disease, producing hypo- natremia and a decrease in extracellular fluid volume.

Patients present with excessive natriuresis and hyponatremic dehydration.

Inappropiate natriuresis with volume depletion.

Etiology

Etiology of 6 % of cases of hyponatremia following aneurysmal subarachnoid hemorrhage [1].

This syndrome can occur in patients who have sustained damage to the central nervous system (e.g. patients with subarachnoid bleeding, bacterial meningitis or after neurosurgery).

Cerebral salt wasting (CSW) has been suggested to precede the development of symptomatic vasospasm.

Patients with aneurysmal SAH may have CSW with hyponatremia which mimics SIADH, however there is usually also hypovolemia in CSW. In this setting, fluid restriction may exacerbate vasospasm induced ischemia [2) 3) 4) 5)].

The mechanism whereby the kidneys fail to conserve sodium in CSW is not known, and may be either a result of an as yet unidentified natriuretic factor or direct neural control mechanisms.

Diagnosis

Cerebral salt wasting diagnosis.

Treatment

Cerebral Salt Wasting Treatment.

1)
Sherlock M, O'Sullivan E, Agha A, Behan LA, Rawluk D, Brennan P, Tormey W, Thompson CJ. The incidence and pathophysiology of hyponatraemia after subarachnoid haemorrhage. Clin Endocrinol (Oxf). 2006 Mar;64(3):250-4. PubMed PMID: 16487432.
2)
Harrigan MR. Cerebral Salt Wasting Syndrome: A Review. Neurosurgery. 1996; 38:152–160
3)
Maroon JC, Nelson PB. Hypovolemia in Patients with Subarachnoid Hemorrhage: Therapeutic Implications. Neurosurgery. 1979; 4:223–226
4)
Wijdicks EFM, Vermeulen M, Hijdra A, et al. Hypo- natremia and Cerebral Infarction in Patients with Ruptured Intracranial Aneurysms: Is Fluid Restriction Harmful? Ann Neurol. 1985; 17:137–140
5)
Wijdicks EFM, Vermeulen M, ten Haaf JA, et al. Volume Depletion and Natriuresis in Patients with a Ruptured Intracranial Aneurysm. Ann Neurol. 1985; 18:211–216