Hyponatremia after aneurysmal subarachnoid hemorrhage

Epidemiology

Hyponatremia after subarachnoid hemorrhage (SAH) has prevalence rates of approximately 30-55%.[1][2][3].

Hyponatremia is the most common electrolyte abnormality in patients with aneurysmal subarachnoid hemorrhage (aSAH), occurring in one-third of aneurysmal subarachnoid hemorrhage (aSAH) patients. This is secondary to Syndrome of inappropriate antidiuretic hormone secretion (SIADH) or Cerebral salt wasting syndrome (CSW).

Distinguishing between these two entities can be difficult because they have similar manifestations, including hyponatremia, serum hypoosmolality, and high urine osmolality. SIADH is much more common than CSW in patients with aSAH. Anterior circulation aneurysms may be associated with a higher rate of SIADH than posterior circulation aneurysms.[4]

Animal data and human cross-sectional studies have established that chronic hyponatraemia predisposes to osteoporosis; the effects of acute hyponatraemia on bone turnover have not been determined. Our objective was to test the hypothesis that acute hyponatraemia leads to dynamic effects on bone turnover.

Bone turnover markers [C-terminal crosslinking telopeptide of type 1 collagen (CTX-1), N-propeptide of type 1 collagen (P1NP) and osteocalcin] were measured prospectively over one week in 22 eunatraemic patients with subarachnoid haemorrhage. Patients treated with glucocorticoids were excluded.

Eight patients developed acute hyponatraemia, median nadir plasma sodium concentration 131 mmol/L (IQR 128-132), and 14 remained eunatraemic, nadir plasma sodium concentration 136 mmol/L (IQR 133-137). Significant main effects of hyponatraemia were found for P1NP (p=0.02) and P1NP:CTX-1 ratio (p=0.02), both fell in patients with acute hyponatraemia, with significant interaction between hyponatraemia and time from baseline for P1NP (p=0.02). Significant main effects of time from baseline (p<0.001) but not hyponatraemia (p=0.07) were found for osteocalcin. For CTX-1, significant main effects of time from baseline (p=0.001) but not hyponatraemia (p=0.65) were found. There was a positive correlation between change in P1NP:CTX-1 ratio and nadir plasma sodium concentration, r=+0.43, p=0.04. Median serum cortisol (measured on day 1, 3 and 7) was higher in the hyponatraemia group than in those who remained eunatraemic, 545 nmol/L (IQR 373-778) versus 444 nmol/L (IQR 379-542) p=0.03.

These data suggest that acute mild hyponatraemia is associated with a reduction in bone formation activity[5].
Treatment

Hyponatremia after aneurysmal subarachnoid hemorrhage treatment.

Outcome

Hyponatremia is significantly associated with poor outcome in patients with SAH. Anticipate hyponatremia in patients with aneurysmal subarachnoid hemorrhage, timely detect and appropriately treat it to improve outcome. It is more common in patients who are more than 50 years old and whose aneurysm is in the anterior communicating artery. Our comprehensive monitoring ensured early detection and efficient surgical and nursing management reduced morbidity and mortality.

References