Cerebrospinal fluid acid-base equilibrium of spontaneously breathing patients with subarachnoid hemorrhage developing hypocapnic alkalosis

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Introduction. The pathogenesis of hypocapnic alkalosis developing in patients with spontaneous subarachnoid hemorrhage (SAH) has not been fully elucidated.

Aim of the present study was to assess the incidence and investigate the pathophysiologic mechanisms of hypocapnic alkalosis developing in spontaneously breathing patients with SAH.

Methods. Spontaneously breathing patients with SAH admitted to the Neuro-intensive care unit were enrolled. A sample of cerebrospinal fluid (CSF) was collected anaerobically from the external ventricular drain simultaneously with an arterial blood sample. Electrolytes, albumin, phosphates, partial pressure of CO_2 (PCO_2) and pH were measured for both samples. The Strong Ion Difference (SID) and total concentration of weak, non-volatile acids (A_{TOT}) were calculated using standard formulae. Furthermore, ΔPCO_2 was calculated as the difference between CSF and arterial PCO_2. Hypocapnic alkalosis was defined as arterial PCO_2<35 mmHg and pH>7.45 and the population was divided according to the presence or absence of hypocapnic alkalosis. T-test or Rank Sum test were used, as appropriate, for comparison between groups.

Results. Eighteen patients with SAH (59±9 years, Fisher 4 [3-4]) were enrolled. Seven (39%) of these patients fulfilled the "hypocapnic alkalosis" criteria. Patients developing hypocapnic alkalosis had significantly lower CSF SID as compared to the other patients (Table 1). While CSF PCO_2 and pH did not differ, ΔpCO_2 was significantly lower in patients not developing hypocapnic alkalosis.

Conclusions. Patients with SAH developing hypocapnic alkalosis had a lower CSF SID. Interestingly, CSF PCO_2 did not differ between groups, while arterial PCO_2 did. The decreased ΔPCO_2 observed in SAH patients who did not develop hypocapnic alkalosis could be explained by a reduced cerebral metabolism, resulting in lower CO_2 production.

References

1. Froman C et al.***Lancet*** 1966; 1(7441):780-2