SPONTANEOUS HYPERVENTILATION AFTER TRAUMATIC BRAIN INJURY: A CASE REPORT

Perini G.[1] ; Pacini C.[1] ; Antolini R.[1] ; Santoni F.[1] ; Vitali E.[1] ; Salvucci Salice A.[1] ; Pesaresi L.[1] ; Carsetti A.[1-2] ; Donati A.[1-2]
1)Department of Biomedical Sciences and Public Health, Università Politecnica delle Marche, Ancona, Italy
2) Clinica di Anestesia e Rianimazione Generale, Respiratoria e del Trauma Maggiore, AOU delle Marche, Ancona, Italy

BACKGROUND

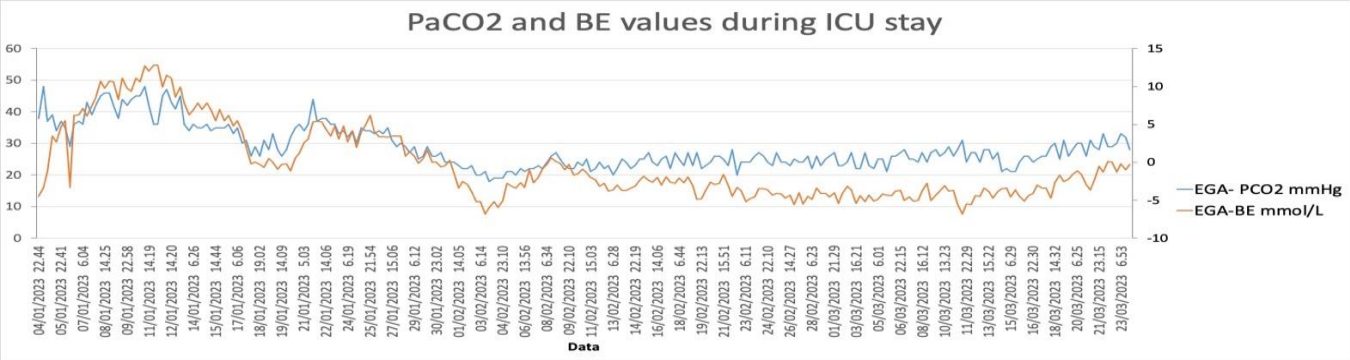
Spontaneous hyperventilation in patients with traumatic brain injury is a common occurrence that should not be underestimated. In patients who are spontaneously breathing or receiving assisted ventilation, it may be due to a compensatory mechanism for a state of cerebrospinal fluid acidosis in the immediate post-acute phase[1].

CASE REPORT

A 64-year-old patient was admitted to our ICU with a GCS score 3 with a head injury resulting in bilateral hemorrhagic spot.
In the acute phase, the patient was mechanically ventilated in controlled mode, in order to maintain a P_aCO_2 of around 38 mmHg. An MRI performed one week later showed the appearance of cerebral and cerebellar ischemic areas. In the post-acute phase, the patient was gradually weaned off the ventilator and transitioned to spontaneous breathing via tracheostomy.
From this point onwards, there was an increasingly marked hypocapnia due to spontaneous hyperventilation, leading to respiratory alkalosis with attempted renal compensation.
We can exclude a renal origin for the alteration of blood gas parameters since during the initial phase, we did not observe any organ dysfunction or pH values indicating acidosis due to a primary loss of bases.

DISCUSSION

In the absence of hypoxia, other causes of metabolic acidosis or brainstem lesions that alter the central generator of the respiratory pattern, we believe that the hyperventilation developed by our patient is associated with a compensatory mechanism for cerebrospinal fluid acidosis due to the brain injury. Unlike the data reported in the literature, where compensation of cerebrospinal fluid acidosis occurs within 7-10 days[2], in our patient, it took about 60 days.
Furthermore, despite the persistence of PaCO2 values <30 mmHg for more than 50 days, we did not observe any deterioration of the cerebral blood flow with worsening of ischemic areas.



References: 1- Froman C. et al Lancet 1966, 1(7441):780-2; 2- Raichle ME, Eur Neurol 1971-1972; 6:1-5