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Episodic Respiratory Failure Due to Focal Epileptic Activity

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Abstract
We report on a patient with a complicated course after surgical abdominal intervention and episodic life threatening respiratory failures successfully treated with carbamazepine after diagnosis of a ponto-medullary lesion in the MRI.

Key words: Carbamazepine, Pontine lesion, Respiratory failure

Introduction

A serious problem after weaning and extubation on a surgical intensive care unit (ICU) is the need for reintubation due to often unexpected respiratory failure with consequently prolonged invasive respiratory therapy. Demling (1988) found a 4 % rate of reintubation in a retrospective study of 400 patients without predisposing factors. Insufficient respiratory drive by the central nervous system is mostly due to anaesthesia or drug overdose, metabolic or inflammatory encephalopathies or lesions of the central nervous system due to stroke (for overview see Glauser et.al. 1987)).

Case Report

A 41-year-old man presented to the emergency room with severe abdominal pain of acute onset, which had started the day before. An appendectomy due to an acute appendicitis had been performed without complications when he was twelve years old. One year after this operation he had undergone re-operation for an adhesion ileus. History revealed that he was under medication for a schizoaffective disorder. Long-time chronic alcohol intake was discontinued a year before current admission.

The patient was explored for a recurrence of an adhesion ileus and a fibrotic subtotal stenosis of the ileum of 42 cm length was found and resected. On the 7th postoperative day the patient was reexplored for an anastomosis leakage and generalized peritonitis. The anastomosis was resected and a terminal ileostomy was implanted. Reoperations had to be performed on days 8 (resection of right hemicolon, lavage), 17 (tracheotomy, lavage), 21 (lavage), 37 (wound adaptation), 68 (wound adaptation) and 73 (wound closure). Between days 8 and 33 hemofiltration had to be done due to anuria. The patient needed mechanical ventilation (Bilevel Positive Airway Pressure [BIPAP], Evita 4, Draeger, Lübeck, Germany) from the day of the first relaparotomy (day 7) until day 49, when the respiration assistance could be changed to Continuous Positive Airway Pressure (CPAP). On day 76 the respirator had to be changed to BIPAP again for 7 hours due to an increase of pCO_2. At this time the patient could communicate and cognitive functions were normal. No paresis or brain stem dysfunctions was found in the neurological examination. Spontaneous breathing assisted by CPAP had been continued unto day 80, when no more mechanical assistance was needed. On day 89 respiratory failure occurred again and the ventilation had to be assisted by BIPAP for 8 hours. The blood gas analysis revealed normal values and the patient recovered completely. The next episode of respiratory failure occurred on day 99, and assistance by CPAP for 5 hours was sufficient. On day 102 the patient had to be reintubated due to another respiratory failure. No paresis of the diaphragm was seen.

The MRI of the brain stem (T2, FLAIR) revealed a discrete but considerable signal enhancement in the pons (Fig. 1A,B). We decided to start a medication with carbamazepine. No further episodes of respiratory failure occurred since then with a follow-up of 24 months. A control MRI 9 months later displayed no lesion anymore (Fig. 1C,D).

Discussion

This is an unusual case with repetitive episodic respiratory failure after complicated abdominal surgical treatment and successful treatment by carbamazepine.

In mammals, the critical sites necessary for respiratory rhythmogenesis sit near the ponto-medullary border, in the parafascial region and in a more caudal region, the preBötzinger complex, just below the compact region of the nucleus ambiguus (Milsom et al., 2004). This archaic central rhythmogenesis is mostly impaired by drugs or by central nervous system lesions, metabolic or inflammatory disorders. The MRI signal enhancement in this patient in the ponto-medullary region provides evidence for focal central nervous system lesion. Clinically these lesions are attributed to brain stem neurological dysfunction and are not responsible for the respiratory failures. Thus, we had to postulate further lesions in the parafascial region or the preBötzinger complex which were not

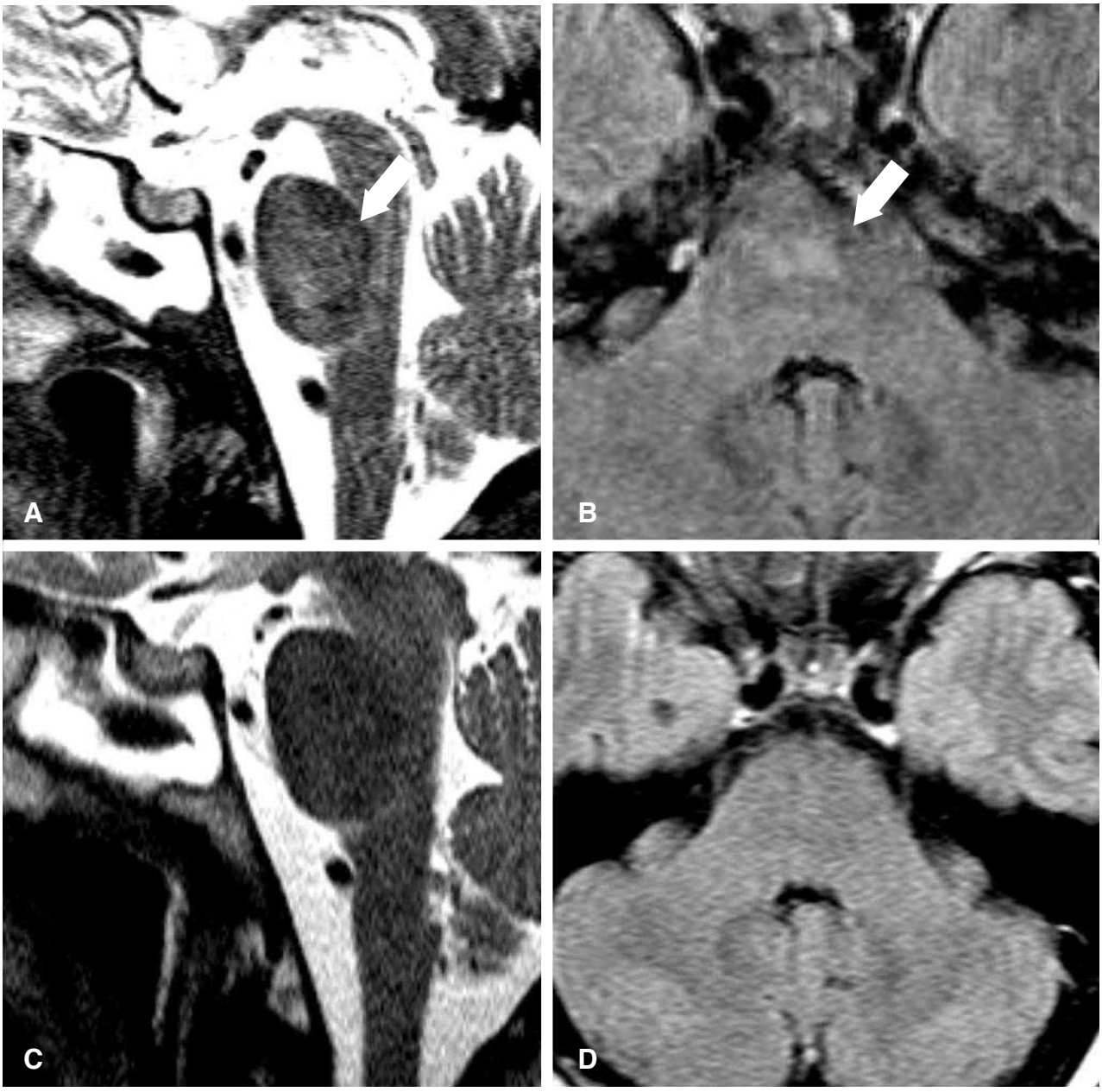

Fig. 1. Signal enhancement (see arrow) in the medullo-pontine region. A: transversal reconstruction, B: sagittal reconstruction before treatment. C,D: on control MRI nine months later the pathological enhancement has resolved.

detected by the MRI. The fact that in control MRI the lesions disappeared argue for unspecific focal oedema due to inflammation or pontine myelinosis (blood sodium was always normal).

Recurrent respiratory failures are known from patients with central sleep apnea or from children with congenital central hypoventilation syndrome (CCHS, Ondine's curse). In these cases the respiration efforts could be critically low in sleeping phases or drowsiness leading to hypoxia and hypercapnia (Abad and Guilleminault, 2004). There are only few cases reported for adults presenting with brain stem lesions (Gupta et.al., 2003). In our case we found no association with sleep. The measured hypercapnic values during the episode may argue for an episodic hyposensitivity to CO_2 but the interictal pCO_2 values were normal.

One probable mechanism of sudden unexplained death in epilepsy is the marked central suppression of respiratory activity after seizures (Abad and Guilleminault, 2004). In this patient toxic side effects (former alcoholic disease, use of neuroleptic drugs before surgery) and additional focal lesions in the brain stem may have generated an epileptic focus. After application of the antiepileptic drug carbamazepine, the episodes stopped promptly providing some evidence for the epileptic hypothesis. We have continued this medication for over one year due to ethical reasons for not to prompt another episode of potentially life threatening respiratory failure.

Although the precise pathophysiology remains unclear, this case highlights the importance to rule out an epileptic focus in patients with recurrent respiratory

failures especially in intensive care patients. In case the level of suspicion for this pathology is high an antiepileptic probatory medication is justified.

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