Radiology Case Reports 13 (2018) 241–243

Available online at www.sciencedirect.com

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RCR RADIOLOGY CASE REPORTS

Neuroradiology

Tension pneumocephalus mimicking septic shock: a case report

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ARTICLE INFO

Article history:
Received 12 September 2017
Received in revised form 4 October 2017
Accepted 7 October 2017
Available online 7 November 2017

Keywords:
Tension pneumocephalus
Systemic inflammatory response syndrome

ABSTRACT

Tension pneumocephalus can lead to rapid neurologic deterioration. We report for the first time its association with aseptic systemic inflammatory response syndrome mimicking septic shock and the efficacy of prompt neurosurgical intervention and critical care support in treating this condition. A 64-year-old man underwent 2-stage olfactory groove meningioma resection. The patient developed altered mental status and gait instability on postoperative day 6. Imaging showed significant pneumocephalus. The patient subsequently developed worsening mental status, respiratory failure, and profound shock requiring multiple vasopressors. Bedside needle decompression, identification and repair of the cranial fossa defect, and critical care support led to improved mental status and reversal of shock and multiorgan dysfunction. Thorough evaluation revealed no evidence of an underlying infection. In this case, tension pneumocephalus incited an aseptic systemic inflammatory response syndrome mimicking septic shock. Prompt neurosurgical correction of pneumocephalus and critical care support not only improved neurologic status, but also reversed shock. Such a complication indicates the importance of close monitoring of patients with progressive pneumocephalus.

Introduction

Pneumocephalus, an accumulation of intracranial air because of communication between intracranial and extracranial compartments, may result from trauma, surgery, tumor, or chronic infection [1]. Tension pneumocephalus occurs when the accumulated air exerts mass effect on the adjacent parenchyma, leading to rapid neurologic deterioration. Although tension pneumocephalus has been associated with cerebrospinal fluid (CSF) pleocytosis [2], a review of the literature did not reveal reports of pneumocephalus eliciting an aseptic systemic

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https://doi.org/10.1016/j.radcr.2017.10.008

inflammatory response syndrome (SIRS). Here, we describe a case of postoperative pneumocephalus that mimicked severe septic shock, and discuss the efficacy of basic resuscitative measures, critical care support, and neurosurgical intervention employed to treat this condition.

Case report

A 64-year-old man with a history of obesity and recurrent olfactory groove meningioma (Fig. 1A) causing left-sided optic neuropathy but no other neurologic deficits underwent a 2-stage surgery to remove the tumor from both above and below, consisting of a bifrontal craniotomy and an endoscopic resection the following day. The patient received intra- and postoperative empiric antibiotics and steroids. Immediately following his second surgery, a non-contrast computed tomography (NCCT) scan of the head showed expected postoperative changes including pneumocephalus (Fig. 1B-C). On postoperative day (POD) 5, physical examination revealed a new palpable collection below the scalp. Repeat imaging demonstrated worsening pneumocephalus and a new large scalp collection. Nevertheless, the patient remained at his preoperative neurologic baseline.

On POD 6, the patient experienced disorientation and unsteady gait. NCCT on POD 7 demonstrated worsening of both the intracranial and the extracranial air below the scalp (Fig. 1D). Shortly thereafter, the patient's mental status and respiratory status acutely declined. Although he remained afebrile, he developed bradycardia (heart rate 40s), tachypnea (respiratory rate 30s), and hypoxia (SpO_2 70%). Neurologic examination was notable for disorientation and restlessness with minimal verbal output. Arterial blood gas testing on 5 L of oxygen via nasal cannula revealed hypoxic respiratory failure (pH 7.34, $PaCO_2$ 43, PaO_2 50, HCO_3 23). The patient underwent emergent endotracheal intubation. Given concern for tension pneumocephalus, he underwent emergent bedside needle decompression of the craniotomy site and was taken immediately to the operating room for endoscopic exploration and repair of the anterior cranial fossa defect.

On transfer to the intensive care unit, the patient developed profound hypotension requiring multiple vasopressors. His chest radiograph demonstrated diffuse pulmonary edema in the setting of an unremarkable transthoracic echocardiogram, raising concern for acute respiratory distress syndrome. Laboratory studies were notable for a white blood cell count of 44, erythrocyte sedimentation rate of 49 mm/h, C-reactive protein of 11.7 mg/dL, procalcitonin of 1.17 ng/mL, platelet count of 521, lactate of 2.25 mmol/L, and creatinine of 2.3 mg/dL. Given this clinical picture, the patient was empirically treated with broad-spectrum antibiotics, but cultures of samples from blood, urine, and bronchoalveolar lavage did not grow any pathogenic organisms.

NCCT on POD 9 indicated improved pneumocephalus (Fig. 1E). Over the course of the next few days, the patient's laboratory parameters normalized, his kidney function improved, the vasopressors were weaned off, and he was extubated on POD 11. CSF sampling on POD 17 revealed a white blood cell count of 25, glucose of 111 mg/dL, protein of 194 mg/dL, and lactic acid of 4.1 mmol/L. All cultures, including CSF, remained negative. The patient improved back to his preoperative neurologic baseline. On POD 54, a repeat NCCT demonstrated near-complete resolution of the pneumocephalus.

Discussion

We report a case of severe tension pneumocephalus resulting in multiorgan dysfunction. We postulate that the patient's tension pneumocephalus triggered a physiological stress response that caused a systemic inflammatory process and

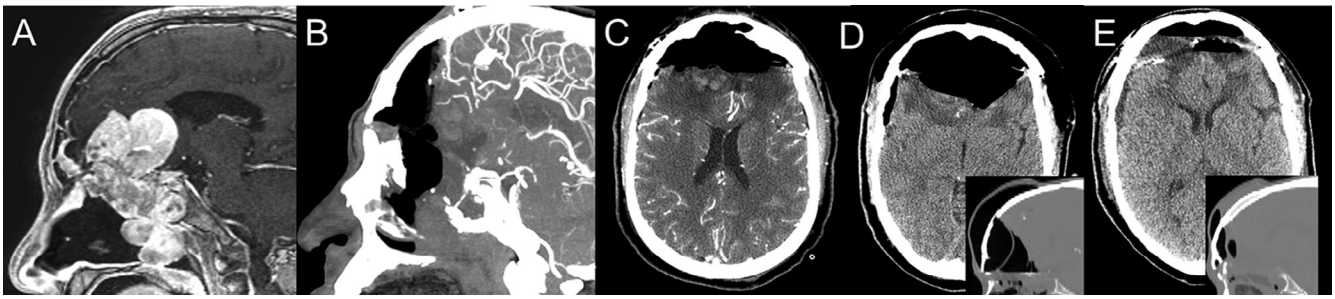

Fig. 1 – Preoperative sagittal post contrast T1-weighted magnetic resonance imaging (MRI) (A) demonstrates a large enhancing meningioma centered on the cribriform plate, extending into the anterior cranial fossa and sinonasal cavity. There is evidence of prior sinonasal surgery performed for prior resection of this recurrent mass. Immediate postoperative sagittal and axial computed tomography (CT) angiogram reconstructions (B, C) show a large amount of extra-axial pneumocephalus anterior to the frontal lobes. Seven days following resection, the axial non-contrast computed tomography (NCCT) with a lower right sagittal reformat inset (D) shows a marked increase in the degree of pneumocephalus. There is increasing mass effect and posterior displacement of the frontal lobes, compatible with a tension pneumocephalus. After needle decompression and endoscopic repair of the anterior cranial fossa defect, a follow-up NCCT with a lower right sagittal reformat inset (E) demonstrates significant interval improvement in the pneumocephalus with resolution of frontal lobe compression.

end-organ injury. This process might be mediated by a catecholamine surge or increased intracranial pressure as seen in severe trauma cases, although the exact mechanisms remain unclear [3,4]. Alternatively, mechanisms of central dysregulation might be at play, as has been postulated in SIRS associated with subarachnoid hemorrhage [5].

Emergent decompression and surgical repair of the cranial fossa defect, along with critical care support, led to reversal of the patient's tension pneumocephalus and associated SIRS response. Infection, the most typical etiology of SIRS [6], seems unlikely in this patient given the abrupt clinical improvement as well as the absence of any apparent infectious source. The postoperative state can also theoretically lead to a SIRS-like response, which is an important consideration in this case given that the patient underwent 2 separate operations. However, the length of time between the initial procedure and his subsequent multi-organ dysfunction makes this less likely. Given the temporal profile of events, the tension pneumocephalus seems the much more likely culprit of the patient's decline.

Our reported findings may be of value to clinicians who provide postoperative care for neurosurgical patients because they indicate the need for close observation and timely neurosurgical intervention, as well as the importance of supportive care, for patients with progressive pneumocephalus.

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