rather than cytotoxic edema [3]. In contrast, another theory hypothesizes that at elevated blood pressures or from endothelial toxins, the autoregulatory system overcompensates, resulting in decreased blood flow, ultimately resulting in ischemia and therefore cytotoxic edema [5].

Though cerebellar and brainstem white matter hyperintensities have been reported, these have been largely asymptomatic features of the disease [6]. A recent review of patients with hypertensive encephalopathy involving the brainstem found that correlated clinical symptoms were present in less than 25% of patients, suggesting a 'clinical radiologic dissociation' [7]. Of the reports in which symptoms were detailed, only eight prior cases describe symptomatic brainstem dysfunction from PRES. These cases are summarized in table 1. Cases of obstructive hydrocephalus and resulting symptoms have also been reported [8]. In our patient, the prominent dysarthria and dysphagia suggesting lower motor neuron involvement could be correlated more directly to the pontine edema.

The differential diagnosis of brainstem encephalopathy with associated MRI T₂-weighted hyperintensities is broad and includes central pontine myelinolysis, autoimmune diseases (systemic lupus erythematosus, Behçet's disease, polyarteritis nodosa), multiple sclerosis, infectious/postinfectious conditions (acute disseminated encephalomyelitis, Bickerstaff's encephalitis, *Listeria* rhombencephalitis, progressive multifocal leukoencephalopathy), neoplastic disorders (lymphoma and glioma), and vascular insults (subacute infarction) [9].

In our case, there was no evidence of metabolic derangements to suggest central pontine myelinolysis, and gadolinium MRI failed to show acute inflammatory changes or neoplasm. The patient's clinical history and laboratory values also helped exclude other diagnoses. Initial DWI sequences did not reveal acute infarction, but could not rule out the possibility of subacute infarction. However, the reversible T₂-weighted hyperintensities on MRI argued against infarction and supported the diagnosis of PRES.

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Fatal Venous Cerebral Air Embolism Secondary to a Disconnected Central Venous Catheter

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Introduction

Venous air embolism is a well-known complication of trauma, central venous (CV) catheterization, pressurized intravenous infusion systems and orthopedic, neurosurgical or cardiovascular surgical procedures [1]. Clinical presentation is mostly dominated by right ventricular dysfunction and pulmonary injury. Systemic presentation and arterial cerebral air embolism can be the result of paradoxical embolism through an intracardiac or intrapulmonary right-to-left shunt [1–4]. We present a fatal case with extensive venous cerebral air embolism due to an accidentally disconnected CV catheter. Diagnosis was confirmed by brain computed tomography (CT) and anatomo-histological examination.

Case Report

A 79-year-old male known with chronic obstructive pulmonary disease was admitted to the hospital because of bronchopneumonia. Antibiotics were administered intravenously via a CV catheter (type Arrow, latex-free, two-lumen, French 7) located in the left subclavian vein. The patient's condition improved gradually. On day 7 of infusion, he shortly lost consciousness while shaving. Because of the excellent recovery, absence of a focal neurologic deficit, normal vital parameters and normal findings for ECG and glyce-

mia, a reflex syncope due to carotid stimulation by shaving was suspected. At that moment, it was noted that the CV catheter was bent at the site of insertion and partially disconnected. Since infusion was still possible, no further attention was paid. The morning after, the patient was found in a comatous state. Brain CT revealed extensive air collections in the venous structures of the neck and brain (fig. 1). The patient deceased shortly after imaging. On autopsy, there was no patent foramen ovale, pulmonary vascular mal-

formation or free air in the thoracic, subdural or subarachnoid space. Cranial entry sites for air could not be identified. Macroscopic examination of the brain showed wedge-shaped cortical ischemic changes in the right precentral gyrus and right occipital lobe (fig. 2). On microscopy, these localizations showed vascular congestion and cavities with variable diameter without inflammatory reaction, compatible with air collections. Cultures for aerobic and anaerobic microorganisms were negative.

Discussion

Insertion, accidental disconnection or removal of a CV catheter may cause cerebral air embolism, which occurs in the arterial vascular bed as a result of paradoxical embolism through a intracardiac or intrapulmonary right-to-left shunt [2–8]. Venous cerebral embolism as a result of CV catheterization has not yet been described in the literature. We hypothesize that in this patient, air has been aspirated through the partially disconnected CV catheter with subsequent expulsion into the cerebral venous system due to raised intrathoracic pressure on expiration. This cycle might have been enhanced by the dyspnea and coughing (forced in- and expiration) that accompanied the bronchopneumonia. Diagnosis of cerebral air embolism can be easily confirmed by brain CT [1, 7]. Treatment of venous air embolism consists of immediate termination of any central line procedure in progress. The patient should be placed in Trendelenburg position and rotated towards the left lateral decubitus position in order to trap air in the apex of the ventricle and to prevent its ejection into the pulmonary arterial system, or retrogradely into the cerebral venous circulation. If a CV catheter is present, aspiration should be applied in an attempt to remove air [1, 9]. Experience with hyperbaric oxygen therapy for venous air embolism is limited, but might be efficient [10].

Fig. 1. Brain CT demonstrating extensive air collections in the superior sagittal sinus and cortical veins.

Fig. 2. Wedge-shaped cortical ischemic changes in the right precentral gyrus on autopsy. Areas with vascular congestion and cavities (insets).

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