associated MRI T2-weighted hyperintensities is broad and includes could be correlated more directly to the pontine edema.

Thria and dysphagia suggesting lower motor neuron involvement a ‘clinical radiologic dissociation’ [7]. Of the reports in which symp-
toms were present in less than 25% of patients, suggesting
ecephalopathy involving the brainstem found that correlated clin-
ocedema [5].

The differential diagnosis of brainstem encephalopathy with associated MRT T2-weighted hyperintensities is broad and includes
central pontine myelinolysis, autoimmune diseases (systemic lupus erythematosus, Behcet’s disease, polycystic nodosa), multiple
sclerosis, infectious/postinfectious conditions (acute disseminated
cerebral angiopathy, Bickerstaff’s encephalitis, Listeria rhomben-
cephalitis, 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 di-
agnoses. 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 infu-
sion systems and orthopedic, neurosurgical or cardiovascular sur-
gical procedures [1]. Clinical presentation is mostly dominated by
right ventricular dysfunction and pulmonary injury. Systemic pre-
sentation 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 bronchopneum-

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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 malformation 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 an 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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