of CCA and/or ECA dissection. Thus, we estimate the prevalence of this type of dissection to be less than 1% of all cervical artery dissections. The reasons for the low frequency might be a different ultrastructure of the vessel wall in the CCA and ECA, but underdiagnosis due to a lack of attention regarding dissection of the ECA is also possible because dissection occurs without severe consequences/sequelae for the patients.

Conclusion
Dissections of the CCA and/or ECA are very rare. We assume that a systematic review of neuroimaging investigations would reveal a higher incidence of dissections at these sites.

References


Ralf Dittrich, MD, Department of Neurology University Hospital of Münster, Albert-Schweitzer-Strasse 33 DE–48129 Münster (Germany)
Tel. +49 251 834 7955, Fax +49 251 834 8181, E-Mail dittrir@gmx.de

Cerebrovasc Dis 2006;21:210–212
DOI: 10.1159/000090794

Hypertensive Encephalopathy with Prominent Bulbar Presentation

Joshua Z. Willey, Jan Claassen, Bernardo Liberato, Shyam Prabhakaran
Department of Neurology, Neurological Institute, Columbia University College of Physicians and Surgeons, New York, N.Y., USA

Introduction
Posterior reversible encephalopathy syndrome (PRES) is a well-described clinical entity, typically presenting with features of parieto-occipital cortical dysfunction and associated with vasogenic edema on neuroimaging. Patients often present with headaches, visual changes, confusion, and seizures. Magnetic resonance imaging (MRI) characteristically shows an increased T2-weighted signal in the white matter of the parietal and occipital lobes, usually without restricted diffusion on diffusion-weighted imaging (DWI). The hallmark of the disease is clinical and MRI reversibility with aggressive treatment and/or withdrawal of the inciting agent. MRI brainstem abnormalities have also been described, but signs and symptoms specific to this location are more rare. We present a case of PRES with prominent bulbar manifestations that completely resolved with treatment.

Case Report
A 77-year-old man with a past medical history of hypertension, a greater than 100 pack-year history of tobacco smoking, and right internal carotid artery 90% stenosis was admitted to our institution after new onset dysarthria and inappropriate behavior. His neurological examination showed left hemineglect, confusion, and a blood pressure of 250/150 mm Hg. In addition, there was prominent dysarthria and dysphagia, with characteristic bulbar features of hypophonia and oropharyngeal weakness. There was no seizure activity, headache, or visual changes. Laboratory studies were unremarkable (sodium 139 mEq/l) except for a creatinine of 2.5 mg/dl (baseline). He underwent MRI (fig. 1c, d) which showed T2-weighted hyperintensities in bilateral posterior cerebral white matter as well as in the pontine and ventral medullary white matter, which had not been observed on prior neuroimaging (fig. 1a, b). DWI showed no evidence of cytotoxic edema (data not shown). The patient was transferred to the cardiac care unit, where with aggressive blood pressure control, his confusion, hemiparesis and neglect promptly improved over days. The dysarthria and dysphagia also improved over the ensuing 2 weeks. Serial MRIs were performed that showed interval resolution of prior white matter abnormalities (fig. 1e, f); these findings correlated with his clinical improvement. On discharge, he no longer required a nasogastric tube for feeding, his speech was no longer dysarthric, and he had returned to his functional baseline.

Discussion
Hypertensive encephalopathy is a disease entity that has recently been well characterized in the literature. A broader category for this neurological syndrome has been coined: reversible posterior leukoencephalopathy or PRES [1]. Besides acute severe hypertension, several other conditions have been associated with PRES, including renal disease, immunosuppressive and cytotoxic drugs, collagen vascular disorders such as systemic lupus erythematosus, eclampsia, and hematological disorders, including thrombotic thrombocytopenic purpura or hemolytic uremic syndrome [2]. On MRI, the most commonly observed characteristics include hyperintensities on the T2-weighted sequences in the parietal and occipital lobes, and isointense or hyperintense signals on apparent diffusion coefficient maps, suggesting a vasogenic edema pattern [3].

While the exact underlying mechanism of PRES is unknown, there are two prevailing theories regarding its pathogenesis and predilection for areas of the brain supplied by the posterior circulation. The most widely accepted theory proposes that the myogenic component of autoregulation in the posterior circulation, with its sparse sympathetic innervation [4], becomes overwhelmed by either elevated blood pressure or endothelial toxins, leading to a capillary leak phenomenon and vasodilatation, resulting in vasogenic
**Fig. 1.** a, b MRI 2 months prior to admission. T2-weighted images showing no significant pontine white matter abnormalities. c, d MRI on admission. T2-weighted images showing evidence of increased T2-weighted signal within the pons. e, f MRI performed 7 weeks after admission. T2-weighted images showing near complete resolution of abnormalities in the pons.

**Table 1.** Prior case reports with predominant brainstem signs and symptoms

<table>
<thead>
<tr>
<th>Age/gender</th>
<th>Diagnosis</th>
<th>Clinical presentation</th>
<th>Brain imaging findings</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>58 M</td>
<td>hypertension</td>
<td>somnolence, left hemiparesis, dysarthria</td>
<td>T2 signal increase in medulla, pons, midbrain, thalamus, and periventricular white matter</td>
<td>Yoshida et al. [9] 2001</td>
</tr>
<tr>
<td>28 M</td>
<td>hypertension</td>
<td>blurred vision, diplopia, bilateral 4th nerve palsies</td>
<td>T2 signal increase in the pons</td>
<td>Drees et al. [10] 2001</td>
</tr>
<tr>
<td>54 F</td>
<td>hypertension</td>
<td>difficulty walking, right leg weakness, headache</td>
<td>T2 signal increase in brainstem, cerebellum, and periventricular white matter</td>
<td>Chang and Keane [11] 1999</td>
</tr>
<tr>
<td>55 M</td>
<td>hypertension</td>
<td>difficulty walking, headache</td>
<td>brainstem hypodensity on CT scan, follow-up MRI after 2 weeks was normal</td>
<td>Chang and Keane [12] 2000</td>
</tr>
<tr>
<td>52 F</td>
<td>hypertension</td>
<td>headache, cerebellar signs, multidirectional nystagmus</td>
<td>T2 hyperintensity in the pons, and periventricular white matter</td>
<td>de Seze et al. [13] 2000</td>
</tr>
<tr>
<td>20 F</td>
<td>eclampsia</td>
<td>coma, absent papillary light response, decerebrate posturing</td>
<td>T2 signal hyperintensity in medulla, pons, midbrain, thalamo, internal capsule, bilateral occipital lobes</td>
<td>Keswani and Wityk [14] 2002</td>
</tr>
<tr>
<td>38 M</td>
<td>hypertension, cocaine use</td>
<td>headache, hemiparesis, hemisensory loss</td>
<td>T2 signal hyperintensity in pons and midbrain, obstructive hydrocephalus</td>
<td>Thambisetty et al. [15] 2003</td>
</tr>
<tr>
<td>57 M</td>
<td>hypertension</td>
<td>lethargy, limb weakness, gait instability, progression to coma with unreactive pupils, improved to normal exam</td>
<td>T2 signal hyperintensity in medulla, pons, midbrain, and internal capsule</td>
<td>Lecei et al. [16] 2005</td>
</tr>
</tbody>
</table>
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.

References

Cerebrovasc Dis 2006;21:212–214
DOI: 10.1159/000090795

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

R. Brouns, D. De Surgeloose, I. Neetens, P.P. De Deyn

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 glyc-