patient’s admission, with a 0.5-T superconducting magnet in T2-weighted spin-echo sequences (repetition time, 2000 ms; echo time, 60, 120 ms), with 9-mm-thick contiguous slices, in the axial plane. Hyperintense images were found in both globus pallidus (Fig 1) and in the subcortical white matter (Fig 2). A skin biopsy in the dorsal part of a metacarpus showed a necrotizing arteritis without granulomatous lesion or extravascular eosinophils.

A diagnosis of probable Churg-Strauss syndrome was made. Cyclophosphamide therapy (0.6 mg/kg m2) and corticotherapy (1 mg/kg) were begun and associated with tiapride (300 mg over a 2-day period). The next day, the patient’s temperature was normal. On day 2, a peak-flow test was 500 L/min. Three days after the beginning of the treatment, chorea, skin lesions, and eosinophilia had disappeared. Four months later, a clinical examination was normal. Cyclophosphamide and corticoids were continued.

Churg-Strauss syndrome is a rare necrotizing arteritis in childhood. International criteria and definitions used for the classification of Churg-Strauss syndrome are asthma, eosinophilia, history of allergy, mononeuropathy or polyneuropathy, nonfixed pulmonary infiltrates, paranasal sinus abnormalities, and extravascular eosinophils.4 Our patient had three of the criteria (asthma, eosinophilia, and paranasal sinus abnormalities). Masi et al4 stated that the combination of only two criteria, asthma and eosinophilia, or the alternative criteria set for patients without asthma or eosinophilia yielded a sensitivity of 90% and a specificity of 99.7% in their population. Churg-Strauss syndrome may be separate from the periarteritis nodosa by extravascular eosinophil infiltration.5 On the other hand, a higher prevalence of asthma and hypertension, an important eosinophilia test5 and negative hepatitis B serological test are more frequent in Churg-Strauss syndrome than in periarteritis nodosa.6,7 Thus, we propose that our patient had Churg-Strauss syndrome.

In our case, chorea may be related to the hyperintense signals in both globus pallidus demonstrated by MRI. They may be consistent with bilateral small, deep infarcts or with an inflammatory process. The hyperintense signals may be due to cerebral vasculitis because no other etiology was found and because immunosuppressive treatment was effective.

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Cerebrovascular Events in Cardiac Catheterization

Neurological complications at cardiac catheterization (CC) are infrequent, occurring in approximately 0.2% of patients.1 Posterior cerebrovascular events (CEs) seem to predominate with the brachial approach.2 However, Keilson et al3 presented 13 patients (0.3%) with CE after femoral CC, and the posterior circulation was involved in seven cases.

To date, little is known about the frequency and vascular topography of CE and its relation to the catheterization route. We carried out a retrospective clinical study in 10,000 consecutive patients with CC between 1977 and 1990. All the patients were studied by the femoral approach, and 12 (0.12%) had CE. There were eight females and four males, ranging from 8 to 61 (mean, 48) years of age. Complications occurred either during catheterization (eight patients) or within 24 hours after the procedure (four patients). The main cardiac diagnosis was valvular heart disease in seven, ischemic heart disease in two, congenital heart disease in two, and infective endocarditis in one. Five patients showed evidence of focal disorder in the vertebrobasilar territory, three in the carotid territory, and four were of uncertain localization. Computed tomographic scans were performed beginning in 1978 (five patients). The neurological findings resolved completely within 24 hours in six patients. At 3 weeks, disability was minimal in three patients and moderate in two. One patient died 7 days after CC.

In conclusion, our incidence of CE in CC (0.12%) is similar to other studies.1,4 In agreement with Keilson et al,3 vertebrobasilar CEs also predominate in our series. The preponderance of posterior CEs is apparently independent from the CC approach (femoral versus brachial).

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Magnetic Resonance Imaging in Basilar Artery Dissection

Primary dissections of the basilar artery are rare. They usually result in brain stem ischemia and have a poor prognosis. We report a benign case in which subarachnoid hemorrhage (SAH) was the only manifestation. The diagnosis was suspected at angiography and confirmed by magnetic resonance imaging (MRI).

A 63-year-old woman suddenly developed a severe occipital headache and posterior neck pain associated with photophobia and vomiting. The headache persisted despite paracetamol treatment. Her temperature, which had been normal during the first 5 days, increased to 38°C (rectal). The patient was referred to the hospital 7 days after the onset of the headache.
On admission, the patient had mild meningismus and sleepiness, without other neurological signs or symptoms. Rectal temperature was 38.1°C. General examination was otherwise normal. Routine laboratory tests were negative. Cerebrospinal fluid analysis revealed xanthochromic fluid that contained 4900 red blood cells and 410 white blood cells per mm³, with 81% polymorphonuclear leukocytes, 16% lymphocytes, and 3% monocytes. Protein level was 76 mg/dL. Cytological examination and culture were negative.

Computed tomographic (CT) scan without contrast showed a hyperdense lesion in the prepontine cistern. There was no enhancement after contrast. Brain parenchyma was normal. A vertebral angiogram showed a narrowing of the basilar artery that was more prominent from the origin of the anterior inferior cerebellar artery to the bifurcation (Fig 1A). Sagittal T1-weighted spin-echo MRI sequences showed a high signal intensity in front of the upper pons (Fig 1B). Axial T1-weighted spin-echo sequences showed a narrowed basilar artery lumen, a circumferentially increased signal suggestive of intramural hematoma, and bleeding in the interpeduncular space (Fig 1C). Brain-stem parenchyma was normal.

![Vertebral angiogram (A) showing narrowing of the basilar artery](http://stroke.ahajournals.org/)

![Sagittal T1-weighted spin-echo MRI (B) showing high signal intensity along the basilar artery](http://stroke.ahajournals.org/)

![Axial T1-weighted spin-echo MRI (C) showing high signal intensity mass expanding the wall and reducing lumen of the basilar artery, and bleeding in the interpeduncular space](http://stroke.ahajournals.org/)

![Sagittal T1-weighted spin-echo MRI (D) showing disappearance of intramural hematoma](http://stroke.ahajournals.org/)
The patient’s headache improved progressively, and 1 week after admission she was asymptomatic. On control MRI 2 months later, the hypersignal was no longer visible and was replaced by the normal flow void of the basilar artery (Fig 1D).

Basilar artery dissection usually causes severe brain stem infarction. In some cases, it is manifested as SAH. A review of the literature discloses 38 cases of basilar artery dissection, 20 of which are dissection of the vertebral artery extending to the basilar artery. In 18 cases, the dissection is limited to the basilar artery. Twenty-seven of the patients presented with brain-stem ischemia, 5 with SAH, and 6 with both.3-5 Thirty of the 38 patients died. The apparently poor prognosis of basilar artery dissection may be partly explained by the difficulty of intravital diagnosis. The demonstration of a double lumen is the only definite angiographic diagnostic sign; however, it is extremely rare and reported in only four cases.3-5 Most often, angiography reveals a diffuse narrowing of the basilar artery similar to that caused by a spasm, with diagnosis in most cases not fully confirmed until necropsy.

In our patient, CT scan suggested an aneurysm of the top of the basilar artery, and angiographic results were consistent with either spasm or dissection. Basilar artery dissection was confirmed by the MRI, which revealed intramural hematoma. The sagittal T1-weighted spin-echo view showed high signal intensity along the basilar artery, and the axial view demonstrated a high signal intensity ring surrounding the central lumen of the basilar artery. Two months later, the hypersignal had disappeared. These characteristic MRI findings have been reported only twice before.3 Basilar artery dissection should be considered in patients presenting with SAH, even without the neurological symptoms of brain-stem ischemia. Magnetic resonance imaging is crucial to the diagnosis of such cases and should be performed before angiography when clinical and CT findings suggest the diagnosis. A wider use of MRI in such cases will allow the detection of less severe varieties of basilar artery dissection, thus extending the spectrum of presentation and prognosis of this condition.

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