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Figure 2  Fluoroscopy images showing lateral (A) and anteroposterior (B) views of the D7—D8 vertebroplasty, using a posterior left intercostals – transverse approach.

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References


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Two cases of vertebral artery dissections with late stroke recurrences

Case 1: a previously fit 30-year-old man was admitted because of sudden cephalalgia with vertigo, nausea and vomiting triggered by defecation. Diffusion-weighted magnetic resonance imaging (DWI) showed bilateral cerebellar infarctions. Both digitalized angiography and magnetic resonance angiography (MRA) showed smooth tapering of the right vertebral artery. No intramural thrombus or double lumen were observed. Analysis of the cerebrospinal fluid was normal. Electrocardiogram (ECG) and transesophageal echocardiography (TEE) were normal. Examination of cervical and cerebral arteries by ultrasound and fat-suppressed T1WI was normal. Symptoms improved quickly and the patient was discharged with a daily dose of aspirin. Three months later, he presented sudden severe cephalalgia, vertigo and vomiting. DWI showed a new recent bilateral cerebellar infarct. Angiography showed the tapering of the right vertebral artery (Fig. 1A) but with a thrombus in the lumen of the artery, which was compatible with an arterial dissection. Intravenous heparin followed by oral anticoagulation was administered. A follow-up angiography (Fig. 1B), performed 4 months later, showed a false aneurysm. The anticoagulant was replaced by an antiplatelet drug and no further neurological deterioration was observed.

Case 2: a healthy 19-year-old man was admitted with repeated transient ischemic attacks during and after a soccer match. He complained of right and left hemianopsia, left hemiparesis and dysesthesia with a mild headache. DWI showed a recent right occipital infarct. Ultrasound examination of cervical arteries and MRA were normal. Fat-suppressed T1WI was not performed. The blood results showed a low platelet count (113,000/mm³) but no other coagulation or auto-immune abnormalities. ECG and TEE were normal. The patient was discharged with a daily dose of aspirin. Five months later, he presented successively right and left dysesthesia, and dysphasia. MRI showed a new left occipital infarction. Angiography showed occlusion of the left vertebral artery and stenosis of the basilar artery. Anticoagulation therapy was initiated. A follow-up angiography after 3 months gave regressive findings. Oral anticoagulation was stopped after 4 years, but shortly afterwards the patient complained of transient vertigo, ataxia, dysarthria and facial paresis. Angio CT showed a new stenosis of the basilar artery. Angiography showed stenosis of the left vertebral artery with pearl and string sign (Fig. 2), compatible with left vertebral artery dissection. Endovascular occlusion of the artery was performed. An antiplatelet drug was administered and there was no ischemic recurrence over the following year.

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Figure 1 Right vertebral angiogram (lateral projection) performed 3 months after the first symptoms showing a tapering of the artery with a defect of opacification of the lumen (arrow). Follow-up right vertebral angiogram (frontal view from three-dimensional angiography) performed 7 months after the first symptoms showing a false aneurysm (arrow).

Figure 2 Left vertebral angiogram (frontal view from three-dimensional angiography) performed 4 years after the index stroke, showing the pearl and string sign (arrow).

Discussion

Prognosis for patients with a first event of symptomatic cervical artery dissection (CAD) is generally good [1] with a low risk of ischemic recurrence [2,3]. Recurrences more often occur within 2 weeks following the index ischemic stroke. Few cases of late ischemic recurrences have been reported [4]. Similarly, the recurrence rate of dissection is low; less than 1% [3]. Recurrences seem to be more often asymptomatic. However, late ischemic recurrences due to CAD are possible. When a patient presents several strokes in the same arterial territory without a clearly identifiable cause, artery dissection should be considered. MRI or angiography should be repeated even if the initial findings fail to reveal specific aspects and especially in case of ischemic recurrence.

References


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Pilocytic astrocytoma arising from the pineal gland

A 36-year-old man presented with a one-month history of diplopia of sudden onset, fluctuating on and off with spontaneous remission. Two weeks prior to hospital admission, he developed mild bitemporal headaches and dizziness. Neurological examination was normal. Magnetic resonance imaging (MRI) revealed a round, well-demarcated cystic lesion arising in the pineal gland, causing it to expand and, thus, compress the mesencephalon, posterior recess of the third ventricle, splenium of the corpus callosum, thalamus and cerebellar vermis, leading to compensated obstructive hydrocephalus. The lesion was hypointense compared with the gray matter on T1-weighted images (Fig. 1A), isointense compared with CSF (cerebrospinal fluid) on T2-weighted images and hyperintense compared with CSF on proton density-weighted images (Fig. 1B), and showed only focal peripheral enhancement (Fig. 1C) after administration of gadolinium diethylenetriaminepentaacetic acid. The lesion measured $2.5 \times 3.2 \times 2.9$ cm. Serum levels of alpha-fetoprotein (AFP), beta-human chorionic gonadotropin ($\beta$-hCG) and carcinoembryonic antigen (CEA) were normal. A suboccipital craniotomy was performed and CSF was collected directly from the cistern of the foramen magnum. The tumor was approached via a supracerebellar infratentorial route and microsurgical gross total resection was achieved by the senior author (A.R.V.) after evacuation of the citrus-yellow cystic content.

Histological examination showed a cystic mass with a wall that was thin in parts, and thick and more cellular in the rest. The cellular area was composed of bipolar astrocytes in a dense fibrillary matrix with scattered granular eosinophilic bodies and Rosenthal fibers, all features of a pilocytic astrocytoma (Fig. 2). There was evidence of previous hemorrhage into the cyst. Residual pineal parenchyma was seen at the periphery of the tumor with occasional pineocytes interspersed among the tumor cells. CSF examination was negative for neoplastic cells. Eight years after surgery without adjuvant therapy, the patient was well, with normal visual acuity and no radiological evidence of recurrence of the tumor (Fig. 3).

Pineal lesions include benign or malignant tumors [5] and non-neoplastic cysts. Tumors at this location comprise 0.4—1.0% of intracranial tumors, and can be classified into two major types: germ-cell tumors and neoplasms derived from pineal parenchymal cells [3]. Although the pineal gland contains a significant number of astrocytes, the occurrence of a primary low-grade astrocytoma of the pineal gland is rare. To our knowledge, there are only 10 cases of well-documented low-grade glial tumors definitively arising from the pineal gland [1—3]. Difficulty in the diagnosis most frequently arises in distinguishing low-grade astrocytomas in the pineal region from the more common, non-neoplastic pineal cysts. Imaging studies are useful, but cannot reliably make the diagnosis. Both lesions typically appear cystic,