Recently Lobato et al.\textsuperscript{2} reported cases of angiographically occult intracranial vascular malformations and proposed several reasons why angiographic identification had not been possible. Their conclusions can also be applied to angiographically occult spinal vascular malformations. In our case the AVM was less than 5 mm in size and was located at the boundary of the territory between the two anterior spinal arteries, one from the deep cervical artery and the other from the Adam-Kiewicz’s artery. We believe that its small size and location at the boundary of the anterior spinal artery territories are the most likely reasons why it was not recognised by angiography.

Smaller intracranial AVMs have a higher risk of haemorrhage,\textsuperscript{1} and we wonder if this can also be applied to spinal AVM lesions. Koos has suggested that a microangioma may be present in cases of haematomyelia in the absence of large spinal AVM.\textsuperscript{2} We suggest that bleeding due to a spinal AVM should be considered, even when angiography is negative.

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Extraneural metastases from brainstem astrocytoma through ventriculoperitoneal shunt.

In 1954 Wolf et al.\textsuperscript{1} reported a case of a 14 year old female with a thalamic glioblastoma who needed a ventriculo-pleural shunt to relieve hydrocephalus. She died 85 months after shunt placement, and at necropsy tumour cells were found in the pleural cavity. There have been at least 43 other cases of CNS primary tumours that have metastasised through shunts used in the treatment of hydrocephalus. We report the case of a patient who had a brainstem astrocytoma with extraneural metastases through a ventriculoperitoneal shunt (VPS) with development of ascites, and spinal cord seeding.

A four year old girl, previously in good health, was admitted to hospital on 27 December, 1985 with a clinical picture of continuous and intense occipital headache, dizziness and vomiting. Her past medical history was normal. Positive neurological findings included vertical nystagmus in upward gaze and horizontal nystagmus in right gaze; palsy of the VI, VII and IX left cranial nerves; right cranial paresis with hypertonia, hyperreflexia and Babinski sign; right limb dysmetria and ataxic gait. A brain CT revealed a left pontomesencephalic hypodense area extending to the left cerebellar peduncle with an irregular enhancing after injection of contrast material. The fourth ventricle appeared displaced backwards with a mild compression in its floor. There was also a moderate supratentorial hydrocephalus. The patient improved considerably after treatment with dexametasone and radiotherapy. Five months after diagnosis she was again admitted because of bilateral action tremor, dysmetria and ataxic gait. A month later she experienced headache and vomiting, and was found to have bilateral papilloedema in addition to the previous neurological signs.

A second brain CT showed a prominent obstructive hydrocephalus. A VPS was implanted and the patient’s symptoms rapidly improved. Ten months after diagnosis, and five months after VPS placement she presented with abdominal distension caused by ascites. An abdominal ultrasound failed to show masses or encapsulated collections. On 7 November, 1986 the abdominal catheter was removed, but the ascites followed a progressive course. Two cerebrospinal fluid (CSF) specimens showed moderate rise in protein content, but all CSF cytologies were negative. Ascitic fluid had normal biochemical parameters and negative cytology. Because of the poor condition of the patient we decided to avoid aggressive diagnostic and therapeutic manoeuvres. She died two weeks later.

Necropsy showed a white mass in the left pontomesencephalic region spreading to the cerebellar peduncles, septum, fourth ventricle walls and periventricular white matter. There was also involvement of the arachnoid of the cerebellum and the whole length of the spinal cord but with sparing of the spinal cord itself and the spinal nerve roots (fig A). Peritumour was diffusely thickened, white in colour and nodular in appearance. Histological examination showed a diffuse astrocytic growth in the CNS but with sparing of the abdominal organs. Glial fibrillary acidic protein (GFAP) stain was strongly positive for samples of the tumour from brainstem,
Isolated hypoglossal nerve palsy and Horner’s syndrome with benign course

The hypoglossal nerve may be affected by various diseases, sometimes together with other lower cranial nerves and sympathetic fibres as in Villaret’s syndrome.1 We describe a 54 year old woman with isolated hypoglossal nerve palsy and Horner’s syndrome which disappeared within several months. We could find no similar case reports.

On 23 May 1988, our patient developed tinnitus and a narrowed palpebral fissure on the left side without any preceding episodes. Two hours later she had a severe throbbing headache in the left parieto-occipital region, and vomited twice. There was no dizziness, watering of the eyes, nasal stiffness or any sensory abnormalities. The next day physical examination was normal except for hypertension and left Horner’s syndrome. Within two days these severe headache had subsided. On 26 May, she noticed difficulty in speaking and in tongue movements, and was referred to the Department of Neurosurgery. Left hypoglossal nerve palsy was revealed. Left vertebral angiography, CT scan, VPS and MRI of the brain and upper cervical region were normal, as were the oto logical examinations. She was referred to the Department of Neurology of Tohoku University on 14 June. She had no personal or family history of recurrent headaches.

She had no lymphadenopathy or organomegaly. The diameter of the right pupil was 3-0 mm, and the left 2-5 mm. Pupillary reactions were normal. The palpated fissures were symmetric. The tongue curved to the right at rest in the mouth. Its left side was atomic with occasional fasciculation, and appeared larger than the right side. However, left-sided palsy was evident only when the tongue was protruded (fig top). Other examinations were non-contributory. An hour after two drops of 25% epinephrine, the left pupil showed super sensitization dilatation, up to 7-0 mm, while the right remained unchanged. Thermal sweating was absent on the left medial part of the forehead. This distribution of anaesthetic area indicated a sudomotor sympathetic lesion distal to the bifurcation of the common carotid artery.3 Routine laboratory data and cerebrospinal fluid were normal. VDRL was negative. Serum antirubella, anti-herpes, anti-treponemal, varicella-zoster, Epstein-Barr (EB), VZV and cytomegalovirus were examined on 14 and 28 June. IgG against cytomegalovirus was 1:11264 on both occasions. No significant increase in titre or temporal changes were noted. No antiviral titres were detected. Her symptoms regressed without specific treatment. On 11 July, the pupils were equal and no longer reacted to 1-25% epinephrine. The tongue was slightly atrophic on the left side, but its movements were normal (fig bottom). Carotid arteriography and jugular venography were not performed because of a spontaneous recovery. During the subsequent 19 months, she had no recurrence of headaches or any other neurological symptoms. Follow up CT and MRI were unremarkable. On the last examination in February 1990, the appearance and movements of the tongue were entirely normal.

Our patient had left hypoglossal nerve palsy and ipsilateral Horner’s syndrome which was shown to be postganglionic by the supersensitivity to 1-25% epinephrine and anhidrosis over the left medial part of the forehead. The association suggests that the causative lesion must be in the left retro aortic space. Clinical and radiological examinations rule out mass lesions. Their acute onset with hemicrania, and subsequent regression suggest focal inflammation or vascul ar disorders. DeSimone and Snyder1 reported hypoglossal nerve palsy with good
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