Arachnoidal Cysts in the Sylvian Fissure of the Brain

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Arachnoidal cysts in the sylvian fissure of the brain are a rare cause of raised intracranial pressure. These cysts are encountered in the course of an exploratory operation for a suspected intracranial neoplasm or haematoma. Excision of these cysts gives good results, with rapid relief of symptoms caused by raised intracranial pressure.

Intracranial arachnoidal cysts fall into two major categories: cysts “secondary” to a definite aetiological factor and “primary” cysts, when no such aetiological factor can be found.1 Tavares and Ransohoff2 have described cysts that followed a fracture of the parietal bone of the skull. Arachnoidal cysts following meningitis and encephalitis3 are seen more commonly as dilations of basal cisterns rather than in other sites. The association of arachnoidal cysts at the cerebellopontine angle with acoustic neurinomas is well known, but this association is uncommon either with other neoplasms or in other sites. “Primary” arachnoidal cysts, in which trauma, infection or association with a neoplasm cannot be invoked as an aetiological factor, probably result from an error of development.4

The few reported cases of arachnoidal cysts in the sylvian fissure of the brain have been of the primary variety. McConnell and Douglas5 reported a case of a “subarachnoidal” cyst of the sylvian fissure, which occurred in the absence of a definite aetiological factor. Oliver6 described a large thin-walled cyst in the sylvian fissure, which was classified as a “primary” arachnoidal cyst. Two cases of “primary” arachnoidal cysts in the sylvian fissure were encountered at this Centre, within 3 months of one another. Both of these cysts were located by angiography and the angiographic features are described below. The rarity of occurrence of “primary” arachnoidal cysts in the sylvian fissure and the absence of any previous descriptions of the angiographic features of this condition have prompted this report.

Case Reports

Case 1. A 25-year-old woman was admitted on March 24, 1963, with a history of headache and deterioration of visual acuity of 1 year’s duration. Two weeks previously a sudden obscuration of vision had occurred, following which her visual acuity was limited to perception of hand movements only. There was nothing relevant in either the family or past history.

Examination. The patient was co-operative and alert. Weakness of the external rectus muscles of both eyes was noted. Left facial weakness of the upper motor-neurone-type with minimal weakness in the left upper limb was detected. Both plantar reflexes were flexor. The fundus oculi revealed severe bilateral papilloedema with optic atrophy. There were no other clinical abnormalities detected in the nervous or other systems.

Radiography of the skull revealed decalcification of the posterior clinoid processes. Radiography of the chest was normal. A right carotid angiogram was performed on March 26, 1963. In the anteroposterior projection, the internal carotid, middle cerebral and anterior cerebral arteries were displaced to the left. A large “bare area” on the right side between the bone and the middle cerebral circulation was seen (Fig. 1). The lateral projection showed the anterior cerebral artery stretched and elevated, while the middle cerebral artery was depressed (Fig. 2).

Operation. On the same day, a right temporoparietal osteoplastic flap was elevated under general anaesthesia. The squamous temporal bone was thinned out by the raised pressure. On reflecting the dura mater, a blue domed cyst was found to have opened the sylvian fissure widely, with marked atrophy of the surrounding cerebral tissue. Xanthochronic fluid, which had a protein content of 500 mg per cent, was aspirated from the cyst. After excision of the outer wall of the cyst, which was thicker than normal arachnoid mater, the bifurcation of the internal carotid artery with the middle cerebral vessels pulsating freely on the insula of Reil was clearly visible. The deeper wall of the cyst was adherent to the cerebral tissue. An attempt to remove this inner wall tended to produce vascular damage and this was left in situ.

Histological Report. The appearance of the excised wall of the cyst was that of arachnoid mater. There was no evidence of inflammation.

Course. The patient made a rapid postoperative recovery. On discharge 3 weeks later, the hemiparesis and weakness of the ocular muscles had disappeared. Papilloedema had subsided but optic atrophy persisted. The patient was free from headache, but had no improvement in her visual acuity. When seen 1 year later, her condition was unchanged from that on discharge.

Case 2. A schoolboy, aged 11 years, was admitted on June 14, 1962 with a history of headache for 1 month and deterioration of visual acuity for 2 weeks.

Examination. The patient had weakness of the external rectus muscles of both eyes and a horizontal nystagmus, more apparent on attempting to look to the left. The fundus oculi revealed bilateral papilloedema. There were no other clinical abnormalities in either the nervous or other systems.

Radiography of the skull and chest was normal. A provisional diagnosis of a space-occupying lesion in the posterior fossa was made, and a Myodil ventriculogram was performed on June 18, 1962. This showed a tilt of the 3rd ventricle to the right, indicating a left supra-
tentorial lesion. A left carotid angiogram then produced similar appearances to that seen in Case 1.

Operation. On the same day, a left parietotemporal osteoplastic flap was elevated under general anaesthesia. The sylvian fissure was seen to be opened out by an arachnoidal cyst, which extended backwards over the parietal lobe of the brain. The outer wall of the cyst was thick. Xanthochromic fluid, which had a protein content of 700 mg. per cent, was aspirated from the cyst. After excision of the outer wall of the cyst, the middle cerebral vessels were seen clearly, pulsating freely on the insula of Reil. The inner wall of the cyst was adherent to the brain and was left intact.

Histological Report. The appearance of the excised tissue was that of arachnoid mater. There was no evidence of inflammation.

Course. The patient made a good postoperative recovery. Headache was relieved. The papilloedema subsided rapidly. When last seen, 1 year after operation, the patient was free from symptoms and back at school.

Discussion

These 2 patients presented raised intracranial pressure and minimal neurological signs. Carotid angiography located the lesion which in each case was avascular, extracerebral and occupying space by opening out the sylvian fissure. These appearances are probably typical of the rare condition of an arachnoidal cyst within the fissure.

At operation, the remarkable feature in both cases was the widely-opened sylvian fissure. The opercula of the insula of Reil were atrophied, forming a large cavity, at the deep end of which the middle cerebral vessels were seen lying on the insula. The outer or lateral wall of each cyst was thickened arachnoid mater. The inner or medial wall and the side walls of each cyst were inseparable from the cerebral tissue and thought to be the pia mater. The fluid of the cyst in each case was xanthochromic, caused by a high protein content.

No history of trauma or radiological evidence of an injury of the skull was available in either case. In both cases, no previous history of intracranial infection was elicited. Microscopically the walls of the cyst were free from any evidence of an earlier inflammation. The appearance of the cysts at operation suggested a long-standing lesion and, in the absence of a definite aetiological factor, they were thought to be “primary” cysts which had arisen as an error of development.

Lewis described a congenital arachnoidal cyst in which choroid plexus-like tissue had been identified in the lining wall of the cyst. There was no evidence of such metaplasia in the 2 cysts described above. It was thought that the 2 cysts found in the sylvian fissure had resulted from incomplete canalisation of the subarachnoid space during development.
Summary

Two cases of “primary” arachnoidal cysts in the sylvian fissure of the brain have been reported. No definite aetiological factor could be found in either case, and the cysts were thought to arise from an error of development. The angiographic features associated with these cysts have been described.

Case 1 was referred by Dr. M. Visvaratnam, physician at Provincial Hospital, Batticaloa, Ceylon, and Dr. G. S. Ratnavale, neurologist, General Hospital, Colombo. Case 2 was referred by Dr. R. Pararajasekeram, surgeon at Eye Hospital, Colombo. Dr. David Jayamanna, neuroradiologist, was responsible for the angiography in both cases.

References