Giant global intracranial aneurysm in an infant

Sir: Nonthrombosed giant global aneurysms rarely occur in the cerebral vessels and seem to be unrelated to saccular aneurysms. They usually arise from the larger cerebral arteries and rarely rupture; instead, they present as a space-occupying lesion.1,2 Giant intracranial aneurysm has a characteristic diagnostic computed tomography (CT) appearance.3 The symptoms and signs are those of an expanding intracranial mass and cardiac failure, rather than those of subarachnoid hemorrhage. We report an infant with a giant global aneurysm of the middle cerebral artery which compressed adjacent brain tissue causing hemiparesis and cardiac failure.

A 10-month-old boy was admitted to hospital in November, 1981, with symptoms of nausea, vomiting and right-sided spastic weakness of 3 months duration. He was born at the end of the 38th week of gestation after an uneventful pregnancy and a normal delivery. Family history was not significant. The child had shown congestive heart failure for one month after development. Development was generally delayed and the infant often manifested cyanosis and respiratory difficulty on crying until 3 months after birth. The right side of the extremity has moved less well than the left since 3 months of age and there was steady enlargement of head. At the age of 10 months, he became irritable with enlarged head and increased weakness of the right extremity and was transferred to our hospital. On admission, physical examination revealed an alert and fairly nourished baby. Growth and development were delayed. The cranial circumference was over 95 percentile and the fontanels were bulging. There was no audible intracranial bruit. Visual acuity was not impaired, extraocular movements were normal and the other cranial nerves were also intact. A spastic right hemiparesis with exaggerated deep tendon reflexes and Babinski sign were present. Cerebellar functions were normal. The CT scan demonstrated a large round mass with a density greater than that of the brain in the right temporal fossa, and the mass measured 6 × 5 cm in size. Following contrast enhancement the density of the mass increased markedly, and a homogenous round mass without perifocal oedema was seen (fig). The mass compressed the left lateral ventricle and the midline was shifted to the right. Carotid angiography showed a large global aneurysm arising from a branch of the right middle cerebral artery, and the aneurysm was situated in the sylvian region.

A right frontotemporal craniotomy was performed. A part of the aneurysm was exposed on the cortex and a corticotomy was performed on the surface of the aneurysm. A large global aneurysm with smooth, purple-reddish wall was encountered at a depth of 1·5 cm and it extended subcortically into the frontal, parietal and temporal opercular areas. The aneurysm was completely resected under induced hypotension after ligation of the dilated parent artery, which was a branch originating from bifurcation of the middle cerebral artery. Postoperative angiogram revealed a complete extirpation of the aneurysm.

At examination 3 months later, the baby was in good condition. There was no evidence of cardiac failure. The right hemiparesis was gradually improving and he was able to walk without support.

Adams and Richardson4 postulated that the giant aneurysm is different from the saccular aneurysm because of its larger size, different location, and clinical manifestation related to its space-occupying nature. Giant aneurysms make up 5% of all intracranial aneurysms5 and are frequently found at the cavernous portion of bifurcation of the internal carotid artery, middle cerebral artery, basilar artery and vertebral basilar artery junction.6,7

Giant aneurysms of global type from the middle cerebral artery cause neurological pictures by at least two mechanisms; local pressure and distal ischaemia caused by arterial steal. These aneurysms have most often shown evidence of an expanding mass effect rather than of a subarachnoid hemorrhage. A considerable volume of blood flows through the aneurysm and may deprive blood flow from the adjacent brain. On the other hand the aneurysms may enlarge to some extent, then progressive thrombosis may take place in the sac. In this situation, with progressive narrowing of the vascular channel in the lumen of the aneurysm, ischaemia may result in the territory of the parent artery. Neurological deficit of this case may have resulted from

Fig 1 Postcontrast enhanced CT scans show a well circumscribed, enhancing giant aneurysm in left sylvian area (A) and restoration of the lateral ventricle to normal position following operation (B).
compression of the adjacent brain by the enlarged aneurysm or ischaemic steal from the main middle cerebral artery owing to the large amount of blood pooling into the giant aneurysm.

It is proposed that such a large aneurysm developed from a small saccular aneurysm by continuous dilation due to blood pressure or turbulent blood flow. The large size of aneurysms of congenital origin seems to favour the thrombiation of a laminated clot thus preventing rupture. Other investigators consider giant serpentine aneurysms originate from fusiform aneurysms which are most likely caused by a degenerative process of the arterial wall from infection or arteriosclerosis. This case suggests that it is probably of congenital origin and the aneurysm may have enlarged slowly.

The possibility of a giant aneurysm must be considered when a nonenhanced CT study shows a well circumscribed high density mass without oedema at the adjacent area of brain and contrast infusion reveals homogenous strong enhancement in the lumen. Sometimes it is difficult to confirm the diagnosis before angiographic study. Giant aneurysms can be divided into three types: partially thrombosed, completely thrombosed and non-thrombosed. Our case belongs to the non-thrombosed group and showed a well circumscribed homogeneous mass without perifocal oedema and diffuse enhancement in the lumen with contrast infusion. For the total excision of a giant aneurysm in early infancy, it should be remembered that the patient may have an acute cardiopulmonary insufficiency. In a situation of poor general condition, it is reasonable to proceed with the surgery using the extracorporeal heart-lung machine and hypothermia. It is important to make efforts to minimise the damage of the adjacent functional area accompanying surgical procedures; this principle is especially important in the surgery for a giant aneurysm deep in the dominant hemisphere. During resection of a giant aneurysm in the infant it is imperative to avoid damage of the adjacent areas surrounding it because the compressed immature brain has strong potential for restoration of function. The postoperative course of giant aneurysm in the infant is quite different from that in the adult or in the elderly group, because the underlying brain, which is compressed by a large aneurysmal sac, is in a relatively immature state. The marked improvement of the neurologic deficit following an excision of the giant aneurysm in this patient may also be attributed to restoration of adequate blood flow and prevention of steal phenomena and the resultant return of intracranial contents to their normal position.

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References


Oclusion of the anterior choroidal artery

SIR: Occlusion of the anterior choroidal artery is seldom reported. Its clinical syndrome is controversial and some authors have even questioned its existence. First described in 1891 by Kolisko (cited by Masson et al.) and later by Foix, the anatomy of the anterior choroidal artery and the consequences of its ligation have been generating considerable dispute. For cases of the syndrome with CT scan documentation were recently reported. We report a fifth case of anterior choroidal artery occlusion, with confirmatory CT scan findings.

A 64-year-old right-handed male was admitted because of left hemiparesis. He was known to have hypertension and diabetes, and to smoke. The evening prior to admission he developed sensory loss of the left hand. Over the next 12–24 hours he developed "pins and needles" over the remainder of his left arm, leg, and the left side of his face. His left hand was weak and "clumsy." He fell due to weakness of his left leg. He denied a history of transient ischaemic attacks. He had no nausea, vomiting, headache, or visual loss. On examination, his mental status was normal. Visual fields were intact. There was a left central facial paresis and dysarthria. He was moderately weak on the left side in a pyramidal distribution. Tone was normal. There was a left pronator drift. Sensation was diminished to all modalities over the entire left side of his body. Romberg sign was not present. Rapid, rhythmic alternating movements were slower on the left. The plantar response was equivocal.

The next day he was much weaker on the left side and manifested anosognosia. There was a new left homonymous hemianopia. A CT scan was obtained and intravenous heparin therapy was initiated.

The patient's condition stabilised and heparin therapy was discontinued after several days. Three months after discharge, he still had a "pins and needles" sensation over the entire left side of his body as well as diminished vibratory sensation. He had a slight left central facial paresis and a left homonymous hemianopia. He had left sided weakness in a pyramidal distribution with a pronator drift and slight unsteadiness of gait requiring a cane.

Comparison of the patient's CT scan with those recently published by Mason et al. and with the template published by Damasio, reveals that his lesion corresponds with the area supplied by the anterior choroidal artery. This vessel most often arises from the internal carotid artery as its last branch, although it is sometimes a branch of the posterior communicating or middle cerebral artery. The small vessel crosses the optic tract, then runs laterally to the inferior horn of the lateral ventricle to supply the choroid plexus. Its anastomosis include the posterior choroidal artery. The