CLINICAL REPORTS

Simultaneous presentation of two cerebral arterial aneurysms

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Summary

A case of subarachnoid haemorrhage due to rupture of a middle cerebral artery aneurysm followed by immediate enlargement of a carotid intra-cavernous aneurysm is described. The enlargement of the second aneurysm was due to a rise in blood pressure caused by the subarachnoid haemorrhage. Although multiple aneurysms are common, this phenomenon is very rare and the reasons for this are discussed.

KEY WORDS: subarachnoid haemorrhage, cerebral aneurysm, computed tomography, cerebral angiography, hydralazine.

Introduction

Multiple cerebral aneurysms are common. In a patient with an aneurysm, the risk of having a second lesion is 20% (Locksley, 1966). Although after one aneurysm has been treated, there is a definite tendency for any second lesion to enlarge (Heiskanen and Markla, 1970), it is unusual for two lesions to be active at one time, and any haemorrhage in the acute phase after an aneurysm has bled is almost always from the original lesion (McKissock *et al.*, 1964). This paper details the very rare occurrence of the simultaneous presentation of two cerebral arterial aneurysms.

Case report

A 40-year-old woman presented with a sudden, severe frontal headache, confirmed on lumbar puncture to be due to subarachnoid haemorrhage (SAH). On examination she was drowsy but orientated, with neck stiffness and photophobia. Blood pressure was 180/115 mmHg. There were severe bilateral vitreous haemorrhages. There was a left incomplete third nerve palsy, sparing the pupillary reaction. There

were no other abnormal neurological signs and no hemiparesis. Computed tomography on admission showed blood in the right Sylvian fissure (Fig. 1). Over the ensuing week, the third nerve palsy became complete and the left eye exhibited a total paralysis of ocular movement and impairment of corneal sensation.

These paradoxical findings were explained by cerebral angiography which showed a right middle cerebral artery aneurysm and a saccular left carotid intracavernous aneurysm measuring 20×10 mm in the lateral projection (Fig. 2). After admission, the patient's diastolic blood pressure intermittently rose above 115 mmHg and was reduced with intermittent low dose hydralazine titrated against her overall neurological condition to prevent further enlargement of the intracavernous aneurysm.

After 8 weeks, the ocular palsies had largely resolved and the patient's overall condition had improved sufficiently to permit clipping of the

FIG. 1. CT scan of the head showing blood in the right Sylvian fissure due to bleeding from a middle cerebral aneurysm.

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FIG. 2. Left carotid arteriogram showing an intra-cavernous carotid aneurysm.

middle cerebral aneurysm. At follow-up 18 months later, she was well with no abnormal neurological signs apart from reduced visual acuity in the left eye. The blood pressure was 140/90 mmHg on no medication. Review of the notes of a previous admission for hernia repair showed an average blood pressure of 150/95 mmHg and no neurological abnormality.

Discussion

In a patient with a middle cerebral aneurysm, the internal carotid is the commonest site of a second lesion (Locksley, 1966). The clinical features of intracavernous carotid aneurysms were described by Jefferson (1938). He noted that posteriorly placed aneurysms tend to affect the whole of the fifth nerve, and also the sixth, and intermediately placed ones affect all the ocular nerves but only the 1st and 2nd divisions of the fifth. Anteriorly placed ones affect only the 1st division of the fifth and all the ocular supply, as was the case in this patient although occasionally only the superior division of the third may be affected. Although it is possible that both aneurysms enlarged in response to a rise in blood pressure from some unrelated cause, the patient's resting blood pressure is normal and it seems likely that the increase in size and consequent clinical

effects of the intracavernous aneurysm followed an acute rise in blood pressure caused by the subarachnoid haemorrhage from the middle cerebral aneurysm. Acute rises in blood pressure are common following rupture of a cerebral aneurysm and are probably a reaction to increased circulating catecholamine levels (Neil-Dwyer *et al.*, 1978). However, there are no reports in the literature of this rise in blood pressure causing rapid expansion and immediate presentation of a second lesion.

Why then did the intracavernous aneurysm present immediately after the initial subarachnoid haemorrhage? The aneurysm was able to expand freely to the limits of the cavernous sinus and in view of its large size considerable tension would have developed in its wall in response to a rise in blood pressure. The wall tension (T) in a structure of principal radii R_1 and R_2 is related to the transmural pressure (P) by the law of Laplace and $P = T(1/R_1 + 1/R_2)$. The expansion in response to a rise in tension is a function of the elastic stiffness of the wall which cannot be directly measured. The calculated wall tension in this aneurysm is 70 Nm^{-1} which compares with published measurements of tension in the aorta of 170 Nm^{-1} (Ganong, 1977). The effect of this was that the small rise in blood pressure after the subarachnoid haemorrhage was sufficient to produce rapid expansion of an already large aneurysm and cause its clinical presentation.

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