DELIBERATE THROMBOSIS OF INTRACRANIAL ARTERIALANEURISMS: PARTIAL OCCLUSION OF THE CAROTIDARTERY WITH ARTERIOGRAPHIC CONTROL

PRELIMINARY REPORT OF A CASE

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Accepted methods of treatment of intracranial arterial aneurisms consist of bed rest, carotid ligation, or direct surgical attack. In general, the best results with aneurisms below the circle of Willis have been attained by carotid ligation.\(^5\) We consider them probably due to thrombosis within the aneurism. Occasionally, carotid ligation has an inadequate effect. In these cases there is subsequent rupture of the aneurism. Sometimes carotid ligation causes so much decrease in blood flow that there results cerebral anemia with\(^5\) or without\(^5\) arterial thrombosis. The ideal result, then, is enough impairment of arterial flow to produce thrombosis of the aneurism alone, without causing either cerebral anemia or thrombosis of the parent artery.

In one case of saccular aneurism of the internal carotid artery near its bifurcation, this goal has apparently been achieved by partial occlusion of the common carotid artery. It has been verified by carotid arteriography. The widespread implications of this procedure seem to justify this preliminary report.

REPORT OF CASE


On March 24, 1950 a 48-year-old engineer was admitted to the Syracuse University Hospital some hours after collapsing while shoveling snow. During the previous 2 years, he had had mild frontal headaches, particularly on the left side.

On admission, he had hyperactive deep reflexes and absent superficial reflexes. There were no definite signs of meningeal irritation or neural dysfunction. Lumbar puncture yielded very bloody CSF under pressure of 220 mm. The following day he was transferred to the Syracuse Memorial Hospital. The neck now was stiff and Kernig's sign positive. Roentgenograms of the skull were normal except for slight backward displacement of pineal body. A chest film demonstrated a round area of increased density in the left upper lobe. An EEG indicated a diffuse mild abnormality without evidence of localization.

A left carotid angiogram on March 27 revealed an aneurism, \(20 \times 15 \times 11\) mm., with a broad base, arising from the internal carotid artery, just above the anterior clinoid process (Fig. 1, A and B). There was poor filling of the vessels beyond the aneurism. Right carotid arteriograms with simultaneous compression of the left carotid, showed good filling of both anterior and middle cerebral arteries with poor filling of the aneurism. These films (in Chamberlain-Towne view) showed some squaring of the left middle cerebral artery, suggestive of cerebral swelling (Fig. 1 C).

The patient was placed at complete bed rest for a month, following which there was no change in the chest films. During this period, there were occasional bifrontal headaches lasting from 1 to 1½ hours.

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Left carotid angiography (2nd exam.) on April 3, 1950, revealed no change in appearance of the aneurism. Both anterior cerebral arteries were well visualized. Ten days later, the EEG was normal, even after left carotid compression for 25 minutes.

We then wished to determine whether carotid compression would produce stasis within the aneurism. If the patient were supine, hyperextending his neck would bring the fundus of the aneurism into a dependent position. Then carotid angiography could be performed with carotid compression below the needle. Since 35 per cent diodrast® has a specific gravity (1.185) greater than that of blood (approximately 1.060), stasis would show as a pool of diodrast® in the dependent aneurismal fundus.

Accordingly, on April 26, 1950, left carotid angiography (3rd exam.) was done during proximal compression of the left common carotid. A polyethylene catheter was inserted into the left internal carotid through a #14 gauge Tuohy needle. The mean blood pressure in the right brachial artery equalled the pressure within the left internal carotid. The catheter could be seen in the angiogram to extend up the internal carotid to the base of the skull. Compression of the left common carotid caused a 40 per cent drop in intracarotid pressure. With digital compression of the left common carotid, 15 cc. of 35 per cent diodrast® were injected. The first film made at the end of injection revealed filling of the internal carotid from the point of injection as well as of the aneurism and adjacent arteries (Fig. 2 A). Further films were made at intervals of approximately 6 secs., 12 secs., and 1 min. after injection. Each of these showed the fundus of the aneurism. About 40 per cent of the projected area of the aneurism remained opaque in the last exposure (Fig. 2 B), proving that considerable stasis in the aneurism had been caused by left common carotid compression.

Fig. 1. (A) Left carotid arteriogram, antero-posterior projection. (B) Left carotid arteriogram, lateral projection, demonstrates size and location of the saccular, broad-based aneurism of left internal carotid. (C) Right carotid arteriogram with simultaneous filling of left side. Chamberlain-Towne projection, demonstrating aneurism and "squearing" of left carotid fork (first parts of anterior and middle cerebral arteries).