

Reconstruction of internal carotid artery in a patient with intermittent attacks of hemiplegia

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In 1914 Ramsey Hunt described the syndrome of internal carotid occlusion and prefaced his paper with the following observations:

“The object of the present study is to emphasise the importance of obstructive lesions of the main arteries of the neck, in the causation of softening of the brain, and more especially to urge the routine examination of these vessels in all cases presenting cerebral symptoms of vascular origin. In other words, the writer would advocate the same attitude of mind towards this group of cases as towards intermittent claudication, gangrene, and other vascular symptoms of the extremities, and never omit a detailed examination of the main arterial stem.”

Efficient carotid arteriography has focused attention upon this syndrome, and the diagnosis of internal carotid occlusion is being made with increasing frequency; during the past year Dr David Sutton has demonstrated such an occlusion by arteriography on six occasions.

Unfortunately treatment has been unsatisfactory. But with the knowledge provided by experience of arteriosclerotic thromboses in other vessels it should, by careful clinical examination and selection, be possible to improve, or cure an occasional patient by surgery. In the past the surgical treatment of this lesion has been along one of three lines: arterectomy (Chao et al. 1938) in the hope that removal of the diseased segment might reduce reflex spasm in the cerebral vessels; cervical sympathectomy (Johnson and Walker 1951) with same object in view; and thrombo-endarterectomy (Strully et al. 1953). All have proved to be ineffective.

In this paper we describe a case in which a partially thrombosed segment of the common and internal carotid arteries was resected and the blood-vessel reconstructed by a direct anastomosis.

Case-record

Mrs A, a housewife, aged 66, was in bed Dec. 26, 1953, recovering from a cold, when she had her first attack. This began with a tight feeling in the chest, a sensation of the heart beating fast, and slight shortness of breath. A few moments later she noticed that she could not use either her right arm or leg and that a film had come over her left eye. She tried to call her son but found she could not speak. Her son gave her a glass of water which she could not swallow and the water gurgled in her throat. When she covered up her right eye with her left hand, she found that she was blind. About half an hour later she found that she could move her hand and soon afterwards the sight started to come back in her eye and she could speak. Recovery was complete in a few minutes.

The next attack came on at 2 a.m. the following morning and was identical with the first. For the next fortnight she had attacks every morning at about 2 a.m., each lasting twenty to thirty minutes. Two to three days after the first attack she had many brief attacks during the day in which the vision in the left eye became misty without any other symptoms. Two weeks after the first attack she got out of bed. That day, while sitting up having tea she had a similar attack lasting ten minutes.

Up to the time of her operation she had, in all, 33 major attacks lasting from ten minutes to half an hour, in all of which there was loss of vision in the left eye, right hemiparesis and aphasia, preceded by this heavy feeling in the chest, associated with rapid palpitation of the heart, which would begin suddenly before the paralysis and end gradually before the attack passed off. Eight of these attacks occurred after her admission to hospital on April 9, 1954.

Examination

Seen on March 22, she was an intelligent, alert woman weighing 138 lbs. and slightly obese. Arterial pressure 245/115 mm Hg; cardiac impulse 4 inches from the midline, in the 5th intercostal space; femoral pulses present; and cardiac rhythm regular with occasional extrasystoles., Normal cranial nerves, no muscular wasting or rigidity; muscular power normal; tendon reflexes brisk and equal; plantar responses flexor, sensation intact. Fundi: optic discs

normal; normal vessels seen. Both common carotid pulses normal; left internal carotid pulse felt only with difficulty; left facial and temporal pulses absent; right internal carotid, facial, and temporal pulses normal.

When admitted to hospital on April 9, there was no change in her physical condition except that the blood pressure was now 190/95.

Special examination

Blood: haemoglobin 95% (14.1 g. per 100 ml.); mean corpuscular diameter 7.5 μ ; white cells 11,000 per c.mm. (neutrophils 61%, basophils 1%; lymphocytes 36%, monocytes 2%); Wassermann reaction negative; Harris test negative; blood-urea 39 mg. per 100 ml. Urine: no albumin, sugar or deposit. Urea-concentration test up to 3.2% urea. Cerebrospinal fluid: pressure 90 mm; protein 50 mg. per 100 ml.; colloidal gold 0000000; Wassermann reaction negative. Electrocardiogram: T-waves somewhat flat over left ventricle; no other abnormality seen.

After consultation with Dr Denis Brinton, a left carotid arteriogram (fig 1 Carotid arteriogram showing atheromatous obstruction at the bifurcation of the common carotid artery) was obtained by Dr David Sutton who made the following report:

“The carotid artery was punctured and three injections of contrast was made. There was fair filling of the internal carotid artery and its branches with exception of the anterior cerebral artery which appears small and is only filled at its origin. The middle cerebral vessels appear normal. The first set of films showed delayed filling of the internal carotid artery and poor intracranial filling so that a thrombosis of the internal carotid artery was suspected. Another set of films was taken to show the bifurcation of the common carotid artery and better filling was obtained. However, the delayed filling of the internal carotid was confirmed and was shown to be due to an atheromatous lesion almost occluding the origin of the vessel.

Conclusion

Atheromatous obstruction of the origin of the internal carotid artery. The non-filling of the anterior cerebral artery is probably due to the fact that it is filling from the other side.”

Further Observations

From this it seemed that the attacks were due to intermittent ischaemia in the territory supplied by the left internal carotid resulting from the constriction at the origin of the artery. In an attempt to reproduce such attacks, three injections of tetra-ethyl ammonium chloride were given on April 28. Arterial pressure before the injections varied from 204/98 to 226/112. The lowest pressure reached was 140/90, this pressure being recorded for approximately two minutes after 2ml. of the solution had been injected. 0.1mg histamine was injected intravenously and produced a fall of pressure to 124/68 covering fifteen seconds, rising quickly to 154/90. No attack was produced.

Most of the 8 attacks that occurred in hospital were at night and no observations were made. When seen at the end of an attack by Dr M.A. Pears, her head and eyes were deviated to the left; her pupils were equal and reacted to light. By the time a detailed examination was made, the power of speech and sight had recovered; the blood pressure was 190/110, the pulse regular and the colour normal.

Operation

On May 19 the patient was anaesthetised by Dr C.A. Cheatle and her body-temperature reduced to 28°C. (82.4°F) by external cooling. The left common internal and external carotid arteries were exposed and found to be adherent to the neighbouring structures in the region of the carotid bifurcation. This was freed, the external carotid artery was ligated and the diseased segment of artery (3 cm long) was resected (fig.2 The carotid bifurcation cut longitudinally). Originally it was intended to insert a blood-vessel graft, but this proved to be unnecessary, a direct end-to-end anastomosis between the common and internal carotid arteries being performed. The carotid artery was clamped for twenty-eight minutes and good pulsation observed in the vessels after removal of the clamps. Anticoagulants were not used.

On return to the ward the patient was gradually warmed; her body temperature had reached 37°C (98.6°F) twelve hours after the induction of anaesthesia. She made a satisfactory recovery. At no time were any abnormal physical signs noted in her central nervous system. She was walking forty-eight hours after her operation and left hospital on June 2.

Present Condition

Seen on Oct. 20 she had been doing her household work from a month after leaving hospital. At no time had she had any further attacks of blindness

or paralysis. She had gained a stone in weight and had not noticed that when she was carrying anything or walking upstairs or hurrying for a bus, she would get a tightness in the sternal region, extending into her neck, which made her feel as though she was being choked. She would become short of breath and her voice husky. The tight feeling in her chest would make her stand still or sit down; but, after a few minutes' rest these sensations would disappear. From time to time she awakens in the night, at about 2 a.m., with rapid beating of her heart. She turns over and in a few minutes the rapid heart action disappears. These attacks of rapid heart action are exactly the same as she used to have before her attacks of paralysis.

Examination showed no signs of cardiac failure. Pulse rate 100 regular; blood pressure 220/115; heart sounds, no abnormalities. Good pulsations could be felt along the whole length of the left common and internal carotid arteries, and the retinal vessels were of equal size on the two sides and of normal calibre. An electrocardiogram showed normal rhythm at 100 and only minor irregularities in the Q.R.S.T. complexes. She walked five times over the steps described by Wayne and Laplace (1933-34) in twenty-four seconds and stopped because of the ache in her chest: the heart rate had risen to 120 but remained regular and the electrocardiogram showed no significant change.

Discussion

This is a beautiful example of what has been called chronic psuedo-ureaemia (Volhard 1931) or chronic hypertensive encephalopathy (Oppenheimer and Fishberg 1928) and attributed to cerebral vascular spasm. In this instance the arteries concerned would be left ophthalmic and middle cerebral. The difficulty of reconciling such an idea with the anatomy and physiology of the cerebral vessels has been pointed out by one of us (Pickering 1948), who showed that transient attacks of cerebral paralysis, identical in every way with those associated with high blood pressure, are found in patients with mitral stenosis and auricular fibrillation when they can only be interpreted as embolic in origin. The frequency with which organic simulates spasmodic vascular obstruction elsewhere has also been shown (Pickering 1951). To imagine that on 32 different occasions, and without any obvious cause, the arteries to the left eye and left hemisphere suddenly contracted enough to produce ischaemia while other arteries did not would constitute a considerable exercise in credulity.

In this instance the attacks were clearly most easily explained by intermittent ischaemia in the territory of the left internal carotid artery. The demonstration of an organic obstruction at the origin of that artery made this explanation more plausible. It is not easy to see how a stable obstruction,

such as this was, could give rise to intermittent ischaemia unless there were periods in which arterial pressure fell low enough for long enough. It is recognised that such attacks of transient cerebral paralysis and of blindness occur in patients with internal carotid occlusion, but in only a few cases is the mechanism precipitating these attacks fully elucidated. Denny-Brown (1951) described the occurrence of paralysis in one of his cases when arterial pressure was reduced by a vasodilator drug. A more striking case was described by Adams (1954) of a 56-year-old man with cirrhosis of the liver who bled and developed a left hemiparesis. He was transfused, the blood pressure rose and his hemiplegia disappeared. He bled again and the hemiplegia returned, death occurring forty eight hours later. At necropsy, the lumen of the right middle cerebral artery was narrowed by a plaque to 0.5 mm. but was patent; the nerve-cells of the hemisphere showed ischaemic changes. Similar cases have been described by Corday et al. (1953) under the term cerebral vascular insufficiency.

The history in our patient of attacks beginning with a tight feeling in the chest, a rapid beating of the heart and breathlessness, strongly suggest paroxysmal tachycardia. This was never proved, for no attack was witnessed until the nervous disturbance was passing off. Nor were we successful in inducing an attack with vasodilator drugs, though we did not give large doses, and never reduced the arterial pressure below 124/68, and that for only a few seconds. However, it is well recognised that paroxysmal tachycardia may cause faintness and even loss of consciousness, and in the presence of a gross obstruction of the internal carotid artery, such as was here found, ischaemic symptoms in the territory it supplied would not be surprising. This conclusion is supported by the persistence of the attacks of rapid heart action after operation. The patient stated that these were exactly the same as had initiated her paralytic attacks before the carotid reconstruction.

We present this case therefore not as proof as to how these attacks of hemiplegia and blindness were produced, but as evidence that when such attacks are associated with internal carotid obstruction it is possible that they may be abolished by removing the obstruction to the internal carotid artery.

Summary

A case in which intermittent attacks of hemiplegia were attributed to partial occlusion of the internal carotid artery is described and the reasons for the patient's attacks are discussed.

Surgical treatment, which consisted in resection of the narrowed portion of carotid artery and reconstruction by a direct anastomosis, has prevented further attacks.

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