BILATERAL INTERNAL CAROTID ARTERY OCCLUSIONS
A CAUSE OF TRANSIENT ISCHEMIC ATTACK

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INTRODUCTION

TRANSIENT ischemic cerebral attacks (T.I.A) is a transient disorder of cerebral function, of less than 24 hours' duration with complete recovery. Commonly it lasts for minutes to an hour or two (Marshall, 1973). It tends to recur. It is a warning of an impending complete stroke as 40% of patients with T.I.A.s will have a major stroke within three years (Pearce, 1978). Treatable causes like hypertension, anaemia, polycythaemia, cardiac arrythmias should be looked into.

T.I.A's involves the internal carotids and vertebrobasilar territory and its clinical features differ accordingly. Lesions of the internal carotid has a greater tendency to produce a stroke than that of vertebrobasilar. Hence the urgent need for angiography is in the latter, not only to localise the site of stenosis but also to rule out subdural haematoma, angiomas and meningiomas which may simulate a T.I.A. (Pearce, 1978).

A case of bilateral internal carotid artery occlusions in a hypertensive patient presenting with transient ischemic attacks and subarachnoid haemorrhage is reported.

CASE REPORT

Patient, a 34 year old male, developed recurrent episodes of unilateral weakness over the past two years. Each episode lasted from a few minutes to a few hours, and was associated with unsteady gait, headaches and occasionally had visual and speech impairement.

He was presently admitted (on July 1978) with the complaints of headaches, neck pains and vomiting.

Past history revealed that he had renal calculi 15 years ago, and had been hypertensive for the past five years. Investigations for secondary causes of hypertension were negative then. No relevant family history of hypertension or migraine was forthcoming.

On examination he was not pale or dyspnoeic but was drowsy. Blood pressure was 230/140 on both arms. Heart was enlarged but patient was not in failure. All peripheral pulses were felt and equal and there was no femoral lag. There was no carotid, subscapular or abdominal bruit. No renal mass was felt. Fundi showed grade II retinopathy. Neck stiffness and Kernigs were positive. The patient was drowsy, otherwise the neurological examination was normal.

Lumbar puncture showed a uniformly blood stained C.S.F. Carotid angiogram revealed bilateral occlusions of the internal carotid about one inch from its origin (Fig. 1a, b). Vertebral angiography revealed collaterals between external carotid and arterial cerebral via the opthalmic artery (Fig. 2) and between the vertebro-basilar and the post cerebral (Fig. 3). An aneurysm at the junction of internal carotid and posterior communicating artery was seen (Fig. 4). Renal angiography revealed stenosis of right renal artery (Fig. 5). Other investigations of transient ischemic attack were

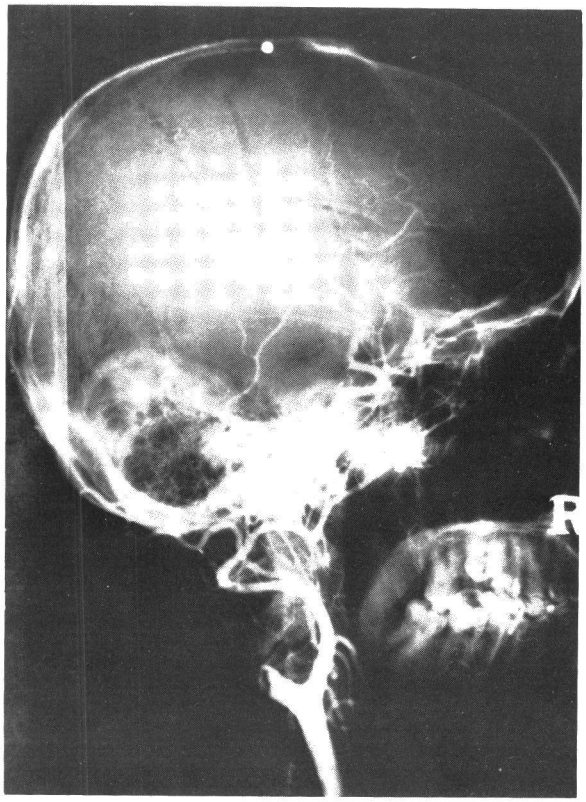

Fig. 1a. Carotid Angiography shows Occlusion of Right Internal Carotid Artery.

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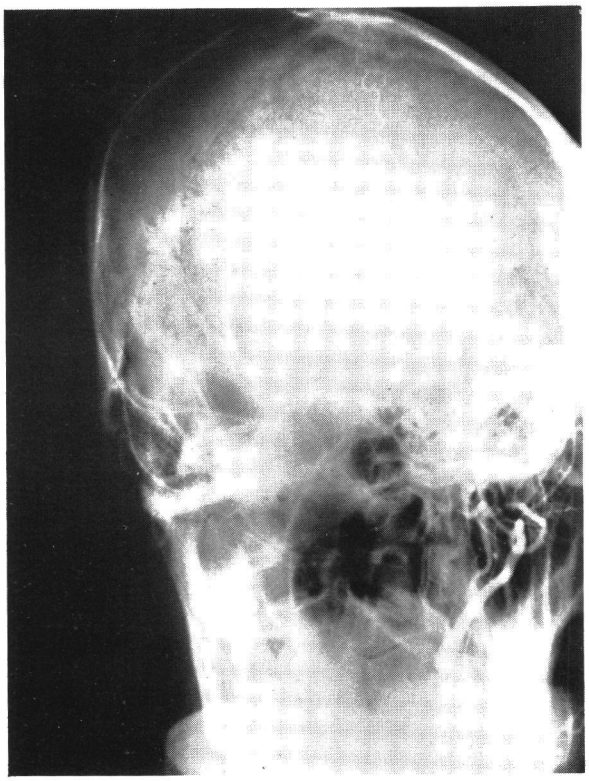

Fig. 1b. Carotid Angiography showing occlusion of Left Internal Carotid Artery.

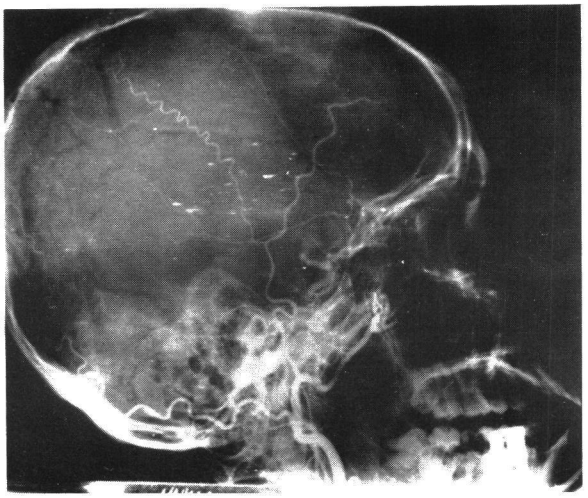

Fig. 2 Vertebral Angiography showing Anastomosis between internal and external carotid via opthalmic artery.

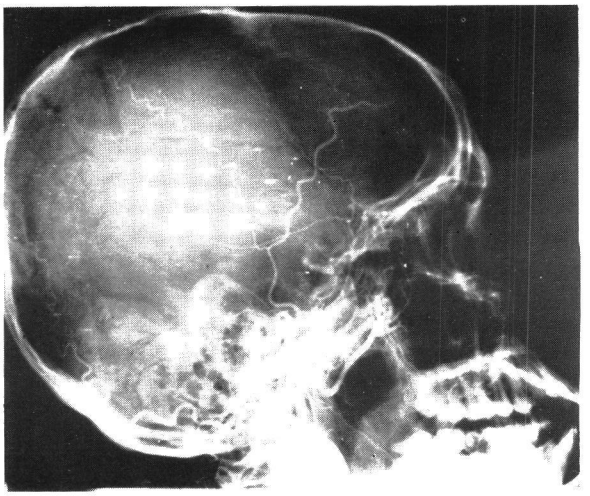

Fig. 3. Vertebrobasilar angiography shows anastomosis via posterior communicating artery.

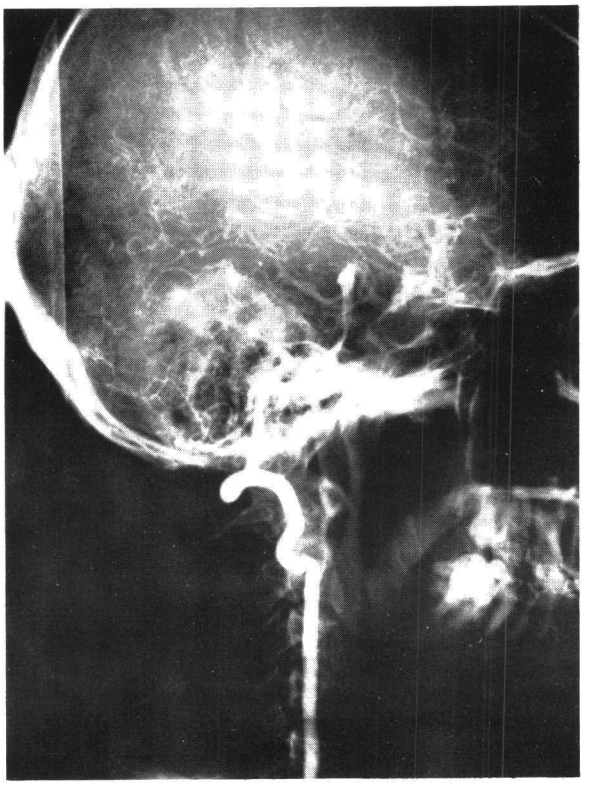

Fig. 4. Vertebrobasilar angiography showing aneurysm at junction of Internal Carotid and Posterior Communicating Artery.

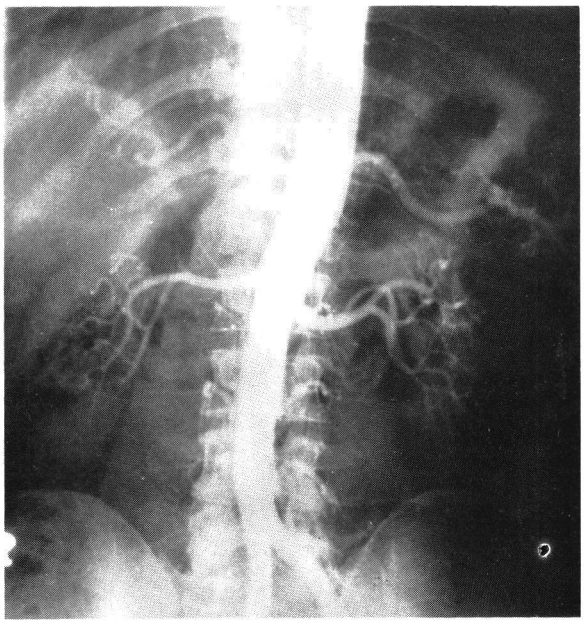

Fig. 5. Renal Arteriography showing stenosis of right renal artery and small right kidney.

normal except the ESR was 55 mm/hr. IVP showed a small right kidney and there was no stones. Urine cultures did not reveal any organism. Patient was treated for his hypertension and he recovered from his subarachnoid haemorrhage.

DISCUSSION

Hypertension is a known cause of T.I.A. Hypertension produces arterial degenerations in large arteries which spreads to smaller vessels as well as produces microaneurysms (Russel, 1973). Hypertension therefore can produce T.I.A. by inducing arterial degenerations, thrombosis as well as subarachnoid haemorrhage. Our patient have both T.I.A. and subarachnoid haemorrhage. Adequate treatment of hypertension reduces the risks of T.I.A. and stroke. Malignant hypertension can produce vasospasm resulting in T.I.A. (Marshall, 1973). In our patient no papilloedema was detected. Stenotic lesions of the extracranial vessels are unlikely to be found if the diastolic pressure is less than 110 mm Hg. (Marshall, 1973). Our patient have both hypertension and occlusion of the internal carotid!!

Recurrent headaches could be due to migraine. Patients with T.I.A. develop headaches due to the dilations of collateral vessels between internal and external carotids. Our patient has such collaterals via the opthalimic vessels and posterior communicating. There was no history of headaches from childhood nor was there any family history of migraine.

Our patient had bilateral occlusions of the internal carotid about an inch from its origin. Du Boulay (1973) found that occlusions occured at the bifurcations of the common carotid; the internal more severely involved than the external. 50% of T.I.A. has tight stenosis or occlusions of the internal carotid artery (Pessin, 1977). Only up to 30% of patient with T.I.A. have signs of extracarotid disease. Hence the importance to do carotid angiography in the young, as lesions of the internal carotid have a greater tendency to produce stroke. 50% of subarachnoid haemorrhage have hypertension (Uttley, 1978). In our patient it is unclear whether the cause is intracerebral haemorrhage or rupture of an aneurysm (congenital or acquired). With the availability of C.A.T. scanner, similar problems, in the future, would be solved.

SUMMARY

Patient, is a young hypertensive due to renal artery stenosis. He presents with five years history of transient ischemic attacks before he developed subarachnoid haemorrhage. Investigations revealed complete occlusions of both internal carotid arteries with collaterals via the opthalmic artery and posterior communicating. Aneurysms either congenital or due to hypertension was probably responsible for his subarachnoid haemorrhage. Patient's hypertension was inadequately controlled, thereby accelerating the artheromatous process and aneurysmal formations. Hence the necessity for regular follow up and adequate control of hypertension.

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REFERENCES

Du Boulay, G. (1973). Radiological investigation of stroke. *Brit. J. Hosp. Med.*, **10,** 258 — 243

Marshall, J.M. (1973). Transient ischemic cerebral attacks. *Brit. J. Hosp. Med.*, **10,** 240 — 243

Pearce, J.M.S. (1978). If I had a transient ischemic attack at the age of 55. *Brit. Med. J.*, **1,** 969 — 971.

Pessin, M.S. *et al.* (1977). Clinical and angiographic features of carotid transient ischemic attack. *N. Eng. J. Med.*, **296,** 358 — 362.

Russel, R.W. and Harrison (1973). The completed stroke. *Brit. J. Hosp. Med.*, **10,** 258 — 267.

Uttley, D. (1978). Subarachnoid haemorrhage. *Brit. J. Hosp. Med.*, **19,** 138 — 155.