

Dissection of the cervical internal carotid artery. The role of Doppler/Duplex studies and conservative management

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Abstract

Cervical internal carotid dissection is not rare. Doppler ultrasound screening of young patients presenting with stroke, identified 10 patients with reduced common and internal carotid blood flow without any evidence of atheroma. Eight, on angiography proved to have a dissection of the cervical internal carotid artery. All were managed conservatively. Seven received anticoagulant therapy, stopping any further neurological symptoms.

Since Jentzer¹ reported a case of a spontaneously dissecting aneurysm of the internal carotid artery in 1954, an increasingly large number of reports have found a higher incidence than had initially been thought.²⁻⁵ Spontaneous resolution, with a good recovery, has been documented.³⁻⁸ This report examines the screening of young patients, with stroke or transient ischaemic attacks (TIAs), using Doppler ultrasonography to decide which patients should undergo angiography to detect internal carotid artery dissection; the results of non surgical management of this condition are also given.

Patients

Eight patients, in whom dissection of the cervical internal carotid artery was diagnosed on angiography, were admitted to the regional unit from a population of approximately three million, during the period September 1986 to May 1988. During this time 700 Doppler/

Duplex examinations were carried out for vascular disease.

The details of the clinical presentations are summarised in table 1. The male:female ratio was 5:3 and the age range 14 to 55 years. Cerebral hemisphere signs were present in all cases. Transient ischaemic attacks occurred in three cases, stuttering stroke (that is, a stroke evolving in a stepwise fashion) in a further three cases, and multiple strokes in the other two patients. One patient began with transient ischaemic attacks and subsequently developed a stuttering stroke (case 8). Of the multiple strokes, one (case 6) had a left hemisphere stroke four years earlier and had undergone an extracranial-intracranial bypass procedure, elsewhere, on the right side in addition to being given antiplatelet agents, and the second case (case 7) experienced progressive visual failure over three years from multiple cerebral infarcts. In only one case was a carotid bruit found on examination.

More than half the patients were or had been smokers. Using the definitions accepted in the Framingham Study,⁹ four patients were hypertensive, three falling into the definite and one into the borderline categories. Two patients (cases 1 and 7) had family histories of vascular disease. Two had experienced recent minor trauma to the neck: case 2 as the result of a kick to the opposite side of the neck during martial arts training and case 4 rotational effects after being struck on the nose by a falling object. Case 8 had sustained a moderately severe head injury 20 years earlier.

Investigations

Computerised tomography (CT) scans confirmed the presence of infarcts in 75% of cases. The majority were in the cortex and the subcortical white matter but in case 1 were restricted to the capsule and the caudate nucleus (fig 1).

Duplex ultrasonic and CW Doppler examinations (using a Dasonics Duplex CV 400 scanner and a Kranzbuhler 761 CW flowmeter) were used as screening tests in all cases. Reduced flow in both the common and internal carotid arteries, upon the side appropriate to the clinical features was found in all cases (fig 2). Atheroma was detected in 3 cases, 2 of whom had bilateral disease. However in none of these three was the atheroma severe enough to account for the reduced flows in the common and internal carotid arteries. Reversal of flow through the supraorbital vessels occurred in

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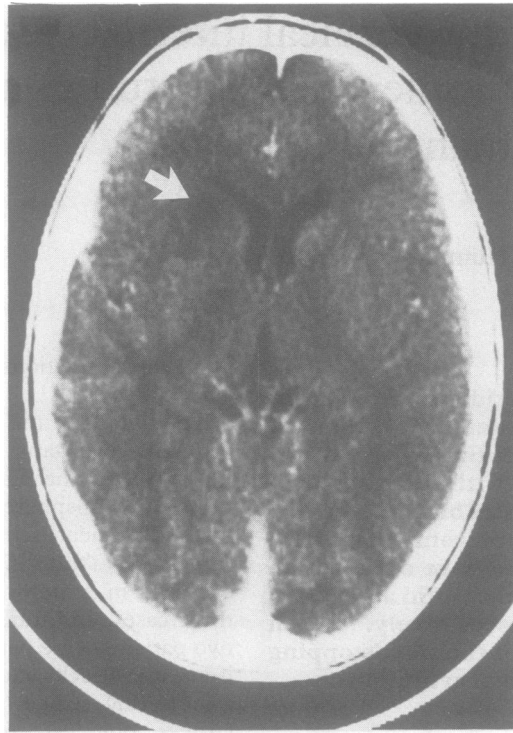
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Table 1 Clinical details

Case no	Patient	Clinical features	Event	Factors
1	14 M	headache L umn paresis	stuttering stroke 10 days	—
2	17 M	headache R umn paresis dysphasia	stuttering stroke 10 days	smoker neck trauma
3	37 F	R umn paresis dysphasia	stuttering stroke 14 days	BP 170/90 oral contraceptive
4	37 F	headache R umn paresis paraesthesia R arm dysphasia	5 TIAs (45 min each) 7 days	BP 160/90 smoker nasal trauma
5	38 M	paraesthesia R face amaurosis fugax carotid bruit	2 TIAs 2 weeks	smoker
6	40 M	R hemiparesis dysphasia	2 strokes 4 yrs & now	smoker
7	47 F	slow visual loss paraesthesia L arm dysphasia	strokes + + + over 3 yrs	BP 180/120
8	55 M	R umn paresis paraesthesia R arm	TIAs 18 mths then progressive stroke 3 mths	BP 150/90 ex-smoker

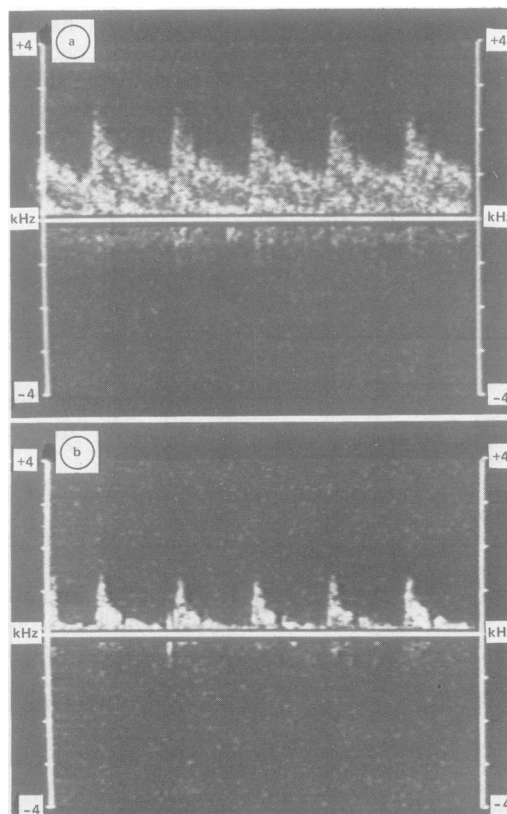
Figure 1 Caudate nucleus infarct on CT (arrow indicates).



only one case. The dissection itself was seen on Duplex scanning (fig 3) in two cases. Five patients had Duplex Scanning within two weeks of onset of symptoms; two within three months of the last symptom and one at a year.

Cerebral digital subtraction angiography was carried out by intra-arterial injection in all cases (table 2). Each case showed the "string sign", (fig 4a) which is considered to be diagnostic for the condition, in the cervical internal carotid artery. The lesion was left sided in five, right sided in two and bilateral in one.

Figure 2 Doppler velocity in internal carotid artery a) normal, b) reduced.



The other angiographic findings were:

- 1) Embolic occlusion of one or more of the distal intracranial arteries in four patients;
- 2) "beading" of the contralateral internal carotid artery compatible with fibromuscular dysplasia was present in two patients;
- 3) an aneurysm arising from the cervical portion of the internal carotid artery, adjacent to the skull base in one patient (fig 4a and b) mild atheroma was demonstrated in only two patients, both of whom had bilateral disease demonstrated on ultrasonic examination.

Two additional young patients, aged nine and 22, who had reduced internal carotid flow on Doppler studies, and no other abnormality in the cervical carotid arteries even on angiography, had multiple intracranial vascular occlusions of the distal branches of the middle and anterior cerebral arteries. These, therefore represent false positives.

One patient, aged 43, had a left hemiplegia and right Horner's sign with reduced right internal carotid artery flow. Angiography was not thought justifiable as his deficit was complete.

Management

Anticoagulation, using heparin initially and warfarin for twelve months, was started within three weeks of the ictus in four patients, three of whom presented with stuttering strokes. The outcome in each was good (table 3), even though two had further minor attacks before full anticoagulation was achieved. Three of the four patients who were initially treated with antiplatelet agents (aspirin in all cases plus persantin in cases 5 and 6) continued to have attacks and were therefore switched to anticoagulants, with symptomatic control. Case 6 had had a right sided extra/intracranial bypass operation carried out following the first stroke (which affected the left cerebral hemisphere) suffered four years before presentation to this unit.

Repeat Doppler studies after anticoagulant treatment was instituted showed an improved flow in four cases, three of whom had been anticoagulated within three weeks of the ictus.

Discussion

Cervical internal carotid dissection is a more

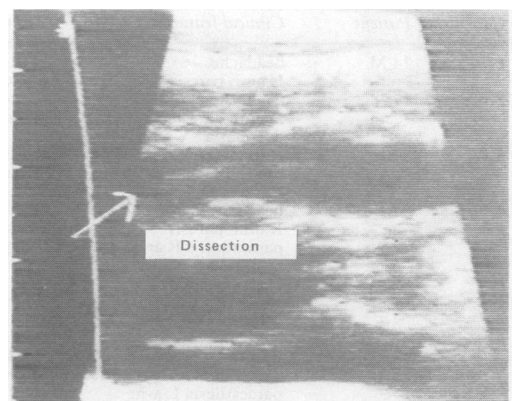


Figure 3 Fixed linear shape (arrow indicates) in lumen of internal carotid artery above bifurcation thought to be secondary to dissection.

Table 2 Investigations

Case no	Side	CT infarct	Doppler*	Angio (int carotid)
1	R	capsule/caudate	flow reduced	"string" sign dysplasia** distal occl ^b
2	L	cortex white matter	flow reduced	"string" sign distal occl ^b
3	L	cortex	flow reduced	"string" sign
4	L	—	atheroma/flow reduced	"string" sign
5	R	—	flow reduced/ reversed supra-orbital flow	"string" sign distal occl ^b ec aneurysm
6	R and L	old cortex/ white matter	L flow reduced	"string" sign mild stenosis
7	L	multiple cortex/ white matter	flow reduced bilat atheroma	"string" sign dysplasia** distal occl ^b bilat atheroma
8	L	multiple cortex/ white matter	flow reduced bilat atheroma	"string" sign bilat atheroma

*Reduced flow was found in the appropriate internal and common carotid arteries.

**Fibromuscular dysplasia

ec—extracranial

common cause of cerebral ischaemia than has been previously recognised.²⁻⁵ Its pathogenesis is not fully understood, but some conditions, which are associated, may be causal agents; these include fibromuscular dysplasia of the internal carotid and the renal arteries which has been reported in 10–20% of cases,^{2,3,10-14} cystic medial necrosis,^{14,15} hypertension, atheroma, a family history of arteriosclerosis, and pheochromocytoma.¹⁶ Two of this current series showed angiographic appearances compatible

with the diagnosis of fibromuscular dysplasia, four were hypertensive (including one patient who was borderline as judged by the Framingham criteria),⁹ two had atheromatous disease seen on angiography and two a family history of arterial disease. Spontaneous internal carotid artery dissection is well recognised. The significance of the patients with minor trauma remains uncertain as there has been no case control study to our knowledge. Most centres in the United Kingdom do not perform angiograms on all young patients with TIAs or stroke; however a history of recent trauma may influence this decision creating a biased impression that dissection usually follows trauma. This study used Doppler/Duplex scanning as the discriminator and a history of minor trauma was elicited in two of the eight patients. Minor trauma in the form of either, a minor direct blunt injury to the neck, or rotation (particularly if combined with an element of flexion/extension) of the neck, cannot be excluded as an initiating factor.¹⁷⁻¹⁹ In centres where all young stroke patients have had angiograms, cervical carotid dissection is the dominant cause.²⁰

Three patients had ipsilateral headache at presentation as described previously.²¹ The literature suggests that carotid bruits are found at the time of presentation in 10–50% of cases whilst occlusory sympathetic paresis (Horner's

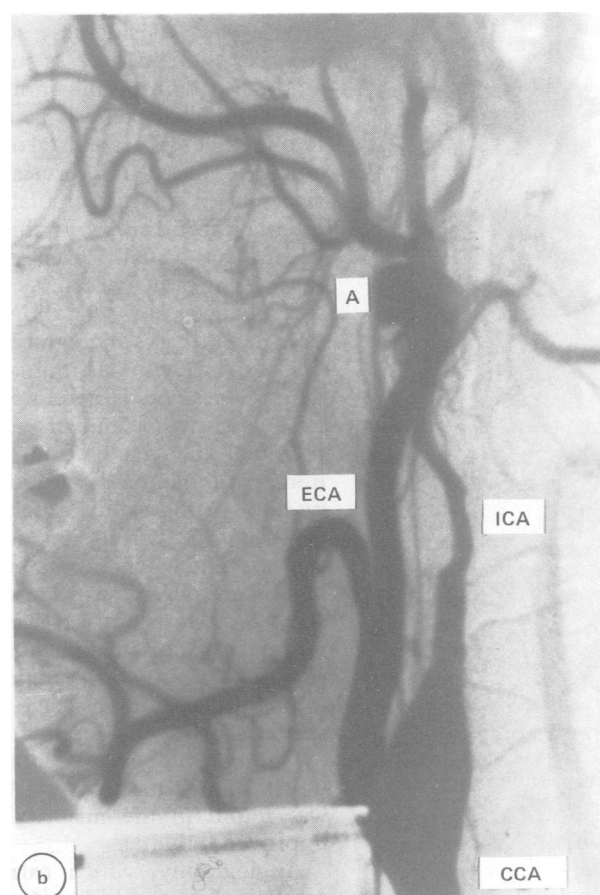
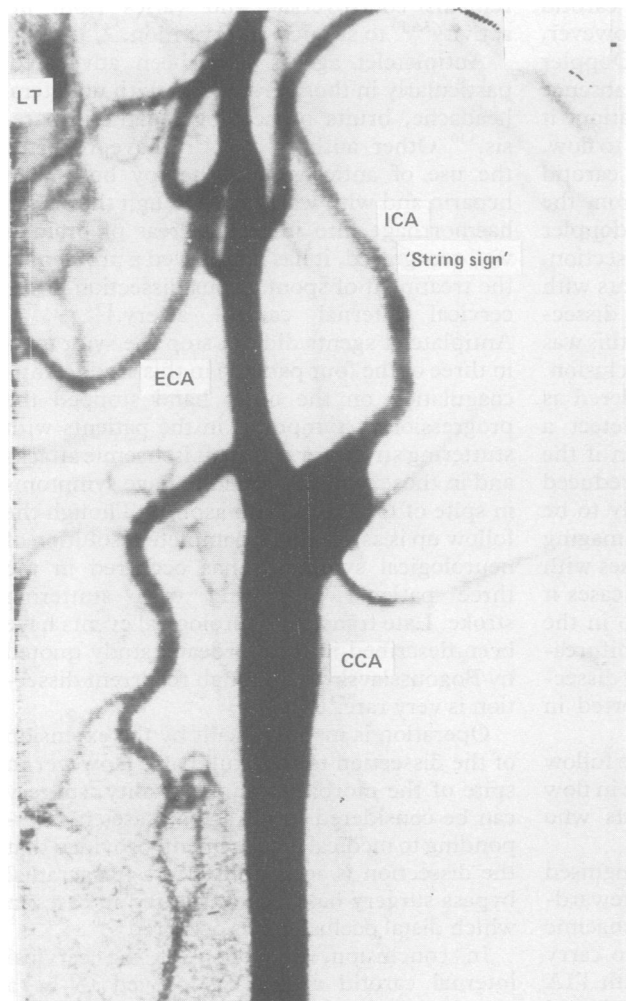


Figure 4 Angiogram a: string sign, b) aneurysm (A). CCA—common carotid artery, ICA—internal carotid artery, ECA—external carotid artery.

Table 3 Treatment and follow up

Case No	Therapy Anti-Coag/Platelet		Time Ictus/ Therapy	Doppler Flow Change	Outcome	Follow up after this therapy
1	+	-	13 days	improved	good	14 mths
2	+	-	10 days	improved	good	31 mths
3	+	-	20 days	none	good	16 mths
4	+	-	1 day	improved	good	15 mths
5	-	+	0 days	improved	good	22 mths
6	+	+	4 mths	none	old R hemiparesis	12 mths
7	+	+	24 mths	none	poor vision	14 mths
8	+	+	2 mths	improved	good	14 mths

Cases 6, 7, and 8 all had further ischaemic events on aspirin and were therefore anticoagulated.

Good outcome = full functional recovery and no further ischaemic events following establishment of therapy.

Cases 6 and 7 continued with their pre-anticoagulation deficit.

syndrome) which is thought to be one of the cardinal signs, is present in 21–58%. Both may spontaneously resolve in the first week.^{3 10 13 14} Carotid bruits were heard in one case but no angiographically proven case demonstrated an oculosympathetic paresis.

The Doppler/Duplex examination is a simple non invasive test, carrying little risk and in experienced hands is a reliable method of screening patients presenting with carotid disease.²² Eight cases of angiographically proven dissection of the cervical internal carotid artery have been discovered in young patients having strokes.

Doppler/Duplex examinations were carried out in all eight cases on presentation before angiography, and showed that the blood flow in the common and proximal internal carotid arteries was considerably reduced. However, the reduction in flow as judged on Doppler studies is a non specific finding. In the absence of a significant stenosis at the bifurcation, it implies that there is a distal obstruction to flow, either in the distal cervical internal carotid artery or in the intracranial run off from the internal carotid artery. In this unit this doppler finding has usually been due to dissection, there being only two other young patients with such reduced flow who did not have a dissection; in both cases angiography showed this was due to intracranial multi-vessel occlusion. These cases could therefore be considered as false positives. Doppler will only detect a proximal flow change from a dissection if the lumen of the internal carotid artery is reduced by 60% or more; this criterion is likely to be fulfilled if the “string sign” is present. Imaging the vessel may help in some of these cases with less severe degrees of stenosis; in two cases it showed a consistent intravascular echo in the internal carotid artery just distal to the bifurcation (fig 3). This was thought to be the dissection. Similar findings have been reported in vertebral artery dissection.²³

Doppler was also used as an objective follow up test to monitor progress. An increase in flow was found in the majority of patients who improved (table 3).

Angiography, which carries a recognised mortality and morbidity,²⁴ is often unrewarding in the young when investigating ischaemic disease. We believe it is not justified to carry out angiography in all young patients with TIA or stroke not attributable to subarachnoid haemorrhage. Nor do we usually angiogram patients with a dense stroke; there was one

other young patient with a left hemiplegia and right Horner’s syndrome with reduced flow in the right carotid artery in whom angiography was not performed, because his deficit was complete. It is therefore not possible to determine a false negative rate for Doppler/Duplex scanning in carotid dissection. All the cases reported here had TIAs or a partial deficit; angiography was performed on the basis of the Doppler/duplex findings before a decision about anticoagulation. Each showed the “string sign” and half, distal occlusion. The frequency of the “string sign” was similar to previously reported series but distal occlusion seems to be two and five times more common in this group.^{2-4 10 11 13 14}

Management of internal carotid dissection remains controversial and varies from inactivity^{3 4 10} to surgical intervention.^{2 4 10 11 14 25 26}

Antiplatelet agents have been advocated, particularly in those presenting with unilateral headache, bruits or oculosympathetic paresis.^{3 10} Other authors^{5 6 10 13 27-29} have reported the use of anticoagulant therapy both with heparin and with warfarin. Though the risk of haemorrhage into infarcted areas of brain is well recognised, it has not proved a problem in the treatment of spontaneous dissection of the cervical internal carotid artery.^{3 5 6 10 13 27-29} Antiplatelet agents did not stop the symptoms in three of the four patients in this study. Anticoagulation on the other hand stopped the progression of symptoms in the patients with stuttering strokes or transient ischaemic attacks and in those who continued to have symptoms in spite of treatment with aspirin. Though the follow up is as yet short, complete resolution of neurological symptoms has occurred in the three patients presenting with stuttering stroke. Late transient neurological events have been described in the Bordeaux study quoted by Bogousslavsky,³⁰ although recurrent dissection is very rare.^{13 19}

Operation is made difficult by the extension of the dissection to the skull base. However in spite of the morbidity and mortality, surgery can be considered in refractory cases not responding to medical management, provided that the dissection is accessible. Extra-intracranial bypass surgery has been advocated in cases in which distal occlusion has occurred.^{3 10}

In conclusion, dissection of the cervical internal carotid artery has proved to be a common cause of stroke or transient ischaemic attack in the young patient presenting to this unit. More minor cases may well go undetected.

ted. Doppler/Duplex ultrasonography would seem to be a useful screening test and helps make the decision as to whether one should proceed to angiography in this young age group. Anticoagulation would seem to be the treatment of choice in those with recurrent or progressive hemisphere neurological symptoms and signs whose deficit is not complete.

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