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Non-invasive diagnosis of internal carotid artery dissections

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Abstract

Arteriography is thought to be mandatory for the diagnosis of internal carotid artery (ICA) dissection. With the introduction of transcranial Doppler sonography (TCD) and magnetic resonance imaging (MRI), however, this is no longer the case. In 13 consecutive patients with ICA dissections the diagnosis was made by means of non-invasive tests including extracranial and transcranial Doppler sonography, contrast enhanced computed tomography (ceCT), and, in five patients, MRI. Intra-arterial digital subtraction angiography used as the gold standard in all cases was confirmative. Extracranial and transcranial ultrasound findings indicative of the diagnosis could be identified. MRI directly demonstrated the intramural haematoma and the false lumen of the dissected artery. These non-invasive techniques also allowed for repetitive follow up examinations. They were, however, unable to demonstrate false aneurysms in the chronic state. Results show that the diagnosis of carotid dissection can be made by means of cerebrovascular ultrasound and MRI.

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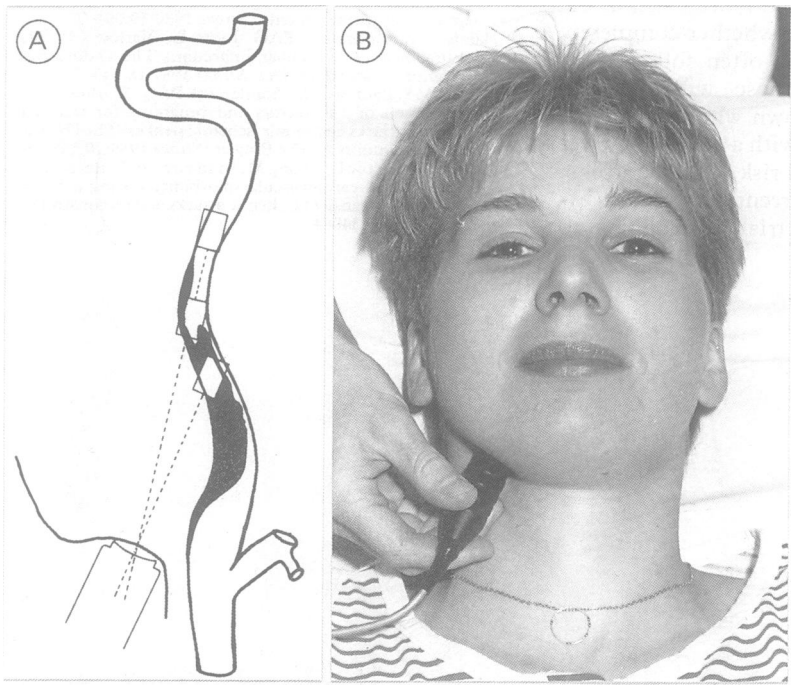


Figure 1 Submandibular approach to distal internal carotid artery by transcranial Doppler sonography. (a) Schematic diagram of dissected ICA. Probe is placed in submental triangle. Three different positions of sample volume are shown (black silhouette indicates false lumen). (b) Practical application.

Spontaneous dissecting aneurysms of the internal carotid artery (ICA) and other brain-supplying arteries are an essential cause of acute stroke (1%) particularly in the young (5%).[1] Clinical signs leading to the diagnosis are well known.[1] Up to now, cerebral intra-arterial angiography was thought to be mandatory for diagnosing dissecting aneurysms,[1–3] while only a few reports referred to the non-invasive diagnosis of ICA dissections by means of ultrasound Doppler sonography,[4–10] contrast enhanced computed tomography (ceCT)[3 11–14] and magentic resonance imaging (MRI).[10 12 15 16] In the past three years we have used these non-invasive methods in 13 consecutive patients with ICA dissections to make the initial diagnosis and to perform close-meshed follow up studies and compared these findings with those of subsequent intra-arterial selective cerebral arteriography. This was done to evaluate non-invasive techniques by the conventional gold standard and to clarify how far angiography may be dispensable.

Patients and methods

Clinical evaluation—History of premonitory complaints and clinical findings were evaluated on admission, and outcome was checked for at least three months. The longest observation period was 28 months after the stroke. Characteristic clinical signs for presumptive diagnosis were ischaemic strokes, either transient or completed, and accessory symptoms due to direct damage of neighbouring structures by the dissected vessel wall such as carotidynia, ipsilateral head pain, Horner's syndrome, cranial nerve lesions, and pulsatile tinnitus.

Sonography—All ultrasound examinations were done before arteriography—that is, investigators were not aware of the final diagnosis. Extracranial cw-Doppler sonography of all brain-supplying arteries in the neck was performed initially in 11 of the 13 patients with a zero-crossing flowmeter with a bidirectional 4 MHz Doppler device (Delalande D-800^{R}). In 10 patients, an initial transcranial pulsed Doppler ultrasound examination was also performed (EME TC 2–64^{R}, 2 MHz) with insonation of the retromandibular ICA segment from a submandibular approach (fig 1), transorbital insonation of the carotid siphon, and transtemporal access to the middle, anterior, and posterior cerebral arteries (MCA, ACA, PCA), as well as the very distal part of the ICA (C1-segment). Transcranial insonation of the vertebrobasilar system from suboccipital

Table Initial and follow up laboratory findings in 13 patients with internal carotid artery dissection

	Side affected	*CT*	*Angiography*	*MRI*	*Doppler sonography*	*Other tests*
Case 1						
Initial	Left	TI	Pseudo OCC	Positive	Retromandibular pseudo-occlusion	Positive Duplex sonography
After 2 weeks				Normal	Retromandibular high flow	
Case 2						
Initial	Right	TI	80% ST		80% ST retromandibular, STA retroflow, siphon ST	
After 3 weeks			VWI		Siphon low flow	
Case 3						
Initial	Left	TI	90% ST		90% ST, siphon ST, anterior crossfilling	Positive ceCT
After 4 weeks					Normal	
Case 4						
Initial	Left	TI	OCC		Retromandibular OCC	Negative Duplex sonography
After 4 weeks			VWI	Positive	Retromand turbulences, siphon ST, anterior crossfilling	
After 9 weeks					Normal	Negative Duplex sonography
Case 5						
Initial	Right	LFI	OCC		Not performed	Negative Duplex sonography
After 4 weeks					Retromand low grade ST, STA retrograde flow	Negative Duplex sonography
Case 6						
Initial	Left	LFI	OCC	Positive	Retromand OCC, STA retroflow, low flow MCA, anterior crossfilling	
After 4 weeks			VWI		Normal	Negative Duplex sonography
Case 7						
Initial	Left	LFI	OCC		Retromand OCC, MCA + ACAs high flow	
After 28 months					Identical	
Case 8						
Initial	Left	Normal			Retromandibular 90% ST, siphon ST	
After 2 weeks			VWI	Positive	70% Retromandibular ST	
After 4 weeks					Normal	
After 13 months				Normal		
Case 9						
Initial	Left	Normal	ST			
After 7 months				Normal	Normal	
Case 10						
Initial	Left	LFI	Tandem ST		Retromandibular 80% ST, siphon ST, MCA low flow, anterior crossfilling	50% ST on Duplex sonography
After 5 weeks					Extracranially idem, transcranially normal	
Case 11						
Initial	Left	TI	OCC		Retromandibular OCC, oscillating flow in siphon	
After 20 weeks					Normal	
Case 12						
Initial	Left	Normal	ST		80% Retromandibular ST, siphon ST, MCA low flow	Positive ceCT
After 6 days		LFI			ICA OCC, STA retroflow, MCA low flow, crossfilling	
After 2 months					Identical	Thrombus on Duplex sonography
Case 13						
Initial	Left	TI		Positive	Retromand high flow, siphon ST, MCA low flow	Negative Duplex sonography
After 2 weeks			VWI	Better	Retromand high flow, transcranially normal	

TI = territorial type of brain infarction.
VWI = vessel wall irregularity.
ST/OCC = stenosis/occlusion.
ceCT = contrast enhanced CT.
LFI = low flow induced brain infarction.
MCA/ACA/PCA = middle/anterior/posterior cerebral artery.
STA = supratrochlear artery.
Retromand = retromandibular.

approach was also performed. Methodological details of these techniques are published elsewhere.[7 16 17] Abnormal flow was diagnosed if flow velocities were either two standard deviations lower or higher than normal values.[16] In five patients B-mode imaging of the carotid bifurcation and proximal segment of the ICA was also performed (Biosound 2000 SA^{R}, 7·5 MHz).

Intra-arterial angiography was performed in all cases, in 11 on admission and in two after two weeks. The common carotid artery was catheterised selectively via a transfemoral approach. The injection volume of a non-ionic contrast medium was 5 to 7 ml. At least two views of the carotid axis were imaged on digital subtraction angiograms. In each case, a biplane vertebrobasilar imaging series was also performed by selectively injecting into the left (10) or right (3) vertebral artery.

Cranial computed tomography (CT) was done in every patient with a Somatom DRH-scanner (Siemens^{R}, matrix 512*512). The infratentorial slice thickness was 4 mm and 8 mm supratentorially. Contrast enhancement was used to visualise the carotid artery with its bifurcation and the siphon.[11-13] Cerebral infarctions visible on CT were categorised according to a classification system recently published elsewhere.[17-19] Territorial infarcts, which were thought thromboembolic in origin and haemodynamically caused low flow infarcts, were differentiated.

MRI with visualisation of the cervical ICA was performed in six patients (Siemens Magnetom^{R}, 1·5T). Images were T1- and

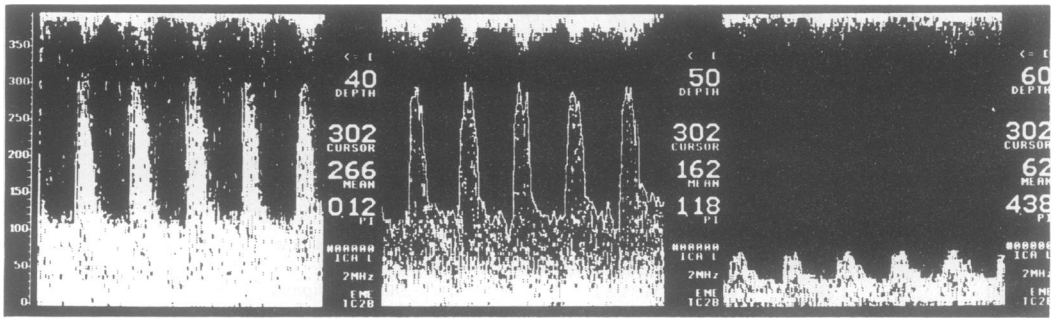


Figure 2 Typical transcranial Doppler sonographic finding in ICA dissection. Left and middle: submandibular insonation reveals high flow velocities (266 and 162 cm/s) at 40 and 50 mm insonation depths, indicating high grade stenosis. Right: poststenotic low flow (62 cm/s) at 60 mm depth.

proton-weighted, as well as T2-weighted. Slice thickness was 5 mm without gap. The internal carotid artery was visualised transversally and also longitudinally.

Sonographic follow up investigations were performed in 12 of the 13 cases by extracranial and transcranial Doppler sonography. In six cases, repeat arteriograms or repeat MRI or both were also taken.

Results

PATIENTS AND CLINICAL SIGNS

Five women and eight men (mean (range) age 46 (20–58) years with isolated unilateral dissection of the left (12) and right (1) ICA were included in the study. Four patients had suffered an unequivocal whiplash injury of the neck or a slight trauma without direct involvement of head and neck one to nine days before the occurrence of suggestive clinical symptoms. In the nine other patients dissections had occurred spontaneously. Vascular risk factors were present in eight cases; five were smokers, two women took contraceptive pills, and one had diabetes mellitus. Three patients had two vascular risk factors; three had common migraine.

Clinical signs and symptoms were quite variable. Six patients had suffered a complete stroke, all of them with premonitory attacks. One patient had suffered transient ischaemic attacks (TIA), five had prolonged reversible ischaemic neurological deficits with preceeding TIA in three of them. While eight patients only had ischaemic events without any accessory symptom, one presented with accessory signs only—that is, she had pulsatile tinnitus, carotidynia and temporal headache. Thus the combination of both was present in four of the 13 patients. Four patients complained only of pain located either temporally, retro-orbitally, or in the submandibular and carotid region. Another patient showed involvement of more than one cranial nerve presenting with trigeminal dysaesthesia, pulsatile tinnitus, and Horner's syndrome. All patients underwent therapeutical heparinisation with PTT 2 to 3 times normal control. Initial and follow up findings are summarised in the table.

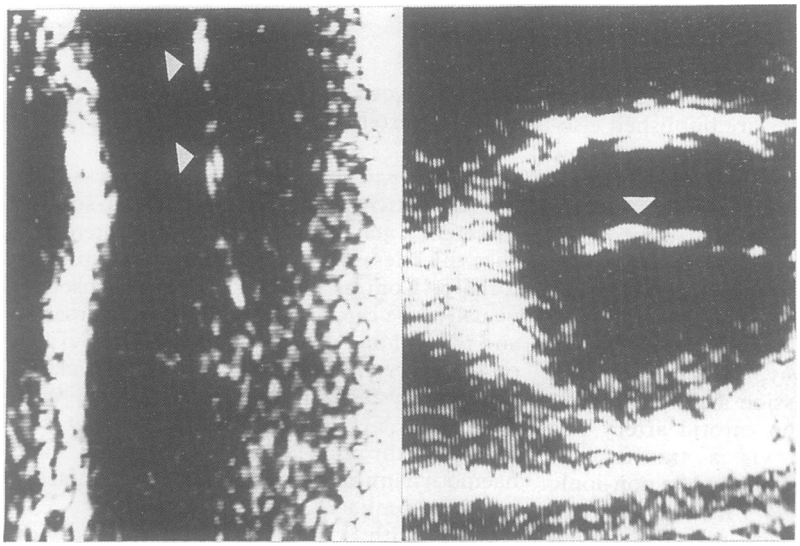
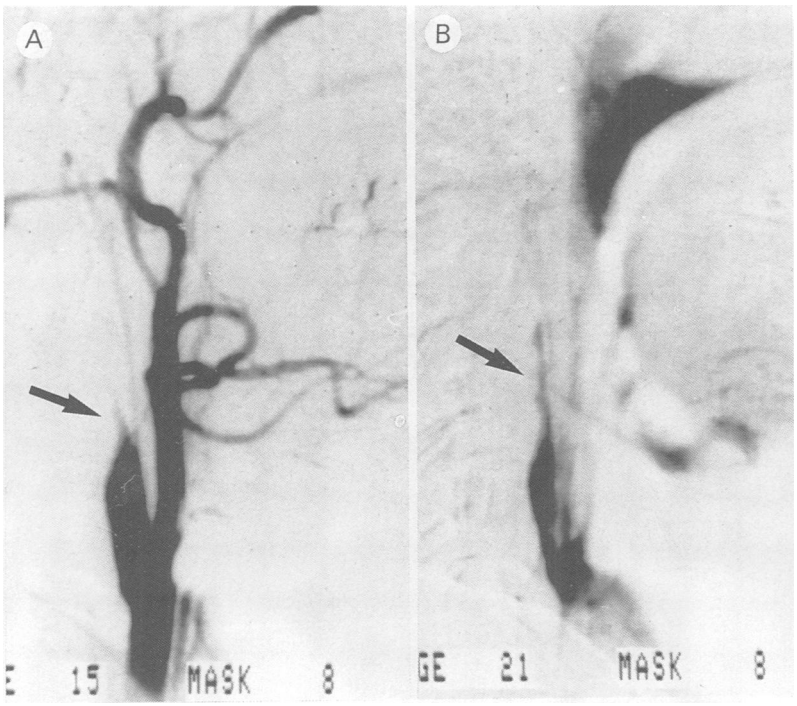


Figure 3 B-mode imaging of ICA dissection in longitudinal (left) and transversal (right) views. Intimal flap (arrows) oscillated within blood stream.

TECHNICAL FINDINGS

Extracranial cw-Doppler sonography performed in 11 patients before cerebral arteriography showed a high-grade ICA stenosis retromandibularly at least 2 cm distal to the bifurcation in six cases. This finding was thought to be characteristic of an ICA dissection in terms of type and location of the lesion. Four other patients showed to-and-fro movement of the blood column in the proximal ICA, possibly indicative of a blind arterial stump due to complete occlusion of the distal ICA.[7 19] Only a tentative diagnosis of ICA dissection was made in these patients as similar findings may also be seen in embolic ICA occlusions. In one case a 90% ICA stenosis was detectable immediately at the carotid bifurcation, suggesting arteriosclerotic disease rather than dissection. During submandibular insonation of the ICA by means of transcranial Doppler sonography each of the seven extracranial stenotic lesions could be traced further distally, some of them even extending up to the pars petrosa of the carotid artery. The typical sonographic finding was a high-flow velocity signal and post-stenotic disturbed flow indicated by bidirectional low frequency components of the flow signal (fig 2). In three cases, pronounced harmonic post-stenotic covibrations of the affected vessel wall segments generated so-called "musical murmurs".[20] In two patients an intracarotid valve mechanism with functional ICA occlusion and stoppage of flow



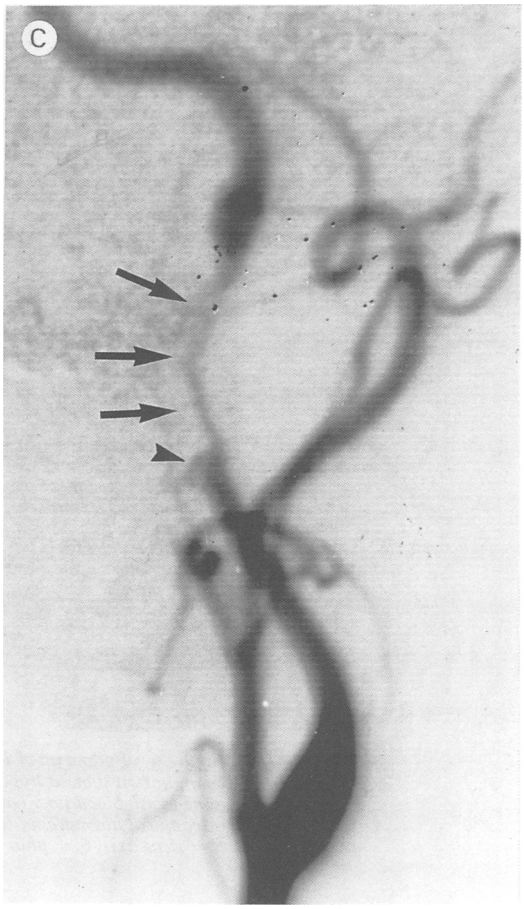


Figure 4 Typical angiographic appearance of ICA dissections. (a/b) Gradually tapering ICA occlusion (arrows) during early and late phase of angiogram. (c) Long filiform ICA stenosis exceeding 2 cm in length at base of skull (arrows) and pseudoaneurysmatic dilatation of artery (arrow head).

during diastole but a short-lasting jet-like flow signal during systole could be detected. During the additional transtemporal insonation of the middle cerebral artery in nine patients, low-flow became obvious in five cases.

B-mode sonography of the ICA in the neck was performed in five cases. A false lumen with an intimal flap could be shown in only one of them (fig 3). In the other cases the carotid bifurcation and the proximal 1 to 2 cm of the ICA appeared normal. In two cases flow signal within the proximal ICA appeared abnormal with lack of diastolic or even reversed blood flow indicating high peripheral resistance. These patients turned out to have either a distal ICA occlusion or a pseudo-occlusion. An initial arteriography was performed after ultrasound investigations in 11 patients within 24 hours after admission and in two within the first two weeks. Angiograms showed five ICA occlusions, four of them with the typical gradually tapering appearance of the proximal stump (fig 4a, b). Eight ICA stenoses of variable extent were also seen (fig 4c). Four of them had an extraluminal pouch of contrast medium close to a tight stenosis beyond the base of the skull. In the other cases the appearance of the lesions was also suggestive of but less specific for ICA dissection, and final diagnosis was based on the rapid resolution of the lesion during follow up. Angiograms were in accordance with Doppler sonography findings in every case. The type of vessel lesion (occlusion *v* stenosis) did not correspond to certain clinical symptoms or the severity of the deficits.

Brain imaging by means of cranial CT showed low flow infarctions in five and thromboembolically induced territorial infarctions in six cases,[18] but was normal in two. (Details are published elsewhere[21]). The type and extent of brain infarctions did not depend on the type of vessel lesion.

Direct imaging of the affected carotid artery by means of contrast enhanced CT revealed highgrade lumen narrowing in only two cases. Both ultrasound and arteriography were confirmative. In five patients dissection of the artery

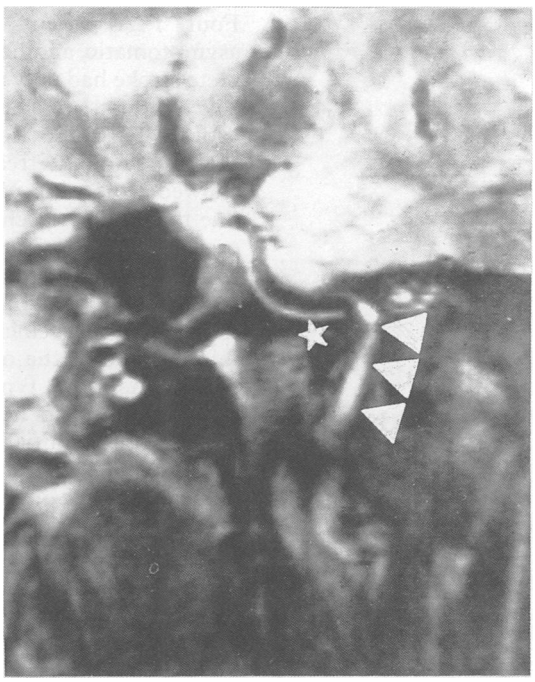


Figure 5 T2-weighted paraxial image of ICA dissection. Hyperintense vessel wall signal (arrow heads) beyond pars petrosa of skull (asterisk) is abnormally broad and indicates intramural haematoma.

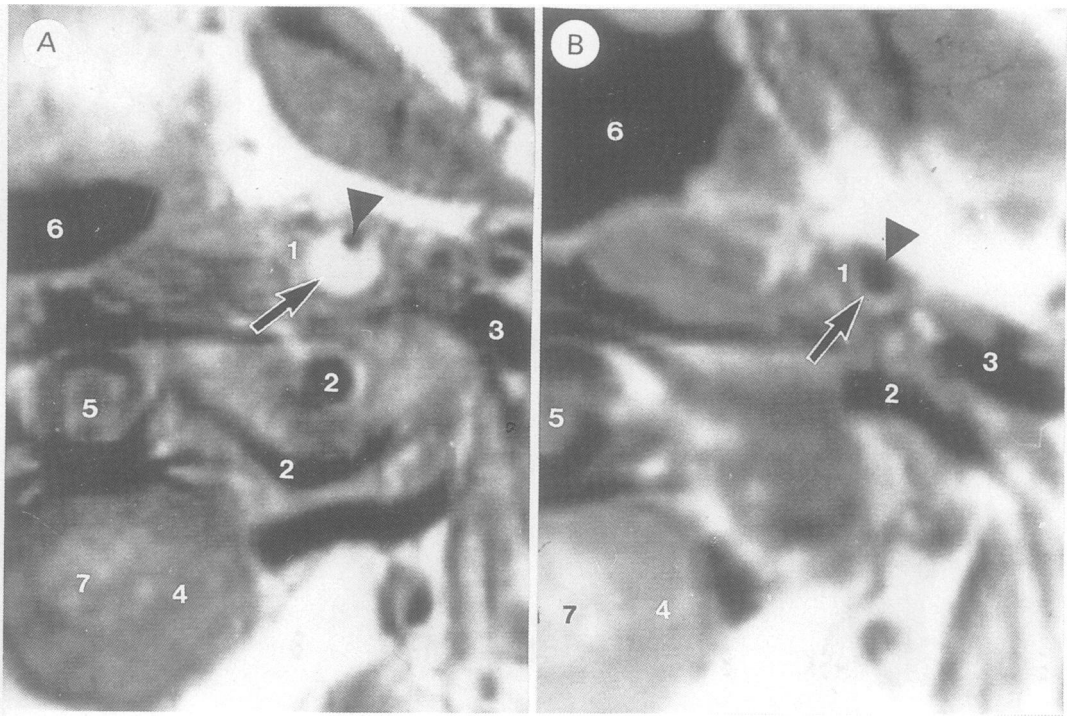


Figure 6 Follow up of ICA dissection by means of MRI imaging (T2-weighted, horizontal cut). (a) Initial examination showed hyperintense sickle-like vessel wall haematoma (arrow) leaving only small residual, hypointense lumen with blood flow (arrowhead). (b) Two weeks later, ICA lumen (arrowhead) nearly restored by resorption of intramural haematoma (arrow). 1 = internal carotid artery, 2 = vertebral artery, 3 = jugular vein, 4 = spinal fluid, 5 = dens axis, 6 = pharynx, 7 = medulla.

could be pinpointed directly by MRI. The intramural haematoma presented as a hyperintense vessel wall signal in both T1- and T2-weighted images (fig 5). Again, findings were in accordance to Doppler sonography and arteriography.

CLINICAL AND LABORATORY FOLLOW UP FINDINGS

Four TIA patients subsequently remained asymptomatic, another four patients with complete stroke had only minimal residual deficits. Five patients remained considerably handicapped by their ischaemic deficit. During the course of the treatment, only one patient showed worsening of the neurological deficit due to complete occlusion of an initially stenosed ICA. Transcranical Doppler sonography showed normalisation in seven cases or, at least, severe reduction of the lesions in five cases. The degree of improvement did not depend on the initial grade of the occluding lesions. In one patient the initial typical occlusion proved by arteriography did not improve. In only one patient did the initial 80% stenosis progress to an occlusion. There was no relation between clinical outcome or angiological follow up findings and the initial severity of the ICA lesion. Day by day transcranial Doppler studies also revealed rapidly improving intracerebral haemodynamics paralleling ICA blood flow normalisation. Repeat angiography in four patients showed normalisation of the vessel lumina. In four other cases follow up studies by MRI also showed normalisation of the vessel wall (fig 6) corresponding to the transcranial Doppler findings.

Discussion

We studied 13 patients with isolated ICA dissection by means of a battery of non-invasive and invasive tests. The causes of the vessel lesion were whiplash injury of the neck and questionable blunt trauma in four patients, the remainder suffering from idiopathic spontaneous ICA dissections. In no patient did arteriography deliver indications for fibromuscular dysplasia, Marfan's syndrome, or cystic medionecrosis as the underlying vessel disease.[22-27] Hypertension, cigarette smoking, migraine, and contraceptive pills are thought to be risk factors for ICA dissections[2 28] and were present in eight of our patients. In one patient, however, the dissection may have been atherosclerotic in origin, as arteriograms showed an ulcerated atherosclerotic plaque immediately next to the proximal end of the tapering stenosing dissection; while the latter disappeared, the plaque remained. Clinically the combination of an acute stroke including TIA with so called accessory symptoms, like Horner's syndrome, pulsatile tinnitus, temporal pain, and carotidynia, strongly suggests an underlying ICA dissection.[1 2 9 10 14 15 28-31] Just one third of our patients, however, showed this combination of symptoms, suggesting why the true nature of the initial signs may be overlooked and that technical investigations are necessary to lead to the correct diagnosis.

Up to now carotid arteriography was thought to be mandatory for the diagnosis of ICA dissection.[1-3] Findings thought diagnostic[32] are an extraluminal pouch filled with contrast material; a double lumen; a smooth or scallopped narrowing of the ICA distal to the

carotid sinus; a long filiform stenosis exceeding 2 cm in length; and a gradually tapered occlusion. Some of these signs, however, are less specific and do not unequivocally prove an ICA dissection in every case.[1 2 27 32 33] Only eight of our 13 patients showed arteriographically pathognomonic tapering occlusion or long filiform stenosis. Other findings were suspicious but in these cases follow up findings proved the diagnosis correct. There is a need for diagnostic tools which are both non-invasive and reliable as arteriography during the acute phase bears a substantial risk of embolic complications.[35] Extracranial Doppler sonography, which was performed in 11 cases, showed a retromandibular high grade stenosis of a typical finding in most of the cases. The site and type of this lesion is highly suggestive of ICA dissection, particularly in the young and if risk factors or signs of atherosclerosis are lacking. Flow abnormalities indicating a blind ICA stump suggest ICA occlusion distal to the bifurcation. This finding is less conclusive as to the underlying vascular disease[11] but rather suggests dissection than distal embolic ICA occlusion. Atherothrombotic ICA occlusion at this site is rare.[35]

Transcranial Doppler sonography turned out to be a sensitive technique for detecting arterial disease inaccessible to conventional Doppler tests[36] and provided quite characteristic findings for detecting ICA dissection. Stenoses were identified in all cases. The location of the lesions, either stenosis or occlusion, was also highly suggestive for the diagnosis. There was no false positive finding, although there were too few cases for statistical analysis. Definite proof of the diagnosis might be achieved during repetitive TCD tests in that both stenoses and occlusions had a strong tendency to improve over time.[1] Day by day ultrasound checks demonstrated rapid ICA recanalisation in all but one case. A systolic valve mechanism with transient occlusion of the distal ICA during diastole also seems to be a specific TCD finding as to our knowledge it has not been described as a transient phenomenon in atherosclerotic ICA stenoses or pseudo-occlusions.[37] ICA dissections were also found to have considerable impact on intracranial haemodynamics in that reduced intracranial perfusion pressure led to severe slowing of blood flow velocity.[21] Duplex scanning allows for rapid visualisation of ICA in the neck by B-mode imaging. There are, however, limitations of this technique. Due to the distal localisation of the lesions[1 7 32 33] the characteristic intimal flap may be shown only in a limited number of patients; we succeeded in only one out of five patients. Sector scanners with small transducers have been reported to be more sensitive for detecting a dissected ICA wall.[4-9]

Early MRI was performed in five cases and led to the correct diagnosis in all of them, as was proven by simultaneous arteriography in four cases and by angiological follow up investigations in all. The intramural haematoma was indicated by a hyperintensive arterial wall signal on both T1- or proton-weighted and T2-weighted images, as has been reported by others.[11] Other stenosing processes like fibromuscular dysplasia, vasculitis, or atherosclerosis may also be differentiated by MRI.[14 15] The false vessel lumen can directly be visualised and separated from the flowing blood column in both the residual and false lumen.[11] In our study, five initial and four follow up studies of ICA dissections exactly corresponded in site and severity to Doppler and angiographic findings, indicating the high diagnostic reliability and validity of this technique. In contrast, enhanced CT imaging of ICA in the neck, as a rule, did not prove to be diagnostic.

The diagnosis of an ICA dissection is undoubtedly confirmed by rapid day by day regression of the arterial lesion.[1 33] We showed this by monitoring lesions by close-meshed, repetitive transcranial Doppler sonography studies. This is also true for progression of a stenosis, which eventually may occur. Repeat MRI scans in four cases also showed normalised or nearly normalised arteries. Whether treatment with therapeutic heparinisation or antiplatelet agents will prompt a better outcome is still a matter of debate.[1 2 9 22 23 32 39] Interestingly, infarctions due to carotid artery dissections are as commonly thromboembolic in origin as haemodynamic.[21] This may influence the type of treatment.

In conclusion, patients with acute strokes, particularly the young, and those presenting with accessory symptoms should be subjected to intense extracranial and transcranial ultrasound checks by means of ECD, TCD, and, if available, B-mode imaging. The submandibular approach of transcranial Doppler sonography is most promising, the characteristic finding in ICA dissection being an extended distal stenosis. In the case of distal extracranial ICA occlusion, with proximal "blind stump" phenomenon, the diagnosis is highly suggestive. Follow up ultrasound examinations often deliver further diagnostic clues as dissections tend to regress (or progress) rapidly. Findings may be further clarified by means of MRI as it may demonstrate the underlying vessel wall disease. Both procedures are non-invasive, reliable, and quick. From our point of view, arteriography should be confined to certain cases in the post-acute period when regression of the lesion has already taken place. At this time, residual aneurysmata spuria may be found, which require surgical removal.[30] Based on our experiences, four further cases of ICA dissection have been diagnosed non-invasively by both transcranial Doppler sonography and MRI. Follow up findings strongly suggested the diagnosis to be correct.

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