

EMBOLIC STROKE DUE TO INTERNAL CAROTID DISSECTION: NONINVASIVE MONITORING OF RECANALIZATION BY COLOR DOPPLER FLOW IMAGING AND TRANSCRANIAL DOPPLER

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SUMMARY – Craniocervical arterial dissection is an under-recognized and uncommon cause of stroke. We describe a 66-year-old hypertensive female patient who developed internal carotid artery dissection at an atypical location, with consequential ischemic stroke. Double lumen was visible by color doppler flow imaging, and high-intensity transient signals were detected. Transcranial doppler ultrasonography was used to monitor cessation of ipsilateral distal microembolization associated with clinical improvement on anticoagulant therapy. In embolic stroke due to embolization from a dissected internal carotid artery, neurosonologic investigations enabled noninvasive visualization and monitoring of the time course of vessel dissection and secondary embolism.

Key words: *Carotid artery diseases, ultrasonography; Aneurysm dissecting, diagnosis; Ultrasonography – aneurysm dissecting, complications*

Introduction

Craniocervical arterial dissection is a relatively uncommon cause of stroke in the general population¹. Internal carotid artery (ICA) dissections seem to be much more frequent than vertebral artery dissections², and multiple artery involvement is quite common (8%-60%)¹. The cited incidence is 1-3 cases *per* year in large referral-based hospitals²⁻⁴. There is a possibility of under-recognized vertebral artery dissections⁵. Early manifestations of dissection may often be misleading, however, permanent neurologic disability and death can ensue if the diagnosis is delayed. Early detection is therefore of utmost importance to minimize the associated morbidity and mortality.

The pathogenesis of spontaneous dissection of ICA includes a broad spectrum of diseases, mostly hereditary,

that affect the vessel wall¹. The history of trauma is often negative^{4,6}, or only a mild trauma is present⁵. Hypertension is a risk factor in the elderly^{5,6}. Most patients had no risk factors^{4,6}.

A traditional method of imaging cervical cephalic arterial dissection was selective angiography, where findings may include smooth or slightly irregular luminal narrowing, pseudoaneurysm, intimal flap, or distal branch occlusion secondary to embolization⁷. The presence of an intimal flap at the proximal margin of the dissection or double lumen is a specific finding, however, it is seen in less than 10% of patients^{7,8}. Slow arterial filling and emboli within the parent artery or distal branches are another two angiographic features that may be seen but are present in less than 20% of cases^{7,8}. Magnetic resonance (MR) images the eccentric or circumferential periarterial rim of intramural hematoma that typically shows hyperintense signal on T1 and T2 weighted images⁷. MR angiography is of a limited value, and duplex sonography has been under-rated⁹. Although color doppler has shown good results in

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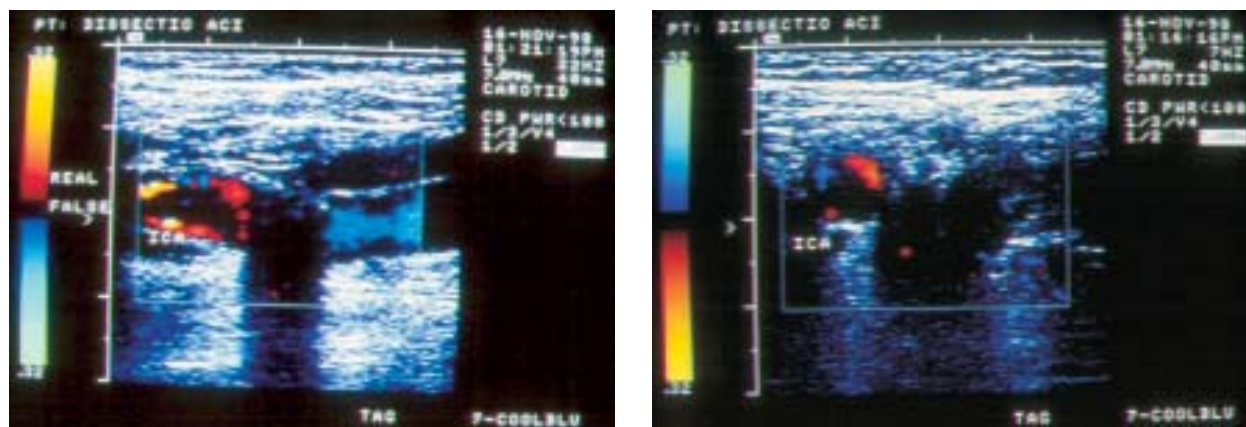


Fig. 1A. Longitudinal (A) and transverse (B) planes show a 2-cm long hematoma stenosing the left internal carotid artery. Under the hematoma, a tiny color coded flow is visible.

the visualization of dissection¹⁰⁻¹², its limitations refer to the localization of dissection, which may be V3, V4 segments of the vertebral arteries, or intracranial segment of ICA¹.

The possibility of detection of embolic signals suspected to cause ischemic symptomatology has been reported in a small proportion of patients¹³⁻¹⁵. Only in the case report of Babikian *et al.*¹⁴, transcranial doppler (TCD) ultrasonography was used to monitor ipsilateral distal microembolization associated with clinical deterioration.

We report on a patient in whom the use of color doppler flow imaging (CDFI) of carotid arteries and TCD monitoring of high-intensity transient signals (HITS) enabled the disease diagnosis and therapy monitoring.

Case Report

A 66-year-old female patient was observed at the emergency room of the University Department of Medicine, for the acute onset of left-sided thoracic pain radiating to the neck. Blood pressure was 220/105 mm Hg. Electrocardiography showed atrial fibrillation (AF) and no signs of cardiac ischemia. The patient had been suffering from hypertension for more than 10 years, and from cardiac disease, however, not specified as AF. During the observation, sensorimotor dysphasia and right arm weakness developed, and the patient was admitted to the University Department of Neurology. Neurologic findings indicated moderate right-sided hemiparesis, weakness of nerve VII and sensorimotor dysphasia. Brain computed tomography

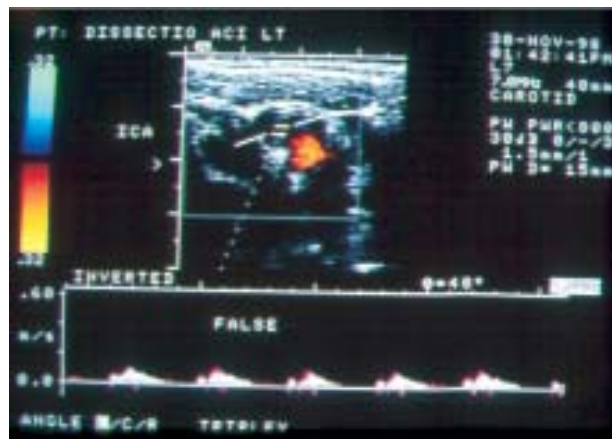


Fig. 2A. Transverse plane shows two color coded lumina in the internal carotid artery. In false lumen, no end-diastolic flow was visualized in hemodynamic spectra indicating distal flow obstruction (PI=2.60).

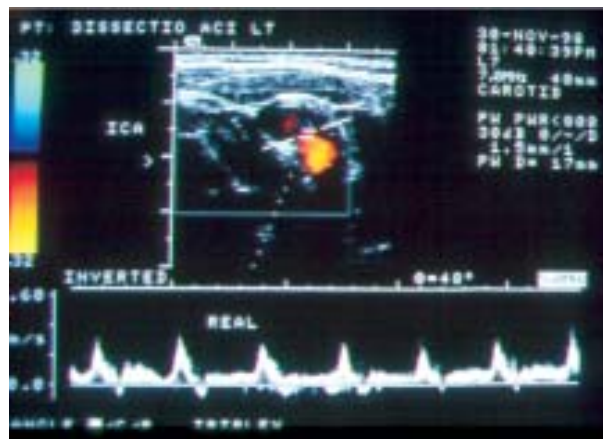


Fig. 2B. Transverse plane showing true lumen which also has a high resistance flow pattern, however, positive end-diastolic flow is present throughout its length although there is a retrograde early diastolic flow.

(CT) scan showed two ischemic lesions, one in the parietal and temporal lobe each, both irrigated by the left middle cerebral artery. CDFI of the carotid arteries showed a 2-cm long homogeneous mass that subtotally stenosed the left ICA, visible in longitudinal (Fig. 1A) and transverse (Fig. 1B) plane. Under the plaque, a tiny string of color coded flow was detected (Fig. 1A-B), with the spectra characteristic of false lumen (attenuated hemodynamic spectra, absent diastolic flow) (Fig. 2A). In the true lumen, a higher resistance pattern was observed (Fig. 2B). TCD monitoring during a 1-hour period showed 8 HITS on the left (12-15 dB) and none on the right side. The ultrasonographic findings were confirmed by selective digital subtraction angiography (Fig. 3). The patient was administered heparin subcutaneously¹⁶. CDFI was repeated at 7, 14, 17 days, then at 1 and 3 months. TCD monitoring was performed on day 3, then at 1 and 3 months.

Control CDFI performed in a week showed regression of the mass within the vessel, almost complete recanalization on day 17 (Fig. 4A-B-C), and complete recanalization in one month. The finding was similar at 3 months. TCD monitoring performed on day 3 revealed 2 HITS (13 dB) on the left and none on the right side. At 1 and 3 months, no HITS were observed.

The patient was discharged on day 17, with warfarin prescribed. Neurologically, there was mild right hemiparesis. At 3 months, mild right hemiparesis was the only finding.

Discussion

We documented the course of the left ICA dissection by use of ultrasonography. The lesion localization was atypical for ICA dissection, as they are usually localized a few centimeters distally to the bifurcation⁹. The diagnosis was facilitated by the presence of the string sign, which is found in 10% of ICA dissections^{1,9}. The patient was aged 66, the main risk factor probably being her longstanding hypertension. There was no trauma in the patient history. The pain localized in the left hemithorax during the hypertensive crisis, radiating to the neck was indicative of anginose pain.

Recanalization was observed by CDFI. It was possible because of the typical visible double lumen and the localization accessible to this investigation. Therefore, CDFI was used on several occasions. The reduction of clinically relevant embolic signals from the dissected ICA was observed by TCD monitoring of HITS. Such a method may prove useful for noninvasive monitoring of the effect of anticoagulant therapy or time course of the disease. Visu-



Fig. 3. Selective digital subtraction angiography shows a filling defect in the internal carotid artery.

alization of vertebral artery dissections by ultrasonography has been reported by Bartels and Flugel¹⁰. Bartels has also reported on a single visualization of the carotid artery dissection¹⁷. Visualization of the lumen and color coded flow in the first days was found insufficient, probably due to hematoma, and hemodynamic analysis revealed a high resistance pattern^{10,17}. In our patient, hematoma could be visualized and was found to have two lumina. The true lumen changed the hemodynamic spectra with a higher resistance pattern. In the false lumen, attenuated hemodynamics with absent diastolic flow was observed. During recanalization, re-expansion of the lumen with normalization of the flow was seen. The false lumen disappeared within one month of observation, and the finding remained stable during the next three months. At the same time, HITS were monitored. On admission, 1-hour monitoring recorded 8 HITS on the left and none on the right side. Anticoagulant therapy was initiated and reduction of the HITS was observed. At one month, as the false lumen visible by CDFI disappeared, HITS vanished too. HITS were the signs of clinically relevant embolic material from the dissected artery, which led to ischemic stroke visible on CT scan. According to Hennerici in Easton's *pro et contra* dis-

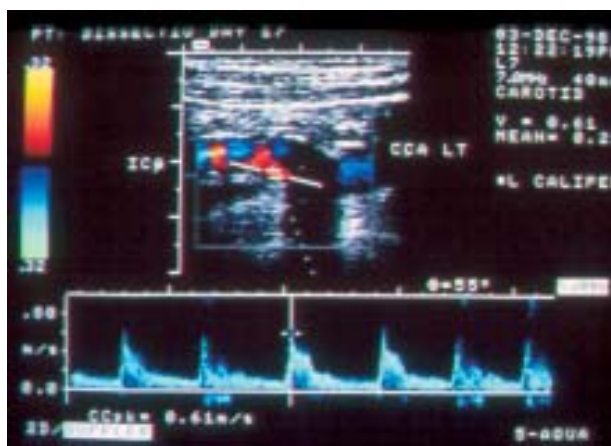


Fig. 4A. Although both false and true lumina were still visualized by day 17, arterial recanalization was in progress since the end-diastolic flow in true lumen increased, leading to improved flow pulsatility. Note the disappearance of retrograde early diastolic flow (PI=1.91).

cussion¹⁸, HITS are not emboli but features from the doppler signal spectrum which, only if associated with appropriate clinical signs/symptoms or if combined with findings from other technical studies such as DW MRI, may sometimes suggest ongoing cerebral embolism. Otherwise, they do not indicate an increased stroke risk. Our previous findings have suggested HITS to be embolic signs that are unilateral in carotid disease and bilateral in atrial fibrillation¹⁹. In our patient, CDFI revealed dissection of ICA and HITS ipsilaterally. Contralaterally, no HITS were recorded. The disappearance of HITS with anticoagulant therapy and regression of dissection suggest that HITS represent microembolic signals.



Fig. 4C. Transverse plane shows two lumina and hypoechoic septum in-between.

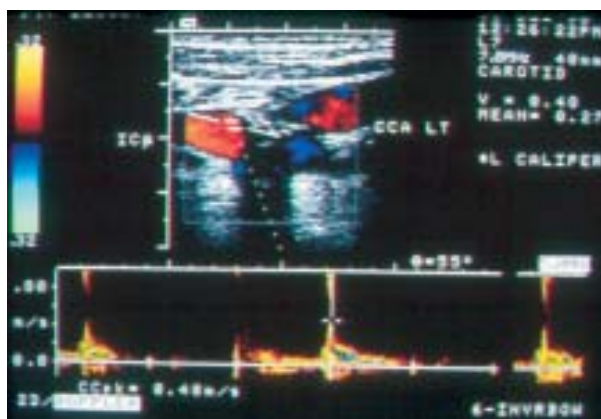


Fig. 4B. Higher resistance pattern in false lumen (PI=1.76).

In their report on six patients with carotid dissection, Koennecke *et al.*¹³ found microembolic signals in three of four patients presenting ischemic symptomatology, whereas no such signals were detected in two patients with other symptomatology. Microembolization associated with clinical deterioration has been reported in one patient with carotid dissection¹⁴. Srinivasan *et al.*¹⁵ describe the use of TCD for initial state evaluation and repeat TCD until cessation of embolic signals with treatment. Their report is based on ten patients with embolic signals out of 17 patients with either traumatic or spontaneous carotid dissection, in whom the diagnosis was made by carotid angiography.

This is the first report on the use of combined CDFI visualization of two lumina in carotid dissection and TCD monitoring of HITS in the follow-up of patient's recovery and regression of ICA dissection in parallel with the disappearance of emboli in a patient with stroke induced by ICA dissection.

References

1. PROVENZALE J. Dissection of the craniocervical arteries. In: TAVERAS JM, FERUCCI JT, eds. Radiology – diagnosis – imaging – intervention. Vol. 3. Neuroradiology and radiology of the head and neck. Philadelphia: Lippincott-Raven, 1995:1-10.
2. HART RG, EASTON JD. Dissection of cervical and cerebral arteries. *Neurol Clin* 1983;1:155-82.
3. MOKRI B, HOUSER OW, SANDOK BA, PIEPGRAS DG. Spontaneous dissection of the vertebral arteries. *Neurology* 1988; 38:880-5.
4. MAS JL, BOUSSER MG, HASBOUN D, LAPLANE D. Extracranial vertebral artery dissections: a review of 13 cases. *Stroke* 1987;18:1037-47.

5. STURZENEGGER M, MATTLE HP, RIVOIR A, RIBS F, SCHMID C. Ultrasound findings in spontaneous extracranial vertebral artery dissection. *Stroke* 1993;24:1910-21.
6. SUE DE, BRANT-ZAWADZKI MN, CHANCE J. Dissection of cranial arteries in the neck: correlation of MRI and arteriography. *Neuroradiology* 1992;34:273-8.
7. HOUSER O, MOKRI B, SUNDT TS Jr, BAKER H, REESE D. Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. *AJNR Am J Neuroradiol* 1984;5:27-34.
8. ANSON J, CROWLL RM. Cervicocranial arterial dissection. *Neurosurgery* 1991;29:89-96.
9. PROVENZALE J. Dissection of the internal carotid and vertebral arteries: imaging features. *AJR Am J Roentgenol* 1995;165:1099-104.
10. BARTELS E, FLUGEL KA. Evaluation of extracranial vertebral artery dissection with duplex color-flow imaging. *Stroke* 1996;27:290-5.
11. BLUTH EI, SHYN PB, SULLIVAN MA, *et al.* Doppler color flow imaging of carotid artery dissection. *J Ultrasound Med* 1989;8:149-53.
12. LOVRENČIĆ-HUZJAN A, BOSNAR-PURETIĆ M, VUKOVIĆ V, DEMARIN V. Sonographic features of craniocervical artery dissection. *Acta Clin Croat* 2002;41:307-12.
13. KOENNECKE HC, TROCIO SH Jr, MAST H, MOHR JP. Microemboli on transcranial Doppler in patients with spontaneous carotid artery dissection. *J Neuroimag* 1997;7:217-20.
14. BABIKIAN VL, FORTEZA AM, GAVRILESCU T, SAMARAWERA R. Cerebral microembolism and extracranial internal carotid artery dissection. *J Ultrasound Med* 1996;15:863-6.
15. SRINIVASAN J, NEWELL DW, STURZENEGGER M, MAYBERG MR, WINN HR. Transcranial Doppler in the evaluation of internal carotid artery dissection. *Stroke* 1996;27:1226-30.
16. DEMARIN V, LOVRENČIĆ-HUZJAN A, ŠERIĆ V, VARGEK-SOLTER V, TRKANJEC Z, VUKOVIĆ V, LUPRET V, KALOUSEK M, DeSYO D, KADOJIĆ D, LUŠIĆ I, DIKANOVIĆ M, VITAS M. Recommendations for stroke management. *Acta Clin Croat* 2001;40:127-54.
17. BARTELS E. Color-coded duplex ultrasonography of the cerebral vessels. Atlas and manual. Stuttgart: Schattauer, 1999:78.
18. EASTON JD. Cerebral embolism and Doppler ultrasound. *Cerebrovasc Dis* 1999;9:188-92.
19. RUNDEK T, DEMARIN V, NIEDERKORN K, VUKOVIĆ V, PODOBNIK-ŠARKANJI S, RUNJIĆ D, LOVRENČIĆ-HUZJAN A, STARČEVIĆ B, RICHTER D, PLANINC D. Prevalence of intracranial emboli signals in patients with carotid and cardiac disease. *Acta Clin Croat* 1995;34:137-43.

Sažetak

EMBOLIJSKI MOŽDANI UDAR UZROKOVAN DISEKCIJOM UNUTARNJE KAROTIDNE ARTERIJE: NEINVAZIVNO PRAĆENJE REKANALIZACIJE POMOĆU OBOJENOG DOPLERA I TRANSKRANIJSKOG DOPLERA

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Disekcija krvnih žila glave i vrata je rjeđi uzrok moždanog udara koji se često ne prepoznaje. Prikazujemo 66-godišnju bolesnicu s hipertenzijom u koje je nastupila disekcija unutarnje karotidne arterije na netipičnom mjestu i posljedični moždani udar. Dvostruki lumen je bio vidljiv uporabom obojenog doplera karotidnih arterija, a zabilježeni su prolazni signali visokog intenziteta. Transkranijskim doplerom praćen je prestanak distalne mikroembolizacije koja je bila povezana s kliničkim poboljšanjem uz primjenu antikoagulantne terapije. U embolijskom moždanom udaru uzrokovanom disekcijom unutarnje karotidne arterije neurosonološko ispitivanje je omogućilo neinvazivnu dijagnostiku i praćenje tijekom disekcije i sekundarne embolizacije.

Ključne riječi: Bolesti karotidne arterije, ultrasonografija; Disekcije aneurizme, dijagnostika; Ultrasonografija – disekcija aneurizme, komplikacije