Microembolization from a carotid mural thrombus detected by transcranial Doppler

Solaro M., Roberti C., Spalloni A., Mancini G., Beccia M., Rasura M.
Dipartimento di Scienze Neurologiche Università “La Sapienza”, Roma, Italy

Over the last few years, many authors have described the possibility of using transcranial Doppler to demonstrate the passage of microemboli in the cerebral arteries. We report the case of a 44-year-old woman with thrombotic diathesis and thrombocytosis who was admitted twice within a short period of time (one and a half months) to a neurological department because of multiple cerebral infarctions. On the occasion of the second admission, a colour-Doppler examination of the epiaortic vessels, which had previously been negative, showed a carotid lesion due to a mural thrombus and, on the same side as the carotid lesion, transcranial Doppler detected short-duration, high-intensity signals in the middle and anterior cerebral arteries, an expression of the passage of microemboli. As already described by other authors in similar clinical situations, our case confirms that transcranial Doppler can identify the passage of microemboli in the circle of Willis.

Key Words: Transcranial Doppler — Emboli — Carotid mural thrombus.

Introduction

The embolic pathogenesis of cerebral infarction has become increasingly important over the last few years, especially since studies of patients observed in the very first hours after symptom onset have revealed a high incidence of intracranial occlusions which in many cases disappeared within the first week [19]. Another recent study has shown that an embolic pathogenesis is possible in about 80% of ischemic stroke cases [3]. These data make the development of methods capable of detecting possible embolisations, even if they are clinically silent, all the more interesting.

It has recently become possible to use non-invasive transcranial Doppler (TCD) to record the passage of clinically silent microemboli in the cerebral arteries as High Intensity Transient Signals (HITS) [14, 20]. These signals have been found mainly in patients with internal carotid artery stenosis [11, 13], in patients with valvular heart disease or the bearers of prosthetic valves [6, 10], and in patients with hypercoagulability conditions [14]. The following is a report of a case in which TCD was used to document the embolicogenic activity of a carotid lesion probably consisting of a mural thrombus.

Case report

The patient, a 44-year-old woman had undergone splenectomy because of a splenic vein thrombosis at the age of 41 years. Two days after surgery, she developed a thrombosis of the vena portae and the upper mesenterium, with subsequent intestinal infarction and the surgical resection of the involved segments. A few years later, she was admitted to our Department of Neurological Sciences because of the appearance of aphasia and a strength deficit in the right half of the body. During her stay in hospital, she underwent colour-Doppler examination of the epiaortic vessels and a transcranial Doppler which were normal. Transesophageal echocardiography was negative, and a cerebral CT scan showed hypodense punctiform areas which were due to ischemic lesions situated at the level of the head of the left caudatum.

Having improved clinically, she was discharged after one month with a diagnosis of "left hemisphere cerebral ischaemia, thrombocytosis and leucocytosis of unknown origin" and a prescribed treatment acetylsalicylic acid 100 mg × 2 and calcic heparin 12,500 UI × 2 s.c.

One month after discharge, the aphasia and strength deficit in the right half of the body reappeared, and the patient was readmitted to our Department. She underwent cerebral CT on the second day after onset which, in addition to the outcome of the previous lesion, revealed the presence of left temporoparietal hypodense areas. Colour-Doppler examination of the epiaortic vessels, performed on the same day, showed a non-echogenic formation at the origin of the left internal carotid, which led to a marked acceleration in blood flow (systolic rate = 250 cm/sec) and an 80% stenosis of the vessel area (Fig. 1). The results of a transcranial Doppler examination of the intracranial arteries were normal but, during an extended scan of twenty minutes on each side, two HITS were observed only on the left in the middle cerebral artery (MCA), (Fig. 2) and three in the anterior cerebral artery (ACA). Immediately after this examination, the patient was given intravenous anticoagulant
treatment (sodic heparin 1000 U.I/h) for the first eight days, associated with and then replaced by oral treatment (warfarin), the dose of which was adjusted on the basis of prothrombin activity and the International Normalised Ratio (INR) values, which were measured daily and kept at respectively 30-40% and 2-2.5.

Blood chemistry tests showed constant thrombocytosis (700,000/mm$^3$) and leucocytosis (25,000/mm$^3$). The search for antinuclear antibodies (ANA) was positive (++).

On the fourth day, left carotid angiography showed a reduction of 70-80% in the diameter of the origin of the internal carotid artery.

Neither Holter-ECG nor transesophageal echocardiography revealed any significant alterations. In addition to the previous lesion of the left caudatum, a cerebral CT control scan performed on the tenth day showed further evidence of the ischemic area, which had extended into the posterior temporal and left cortico-subcortical parietal sites (Fig. 3).

Subsequent colour-Doppler scans of the epiaortic vessels performed every few days (while the patient was receiving anticoagulant treatment), showed a gradual reduction in the size of the non-echogenic formation described above. No more HITS were revealed by transcranial Doppler examinations. In this period, prothrombin activity and the INR remained at around the values mentioned above.

Discussion

Over recent years, a number of authors have described the possibility of using Doppler equipment to record the passage of solid or gaseous microembolic particles in specially designed in vitro models [9] and, in vivo, in the circle of Willis [5, 8]. A microembolic particle passing through a vessel is detected as an audible monodirectional signal, superimposed on the flow signal but distinguishable because of its high intensity (3 dB more than the Doppler flow signal) and short duration (less than 300 ms) [1, 2, 9]. The ability of Doppler methods to record the passage of microemboli in the cerebral arteries has important prognostic and therapeutic implications in is-