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Changes in Vessel Stenosis due to Internal Carotid Artery Dissection during Anticoagulant Treatment

Peter Höllinger, Matthias Sturzenegger

Department of Neurology, Inselspital, University of Berne, Berne, Switzerland

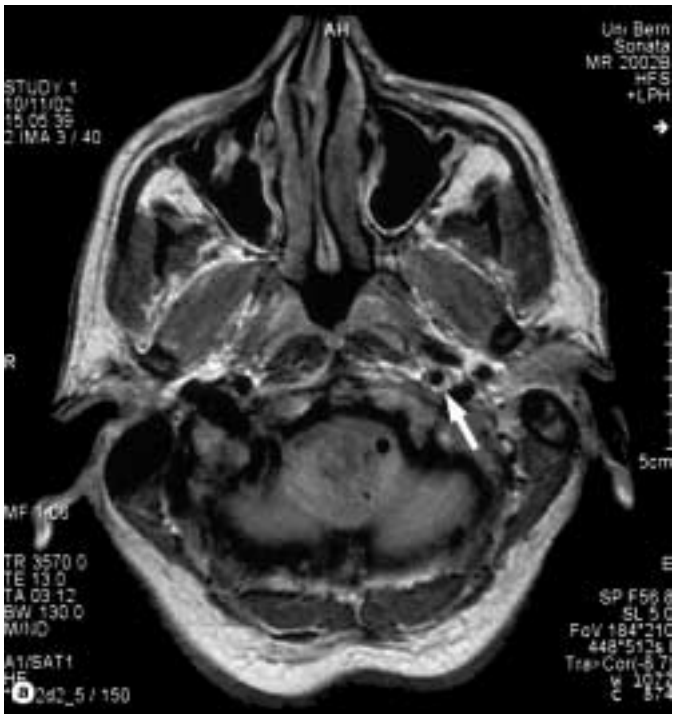
Introduction

Recurrence of internal carotid artery dissection (ICAD) is generally considered a rare event with an estimated annual incidence of approximately 1% in the patient population affected [1–3]. Herein, we describe 2 patients with early recurrence or, probably more correct, changes in ICAD in the same vessel which was initially affected while on adequate anticoagulation, a constellation hitherto not reported in the literature.

Case Reports

Patient 1. A 49-year-old tourist suddenly developed right frontal headache, followed by slurred speech, disorientation and clumsy left hand several hours later. On admission, he suffered a generalized seizure. After the postictal period he showed dysarthria, left facial palsy, paresis of the left arm and tactile extinction on the left. There was no oculosympathetic palsy. CT showed hypodensity of the right basal ganglia. Catheter angiography disclosed flame-shaped occlusion of the right internal carotid artery (ICA). Anticoagulation with high-dose intravenous heparin was initiated. Ultrasound (US) examination 4 days later showed recanalization of the right ICA with slight residual stenosis below the skull base (systolic flow velocity 127 cm/s right, 78 cm/s left). Meanwhile dysarthria and hemiparesis had improved. Since the patient's transfer back to his country was planned, a switch in anticoagulation to aspirin seemed desirable. However, repeat US 10 and 16 days after the initial event showed an occluded right ICA, when the patient was still under heparin treatment. Therefore, anticoagulation with a vitamin K antagonist was started. Two months later US findings were normal.

Patient 2. A 52-year-old man was admitted for sudden nausea and right hemiparesis. On examination, he additionally had left Horner's syndrome. MRI showed no cerebral ischemia, but a semilunar-shaped hyperintensity around the ICA lumen below the skull base (fig. 1). US 2 days later was normal. The patient was discharged



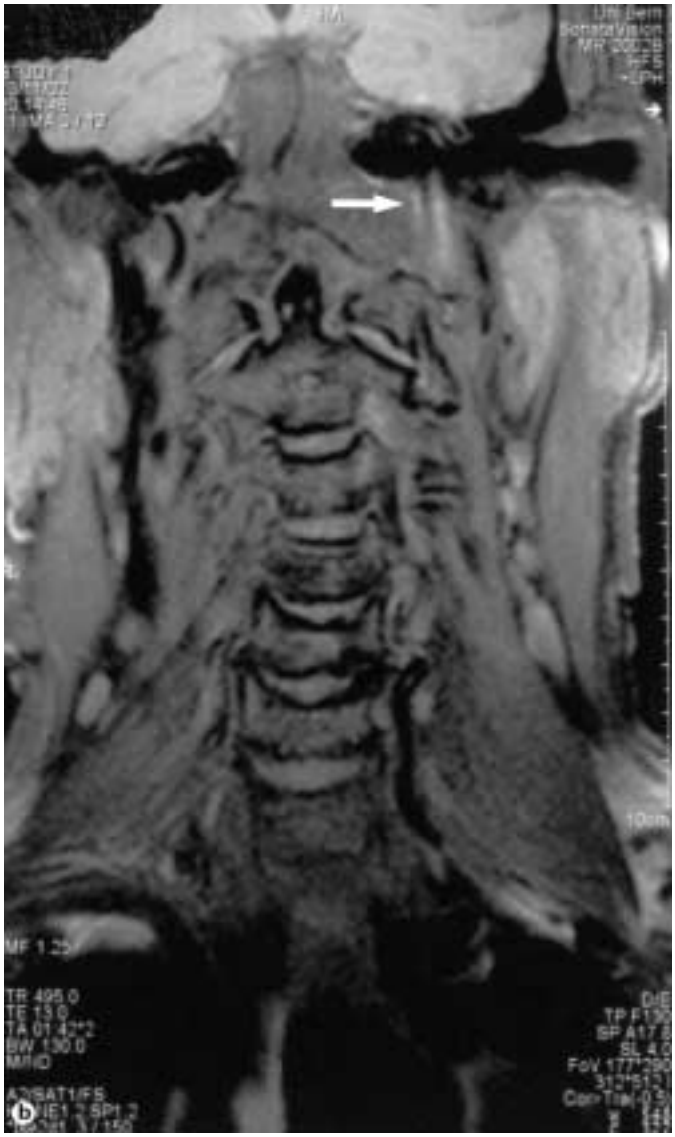


Fig. 1. Axial proton-density weighted MRI in patient 2 shows semilunar-shaped hyperintensity (mural hematoma) around the ICA lumen below the skull base (**a**, arrow). The longitudinal extension is shown on the coronal image (**b**, arrow).

without neurological deficits on oral anticoagulation. 11 weeks later, still under optimal anticoagulant treatment (INR 3.2), he had a transient blurring of vision on the left eye. US examination now disclosed left ICA occlusion. MRI and MRA on week 13 showed narrowing of the vessel with coiling in the neck (fig. 2). US on week 19 showed complete recanalization of left ICA, a finding which was corroborated by MRI and MRA on the same day. Oral anticoagulation was continued. US follow-up showed normal findings on week 25 and 39.

Discussion

Recurrent ICAD has been reported in several case series and case reports, with an estimated incidence of 1% per year [2, 3]. The majority of patients reported had recurrent dissection in a previously undissected vessel, only 9 patients with ICAD in the previously dissected artery are mentioned in the English literature [3–5]. In those 9 patients, the interval between both events ranged from 4 months to 14 years. Recurrent ICAD has been described irrespective of the antithrombotic treatment, although such an event was considered to be unusual with anticoagulation [5, 6]. However, follow-up examinations in search of recurrent ICAD are usually prompted by new symptoms and/or signs, so that a clinically silent recurrence of ICAD has only been described in 1 patient [2].

In both of our patients, clinical, US and MRI follow-up was quite close-meshed, thus even a clinically unrecognized reocclusion of the ICA in patient 1 was detected. Our patients demonstrate that ICAD is a highly dynamic process of vessel wall pathology, which can change rapidly (within a few days) from occlusion to recanalization and vice versa. Therefore, in our opinion, both patients did not really suffer from recurrent ICAD, but they rather exhibited changes in the disease process, i.e. in the extent of vessel wall hematoma, a finding not described before. Probably such a rapid change in an intramural hematoma would be detected more often, if closer follow-up examinations were performed in a systematic and prospective manner, e.g. twice a week during the 1st month. Finally, our findings have implications for antithrombotic treatment of ICAD, since it has become general practice to guide therapy according to US and/or MRI follow-up findings [1]. Patient 1 demonstrates that an early change in treatment from anticoagulation to aspirin based on US showing recanalization already 4 days after stroke might be precipitate. On the other hand, patient 2 shows that anticoagulation is not dangerous for the intramural hematoma, since occlusion recurred and cleared again despite ongoing adequate oral anticoagulation. Therefore, anticoagulant treatment should not be held responsible for the extent of the mural hematoma. It should be kept in mind that most strokes in the ICAD are due to embolism from the dissection site, and only rarely is hemodynamic insufficiency due to occlusion the cause [7].

Furthermore, the two non-invasive monitoring tools must be cautiously interpreted. Direct monitoring with duplex or color duplex is usually not possible due to the high cervical, retromandibular location of the ICAD. One has therefore to rely on Doppler blood flow velocities. They are definitely increased only when vessel stenosis results in >50% reduction in the diameter of the vessel area. Thus this method is sensitive for monitoring the stenosis range between 50 and 100%, which usually occurs with subintimal dissection [1, 8]. MRI on the other hand can detect slightly or non-stenosing ICAD, which is usually the case with subadventitial dissection, especially when using fat suppression T_1-weighted imaging to demonstrate the typical intramural hematoma. MRA can demonstrate the longitudinal extent of a stenosis and a predisposition to vessel tortuousity [9]. Thus the adequate non-invasive monitoring methods must be individually selected and in some patients combined.

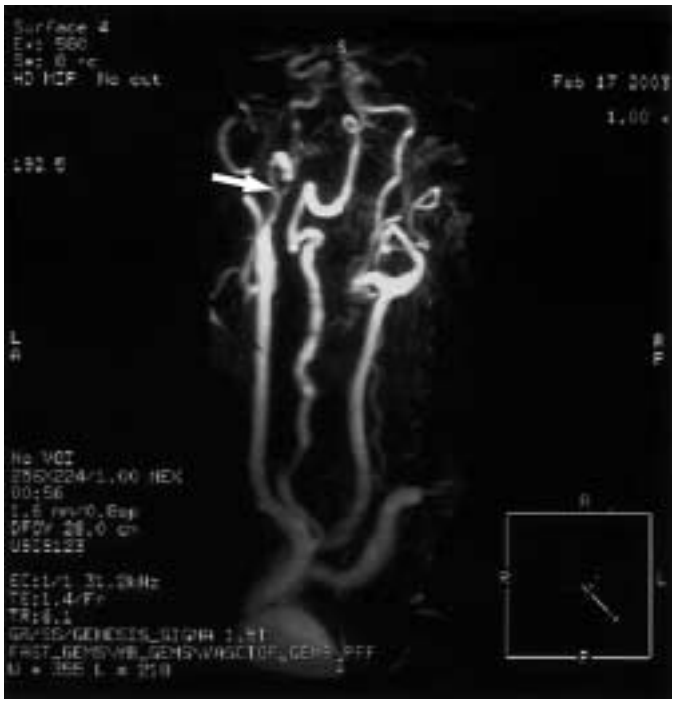


Fig. 2. MRA in patient 2, 13 weeks later, shows stenosis and coiling of the cervical ICA (arrow).

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M. Sturzenegger, MD, Department of Neurology, Inselspital
University of Berne, Freiburgstrasse, CH–3010 Berne (Switzerland)
Tel. +41 31 632 33 81, Fax +41 31 632 48 37
E-Mail matthias.sturzenegger@insel.ch