

Reversible intracranial parenchymal changes in MRI after MCA aneurysm treatment with stent-assisted coiling technique; possible nickel allergy

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Dear Sir,

We would like to bring to your attention our experience with a 41-year-old female with reversible cranial parenchymal changes in magnetic resonance imaging (MRI) of a possible hypersensitivity reaction presenting with headache and visual disturbance of flashing lights, occurring 1 month after endovascular stent assisted coil embolization with the Y-stent technique for an unruptured saccular aneurysm in the left middle cerebral artery (MCA). The aneurysm had a wide neck and saccular sizes of the aneurysm were $5 \times 4 \times 3.5$ mm. One stent was placed in the upper and the other in the lower truncus of the left MCA (Enterprise stent, Codman Neurovascular, Raynham, MA, USA), and stent-assisted coil embolization was performed. Ten GDC platinum coils were used (Boston Scientific, USA).

Embolization was successful with no complications (Fig. 1). One month later, the patient presented to our hospital with headache and visual disturbance of flashing lights. MRI

revealed multiple subcortical and cortical, noncoalescent, maximum 4 cm and nonhomogeneous in size, patchy parenchymal lesions in the left frontal, parietal, temporal, and occipital lobes, i.e., in the watershed areas. The lesions were inhomogeneously hyperintense on T2-W and fluid attenuation inversion recovery (Fig. 2a) images and hypointense in T1-W images. Since the lesions showed no diffusion restriction, they most probably corresponded to vasogenic edema. Some of the lesions showed nodular contrast enhancement especially in the cortical part with a subtle leptomeningeal contrast-enhancement adjacent to the lesions (Fig. 2b). Cerebral digital subtraction angiography (DSA) showed total occlusion of the aneurysm. In the distal arterial segments there were no signs of vasculitis or occlusion. Blood tests for the exclusion of vasculitis were normal except for a mild increase in the erythrocyte sedimentation rate (33 mm/h) and C-reactive protein (CRP; 0.62 mg/dl) values. No specific treatment was given and patients' symptoms gradually decreased over the next 2–3 weeks and finally resolved completely. The control MRI performed after 2 months showed almost complete regression of the signal changes defined in the previous MRI (Fig. 3). Parenchymal and leptomeningeal enhancement had resolved completely. Sedimentation and CRP values were back to normal. Another control MRI performed 6 months later was likewise normal.

The "Y" stenting technique is a safe and effective option in the endovascular reconstruction of unruptured complex intracranial aneurysms [1, 2]. In our case, "Y" stenting technique was used because of the wide neck of the aneurysm. Enterprise stent is a widely used, self-expanding nitinol stent with a closed-cell design, designed for the treatment of wide-necked aneurysms [3, 4]. Nitinol is composed of 55 % nickel and 45 % titanium, and there has been a great deal of concern in the medical industry regarding the release of nickel, a known allergen [5]. When properly treated, nitinol forms a very stable protective TiO_2 layer that

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Fig. 1 Unsubtracted DSA shows the left MCA aneurysm treated with Y-stent assisted coiling technique

acts as a very effective and self-healing barrier against ion exchange. The currently used vascular self-expandable metallic stents show no evidence of corrosion or nickel release. However, adverse events associated with nickel allergy in patients undergoing percutaneous heart procedures or after deployment of intracoronary stents have been reported [6–12]. It has been postulated that the nickel ions in the intracranial stents may induce hypersensitivity reactions and it should be of concern since the amount of nickel in the nitinol stents used in the cerebral circulation is higher than in the stents used in the coronary system [12]. There have been reports of nickel release after the use of device implantation for the occlusion of atrial septal defects, causing a systemic rise in serum levels of nickel [6]. It was also shown that the mean serum nickel level significantly increased at 24 h, had a maximum level at 1 month, and declined to the baseline at 12 months. New onset or an increase in frequency and severity of migraine headaches for several weeks following percutaneous closure of patent foramen ovale or atrial septal defect with devices composed of nitinol have also been reported [7–9].

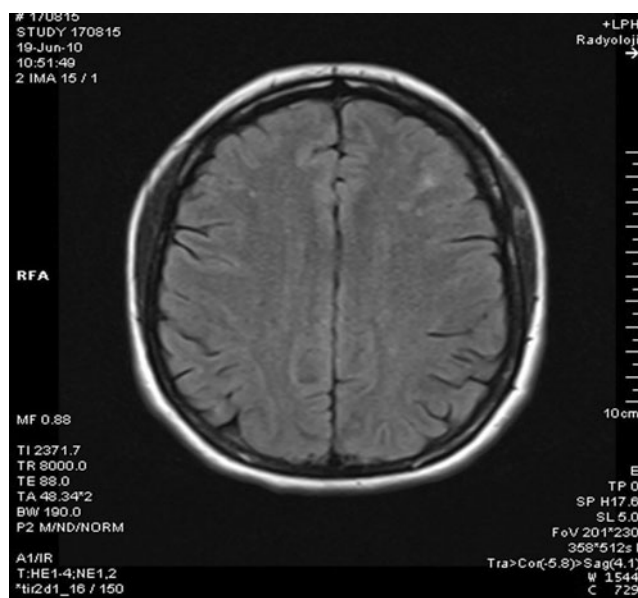
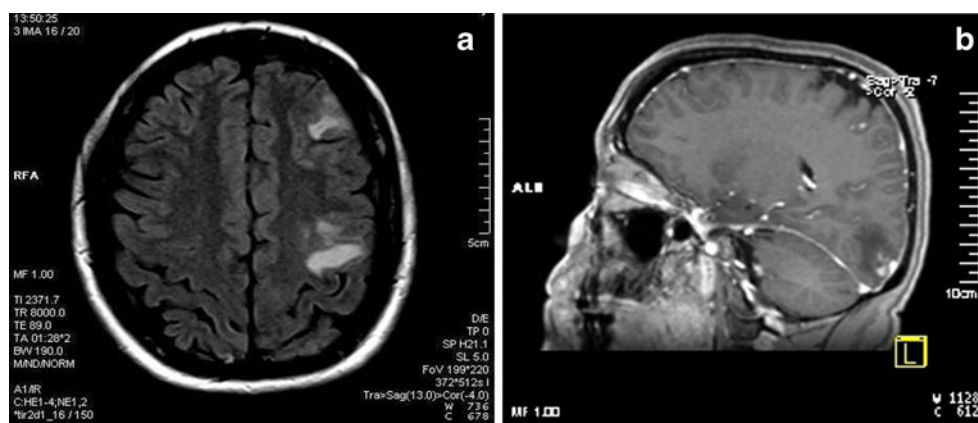


Fig. 3 Control MRI after 2 months. There is almost complete regression of the signal changes in the control axial fluid attenuation inversion recovery image

In our case, we first eliminated possible arteriovenous pathologies. Vasculitis was ruled out, since all the associated blood workup was normal, and no vascular pathology within the DSA resolution was detected. Secondly, we eliminated possible MS-like conditions, including parenchymal changes in migraine syndromes. In the follow-up of the patient, even though we did not obtain the serum nickel level at the time of her symptoms, a metallic hypersensitivity reaction was considered as the possible diagnosis. In conclusion, it should be kept in mind that nitinol containing intracranial stents may cause transient migrainous symptoms, most possibly due to a hypersensitivity reaction to the nickel in the stent. Particularly interventional neuroradiologists should be aware of this phenomenon.

Fig. 2 Axial (a) fluid attenuation inversion recovery image reveals hyperintense lesions in the fronto-parieto-temporo-occipital lobes. Nodular contrast enhancement at corticosubcortical regions is seen in the postcontrast sagittal image (b)



Conflict of interest We declare that we have no conflict of interest.

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