Giant Basilar Apex Aneurysm Presenting as Bilateral Thalamic Compression with Neuropsychological Disorders

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Dear Sir,

Bilateral lesions of the antero-medial thalamus may result in a neuropsychological syndrome characterized by severe and persistent anterograde and retrograde memory deficit, apathy, inappropriate social behaviour, impulsive aggressive outbursts, emotional blunting, loss of initiative and a reported absence of spontaneous thoughts or mental activities, conceptualized as loss of psychic self-activation [1]. We present here a rare case of giant unruptured basilar bifurcation aneurysm presenting as neuropsychological disorders due to antero-medial bilateral thalamic compression.

Three months before admission to our department, a 42-year-old obese male with hypercholesterolaemia and hypertension had developed memory disturbances such as severe impairment in explicit recall of new facts and events, behavioural disorders including lack of inhibition, occasional aggressiveness, apathy, indifference, poor motivation, flattened affect and drowsiness. However, remote memory was intact. The symptoms gradually worsened. On admission, neurological examinations were negative for focal deficits, but the symptomatology did not permit the patient to be autonomous because he was abulic. Furthermore there were no clinical signs of intracranial hypertension. Despite

a clinical history of migraine without aura, headache was not reported by the patient in the 3 months prior to hospitalization. The hormonal status excluded a hypothalamic dysfunction, and the patient's obesity was considered to be due to a hypercaloric and hyperlipaemic diet. The Mini Mental State Examination (MMSE) was given, and the score was 20. Cerebral magnetic resonance imaging (MRI) disclosed a giant aneurysm at the basilar bifurcation, producing bilateral antero-medial thalamic compression and interruption of the mammillo-thalamic tract (fig. 1a). A vertebral digital subtraction angiography confirmed the presence of a large aneurysm with a wide neck, where fortunately the posterior cerebral arteries did not originate (fig. 1b). The aneurysm was not thrombosed on either the MRI or angiography images, and it was considered at high risk for rupture. The treatment was intravascular coil embolization that completely excluded the aneurysm preserving both posterior cerebral arteries (fig. 1c). After a 12-month follow-up, there has been an improvement in the neuropsychological symptoms, which allowed the patient to regain his autonomy. A newly performed MMSE evidenced a significant improvement with a score of 27. A second cerebral digital subtraction angiography

confirmed the complete exclusion of the aneurysm.

To date several reports have described bilateral thalamic lesions presenting as memory disturbances and behavioural disorders such as apathy, indifference, poor motivation and aggressiveness, but the lesions described have usually been due to haemorrhage, ischaemic damage or infiltrating mass such as bilateral thalamic glioma [1, 2]. Moreover, several reports have described giant basilar aneurysm as neurological progressive deterioration due to obstructive hydrocephalus, subarachnoid haemorrhage, transient ischaemic attack, ischaemic stroke or a posterior cerebral fossa tumour with vertigo, velo-palataline paralysis or trigeminal neuralgia [3–5]. To our knowledge, this is the first ever report of a patient whose neuropsychological thalamic syndrome was due to a compression by a basilar artery aneurysm. A giant basilar artery aneurysm has a bad prognosis due to the risk of either rupture of the malformation or acute occlusion of the basilar artery [3, 5]. Several procedures have been described in the literature to treat the aneurysm, and for the presence in this case of a wide neck, where the posterior cerebral arteries did not originate, the treatment given was intravascular coil embolization that completely elim-

Fig. 1. A giant aneurysm in the basilar artery on MRI (a), and angiography before (b) and after embolization (c).

inated the aneurysm but still preserved both posterior cerebral arteries with a good long-term outcome.

In conclusion, to the best of our knowledge, this is the first ever report of a patient whose thalamic syndrome was due to a compression by a basilar artery aneurysm and whose memory and behavioural disorders improved after the aneurysm embolization.

References

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