Journal of Clinical Neuroscience xxx (2017) xxx–xxx

Contents lists available at ScienceDirect

Journal of Clinical Neuroscience

journal homepage: www.elsevier.com/locate/jocn

Case report

Intraoperative neurophysiologic monitoring (IONM) changes associated with a case of delayed thalamic infarct: Implications for postoperative management

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ARTICLE INFO

Article history:
Received 25 January 2017
Accepted 9 October 2017
Available online xxxx

Keywords:
Intraoperative neurophysiologic monitoring
Endovascular treatment
Cerebral aneurysm
Delayed deficit
IONM

ABSTRACT

Treatment of cerebral aneurysms poses a risk of cerebral hemorrhage and/or ischemia; these potential sequelae are usually associated with changes in intraoperative neurophysiologic monitoring (IONM) modalities. Our case demonstrates a patient with significant changes in IONM during the treatment of a right posterior cerebral artery aneurysm who did not develop neurologic deficits until three days post-operatively. IONM changes can represent a guide in postoperative patient management and may pose grounds for closer monitoring of patients with IONM changes that do not develop immediate postoperative deficits.

1. Introduction

Treatment of cerebral aneurysms carries the risk of intraoperative cerebral ischemia. Persistent intraoperative neurophysiologic monitoring (IONM) changes typically correspond with immediate deficits. Reports have been made of delayed deficits without IONM changes, likely associated with postoperative hypotension or edema [1–4]. However, our literature search did not show any cases with delayed deficits and concomitant IONM changes.

2. Case report

A healthy 38-year-old man presented with one week of headache. Neurologic examination was normal and imaging revealed a right posterior cerebral artery (PCA) aneurysm (Fig. 1) that was amenable to endovascular treatment.

Baseline somatosensory evoked potentials (SSEPs) were symmetric (Fig. 2A). After detachment of 8 coils, ~75% amplitude reduction in the left upper and loss of the left lower extremity cortical SSEPs were noted (Fig. 2B). Anesthetics and transcranial motor evoked potentials (TcMEPs) remained stable. Interventions in response to the IONM changes included: increasing the mean arterial blood pressure (MAP), removing the guide catheter, and cessation of coiling; though decreased left-sided cortical SSEPs persisted.

Immediate post-treatment examination was normal. The patient was sent to the ICU with close monitoring and blood pressure support (MAP 90–110). On post-procedure day (PPD) 2 the MAP goal was relaxed and he developed mild, transient left arm weakness which resolved with increased MAP. On PPD 3, when out of the ICU, acute left-sided weakness and numbness developed. Emergent MRI showed an acute right thalamic infarct (Fig. 3D). The patient ultimately regained normal strength but thalamic pain syndrome remains.

3. Discussion

As shown in the past, SSEP changes correlate strongly with regional cerebral blood flow and ischemia [4]. A review by Thirumala et al. further supports the high specificity of intraoperative SSEP monitoring for predicting neurological deficits [5]. Reversible IONM changes typically do not result in permanent deficits while non-reversible/persistent changes

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https://doi.org/10.1016/j.jocn.2017.10.035
0967-5868/

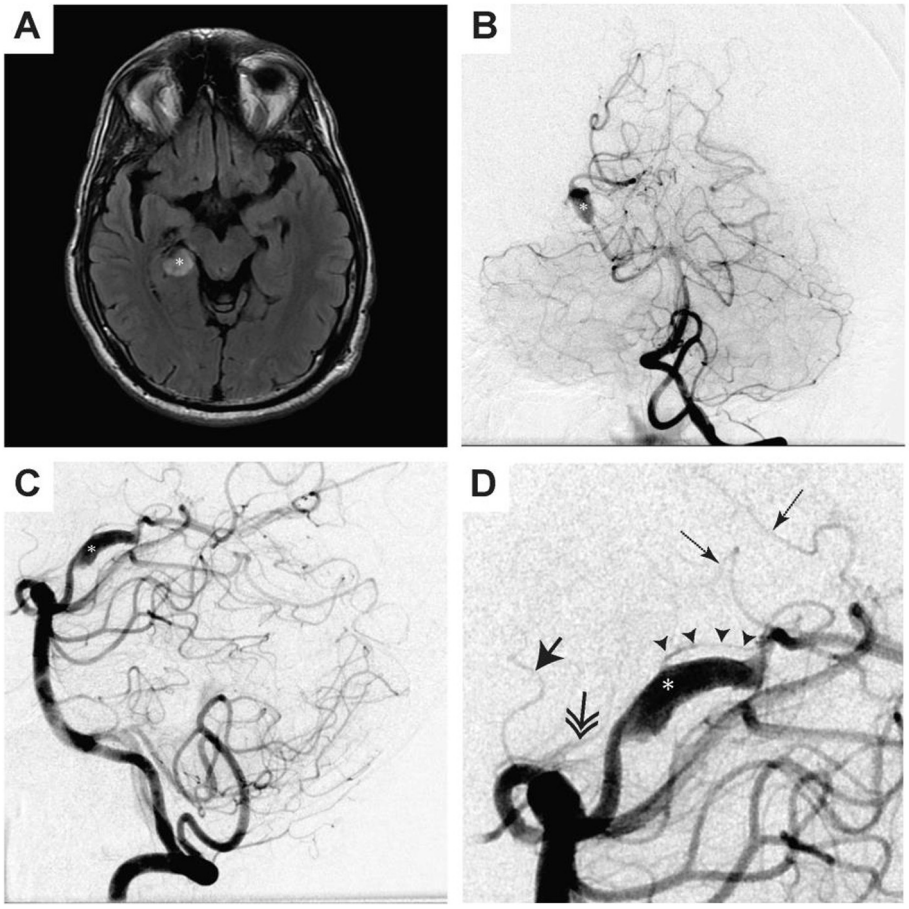

Fig. 1. Pre-treatment Imaging. A. Axial FLAIR MRI demonstrating a rounded lesion in the right ambient cistern corresponding to a right PCA P2 segment aneurysm. B. Left vertebral artery angiogram demonstrating a partially thrombosed right PCA P2 segment aneurysm. C. Lateral projection left vertebral artery angiogram demonstrating the aneurysmal filling again. D. Magnified lateral projection left vertebral angiogram identifying a fusiform aneurysmal segment (asterisk) having a small thalamogeniculate perforator (arrowheads) originating from the aneurysmal segment; nearby perforators are also seen-the tuberothalamic artery (arrow), paramedian artery (double arrow), and the posterior choroidal arteries (dotted arrows).

predict postoperative deficits [6]. Our case suggests that the significant SSEP changes indicate cerebral ischemia which we postulate did not progress clinically due to the intraoperative intervention of increasing the MAP. Given the persistent IONM changes, the patient was at higher risk for postoperative cerebral ischemia, and the IONM team recommended close patient monitoring and optimizing cerebral perfusion postoperatively. Infarction did not occur until blood pressure support was removed, causing worsening focal ischemia.

Further raising the risk of ischemia is that the thalamic territory perforator vessels are primarily end-vessels with minimal collateralization [7]. This thalamic territory ischemia can be predicted by intraoperative SSEPs as seen in our case. The SSEP changes were treated with increased MAP, which we theorize resulted in recruitment of tuberothalamic vessels to supply the compromised thalamogeniculate territory. This collateralization is tenuous and once pressor support was removed cerebral blood flow was compromised, resulting in an inferolateral thalamic infarct.

Though the patient also developed left-sided weakness on PPD 3, this resolved and only altered sensation persisted. Although we would expect TcMEP changes with subcortical ischemia [1,8], the lack of TcMEP changes likely indicates more significant ischemia in the thalamic sensory pathways. DWI changes seen in the left occipital lobe (Fig. 3) represent emboli during or after coiling. Emboli may have staggering courses and result in infarction with exacerbating effects from hypotension, which could have occurred in our patient, further indicating the predictive value of IONM changes.

4. Conclusion

IONM changes may indicate postoperative ischemia and alter postoperative neurological care. It is critical to utilize IONM in identifying patients at higher risk for post-treatment deficits; this has implications for subsequent management and demonstrates that patients may need longer periods of blood pressure monitoring and support after treatment.

Disclosures

None.

Financial support

None.

Please cite this article in press as: Ghavami F et al. Intraoperative neurophysiologic monitoring (IONM) changes associated with a case of delayed thalamic infarct: Implications for postoperative management. J Clin Neurosci (2017), https://doi.org/10.1016/j.jocn.2017.10.035