Perfusion and Diffusion MRI as Tools for Monitoring Anaesthetic Regimes in Patients Subjected to Craniotomy for Brain Tumors

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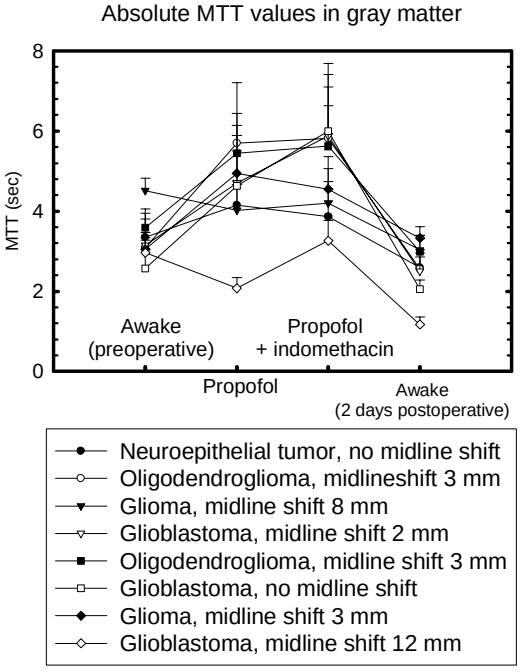
Introduction

Indomethacin, a fatty acid cyclooxygenase (COX) inhibitor, is a cerebral vasoconstrictor and reduces cerebral blood flow (CBF) without affecting cerebral oxygen metabolism ($CMRO_2$) in clinical studies[1]. Indomethacin effectively reduces ICP and improves cerebral perfusion pressure (CPP) in tumor patients during isoflurane anesthesia[2]. In spite of the beneficial effects in lowering ICP and improving CPP, the use of indomethacin is still controversial due to the presumed risk of inducing severe cerebral ischemia in patients with brain pathology. Propofol has been suggested as the drug of choice for brain tumor surgery [3]. However, several reports have demonstrated low jugular bulb oxygen saturations believed to indicate global cerebral hypoperfusion or cerebral ischemia during brain tumor surgery in propofol anesthesia [4-5]. Although clinical signs of propofol-induced cerebral ischemia in these patients have never been reported[6], no study has evaluated whether propofol induces regional ischemic damage in patients with space-occupying brain pathology.

Diffusion weighted imaging (DWI) is highly sensitive technique in the detection of acute ischemic damage. Furthermore, the mean transit time (MTT) derived by perfusion weighted imaging (PWI) is inversely related to the CPP. We hence hypothesize that PWI and DWI before, during and after injection of indomethacin and propofol may be a powerful paradigm for ruling out ischemic damage and non-invasively studying perfusion pressure changes induced by anaesthesia in patients subjected to craniotomy for cerebral tumors.

Materials and Methods

Nine patients subjected to craniotomy for supratentorial brain tumors (>3 cm) in propofol/fentanyl anesthesia were studied on a 1.5 T GE Signa LX NVi. MRI examinations including DWI with spin echo EPI (TR/TE=5000/81.5 ms, b=1000 along three orthogonal directions and one unweighted scan in a 96 by 96 resolution, 240 mm FOV, 17 slices), PWI with bolus injection of 0.2 mmol.kg^{-1} Gadolinium-DTPA (Magnevist®, Schering AG) during gradient echo EPI (TR/TE=1500/45, slices as DWI), and structural MRI were performed i) the day before surgery, ii) before and iii) after i.v. administration of indomethacin (bolus of 0.2 mg·kg-1 followed by infusion of 0.2 mg·kg^{-1}·h^{-1}) in the propofol/fentanyl anesthetized patient and again iv) two days after surgery. Maps of ADC, MTT, relative CBF and CBV were calculated[7], and gray and white matter, tumor and peritumoral tissue outlined (ALICE™, Hayden Imaging solutions) on structural images and transferred to the functional maps. DWI, ADC and structural images were inspected by a trained neuroradiologist. An acute ischemic lesion was defined as a DWI lesion with low ADC not present on the first MRI. Midline shifts were recorded to allow assessment of tumor mass effect.



Results

No ischemic lesions were detected in the DWI, ADC or structural images. The average, absolute MTT values for contralateral gray matter are displayed in the figure along with tumor type and midline shifts (Peritumoral gray showed similar behaviour). All tumors except two showed reduction in CPP (increase in MTT) upon propofol and subsequent indomethacin injection, while postoperative CPP (with the tumor removed) was slightly higher (MTT lower) than preoperative values. The two remaining tumors showed a paradoxical increase in CPP upon propofol, followed by a CPP reduction by indomethacin. These two tumors displayed the largest mass effects and also the largest postoperative increase in CPP (reduction in MTT) after tumor removal.

Discussion

DWI demonstrated that administration of indomethacin and propofol was not associated with ischemic damage in investigated patients with cerebral tumors. PWI showed the expected CPP reduction in tumors with little mass effect, whereas tumors with mass effect showed an initial CPP increase with propofol injection, possibly because preoperative CPP was affected by the tumor mass effect. PWI and DWI seems valuable tools for non-invasive monitoring of CPP and the emergence of ischemic lesions during various anaesthetic regimes.

References

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