Intraoperative electroencephalography changes in unilateral moyamoya phenomenon

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INTRODUCTION

Moyamoya phenomenon is the proliferation and profuse collateral formation in distal branches of occluded unilateral or bilateral distal internal carotid artery (ICA) in presence of some associated condition like atherosclerosis, meningitis, vasculitis, sickle cell disease, etc., In the absence of any associated condition, the term used is moyamoya disease.[1] We report a case of moyamoya phenomenon due to encasement of ICA by an optochiasmatic glioma and characteristic electroencephalography (EEG) changes observed intraoperatively and postoperatively.

CASE REPORT

Consent for publication of this report was obtained from guardians of the patient.

Patient was a 10-year-old boy with history of progressive vision loss in left eye for 4 years, followed by right eye of 3 years duration. Magnetic resonance imaging revealed an optochiasmatic glioma encasing the left ICA. Multiple flow voids were visible within left hemisphere parenchyma and normal vasculature in the right hemisphere [Figure 1a]. Cerebral angiography revealed left ICA narrowing and multiple "puff of smoke" collaterals in ICA distribution territory [Figure 1b]. Patient was posted for decompression of the lesion via left pterional craniotomy.

Anaesthetic plan was devised based on moyamoya picture, primarily including maintenance of baseline blood pressure and normocapnia. Intraoperative EEG was placed for ischaemia monitoring, due to the possibility of ICA handling. Due to the agitated state of child, EEG electrode was placed after induction of anaesthesia. EEG monitoring was instituted with P4-T6 and P3-T5 bipolar montages. Electrodes were placed, and secured carefully using gauze and waterproof adhesive tape so as to avoid hindering the surgical field and to prevent blood soakage. Induction with thiopentone, fentanyl and rocuronium was, followed by maintenance with sevoflurane and nitrous oxide 50% targeted to maintain 1.2 age-adjusted minimum alveolar concentration (MAC).

During intraoperative course, EEG waveform was indistinguishable bilaterally, however quantitative EEG processing revealed spectral edge frequency (SEF), median frequency (MF) to be consistently lower on affected side, and relative delta band power (RDP) to be higher. The differences were small (2 Hz difference in SEF and 1.5–2% in RDP) but consistent throughout intraoperative course [Figure 1c]. Intraoperative course was uneventful and patient was extubated at the end of surgery. Partial tumor resection was accomplished with persistence of ICA encasement. Postoperatively, the EEG showed attenuation of waveform amplitude on the affected side with SEF 5–6 and MF 0.5–1. Normal side showed maintained waveform amplitudes with SEF 16–20 and MF 2–3. RDP was marginally higher on the affected side with no α/β activity, and normal side showed small band powers in α and β frequency range [Figure 1d]. The EEG readout was taken when patient had clinically recovered from anaesthesia (end tidal MAC ≤0.2 and obeyed commands). The patient maintained preoperative neurological status. Due to persistence of ICA encasement postoperatively and thus no change in cerebral haemodynamic status, postoperative EEG was assumed to reflect preoperative EEG findings.

DISCUSSION

Moyamoya phenomenon occurs when there is nonvasculitic, nonatherosclerotic narrowing or stenosis of the terminal segment of ICA or proximal segments of anterior or middle cerebral arteries, in the presence of certain conditions. Optic glioma has been previously described as a cause of moyamoya phenomenon. Gradual occlusion of the major intracranial arteries allows extensive anastomotic collateral formation to supply the relatively ischemic brain parenchyma. Clinical presentation is varied, with paediatric moyamoya presenting with motor or sensory deficits, headaches, involuntary movements or seizures. Adults commonly present with haemorrhagic complications.[1]

Characteristic EEG abnormalities described in moyamoya disease include "rebuild-up phenomenon," that is,

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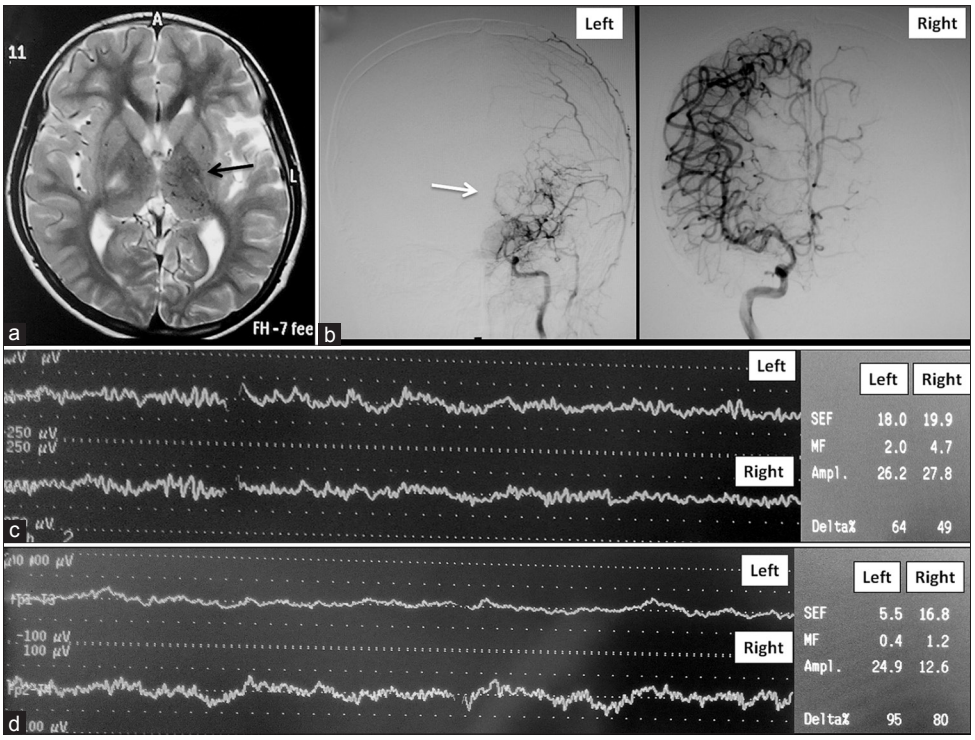

Figure 1: (a) Multiple flow voids in left hemisphere (black arrow). (b) "Puff of smoke" collaterals in left internal carotid artery angiography (white arrow) compared with normal vasculature in right. (c) Intraoperative, indistinguishable electroencephalography (EEG) waveform of left and right sides with reduced spectral edge frequency (SEF), median frequency and higher relative delta power on the left side. (d) Postoperative, attenuated waveform with predominant slow activity of left compared with normal EEG waveform of right. SEF markedly reduced on the left side

return of high voltage slow waves after a period of hyperventilation. Other findings include posterior slow, centrotemporal slow and diffuse low voltage slowing of EEG activity after mean of 10 months, 28 months and 56 months after clinical onset of disease, respectively.[2]

Frequency domain processing of raw EEG yields many parameters such as SEF, MF and RDP. SEF and MF relate to the power distribution of the power density spectrum of the EEG. They are the highest frequencies below which 95% and 50% of power, respectively, is located. Thus, as the overall slowing of EEG occurs, the power in the higher spectrum decreases and SEF and MF both decrease. RDP is the ratio of power in the delta band to the power of the whole EEG spectrum.

Based on the findings from our case, following conclusions can be drawn:

- The ICA compression led to relative hypoperfusion of the affected side with reduced neuronal electrical activity. This is in concurrence with centrotemporal slowing of EEG activity discussed by Kodama *et al*.
- Intraoperative anaesthetic regimen (inhalational agent-based) led to near equalisation of bilateral neuronal activity, probably by improvement in perfusion due to cerebral vasodilation.[3]

Sato *et al*. showed increased Jugular venous oxygen saturation and regional reductions in cerebral blood flow with inhalational anaesthetic agents in 13 bilateral moyamoya disease patients and suggested intracerebral steal phenomenon to be the cause.[4] Findings in our patient seem to contradict the known "steal" phenomenon of inhalational agents. However, the effect of anesthetics on unilateral moyamoya phenomenon has not yet been described in the literature. The complex interaction of cerebral metabolic suppression, vasodilation, inherent metabolic requirements of the relatively ischaemic tissue on the affected side cannot be deduced without evidence. It is probable that global cerebral vasodilation coupled with metabolic suppression in the normal side might produce improved perfusion of affected side, leading to near equalization of neuronal electrical activity. Robust conclusions cannot be drawn from our experience; however, this does point to the need for formal study on the effect of anaesthetic agent on EEG and cerebral blood flow in patients of unilateral moyamoya phenomenon.

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Conflicts of interest

There are no conflicts of interest.

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