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Case report

Cerebral embolism during elective carotid endarterectomy treated with tissue plasminogen activator: Utility of intraoperative EEG monitoring

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ABSTRACT

Electroencephalography (EEG) is routinely used during elective carotid endarterectomy (CEA) for monitoring cerebral perfusion. The period most frequently associated with cerebral hypoperfusion is the one during the clamping of the carotid artery. We present a case whereby acute hypoperfusion, as detected by ipsilateral hemispheric slowing and attenuation of the fast frequencies on EEG, was detected in the period *prior* to clamping of the carotid artery. The acute changes were caused by a cerebral embolism. Following emergent treatment with intraoperative thrombolytic therapy with intra-arterial tissue plasminogen activator (t-PA) the EEG changes reversed fully. We discuss the utility of intraoperative EEG monitoring in the detection and treatment of cerebral embolism. The ability of EEG to intraoperatively measure the function of the at-risk cerebral cortex makes it not only a useful tool in detecting acute changes such as from a large embolism, but also in guiding necessary treatment by offering direct feedback in the absence of reliable imaging and clinical examination.

1. Introduction

Electroencephalography (EEG) is a sensitive and reliable method for monitoring cerebral activity during carotid endarterectomy (CEA) [1,2]. Its ability to detect cerebral ischemia is due to the good correlation among regional cerebral blood flow decrease, neuronal dysfunction and EEG parameters [2,3]. EEG is particularly useful in detecting the cases of cerebral hypoperfusion due to clamping of the carotid artery [2] and selecting those cases that need temporary bypass shunting. However, embolic events are the major cause of perioperative strokes [4]. Transcranial Doppler (TCD) is considered to be a sensitive tool in detecting microemboli [5,6], but it does not offer a direct intraoperative assessment of the possible impact of such emboli on neurophysiologic function. Despite the notion that EEG is not sensitive in the detection of embolic events [7,8] we report here a case whereby EEG monitoring identified acute cerebral embolism during elective CEA and successfully guided treatment with intra-arterial tissue plasminogen activator (tPA).

2. Case report

An 80-year old, asymptomatic, right-handed woman, with significant cardiovascular risk factors was found to have a high-grade stenosis of the left common carotid artery (L CCA) by carotid duplex and magnetic resonance angiography (MRA) of the neck. These imaging investigations showed that the stenosis was at 90% by MRA, and at 2 cm proximal to the left carotid bifurcation. The neuroimaging studies also revealed a 40–50% stenosis of the middle segment of the right internal carotid artery (ICA) and of the origin of the left subclavian artery. Brain MRA revealed atherosclerotic, non-occlusive changes of the vessels of the circle of Willis. A left CEA was recommended.

Digital EEG recording with the Standard International 10-20 system, using a 16-channel array was performed during the entire CEA. The Cz channel was used as reference and the EEG was monitored using a bipolar montage. With the patient awake, the recording showed a symmetric background with a posterior alpha rhythm and superimposed muscle activity (Fig. 1-left half).

During induction of anesthesia with propofol and remifentanyl, followed by nitrous oxide and isoflurane the EEG showed diffuse, symmetric slowing with superimposed symmetric fast frequency activity. The post-anesthetic induction EEG was analyzed at a slower paper-speed and relatively increased amplitude. Both changes allow for the easier detection of potential hemispheric asymmetries following anesthetic induction. During induction the

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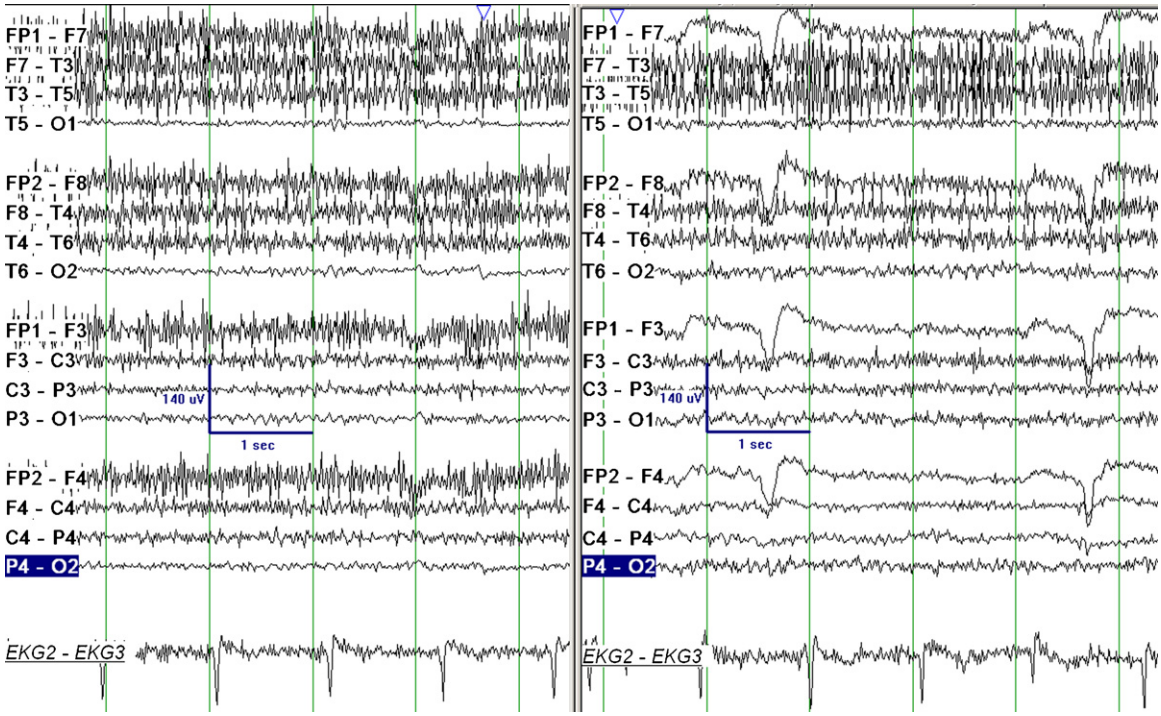

Fig. 1. The left half of the page shows the baseline 16-channel EEG recording, in the International 10-20 system, prior to the induction of anesthesia with a symmetric awake background, an unsustained but symmetric posterior alpha rhythm at 8.5–9.5 Hz and superimposed muscle artifact. The right half of the page shows the EEG after completion of the procedure and recovery from anesthesia showing a return to the baseline awake EEG. (Sensitivity 7 μV/mm; timebase 30 mm/s; HFF 70 Hz; LFF 1 Hz.)

systolic blood pressure varied between 120 and 160 mmHg. During dissection and artery exposure, the EEG acutely became asymmetric, showing increased slowing and more than 50% attenuation of the fast frequency activities over the anterior-central left hemisphere regions (Fig. 2). No changes in blood pressure, cardiac rhythm or anesthetic doses had preceded this sudden EEG change.

A decision was taken to proceed with emergent CEA. The clamp was placed on the CCA. Despite emergent placement of an intra-arterial bypass shunt from the CCA to the distal ICA and induced hypertension to a systolic blood pressure of 190 mmHg, there was no substantial EEG improvement (Fig. 3, left half). Subsequent arteriotomy of the left CCA revealed an ulcerated plaque tapering

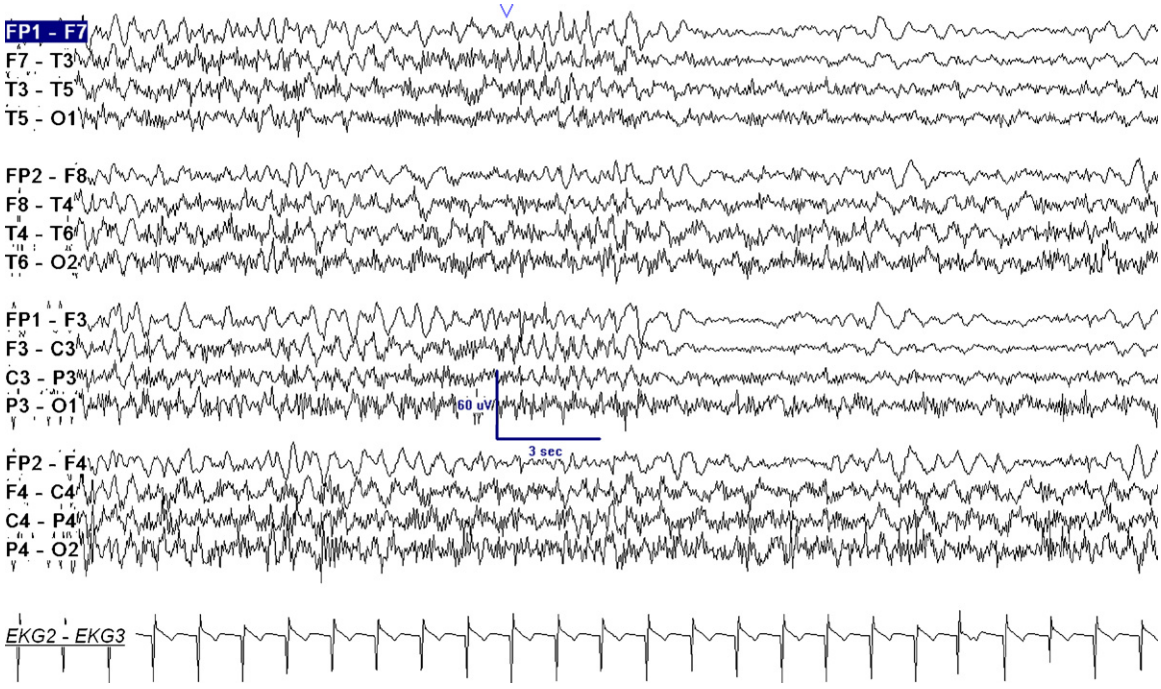

Fig. 2. With the EEG compressed to 10 mm/s, an abrupt development of an asymmetric background is seen, with increased slowing and attenuation of the fast frequencies over the anterior-central left hemisphere. (Sensitivity 3 μV/mm; timebase 10 mm/s; HFF 70 Hz; LFF 1 Hz. Note: changed timebase to facilitate the detection of asymmetries in the background due to the suppression of the background because of the anesthetic agents.)