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Transcranial Doppler for brain death after decompressive craniectomy: persistence of cerebral blood flow with flat EEG

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Dear Editor,

Brain death (BD) diagnosis is made based upon clinical criteria—unresponsive coma with absence of brainstem reflexes and persistent apnea—and upon neurophysiologic observation of persistent "flat EEG." Cerebral circulatory arrest (CCA) must also be assessed with "ancillary tests," e.g. conventional angiography, transcranial Doppler (TCD), and other neuroimaging techniques, in infants younger than 12 months, when EEG flattening may be related to sedative treatment, and in BD of uncertain origin [1]. CCA may indeed happen both when intracranial pressure (ICP) overrides mean arterial blood pressure (MAP) as well as in diseases affecting cerebral tissue at a cellular level, with ICP not exceeding MAP. TCD is a sensitive, specific, and noninvasive technique to detect CCA in BD by identifying specific patterns [2]. However, it may lead to false-negative results in cases of skull defects (decompressive craniectomy, external drains, and in infants), because in these cases the increase in ICP may partially be compensated for. For these reasons, we recently described the TCD modifications of the CCA patterns in infants with BD, confirming that CCA detection for BD confirmation should be done cautiously in these cases [3]. Here we describe the different TCD findings observed in two adults with BD who were subjected to decompressive craniectomy.

Case 1 was a female, 62 years old, with intracerebral right temporo-parietal hemorrhage, middle cerebral artery aneurism rupture. She underwent large right fronto-temporal craniectomy and presented with unresponsive coma and flat EEG (Fig. 1). TCD, performed under stable hemodynamic conditions (BP 140/70 mmHg), showed the typical CCA pattern. Short compression of the dural expansion induced a further reduction of the signal, promptly returning to the basal conditions at the end of compression (Fig. 1).

Case 2 was female, 56 years old, with a small deep right basal ganglia hemorrhage and aneurysm of the right intracranial internal carotid artery. She was treated with an endovascular procedure. Following sedation withdrawal, 2 days after the procedure, she was conscious but agitated and uncooperative, and she was again sedated. TCD performed under stable hemodynamic conditions (BP 150/100 mmHg) (Fig. 2a) showed a very high-resistive pattern with diastolic reduction in both the middle cerebral arteries and expression of elevated ICP, confirmed by invasive

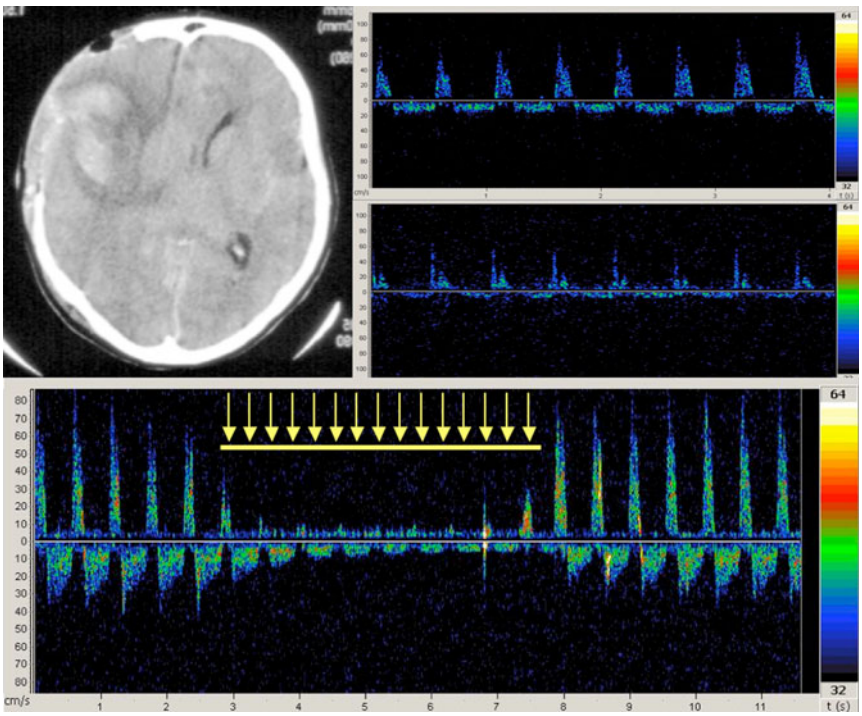

Fig. 1 Cerebral CT scan (*top left*) and transcranial Doppler (TCD) findings (*top right*) in case 1. Cerebral CT shows the extensive intraparenchymal hemorrhage with the right decompressive craniectomy, and TCD shows the typical pattern of absence of cerebral blood flow. Short compression on the dural expansion (*yellow arrows*, *bottom*) induces the further disappearance of TCD signal, which promptly returns to basal conditions once compression is removed

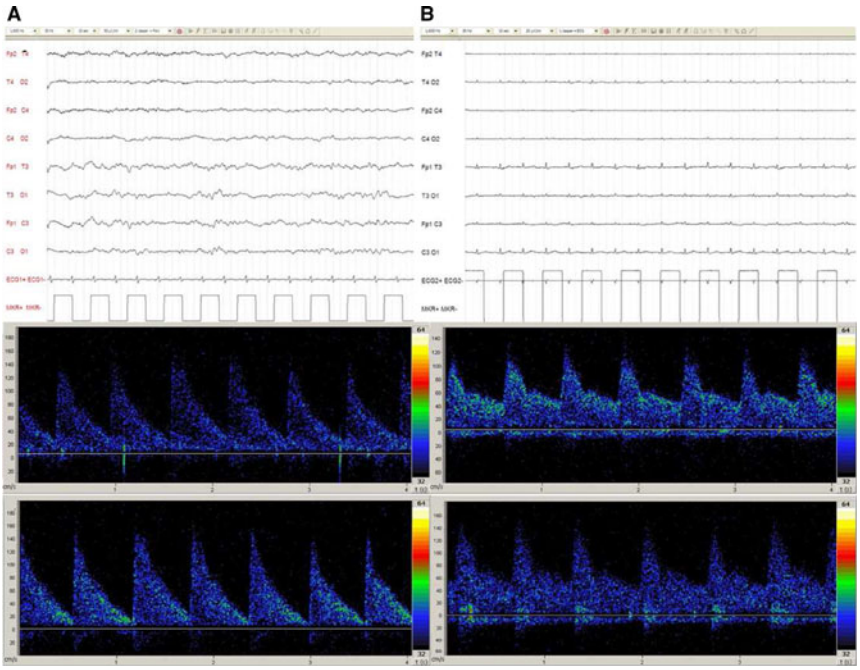

Fig. 2 EEG (*top*) and transcranial Doppler (TCD) findings (*below*), before (**a**) and after (**b**) decompressive craniectomy in case 3. EEG before craniectomy shows the presence of a diffuse slowing of the background activity, predominantly on the right side and, after skull fenestration and sedation withdrawal, the disappearance of electrical activity. TCD before the craniectomy (**a**) shows a marked increase in the resistive indices with a diastolic component reduction and spectrum deformation, suggestive of increased intracranial hypertension. After decompressive surgery (**b**), diastolic flow velocity increases and resistive indices return to normal, but with flat EEG

measurement (90 mmHg). EEG showed right hemisphere slowings, compatible with sedation. CT scan revealed an extensive right intraparenchymal hemorrhage, and the patient underwent large decompressive craniectomy. A second TCD showed the persistence of cerebral blood flow, with a "paradoxical" amelioration of the resistive indices, but with the occurrence of unresponsive coma and flat EEG, 2 days after sedation withdrawal (Fig. 2b).

Diagnosis of BD has to be made with caution. The preservation of CBF in cases of skull defects and BD may exist and should be considered as a "false negative" result, not excluding BD [4]. It is important that both the sonographer and the attending physicians bear in mind all the conditions in which "false negative" results may arise [5], in order to evaluate results in the whole context and consider other options for BD confirmation, without losing precious time for BD declaration in cases of organ transplant.

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