ABSENCE OF DOPPLER SIGNAL IN TRANSCRANIAL COLOR-CODED ULTRASONOGRAPHY MAY BE CONFIRMATORY FOR BRAIN DEATH: A CASE REPORT

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ABSTRACT

Transcranial Doppler ultrasonography (TCD) is a valuable tool for demonstrating cerebral circulatory arrest (CCA) in the setting of brain death. Complete reversal of diastolic flow (to-and-fro flow) and systolic spikes in bilateral terminal internal carotid arteries and vertebrobasilar circulation are considered as specific sonogram configurations supporting the diagnosis of CCA. Because of the possibility of sonic bone window impermeability, absence of any waveform in TCD is not confirmatory for CCA unless there is documentation of disappearance of a previously well detected signal by the same recording settings. Transcranial color-coded sonography (TCCS) with B-mode imaging can reliably detect adequacy of bone windows with clarity contralateral skull and ipsilateral planum temporale visualization. Therefore, absence of detectable intracranial Doppler signal along with available ultrasound window in TCCS can confirm clinical diagnosis of brain death. We herein discuss this entity from the frame of a representative case.

Key Words: Brain death, Doppler, temporal bone, ultrasound penetrance.

INTRODUCTION

Utility of transcranial Doppler ultrasonography (TCD) in confirmation of brain death is well established. The reported sensitivity of TCD for diagnosing brain death is about 90% at average, ranging from 53.5% to 100% (1-5). Its specificity is much higher and actually is very close to 100% (99.6% on average) (3-6). False-positive diagnosis of brain death by TCD was reported in 0.46 % of cases published in the literature; (7-11) however, the significance of this finding is questionable because brain death developed in all of these cases after a short duration following the Doppler examination (7,12).

TCD diagnosis of brain death requires...
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demonstration of cerebral circulatory arrest (CCA) by recording specific sonogram configurations in terminal, or intra-cranial, internal carotid arteries (ICA) bilaterally and also in the basilar artery or in both intracranial vertebral arteries. The CCA specific sonogram patterns are complete reversal of diastolic flow (reverberating or to-and-fro flow) and systolic spikes, indicating absence of any antegrade flow providing a meaningful degree of cerebral perfusion (9,12-14). Unless disappearance of a previously well detected signal is clearly established, absence of any TCD waveform is not considered as diagnostic for CCA because of the possibility for a false positive result due to impermeability of sonic bone windows, which can be present in up to 40% (in average: 7.6%) of these patients (4,12,15,16). In such cases, submandibular insonation of cervical ICAs, transorbital insonation of carotid siphons, and though arguable, contrast-enhanced transcranial color-coded sonography (TCCS) can be used as alternatives for neurosonological confirmation of brain death (5,12,15,17,18). TCCS, when compared to TCD, has the advantage of visualizing cerebral tissue and therefore can provide valuable information on the adequacy of sonic bone Windows (19,20). The case presented herein led us to suggest that TCCS demonstration of absence of any intracranial Doppler signal along with apparently adequate sonic bone windows can be used as a confirmatory tool in diagnosis of brain death.

CASE

A poor 20-year-old gentleman developed hemorrhagic type posterior leukoencephalopathy (PRES) in the setting of thrombotic thrombocytopenic purpura and acute rejection of renal transplantation. The clinical course was complicated by brachial artery thrombosis, necessitating forearm amputation, severe pneumonia, sepsis and secondary acute respiratory distress syndrome. He suffered from two cardiac arrests, but was resuscitated successfully. Following cardiac arrests, he was found have deep coma and absent brain stem reflexes. Because apnea test could not be completed due to occurrence of significant desaturation and hypotension, a confirmatory test was ordered to establish brain death diagnosis. TCCS failed to record any Doppler signal despite modification of ultrasound settings suitably for low flow status. However, brain parenchymal structures and contralateral skull were unequivocally identifiable in this TCCS study (Figure 1).

Figure 1. Transcranial color-coded ultrasonography shows absence of flow signal in any intracranial artery. Hyperechogenicity of the middle cerebral artery (arrow) is discernible. (Siemens Acuson X150®, pulse repetition frequency: 488 Hz, persistence: 3; priority:4; gain 80 dB; dynamic range:50; gate: 10-mm).

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A computerized tomography angiography was then performed at the discretion of treating intensivists, and showed extensive brain edema (Figure 2) along with absence of intracranial arterial and venous flow compatible with brain death (Figure 3).

Figure 2. Brain CT shows massive edema along with Duret’s type hemorrhage in the pontomedullary junction and subacute PRES-related hemorrhage in the left occipital lobe.

Figure 3. CT angiography shows no flow in intracranial arteries. A prominent extracranial vascular enhancement along with tentorial and meningeal hyperdensity (pseudo-subarachnoid hemorrhage appearance) are evident in these images. The blood pressure and oxygen saturation were maintained within normal levels (systolic blood pressure >100 mmHg and oxygen saturation >96%) during both TCCS and CT examinations.
DISCUSSION

Experience with TCCS, albeit substantially lower than those with TCD, shows comparable sensitivity and specificity rates in terms of brain death confirmation (21,22). The availability of acoustic window within the temporal bone does not significantly differ between TCD and TCCS (20). However the detection of window quality is easier and more rapid with TCCS through depiction of the main parenchymal and ventricular structures (23,24). Furthermore, the temporal sonic window can be considered as sufficient when contralateral skull and ipsilateral lesser sphenoid wing are insonated as a thick and continuous hyperechogenic structure (24) as seen in the case presented herein. The other advantages of TCCS over TCD include, but are not limited to, easier vessel localization and discrimination, Doppler angle correction, and direct imaging of cerebral parenchyma for other pathologies (25). The presented case highlights an additional aspect of TCCS in brain death diagnosis, the verification of sufficiency of an acoustic bone windows in the setting of absent intracranial Doppler signals. It is important to note that point-of-care ultrasonography devices routinely equipped with phased-array 2- to 3-MHz sector transducers and proper software that enable performing TCCS but not TCD, are becoming increasingly available in ICU environments. Therefore, familiarity of ICU physicians to this additional potential for TCCS in brain death confirmation is important. Properly-organized studies are now required to test the sensitivity and specificity of our observation in confirming brain death diagnosis.

REFERENCES


