The Role of Transcranial Doppler in The Confirmation of Brain Death

Beyin Ölümünün Doğrulanmasında Transkraniyal Doppler'in Yeri

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Abstract: We investigated the value of transcranial Doppler (TCD) examinations of intracranial arteries as a diagnostic test for the confirmation of brain death. Twenty-nine patients with intracranial hypertension who subsequently developed brain death according to clinical criteria were evaluated by TCD ultrasonography during a 16-month period. Typical TCD findings in the arteries of brain-dead patients consisted of either absent or reversed diastolic flow or small early systolic spikes in at least 2 intracranial arteries. This waveform abnormality was found in 26 (89.7 %) of the 29 patients confirmed to be brain dead by clinical criteria, and was absent in 3 (10.3 %). This abnormality does not appear in other patients with coma. All patients with waveform abnormality in TCD examination died. TCD, a practical, noninvasive, early and reliable method for the confirmation of brain death, can be a rapid and convenient alternative to confirmatory tests about vascular circulation.

Key Words: Brain death, transcranial Doppler (TCD)

Özet: Beyin ölümünün tespitinde bir tanı yöntemi olarak kafa içi arterlerin transkranial Doppler (TKD) incelemesinin değeri araştırıldı. Onaltı aylık dönem esnasında kafa içi basınç artışı olan ve sonradan klinik ölçütlere göre beyin ölümü gelişen 29 hastanın TKD kayıtları incelendi. Beyin ölümü için tipik TKD bulguları; en az 2 kafa içi arterde diastolde kan akımının yokluğu veya geriye dönmesi veya küçük erken sistolik diken olarak kabul edildi. Klinik olarak beyin ölümü saptanan 29 hastanın 26 'sında (% 89.7) TKD'de beyin ölümü dalgaları izlenirken, 3'ünde ise (%10.3) TKD bulguları beyin ölümünü doğrulamadı. TKD incelemesinde beyin ölümü dalgaları saptanan tüm hastalar kaybedildi. TKD'in beyin ölümünün erken dönemde ve güvenle tespit edilmesinde pratik, noninvaziv ve hasta başında uygulanabilir bir yöntem olarak damar dolaşımını değerlendiren incelemelere seçenek olduğu kanısındayız.

Anahtar Sözcükler: Beyin ölümü, transkranial Doppler (TKD).

INTRODUCTION

The early diagnosis and confirmation of brain death is one of the most difficult medical tasks which physicians face and carries great legal, economic and ethical responsibility. Brain death is considered to have occurred when cerebral and brainstem functions have both irreversibly ceased (16), as documented by clinical and corroborative studies (7,8,16). The absence of cerebral function is recognized clinically as the lack of cortically mediated responses to any stimuli. Absent brain stem function requires the loss of pupillary, corneal, oculocephalic, oculovestibular, oropharyngeal, and respiratory reflexes (6).

There are different recommendations for objective verification of brain death in addition to the clinical examination prior to withdrawal of life support. These confirmatory tests include a) those that evaluate brain electrical activity, and b) those that evaluate brain blood flow (2,16,19,20). Although

these confirmatory tests provide the clinician with objective evidence for brain death independent of the clinical examination, they have certain disadvantages. Imaging studies are expensive, technology-intensive, invasive, and commonly require transporting critically ill patients to the radiology department.

Some investigators have used transcranial Doppler (TCD) ultrasound to document, noninvasively, arrest of the cerebral circulation in brain-dead patients (9,10,12,13,17). Typical TCD findings in the arteries of brain-dead patients are absence or reversal of blood flow in diastole (oscillating waveform) or small early systolic spikes (9,17). These waveform abnormalities of brain death occur as the intracranial pressure rises above the mean arterial pressure and are indicative of arrest or near-arrest of cerebral circulation (5).

TCD ultrasound may be an alternative to current methods for confirmation of brain death because it is lightweight and portable, and can be performed and interpreted in a matter of a few minutes at the bedside. We reviewed TCD findings in 29 brain-dead patients by clinical criteria to determine the sensitivity and specificity of TCD in the confirmation of brain death.

MATERIALS AND METHODS

During the period between July 1994 to November 1995, TCD recording of 29 patients with intracranial hypertension and subsequent intracranial circulatory arrest were evaluated. There were 12 females and 17 males with an age distribution of 2 to 70 years (mean 20.8 years). Table I shows a summary of underlying diseases of these patients.

Table I. Underlying diseases in 29 brain dead patients.

Diagnosis	No. of Cases
Head injury	23
Subarachnoid hemorrhage	3
Intracranial hematoma	2
Brain tumor	1

The clinical diagnosis of brain death was made according to a protocol established at our institution utilizing guidelines suggested by an ethical comission (16). This protocol includes the lack of cerebral receptivity and responsiveness, absence of brain stem reflexes, flaccid tone, and lack of spontaneous respiration. At the time of clinical examination, the patients were normothermic, were not under the influence of sedative or paralyzing agents, and had no evidence of metabolic nor respiratory disorders, as determined by laboratory testing.

To confirm brain death, TCD evaluations alongside clinical examination were carried out at least four times in a day. TCD examinations were performed at the bedside using a transcranial Doppler device with a 2 MHz probe (Multi-Dop X, DWL Elektronische Systeme GmbH, Uberlingen, Germany). Examinations were considered technically adequate if at least 2 intracranial arteries (1 artery on the right and 1 on the left in the anterior circulation, or the basilar artery and 1 artery in the anterior circulation) could be insonated. Standard technique was used to insonate and identify arteries using either the temporal or suboccipital transcranial ultrasonic windows (1). Net flow velocities could then be determined by calculating the difference between the positive and negative mean velocity values.

The patients were usually positioned with the head elevated 30^0. Artificial respiration was applied with $PaCO_2$ values averaging 35 mmHg. Mannitol was used for the treatment of acutely raised intracranial pressure.

RESULTS

In twenty-nine patients diagnosed brain dead by clinical criteria, technically adequate TCD examinations were carried out. Twenty-six of those patients had waveform abnormality of brain death, consisting of either absent or reversed diastolic flow (Figure 1) or small early systolic spikes (Figure 2), but not in any of other patients with coma.

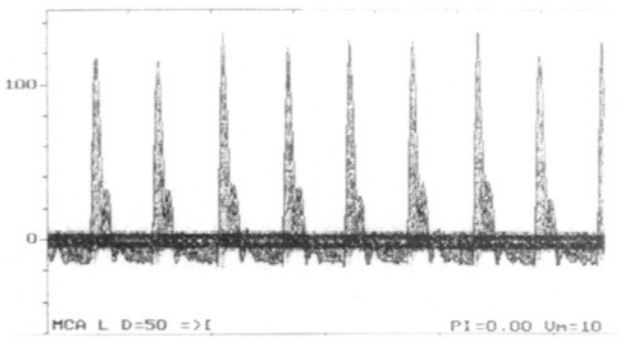

Figure 1. Transcranial Doppler recording demonstrating reversal of blood flow in diastole in the middle cerebral artery. Velocity scale is in cm/sec.

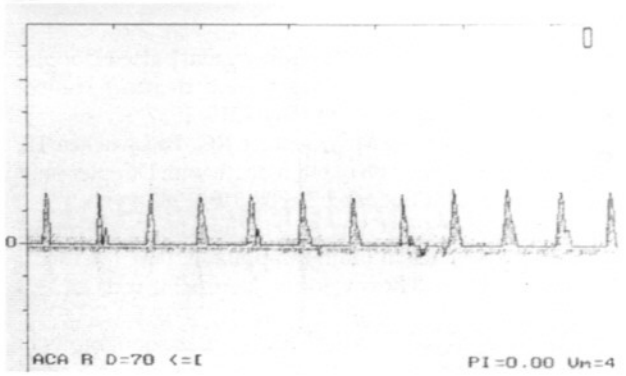

Figure 2. Transcranial Doppler recording demonstrating small early systolic spikes in the anterior cerebral artery. Velocity scale is in cm/sec.

TCD examination failed to demonstrate the waveform abnormality in only three brain-dead patients. All had evidence of brain stem destruction in computerized tomography.

DISCUSSION

The early diagnosis of irreversible cessation of cerebral function has become an important need due to the widespread practice of transplant surgery, and the optimal allocation of limited resources (21).

The characteristic and diagnostic phenomenon of intracranial circulatory arrest is an oscillating movement of the blood column within the arteries (4,18). This arrest causes brain death because of a complete and irreversible cessation of cerebral perfusion and this occurs when the arterial pressure is the same as the intracranial pressure. Reverberating flow pattern in the intracranial arteries are a reliable indication of arrest of circulatory cerebral flow (11,21). Evaluation of net flow velocity is a more sensitive determinant of brain death and closely parallels the patients' neurological function (15).

Hassler et al. (5) reported that with increasing intracranial pressure, the TCD waveforms exhibited different characteristics; high-resistance profiles with first low, then zero, and than reversed diastolic flow velocities, depending on the relationship between intracranial pressure and blood pressure. In the study of Petty et al. (14), TCD waveform abnormality occurred in brain-dead patients by clinical and EEG criteria or clinical criteria alone, but in none of the other patients in coma. These TCD waveform appearances had a sensitivity of 91.3 % and specificity of 100 % for brain death. They suggested that TCD could be an alternative to EEG for confirmation of brain death.

In our series, the finding of either absent or reversed diastolic flow, or small early systolic spikes, in more than 1 intracranial artery (as described in methods) in TCD investigation of a comatose patient was highly specific and sensitive for brain death. This characteristic TCD waveform abnormality was seen in 26 of the 29 patients who fulfilled the clinical criteria of brain-death, and in none of the comatose patients who were not brain dead. This flow pattern which is a systolic forward flow and an early diastolic reflux phenomenon is essential for the diagnosis of brain death. Our data demonstrate that, when patients meet established clinical criteria for brain death, TCD investigation is a valid method to confirm this diagnosis when properly incorporated into institutional protocols. TCD examination can be performed and interpreted rapidly by qualified physicians at the bedside within a matter of minutes and may therefore be preferable to other methods in the confirmation of brain death.

Three of our patients who met the clinical criteria of brain death had false-negative TCD studies with preservation of forward flow throughout diastole. Although rarely documented, there are a number of cases in literature in which radiologic studies have demonstrated preservation of cerebral perfusion in brain-dead patients (3). Petty et al. (14) reported that TCD examination failed to demonstrate the waveform abnormality in 2 (8.7%) of his 23 brain-dead patients. It is probable that the existance of a skull defect or ventricular drain permits preservation of cerebral perfusion in the presence of brain death by partially relieving the markedly increased intracranial pressure. Alternatively, massive brain stem injury might explain preservation of forward blood flow in the anterior circulation despite a clinical examination consistent with brain death (3). Whatever the mechanism of preservation of forward flow throughout diastole in these patients, such a finding in TCD clearly would not suggest the diagnosis of brain death.

The absence of a signal from any intracranial artery, even in patients who previously had obtainable signals when they were not brain dead, should not be considered confirmation of brain death, because of differences in technical skill from examiner to examiner. In a small number of patients, signals are unobtainable under any circumstance due to variations in anatomic configurations of the temporal bone and in its ossification. Furthermore, more authors believe that for the purpose of confirmation of brain death, the characteristic TCD signals should be detected in more than a single

artery (14). Failure to insonate more than one artery should be considered "nondiagnostic", and other diagnostic methods may then be required to confirm the diagnosis of brain death.

TCD ultrasonography was found to be a practical, non invasive, early and reliable method for the diagnosis of arrest of the cerebral circulation.

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