ELSEVIER

Transcranial Doppler Sonography in Fulminant Hepatic Failure

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ABSTRACT

The clinical course of patients with fulminant hepatic failure (FHF) is often worsened by the presence of cerebral edema and endocranial hypertension. In spite of the multiple studies using Transcranial Doppler Sonography (TCDS), few have shown the cerebral blood flow (CBF) pattern among patients with encephalopathy resulting from FHF.

Objective. Our objective was to characterize the CBF pattern in these patients through the use of TCDS to provide therapeutic strategies.

Method: The TCDS pattern was assessed in five patients diagnosed with FHF and compared with a control group who displayed critical neurologic conditions not associated with FHF. Pulsatile index, systolic, diastolic, and mean velocity of the middle cerebral artery were measured.

Results: The mean age of patients with FHF was 45.4 years. One hundred percent were women, with viral hepatitis as the predominant etiology. A cerebral hypoperfusion pattern was found in 80% of the FHF group and 40% of the control group. In the former group there was no evidence of hyperemia, as there was among 20% of the control group. The mean values of velocity and pulsatile index were 36.6 cm/sec and 2.4, respectively, in the FHF group and 47.8 cm/s and 1.8 in the control group ($P = 0.268$, $P = 0.402$).

Conclusions: FHF patients show a predominance of cerebral hypoperfusion pattern with mean velocities lower than normal values and an increased pulsatile index. We recommend that clinicians take appropriate measures to improve cerebral perfusion and avoid hypoxia. Hyperventilation as a first level measure is contraindicated.

THE CLINICAL COURSE of patients with fulminant hepatic failure (FHF) is often worsened by the presence of cerebral edema and endocranial hypertension. Cerebral edema is reported in more than 80% of patients who reach stage IV encephalopaty, which is associated with a high mortality rate.[1,2]

At present the treatment for the critical neurologic patient with endocranial hypertension is becoming more individualized and guided by a complex neuromonitoring system. Among these techniques is Transcraneal Doppler Sonography (TCDS), a noninvasive, bedside procedure, which was introduced into clinical practice in 1982 and validated for measurement of cerebral blood flow (CBF).[3]

In this paper, TCDS is described in groups of patients with versus without FHF. The control group included critically neurologic patients. We sought to characterize the CBF pattern in these patients, offering therapeutic strategies according to the pathophysiologic condition.

PATIENTS AND METHODS

The general data including age, sex, etiology, and encephalopathy degree, were obtained from five clinical records of patients admitted to intensive care unit (ICU) at Centro de Investigaciones Medico Quirurgicas and diagnosed with FHF from January 2001 to July 2002. For the first TCDS performed at admission to ICU, when blood pressure was normal, the data collection included systolic velocity (SV) and diastolic velocity (DV) values, mean velocity (MV) of the middle cerebral artery (MCA), its pulsatile index (PI). The values were compared with a control group of five patients with other neurologic critical conditions: Glasgow coma

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Table 1. Mean Values and Their Variable Ranks Reached Through Transcraneal Doppler Sonography

	Group I Fulminant Hepatic Failure (range)	Group II Control (range)	*P*
Systolic velocity (cm/sec)	99.60 (89–112)	104.20 (85–112)	.540
Diastolic velocity (cm/sec)	22.80 (10–44)	26.80 (9–30)	.846
Mean velocity (cm/sec)	36.60 (25–59)	47.80 (33–70)	.334
Pulsatile index	2.41 (0.79–4.08)	1.81 (1.05–2.94)	.597

scale ≤ 8, admitted to the ICU at the same time, and selected at random. A DWL, Smart Doppler with a 2-Mhz probe was used to explore the MCA through temporal windows. TCDS patterns of cerebral hypo, normal, and hyperemic perfusion, have been described in literature.[4]

Results are presented as means and their ranks. Mann-Whitney test was used. $P < .05$ was considered as statistically significant.

RESULTS

The five patients diagnosed with FHF were all women, with a mean age of 45.4 years (31–55 years), and with likely viral etiology. Among the five patients, one patient was successfully transplanted, two died of bleeding during the surgical intervention, and two died without transplantation. There were two patients with hepatic encephalopathy level II, two patients in level III, and one patient was level IV.

The mean age of the control group was 54.2 years (36–79 years), and the diagnoses were: two patients with hypoxic encephalopathy, and single cases of cranioencephalic trauma, intracranial hemorrhage, and barbiturate coma.

Variables obtained with TCDS in both groups are shown in Table 1.

In the group with diagnosis of FHF, there were four patients with a TCDS suggestive of hypoperfusion (80%) and one with a normal pattern (20%). In the control group, there were two patients with hypoperfusion pattern (40%), two with normal pattern (40%), and one with hyperemia pattern (20%).

DISCUSSION

TCDS revealed that the majority of patients with FHF showed a pattern of cerebral hypoperfusion characterized by MV values below normal and increased PI, even at more pathologic levels than the ones displayed by the control group of neurocritical patients experiencing diagnoses different from hepatic encephalopathy of FHF.

These findings are similar to those described in literature, some using other measurement methods for CBF.[5]

These results and the previous experiences with neurotrauma patients, using multimodal neuromonitoring, which includes TCDS, suggests the utility of the method for early detection of conditions that favor cerebral hypoxia.[6,7]

Hyperventilation as a first level measure to lower intracranial hypertension must not be used in these patients, until a hyperemia pattern is proven due to the CBF that it produces.[2,5]

Our results suggests that mannitol should be the drug of choice after general measures are taken. Systemic monitoring is needed to avoid hypotension, hypoxemia, or any other condition that favors cerebral hypoxia. On the other hand, high blood pressure should also be avoided because of the damage described in the cerebral self-regulation of these patients.[8,9]

Summing up, TCDS is a useful tool for the management of patients with FHF. The pattern mostly found is that of cerebral hypoperfusion. Therefore we recommend to exert measures that improve cerebral perfusion and avoid hypoxia. Hyperventilation is contraindicated as a first level measure.

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