Transcranial Doppler for evaluation of idiopathic intracranial hypertension


Objectives – The value of transcranial Doppler (TCD) ultrasonography in assessing patients with Idiopathic Intracranial Hypertension (IIH) is uncertain. We sought to determine the contribution of TCD to their evaluation.

Materials and methods – Twenty-three patients with suspected IIH underwent TCD. Mean blood flow (BFV), peak systolic (PSV) and end-diastolic (EDV) velocities, and pulsatility (PI) and resistance (RI) indexes were obtained in the middle cerebral (MCA) and vertebral (VA) arteries and compared (Student’s t-test) between patients with confirmed IIH and controls. IIH patients and controls were comparable in terms of age, gender and weight.

Results – The mean ± SD BFV MCA, PSV MCA, EDV MCA and PI VA in the 13 IIH patients were higher than in the ten controls (59 ± 6.8, 94 ± 28.5, 43 ± 12.4, 0.86 ± 0.16 and 50 ± 8.6, 72 ± 25.8, 32 ± 11.5, 0.58 ± 0.45 respectively, \( P < 0.05 \)) but still within normal values. The mean ± SD PI MCA, RI MCA and RI VA values in the IIH patients and controls were similar.

Conclusions – TCD parameters had no useful unique features for monitoring IIH patients.

Introduction

Pseudotumor cerebri or Idiopathic Intracranial Hypertension (IIH) is a condition characterized by the signs and symptoms of elevated intracranial pressure (ICP), without evidence of underlying primary structural or systemic causes. The mechanism for the development of elevated ICP resulting in primary IIH is still not fully understood. There are a number of theories for explaining the underlying pathophysiology, among them are elevation of ICP that may arise from resistance to cerebrospinal fluid (CSF) outflow, a decrease in CSF absorption, an increase in CSF secretion, altered cerebral hemodynamics and increased brain capillary permeability (1–3). Monitoring of patients with IIH could be of vital importance in ensuring the efficacy of therapeutic measures. Until now, the assessment and monitoring of patients with IIH required invasive methods, such as lumbar puncture (LP).

The most devastating effect of ICP elevation is a reduction in cerebral perfusion. Cerebral perfusion pressure (CPP), which is the driving force of brain perfusion, is determined by the differences between systemic arterial blood pressure and ICP (4). CPP is related to cerebral blood flow (CBF) as a ratio to cerebral vascular resistance. Previous studies have shown that CBF decreases at ICP levels > 35–45 cm H₂O (5). With the invention of transcranial Doppler (TCD) ultrasonography, it has become possible to perform non-invasive continuous monitoring of the blood flow velocities (BFVs) in the major basal arteries of the brain (6). Although TCD is not a CBF monitor, the CBF of the basal brain arteries can be directly correlated to BFV if the diameter of the artery at the point of flow velocity measurement remains constant. Therefore, BFV might be a useful quantitative indicator of cerebral perfusion (7). Recent studies have demonstrated specific patterns of TCD in patients with elevated ICP (8–12), but most of these findings were obtained on patients with critically elevated ICP after severe head injury, intracranial hemorrhage, brain tumors, and hydrocephalus. TCD also became an important supplementary tool for confirming brain death as a result of elevated ICP followed by a total cerebral...
circulatory arrest (13, 14). These data demonstrate a significant correlation between TCD parameters and elevated ICP regardless of causative factors. The value of TCD in assessing patients with IIH, however, is uncertain. In the current work, we sought to determine whether TCD could be useful in non-invasive evaluation and monitoring of patients with IIH.

**Materials and methods**

Twenty-three consecutive patients with suspected IIH who were hospitalized in the Department of Neurology of the Tel Aviv Sourasky Medical Center were recruited to the study. IIH was suspected because of the presence of clinical signs and symptoms of elevated ICP (headache, nausea, vomiting, visual disturbances, VIth nerve paresis) and positive findings on funduscopy (swollen optic disk). Before undergoing LP, all the eligible study patients underwent a computed tomographic (CT) brain scan and/or magnetic resonance imaging (MRI) of the head in order to exclude underlying structural lesions or sinus vein thrombosis. Inclusion criteria for the diagnosis of IIH were signs and symptoms of elevated ICP, a normal neurological examination apart from swollen optic disk or a VIth nerve palsy, the absence of structural abnormalities on neuroradiological studies, elevated opening CSF pressure > 20 cm H₂O in non-obese or > 25 cm H₂O in obese persons, with normal composition and microbiological results [according to Dandy’s Modified Criteria (15, 16)]. Body mass index (BMI) was calculated in all patients.

The intracranial arteries were evaluated by TCD (Rimed Trans-link 9900 TCD, Herzliya, Israel) before LP. The TCD examination was carried out with the patient in a supine position. It included transtemporal insonation of the middle cerebral artery (MCA) at a depth of 50–55 mm and transoccipital insonation of the vertebral artery (VA) at a depth of 60–90 mm using a 2-MHz handheld probe (17). The most powerful signal during a 10 s period was used for the measurements of the BFV. The TCD parameters consisted of peak systolic velocity (PSV), end-diastolic velocity (EDV) and mean BFV. The mean BFV was calculated as the time-averaged value of the envelope of the Doppler sonogram. Dimensionless variables, such as the pulsatility index [PI or Gosling index (18)] and the resistance index [RI or Pourcelot Index (19)] are not dependent on the insonation angle as is the mean BFV, and they were calculated as PI = (PSV–EDV)/mean BFV and RI = (PSV–EDV)/PSV, respectively, for each of the studied arteries. These parameters reflect the flow velocity waveform and indicate the cerebrovascular resistance. The normal limits of PI and RI were set at ≤ 0.70 ± 0.3 and ≤ 0.6, respectively (20, 21). Since there was no significant difference between the PSV, PI and RI values for the left and the right MCA and VA, the mean values were calculated for each patient and used throughout the study.

Statistical analysis of the TCD parameters obtained in patients with IIH and in the control group was performed using the paired Student t-test. Differences were established as being significant at P values of less than 0.05.

**Results**

Thirteen of the 23 eligible patients fulfilled the study inclusion criteria for the diagnosis of IIH and formed the study group. The control group consisted of the 10 remaining patients whose CSF pressure measurements were within normal limits based on the results of their LP. The demographic data, BMI, arterial and CSF pressures of all study participants are shown in Table 1. The two groups were comparable in terms of age, gender, weight and arterial pressure. The TCD parameters of patients with IIH and the control group are presented in Table 2. Although the mean BFV, PSVs and EDV values in the MCA of the patients in the study group were significantly higher than in the controls (P < 0.05), all these values were significantly lower than the normal limits.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Demographic data and results of lumbar puncture examinations</th>
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<tr>
<td></td>
<td>Study group n = 13*</td>
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<tr>
<td>Mean age ± SD, years</td>
<td>24.5 ± 7.4</td>
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<tr>
<td>Gender (female/male)</td>
<td>11/2</td>
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<tr>
<td>Body mass index (mean ± SD)</td>
<td>33.5 ± 5</td>
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<tr>
<td>Opening CSF pressure mm H₂O (mean ± SD)</td>
<td>320 ± 60</td>
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<tr>
<td>Systolic/diastolic blood pressure mmHg (mean ± SD)</td>
<td>130 ± 12.5/80 ± 10.5</td>
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*Patients with idiopathic intracranial hypertension.
†Patients with normal pressure on lumbar puncture.
within normal limits of BFV for all the studied arteries. The mean PI values in the VA were significantly higher in the study group but these also stayed within normal limits. All other TCD parameters were similar between the study and control groups.

**Discussion**

We looked for specific parameters of TCD for non-invasive evaluation of patients with IIH. Our results showed that TCD parameters, i.e., BFVs, PIs and RIs, remained within normal values in the IIH patients in both anterior and posterior circulations (MCAs and VAs). Several earlier studies had investigated a relationship between elevated ICP and TCD features. Homburg et al. (8) measured BFV and PI by TCD in neurosurgical patients with head injuries and normal subjects and found a positive exponential correlation between ICP and PI in the group of brain-injured patients. Those authors suggested that the non-invasive bedside TCD registration may be a useful marker of ICP and could probably replace the former invasive methods of measuring ICP. Using TCD for studying elevated ICP was also investigated in various types of patients (subarachnoid hemorrhage, head injury, hydrocephalus) and the results revealed a strong correlation between PI, RI and ICP (9, 11, 21–24). In contrast, other investigators failed to demonstrate any useful TCD parameters in their attempts to evaluate ICP by means of TCD. Krauss and Droste (25) simultaneously monitored ICP and BFV in patients with symptomatic normal pressure hydrocephalus in order to identify patients who might benefit from subsequent shunting. They found no significant relationship between ICP and RI and concluded that TCD monitoring is not suitable for replacing ICP monitoring. Hanlo et al. (26) investigated TCD/ICP measurements in infantile hydrocephalus and noted that the correlation between PI or RI and ICP was generally poor and that TCD indices were inadequate for monitoring the complex intracranial dynamic responses in patients with elevated ICP. What has been universally accepted is the fact that TCD has unique patterns, such as systolic spikes, oscillating flow waveform (‘to-and-fro’), and no flow signals if the ICP rises over the systolic arterial pressure with cessation of cerebral perfusion (27, 28). As such, TCD was recognized as being a highly specific and sensitive supplementary confirmatory test in the protocol for the assessment of brain death (29, 30). Our hypothesis was that patients with IIH might also have specific intracranial blood flow patterns, although not as prominent as in critically high ICP, but which could be detected by TCD. The proposed relationship between TCD parameters and different conditions with elevated ICP can be used in clinical settings only if there is a clear demarcation between abnormal and normal ICP values because of the high variability of TCD parameters. Therefore, we chose a group of patients with suspected IIH, and not healthy persons, to serve as controls, and tested the relevant TCD parameters in order to evaluate IIH in the setting of clinical practice.

Our literature search elicited only one short report on the TCD measurements in patients with IIH (31). TCD was performed immediately before and following LP in five female patients whose LP results showed that the CSF opening pressure was elevated (mean 32.7 cm H$_2$O). The mean BFV values and PI were recorded by means of TCD, and PI values were found to be in range between normal to even below normal, with an inverse correlation between the CSF opening pressure and the PI. The post LP PI values and the mean BFV did not change significantly despite the reduction of CSF opening pressure. TCD parameters in IIH patients were not compared with those of a control group, and the vessels studied by TCD were not mentioned in that study. Our current findings indicate that TCD provides no specific patterns for evaluating patients with IIH. These data support Giulioni et al. (32) who concluded that TCD parameters are not significantly affected by ICP changes during moderate ICP elevation when cerebral regulatory mechanisms are functioning effectively.

**Table 2** Transcranial Doppler parameters: mean PSV cm/s, mean BFV cm/s, EDV cm/s, PI and RI values ± SD

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Study group n = 13*</th>
<th>Control group n = 10†</th>
<th>P-value</th>
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<tr>
<td>PSV MCA</td>
<td>94 ± 28.5</td>
<td>72 ± 25.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>EDV MCA</td>
<td>43 ± 12.4</td>
<td>32 ± 11.5</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean BFV MCA</td>
<td>59 ± 6.8</td>
<td>50 ± 8.6</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>PSV VA</td>
<td>48 ± 18.2</td>
<td>36 ± 14.5</td>
<td>NS</td>
</tr>
<tr>
<td>EDV VA</td>
<td>24 ± 6.5</td>
<td>29 ± 5.8</td>
<td>NS</td>
</tr>
<tr>
<td>Mean BFV VA</td>
<td>28 ± 9.5</td>
<td>27 ± 6.4</td>
<td>NS</td>
</tr>
<tr>
<td>PI MCA</td>
<td>0.86 ± 0.09</td>
<td>0.82 ± 0.18</td>
<td>NS</td>
</tr>
<tr>
<td>PI VA</td>
<td>0.86 ± 0.16</td>
<td>0.58 ± 0.45</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>RI MCA</td>
<td>0.54 ± 0.02</td>
<td>0.59 ± 0.14</td>
<td>NS</td>
</tr>
<tr>
<td>RI VA</td>
<td>0.52 ± 0.06</td>
<td>0.48 ± 0.12</td>
<td>NS</td>
</tr>
</tbody>
</table>

*Patients with idiopathic intracranial hypertension.
†Patients with normal pressure on lumbar puncture.

PSV, peak systolic velocity; MCA, middle cerebral artery; EDV, end-diastolic velocity; BFV, blood flow velocity; VA, vertebral artery; PI, pulsatility index; RI, resistance index.
Gur et al.

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References