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ORIGINAL ARTICLE

Transcranial Doppler in Patients with Severe Traumatic Brain Injury: A Cross-Sectional Study

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ABSTRACT

Aim: To assess the role of transcranial Doppler in patients with severe traumatic brain injury
Study design: A cross-sectional study
Place and duration: This study was conducted at Mekran Medical college Turbat Pakistan.
Methodology: Transcranial Doppler **(**TCD) was performed on 255 patients with severe traumatic brain injury (TBI) who had a Glasgow Coma Scale of 8 or less on admission. All the patients were on a ventilator. TCD was performed on the first, second, third, and seventh days of the hospital stay. Two out of three of the following were used to define hypoperfusion: 1. A mean velocity of less than 35 cm/sec in the middle cerebral artery 2. The diastolic velocity of the middle cerebral artery is seen to be lesser than 20 cm/sec and 3. A pulsatility index of more than 1.4 was recorded.
The following criteria were used to identify vasospasm: mean velocity of the middle cerebral artery more than 120 cm/sec and Landegaarde index greater than 3. The Lindegaard index is the ratio of middle cerebral artery blood flow to internal carotid artery blood flow.
Results: The results showed that 141 people (45 percent) had normal readings. Out of the 141 patients, 55 were admitted to a long-term care facility, 43 patients were discharged. Ten of the patients were unconscious or vegetative, and their relatives pleaded for their loved ones to be removed from them. Four people died as a result of brain death, while two others died as a result of injuries. Seventy-two patients (28%) had hypoperfusion, and 71 died, 65 through brain death, 6 from stopping care, and one with mild disability. Vasospasm was present in 69 individuals (27%).
Conclusion: Patients with normal measures should have a good chance of survival. Hypoperfusion patients had a bad prognosis. The use of nimodipine in patients with vasospasm should be examined, but further research is needed to determine its safety and effectiveness. TCD is helping to assess early prognosis.
Keywords: Prognosis, Transcranial Doppler, traumatic brain injury, hypoperfusion, vasospasm

INTRODUCTION

A traumatic brain injury (TBI) is predicted to affect 1.7 million persons in the United States each year. About 275,000 of them are admitted to hospitals, and 52,000 of them die. In 30.5 percent of all injury-related fatalities, a TBI is a contributing cause. (1-5) Although the initial damage occurs at the time of impact, secondary injuries might arise as a result of hypoxia, hypotension, and hemodynamic abnormalities in the brain. The capacity to quantify cerebral perfusion is critical for TBI monitoring. This may be done by monitoring cerebral perfusion pressure (CPP), however, an invasive intracranial pressure monitor is required (ICP). TCD (transcranial Doppler) is a non-invasive procedure. Basal Cerebral Arteries help assess the blood velocity. Changes occurring to the flow velocity of the middle cerebral artery (MCA) are closely linked to the changes that take place in the cerebral blood flow (CBF). Measurements of flow velocity in the MCA can thus detect hypoperfusion or vasospasm (6-8). In the treatment of severe TBI, TCD is not widely used. The purpose of this research was to discover if TCD can benefit those who have had a severe TBI.

METHODOLOGY

The study looked at patients who received TCD between June 2019 to June 2021who had a severe TBI characterized as a Glasgow Coma Scale score of 8 or below. Permission was taken from the ethical review committee of the institute. To determine the severity of Traumatic brain injury, CT scans brain were used (6). Patients who were either sent to for emergency surgery or to the ICU. This depended on how badly the patient was injured. To avoid seizures, the victims were given either fosphenytoin or phenytoin along with mannitol. Mechanical ventilation was provided to all the victims. Normocapnia and hypocapnia, adjustments were needed in the mechanical ventilation. Blood or crystalloids were given to the patients for resuscitation. Both propofol and fentanyl were administered to maintain sedation. To decrease neurological status, craniotomy was done. (9) On the 1st, 2nd, 3rd, and 7th day of being hospitalized, TCD was done on the patient. Temporal windows were used to insonate both the left and right middle cerebral arteries. Whereas in the upper cervical area, the ICA was insonated. High mean velocity was chosen with a depth of insolation. Mean velocities (Vm), peak systolic (Vs), and end-diastolic (Vd) were measured. The Pilsating index was calculated automatically with the help of each recording. The velocities of MCA/ICA ratios were computed (Lindegaard index). (10)

Hypoperfusion was identified if two of the following symptoms were present, according to prior investigations.

The investigations recorded, mean middle cerebral artery velocity (Vm) as 35 cm/sec, the pulsatility index (PI) is more than 1.4, and lastly velocity diastolic (Vd) 20 cm/sec. Vasospasm was identified based on prior research if the VM was more than 120 cm/sec and/or the index was greater than 3. The Glasgow Outcome Scale was used to determine the outcome (GOS). (11-13)

RESULTS

A total of 252 patients were investigated in this study period . The mean recorded was 40.3(SD 18.06), with the age of the participants ranging from 16 to 85 years. With a mean of 28.3 and a standard deviation of 5, with 5 to 66 (1.104 SD) ranging as the (ISS) injury severity score. There were a total of 209 males and 46 females in the group. The mechanism of injury is shown in table 1

Table 2 shows the results with normal readings at 45%, Vasospasms at 27%, and Hypoperfusion at 28% among 255 participants. Initially, 11 patients have been recorded to have normal readings but all of them died due to the development of hypoperfusion. Patients that had hypoperfusion were 18 and were seen to improve however, 8 died, 6 from withdrawal, and 2 from brain death. A subarachnoid hemorrhage was a prominent component of the TBI in 86 individuals (34%), whereas Hypoperfusion affected 21 (29 percent), 40 (35%) had normal measurements, and vasospasm affected 25 (36 percent).

Table 1: Mechanism of injury

Motor vehicle accident	87 patients
Fall	52
Motorcycle	32
Assault	19
Gunshot wound	18
Auto-pedestrian	17
Fall/jump from MV	8
Horse	6
Other	16

(ATV, bicycle, construction site)

Table 2: shows the age, severity of the injury, vasospasm outcome

	Normal	Hypoperfusion	Vasospasm	Total
No. of patients	114 (45%)	72 (28%)	69 (27%)	255
Age	41.39±17.7	40.3±18.1	38.88±17.03	40.3±18.1
Injury severity score	26.2±10.2	29.8±11.4	30.3±11.5	28.3±11.0
Died	16 (14.0%)	71 (98.6%)	22 (31.9%)	109 (42.7%)
Brain death	4 (3.5%)	65 (90.2%)	13 (18.8%)	82 (32.1%)
Moderate disability GOS 2-3	6 (5.2%)	1 (1.4%)	16 (23.2%)	23 (9.0%)
Good - GOS 4	40 (35.1%)	0	21 (30.4%)	61 (23.9%)
Normal GOS 5	52 (45.6%)	0	10 (14.5%)	62 (24.3%)

Nimodipine was not given to 50 of the individuals who had vasospasm. 17 of them died (34 percent), with 11 dying of brain death, four dying of withdrawal of treatment, and two dying of other reasons. Their GOS averaged 2.78. Nimodipine was given to 19 patients. Five of them died (26 percent), two from brain death, and three after being taken off life support. Their GOS averaged 3.2.

Table 4 shows the average mean and diastolic velocities for the middle cerebral artery, the pulsatility index, and the Lindegaard number for the right and left sides of the normal and vasospasm groups on hospital days 1, 2, 3, and 7: In individuals with normal velocities, the table indicates growing hyperemia after the first 24 hours. The Vm and Vd have increased, but the PI and Landegaarde ratios have not. There is an increase in velocity in the vasospasm group, but no change in the pulsatility index.

Table 3: Discharge status for patients with normal, hypoperfusion, and vasospasm.

	Normal	Vasospasm	Hypoperfusion
Home	43	12	0
LTAC	55	35	1
Died	16	22	71

Table 4: displays average right and left mean velocities (VMR & VL), average left and right pulsatility index (PIL & PIR), average right and left diastolic velocities (VDR & VDL), and average Lindegaard index (LL 7 LR) for vasospasm groups on hospital days 1, 2, 3, and 7.

	Day 1	Day 2	P value	Day 3	Day 7
VML	59.7 + 20.7	70.7 + 23.7	<0.001	78.8 + 23.76	74.4 +21.58
VMR	58.9 + 18.5	70.3 +21.1	<0.001	74.7 + 22.2	72.4 + 22.53
VDL	39.1 + 14.9	45.6 +16.95	<0.001	52.1 +18.31	48.7 + 14.63
VDR	39.1 + 13.5	47.0 + 15.68	<0.001	49.3 + 16.68	48.0 + 16.6
PIL	0.99 + 0.24	0.96 + 0.21	0.4	0.94 +0.26	1.0 + 0.28
PIR	0.97 + 0.24	0.93 + 0.22	0.02	0.95 + 0.26	1.0 + 0.38
LL	1.84 + 0.63	1.96 +0.62	0.046	2.14 + 0.54	2.1 + 0.62
LR	1.79 + 0.48	1.96 + 0.56	<0.001	2.03 + 0.56	2.0 + 0.52

Table 5: Vasospasm on days 1, 3 and 7

	Day 1	P-value	P-value	Day 3	Day 7
VML	89.1 + 29.25	103.7 + 33.49	<0.001	120.7 + 31.16	115.2 + 35.48
VMR	86.1 + 33.57	101.1 + 35.54	<0.001	119.3 + 34.39	107.0 + 37.2
VDL	59.8 + 22.4	72.0 + 26.68	<0.001	82.5 + 28.34	79.9 + 28.72
VDR	59.97 + 26.58	71.2 + 27.52	<0.001	81.7 + 29.11	75.7 + 31.75
PIL	0.88 + 0.24	1.0 + 0.95	0.27	0.94 + 0.68	0.997 + 0.97
PIR	0.85 + 0.23	0.94 + 0.78	0.296	0.89 + 0.8	0.87 + 0.22
LL	2.28 + 0.88	2.66 + 1.09	0.002	2.79 + 0.83	2.79 + 0.83
LR	2.33 + 1.17	2.55 + 0.88	0.236	2.78 + 0.77	2.78 + 0.77

VML is the mean velocity of the left side of the middle cerebral artery in centimeters per second.

VMR stands for the mean velocity of the right side of the middle cerebral artery in centimeters per second.

VDL = diastolic velocity of the left side of the middle cerebral artery in cm/sec.

VDR = diastolic velocity of the right side of the middle cerebral artery in cm/sec.

PIL = left side pulsatility index.
Right side PIR = pulsatility index.
LL stands for Landegaarde number on the left side.
LR stands for Landegaarde number on the right side.

DISCUSSION

This analysis is based on prior classification research on the occurrence of hypoperfusion, vasospasm, and normal velocities and how they implicate each other. Santbrink et colleagues discovered a 63 percent incidence of hypoperfusion with a low flow velocity condition in 57 patients within the first 72 hours after admission, with 26 percent of them dying. (15) Using the same criteria as the Santbrink research, 90 patients (35%) experienced hypoperfusion with a low-velocity flow condition in our investigation, however, our death rate in hypoperfusion patients was 88 percent (79/90). A Vd of less than 25 cm/s and a PI of more than 1.3 were linked to a bad result in children, according to Trabold et al. Adults, according to our research, are on the same page. Patients who suffer hypoperfusion at first and later correct their measures nevertheless have a high death rate. Eighteen patients had hypoperfusion initially, which brought their readings back to normal, but two from brain death eight of them died, and six from therapy withdrawal. Hypoperfusion is almost usually fatal if persistent.

With a GOS score of 4-5, 40% of the patients in the Santbrink trial had a positive result. (16) In 54 percent of cases, Moreno et al reported a positive result. In our investigation, 48 percent of the patients (123/255) had a positive outcome. Patients with normal readings saw a significant increase in flow measurements from day one to day two, going from Vm 59.7 cm/s to 71.0 cm/s.

TCDs report that the rate of post-traumatic vasospasm ranges from 26.7 to 40%. Vasospasm was detected in 27% of our patients. The beginning of vasospasm can occur anywhere from day one to day thirteen of hospitalization, with days two and three being the most prevalent. Vasospasm usually resolves after 14 days of a traumatic brain injury, while instances have been documented to continue up to 30 days. Most resolve after 5 days, according to Oertel et al. We did not routinely maintain TCD monitoring until the vasospasm was resolved.

Subarachnoid hemorrhage, which can affect up to 60% of TBI patients, is a poor predictive risk factor. In our study, 86 people (34%) experienced a SAH as a significant component of their TBI, with 34 (40%) dying. Blood clot load is thought to be linked to the development of vasospasm after aneurysmal SAH. Even if there is no SAH, post-traumatic vasospasm can develop. This raises the question of whether aneurysmal SAH and post-traumatic SAH have comparable pathologies. (17)

Vasospasm in aneurysmal is often treated with the help of 'Triple H therapy' (hypertension, hypervolemia, and hemodilution) however the treatment for vasospasm induced due to a TBI is still challenging. Triple H therapy has the risk of increased bleeding throughout the body not just the brain in an event of trauma. Morbidity can be minimized in aneurysmal SAH through calcium channel blockers. Calcium channel blockers, on the other hand, can influence cerebral perfusion pressure and cause hypotension, which can lead to secondary brain damage in trauma. (18) A random trial was taken to understand the impact of medications of nimodipine in comparison with placebo vasospasm and traumatic SAH patients. The results that were collected after 6 months proved that the nimodipine group had a lower rate of cerebral infarction on poor clinical outcomes, CT, vasospasm, and radio-graphic. (19) A second double-blind placebo-controlled experiment found that Nicardipine therapy improved Doppler flow but had no effect on clinical outcome. Other randomized controlled studies have shown inconsistent outcomes, however, there are some overall tendencies toward clinical improvement. A Cochrane Review meta-analysis, on the other hand, discovered that calcium channel blockers are unsuccessful in the treatment of TBI in general. Studies reveal that patients suffering from Traumatic SAH and TBI had a modest benefit. (20) When comparing calcium channel blocker therapy to placebo treatment (OR 0.95, 95 percent CI 0.71-1.26), research has proven that traumatic SAH patients revealed no difference in mortality or poor outcome (OR 0.88, 95 percent CI 0.51-1.54).

CONCLUSION

TCD is beneficial to people who have had a severe TBI. It aids in outcome prediction and management. With a GOS 4-5 and minimal mortality from brain death, almost 80% of patients with normal measures should predict a positive result. Patients that received hypoperfusion measures had a high mortality rate, with 90 percent dying from brain death and an overall mortality rate of 98.6 percent. Patients with normalized hypoperfusion readings had a mortality risk of 44%. Patients who had vasospasm measurements died at a 31.9 percent greater rate, with 18.8 percent dying from brain death. They also had a worse result, with 44.9 percent receiving a GOS of 4-5. Vasospasm is a difficult condition to cure. Nimodipine should be examined, however, further research is needed to evaluate its effectiveness.

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