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TRANSCRANIAL DOPPLER STUDY IN TRAUMATIC SUBARACHNOID HEMORRHAGE

Neurosurgery

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ABSTRACT

Purpose of article: to review the practical aspect of TCD study in tramatic subarachnoid hemorrhage as a non invasive imaging modality.
Findings: TCD measures peak systolic (PSV), mean (MFV), and diastolic (EDV) cerebral blood flow (CBF) velocities and calculates the pulsatility index from basal intracranial arteries. These variables reflect the brain circulation, provided there is control of potential confounding factors. TCD can be useful in patients with severe TBI to detect low CBF, for example, during intracranial hypertension, and to assess cerebral autoregulation. In the emergency room, TCD might complement brain computed tomography (CT) scan and clinical examination to screen patients at risk for further neurological deterioration after mild-to-moderate TBI. Early bedside management of moderate head injury can be possible if vasospasm and low flow velocity state detected by TCD.

KEYWORDS

TCD, LR, post traumatic vasospasm, low flow velocity state

INTRODUCTION:

The incidence of vasospasm in aneurysmal subarachnoid hemorrhage (aSAH) has been extensively studied; less is known regarding the epidemiology of posttraumatic vasospasm (PTV).[1] PTV is not routinely assessed unless there are signs or symptoms suggesting its presence, which makes determining its true incidence a difficult endeavor. Wilkins and Odom asserted in 1970 that PTV was mechanistically similar to vasospasm in aSAH, in which the subarachnoid blood irritates the cerebral vessels.[2]Current literature shows that PTV typically occurs earlier [after TBI] than does aneurysmal vasospasm (2–3 days post-trauma vs 3–5 days post–aneurysmal vasospasm).[3] Transcranial Doppler Ultrasound introduced by Aaslid and colleagues[4]in 1982. Transcranial Doppler (TCD) used for measuring cerebral blood flow through basal cerebral arteries and various parameters can diagnose cerebral vasospasm and low flow velocity state, on which basis management of traumatic brain injury patients can be modified.

MATERIAL AND METHODOLOGY:

TBI patients admitted in neurosurgical emergency and kept under observation, monitoring, supportive and medical treatment. The monitoring involved vitals, pupils and GCS assessment or symptoms of Traumatic SAH(persistent vomiting, severe headache). The study stratified head injury patients into mild, moderate and severe. On admission and after 48 hours of admission TCD parameters like MFV (mean flow velocity), EDV (end diastolic velocity), PSV (peak systolic velocity), LR (Lindegaard Ratio), PI (Pulsatality index), RI (resistive index) checked and statistical analyses done. Total 70 patients studied after proper informed consent and analyses done on prospective basis.

RESULTS :

We successfully insonated basal arteries of 70 patients with traumatic brain injury with CT scan finding showing presence of SAH either isolated or associated with other intracranial abnormalities. Middle cerebral artery detected in all 70 patients through temporal window with average angle of insonation 30°±13° bilateral side. We did not find any gross difference between parameters of right and left side. We collected data from 70 patients (51 males and 19 females). The mean age was 42.6±17.72 years. Traffic accidents was the most common cause of trauma (48 cases) followed by fall (18 cases), assaults (4) cases. Ct scan findings shown in table 1.

Table 1

Findings in CT Scan	Frequency	Percentage
Acute SAH exclusively	18	25.7
Acute SDH with SAH	23	32.9
Calvarial fracture with SAH	4	5.7
Hemorrhagic contusion with SAH	11	15.7
DAI with SAH	11	15.7
EDH with SAH	3	4.3

We calculated approximate day of admission after patients had head injury. 47% patients admitted at second day after injury followed by 27.1% within 1-day after injury. Only 2.8% patients each admitted after 5 days of injury. We have kept admitted patients till 48 to 72 hours of admission and studied TCD parameters. As per Morris- Marshal grading of traumatic SAH (tSAH), our study patients CT scan on admission show following patterns of SAH as per table 2.

Table 2

Grade	CT scan findings	Frequency	Percentage
0	No CT evidence of tSAH	0	0
1	tSAH present at only one location	15	21.43
2	tSAH present at only one location but quantity of blood fills that structure or tSAH is at two sites, filling neither of them	20	28.57
3	tSAH at two sites, one of which is the tentorium, filled with blood	16	22.86
4	tSAH present at three or more sites, any quantity	19	27.14

As per severity of head injury our patients had following pattern of head injury on admission . On admission, majority of the patients (64.3%) had moderate injury while 10% patients had severe injury. At 48 hours, 40% of the patients had severe injury while 33% patients had mild injury. After 48 hours again GCS of patients assessed and categorized them accordingly. After 48 hours for GCS and severity of injury, we found that 40 patients who had acute SAH as a component of TBI had deterioration of general condition on the basis of GCS in spite of conservative treatment given to them in form of Inj mannitol, InjEptoin, diuretics, fluid management and proper input output charting.

Out of 70 patients, 21 patients showed improvement in general condition on conservative basis, and 9 patients showed same status after 48 hours

Mean flow velocity

We studied mean flow velocity of bilateral side MCA on admission, which shows 3 patients had MFV ≥120cm/sec on admission, so suspected vasospasm on admission present.

On the basis of MFV of B/L MCA, 12 patients had low flow velocity state (MFV <40 cm/sec), 2 patients had MFV between 100 and 120 cm/sec, and 53 patients had normal MFV between 40 to 100 cm/sec. At 48 hours, 16 patients had low flow velocity, 34 patients between 41-100 cm/sec, 5 patients between 100-119 cm/sec, and 15 patients' ≥120cm/sec. (figure 1)

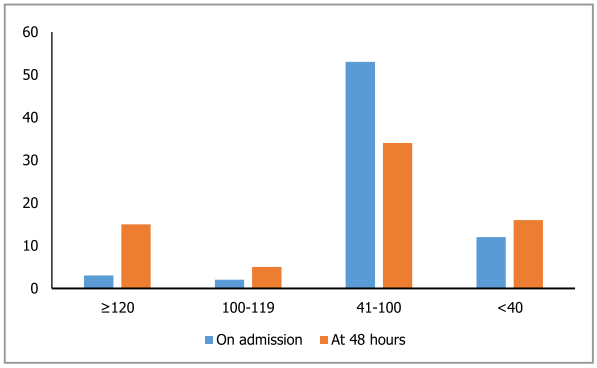

Figure 1

On admission, only 3 patients out of 70 had vasospasm detected by TCD, but over the period of 8 hours, 15 patients developed vasospasm, and MFV of MCA increased >120cm/sec. Degree of vasospasm dependent on caliber of MCA, and it is inversely related to it. Hence, 15 patients who developed vasospasm, out of which 8 had mild vasospasm, 3 had moderate vasospasm and 4 had severe vasospasm

Post-traumatic period of vasospasm
Total 15 patients developed vasospasm after 48 hours of admission post traumatic brain injury in mild, moderate or severe pattern. These 15 patients developed vasospasm after 2 to 7 days of injury. Average post trauma day of developing vasospasm was 3.53 days

Lindegaard ratio [MCA MFV/extracranial ICA MFV]
We found that after 48 hours of admission, 17 patients had Lindegaard ratio >3, out of which 13 patients had ratio between 3-6, and 4 patients had ratio more than 6

Resistive index [(PSVEDV)/PSV]
After 4 hours of admission, total 15 patients had low flow velocity, out of which 5 patients showed resistive index more than 8 which shows that there was increased downstream resistance

Pulsatality index [(PSVEDV)/MFV]
Out of 70 patients, 26 patients found pulsatality index in normal range at admission and 44 patients had pulsatality index >1.2 which means on admission, 44 patients had cerebrovascular autoregulation in such a way that they developed distal vascular increased resistance. After 48 hours, 25 patients had pulsatality index >1.2, it means gradually distal flow resistance decreased in rest of the patients and they had low flow stat in MCA after 48 hours.

DISCUSSION

TCD may play a role in monitoring the early development of cerebral VSP after traumatic subarachnoid hemorrhage (SAH) and the early or delayed development of increase ICP following TBI.[7] This is achieved by daily noninvasive monitoring of CBF, MFV, and ICP of the intracranial vessels.[8]

We measured MFV at admission and after 48 hours and we found that patients with MFV of approximately 50 to 100cm/sec are less likely to develop vasospasm afterwards, but patients with MFV >100 cm/sec are more likely to develop vasospasm, but deterioration of GCS was not correlated with degree of vasospasm. Vasospasm once sets in,it remains for a long time, and affect the consciousness level of patient also.

In the study Zeigler et al.[9], vasospasm group comprised of 69(27%) patients, out of which 22(31%) died and 31(45%) had good outcome. Prasad et al.[6] have recently studied role of TCD in TBI. They observed that 21 patients (28%) had vasospasm. Six out of these twenty-one patients died, 11 patients had severe neurological disability and four patients were discharged in good neurological status.

As per van Santbrinket al.[10], statistically significant relation was found between the occurrence of a LFVS and the severity of injury, age, occurrence of traumatic subarachnoid hemorrhage (tSAH), the degree of midline shift, CT classification and outcome. Mean GCS of patients with LFVS was 5.6±1.7 and in patients without a LFVS 6.9±1.1 (p = 0.008). Mean midline shift in patients with LFVS was 4.9±5.7 mm versus 1.71±3.26 mm in patients without LFVS (p = 0.01). A strong correlation was noted between LFVS and unfavorable outcome (p = 0.01). Univariate analysis showed a strong association between LFVS and death (odds ratio 4.8; CI: 0.96–24) and between LFVS and unfavorable outcome (odds ratio 3.9; CI: 1.2–13).

As compared to this, our study showed Low flow velocity on admission in 12 patients out of 70. In these 12 patients, CT scan findings showed unilateral or bilateral pathology with significant midline shift (average 5.2 mm) opposite to side of lesion if unilateral pathology, and uncal herniation was seen in CT scan.

In our study, we found 3 patients had MFV >120cm/sec with GCS of average 10.3 on admission, and after 48 hours total 15 cases diagnosed to have MFV >120cm/sec. and out of 15 who had HFVS, 13 patients had deterioration of GCS with mean GCS of 6.66 (P<0.0001). Two patients improved after HFVS at 48 hours of admission by conservative management. 13 patients who had GCS reduction at 48 hours, their GCS reduction calculated and average reduction of GCS was 3.3 points. But in present series, no clinical outcome correlation found significant with HFVS.

Lindegaard Ratio
Weber et al.[5] calculated LR by MFV of MCA with MFV of same side ICA, he found that LR <3 was not associated with vasospasm, but LR >3 was related to significant vasospasm, and they monitored outcome of patients till 6 months who has raised LR >3, they found Ischemic damage on ipsilateral side over the period of times.

In our study, we found 17 patients developed LR >3 after 48hours of admission, out of which 13 had ratio between 3-6, and 4 had ratio >6, which is related to severe vasospasm. We correlated deterioration of GCS in patients with LR >3.We found 12 patients out of 17 had deterioration of GCS at 48 hours of admission due to vasospasm mainly. And 2 patients out of 17 improved GCS to one point and 3 patients remained same as far as GCS concerned till 48 hours. By measuring LR, we can identify actual vasospasm, and we can intervene at earlier stage.

CONCLUSION

TCD allows a noninvasive assessment of real-time monitoring of ICP and cerebral perfusion pressure (CPP). TCD ultrasonography can be performed at the patient's bedside to determine and monitor CB), measured by MFV, and ICP can be measured by PI values of the MCA, and the rest of major intracranial vessels, regardless of patient having an altered level of consciousness or being sedated. In addition, TCD may play a role in monitoring the early development of cerebral VSP after traumatic SAH and the early or delayed development of increase ICP following TBI. This is achieved by daily noninvasive monitoring of CBF, MFV, and ICP of the intracranial vessels. The diagnostic and prognostic role of TCD in TBI has not been fully established since available data are limited due to unavailability of any randomized controlled trials, and a small number of prospective cohort study.

A total of 70 patients who fulfilled the criteria were consecutively enrolled in the study who presented with TBI to the emergency department after informed consent. The aim of the study was to outline management and outcome of patient with traumatic brain injury either having isolated subarachnoid hemorrhage (SAH) or SAH associated with Contusions, laceratios, diffuse axonal injury, EDH on the basis of TCD examination at tertiary care centre.

Three patients had vasospasm at the time of admission and 15 patients developed vasospasm after 48hours of admission. The patients with higher MFV (>100cm/sec) on admission has high chances of development of vasospasm after 48hours. Mortality rate and proportion of patients with poor outcome was significantly higher in patients with severe vasospasm. The patients with MFV <50cm/sec are more prone to go into low flow velocity state after 48 hours. The patients with High grades of SAH on admission has more chances of development of Vasospasm after 48 hours. By use of TCD we can easily and non invasively detect vasospasm at early stage and modify our ways of management.

There are various ways of diagnosing Post traumatic vasospasm, by CT angiography, cerebral blood flow study, Xenon study, digital subtraction angiography, but all these modalities are invasive and none of them are available bedside. TCD is a modality of examination of blood vessel status of intracranial compartment at bedside, and at

feasible manner as and when required without shifting patient, and it is also non invasive. Mean flow velocity (MFV), Lindegaard ratio (LR),pulsatality index (PI) and resistive index (RI) can be measured by bedside TCD in post traumatic period. Vasospasm or low flow velocity state at an early period can be diagnosed by TCD examination frequently.

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