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# The Safety and Efficacy of Ultrasound-Guided Stellate Ganglion Block and Nimodipine for Treating Cerebral Vasospasm Following Trauma

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## ABSTRACT

**Background:** Cerebral vasospasm (CV) is a common complication after traumatic brain injury (TBI), with its underlying mechanisms not well understood, complicating the development of effective management strategies.

**Objectives:** The study aimed to compare the safety and efficacy of ultrasound-guided stellate ganglion block (SGB) and nimodipine therapy (NT) as preventive measures against CV after TBI.

**Patients and methods:** The study examined adult patients diagnosed with cerebral vasospasm (CV) following TBI. Evaluation methods included trans-cranial Doppler (TCD) and neurological assessments. Patients in the NT group received symptomatic treatment via ultrasound-guided stellate ganglion block (SGB) daily for one week. Therapeutic outcomes were assessed through mean cerebral blood flow velocity (MBFV), Glasgow Coma Scale (GCS), and the Modified Rankin score.

**Results:** No procedure- or medication-related deaths were documented throughout the course of the study. With a $P < 0.001$, the MBFV of the middle cerebral artery (MCA) showed a significant decrease in both groups as compared to the baseline. Following treatment, the SGB group's MBFV of MCA was significantly higher than the NT group's, with a p-value of 0.047. There was no significant difference in complication rates between the two groups. The modified Rankin score was observed to be significantly higher in the SGB group compared to the NT group ($P < 0.01$).

**Conclusion** Ultrasound-guided SGB and NT demonstrated comparable efficacy in mitigating CV risk among patients with TBI. SGB can serve as a supplementary treatment alternative, particularly in cases where conventional approaches are inappropriate or yield insufficient results.

**Keywords:** Cerebral vasospasm (CV), traumatic brain injury (TBI), Transcranial Doppler (TCD), Stellate ganglion block (SGB), Nimodipine therapy (NT).

## INTRODUCTION

Following a TBI, CV can considerably affect the neurological and functional recovery of injured patients. Clinical impairments are observed in only 3.9% to 16.6% of individuals, even though the reported prevalence varies from 19% to 68% [(1)].

Nimodipine, a pharmacological agent classified as a calcium channel blocker, holds the unique clinical distinction of being the sole medication that has received regulatory approval for the specific management and treatment of cerebral vasospasm (CV). This designation highlights its pivotal and essential role in the current neurocritical care protocol, primarily due to its mechanism of reducing the aberrant smooth muscle contraction in cerebral arteries. This medication facilitates the relaxation of arterial smooth muscles and is widely recognized for its effectiveness in reducing blood pressure [(2)]. The favorable results observed in cerebral arteries are most likely due to the neuroprotective effects [(3)]. Numerous clinical studies have demonstrated that individuals with CV had improved neurological outcomes and a decreased incidence of ischemia consequences after receiving NT. Nevertheless, little is known about the exact processes by which nimodipine provides neuroprotection [(4)]. The non-invasive diagnostic modality of Transcranial Doppler (TCD) sonography has been recently employed to objectively assess the acute effects of stellate ganglion block (SGB) on the cerebrovascular system [(5)]. TCD is specifically utilized to measure changes in cerebral hemodynamics, providing a real-time, quantifiable assessment of blood flow velocities in the basal cerebral arteries. This application offers an important means to gauge the physiological impact of the SGB intervention on cerebral perfusion. This therapeutic procedure operates through the pharmacological blockade of the cervical ganglia, which are the source of the sympathetic fibers innervating the pial vessels of the brain. This blockade consequently leads to a profound reduction in the sympathetic tone exerted on the cerebral vasculature. This physiological alteration promotes a consequential and significant increase in cerebral perfusion pressure (CPP) and contributes to the improvement of CV. Despite these established hemodynamic benefits, clinical uncertainty persists regarding whether the advantages conferred by SGB are sufficient to effectively mitigate the incidence of delayed ischemic neurological deficits in individuals with established CV [(6)]. Therefore, the primary objective of the current investigation was therefore to rigorously compare the safety profile and therapeutic efficacy of two distinct preventive interventions: Ultrasound-guided SGB and NT,



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both employed as prophylactic measures against the development of CV following TBI.

## PATIENTS AND METHODS

**Study design and data collection:** This investigation employed a retrospective cohort study design to systematically analyze clinical outcomes over a defined period, spanning from January 2021 to April 2023. The patient population was specifically identified by querying the institutional critical care patient data management system for individuals who developed post-traumatic CV and subsequently received either SGB or Nimodipine therapy (NT) as part of their management protocol. Due to the inherent nature of a retrospective review, which involves the analysis of pre-existing de-identified clinical data, the requirement for obtaining individual written informed consent from the patients was formally waived by the Institutional Review Board, a standard procedure for this classification of minimal-risk research.

The baseline observations encompassed various information, including age, gender, heart rate, and hemodynamic parameters such as mean arterial pressure (MAP), random blood sugar (RBS), and blood oxygen saturation ($SpO_2$). These data were sourced from patient and medical records.

**Inclusion criteria:** Patients aged ≥ 18 years. Subjects who met the established TCD sonography criteria for the definitive diagnosis of CV. Furthermore, must be a direct sequel of TBI. This selective and stringent inclusion criterion was implemented to ensure that the investigated population was homogenous and highly relevant to the study's specific focus on CV diagnosis in the context of TBI.

**Exclusion criteria:** Individuals who had developed cerebral infarction, whether it was directly attributable to CV or secondary to other etiologies. Patients exhibiting clinical deficits stemming from alternative causes, such as acute or chronic hydrocephalus. The presence of these conditions was definitively determined through the review of Computed Tomography (CT) scans.

Percutaneous ultrasound-guided SGB is generally considered for patients who are not optimal candidates for NT because of one or more of the following:

- Patients with a previous history of cardiac disease or unstable angina.
- Hypotensive patients (systolic blood pressure lower than 100 mm Hg).
- Patients with known renal impairment and/or chronic liver disease.
- History of allergy to nimodipine

**Clinical assessment:** The emergence of novel neurological abnormalities, potentially manifesting as hemiplegia, dysphasia, or cognitive impairment, was the principal indicator of cerebral ischemia and was factored into clinical assessments. A decrease of no less than two points on the Glasgow Coma Scale (GCS) was also considered, and the symptoms were required to persist for a minimum duration of one hour. A standard CT scan of the brain was carried out to identify CV-related cerebral ischemia and rule out other possible reasons for deterioration.

**TCD criteria for cerebral vasospasm:** All measurements were performed with the same device, Toshiba Aplio 300 US (Toshiba Medical Systems, Tokyo, Japan), using a 2 MHz frequency ultrasound probe. The measurements were mainly carried out by a single qualified radiologist (ten years of experience) in performing TCD. The transtemporal window was used to perform TCD sonography of the middle cerebral arteries (MCA), and mean blood flow velocities (MBFV) were noted for each patient. Using the following formula, the MBFV was determined using peak systolic velocity (PSV) and end-diastolic velocity (EDV): [PSV + (EDV x 2)]/3 [7], see figure (1).

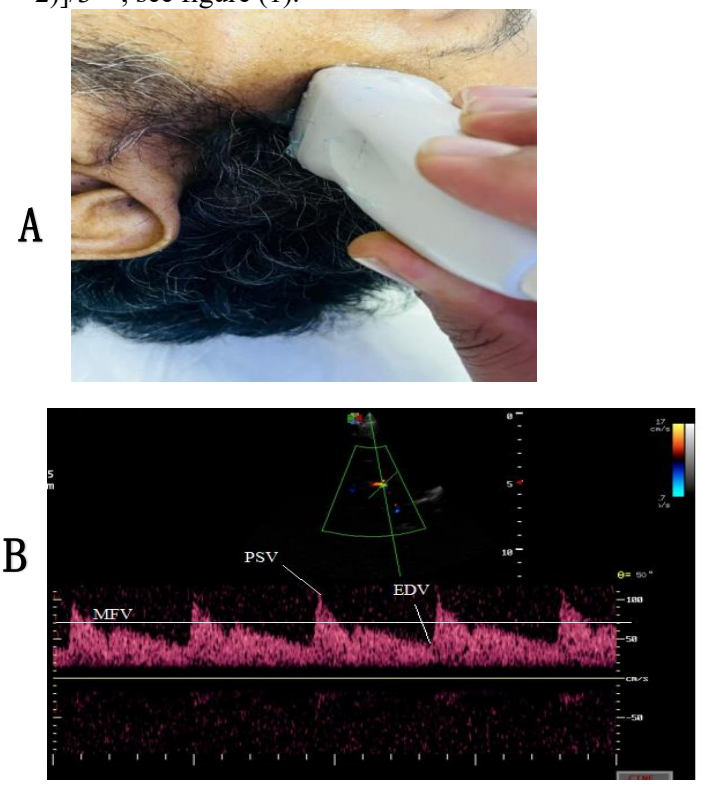
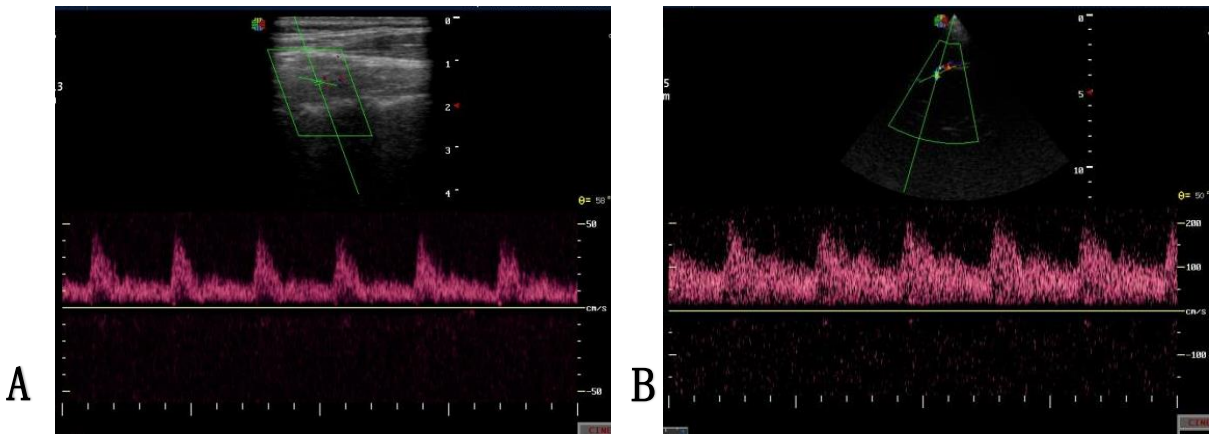



**Figure (1):** The position of the trans-temporal window demonstrates the Doppler effect of the MCA. A. The orientation of the probe index mark is directed towards the patient's anterior/front. B. A typical spectral Doppler velocity waveform from the MCA showed a sharp peak systolic velocity (PSV) and a gradual decrease in end-diastolic velocity, with a mean flow velocity (MFV) of 64.3 cm/s.

The criteria for CV diagnosis include a MBFV of 120 cm/s in the MCA or an increase of over 40 cm/s in 24 hours [8]. The Lindegaard ratio, calculated as MBFVMCA / MFVEICA, differentiates between cerebral vasospasm and hyperemic flow. A ratio between 3 and 6 indicates mild to moderate vasospasm, while a ratio over 6 indicates severe vasospasm (table 1 and figures 2 & 3). A ratio below 3, despite increased blood flow velocity, suggests hyperemia or other abnormalities [9].

**Table (1):** Transcranial Doppler criteria for vasospasm of middle cerebral artery

| Severity of vasospasm | MFV (cm/sec) | $MBFV^{MCA}$/ $MBFV^{EICA}$ ratio (Lindegaard ratio) |
|---|---|---|
| Hyperemia | < 120 | < 3 |
| Mild | 120-149 | 3-6 |
| Moderate | 150-200 | 3-6 |
| Severe | > 200 | >6 |




MCA; middle cerebral artery, MBFV; mean blood flow velocity, EICA; extracranial internal carotid artery.

**Figure (2):** TCD monitoring of MCA vasospasm. Transcranial Doppler US images and spectral waveforms obtained in the right (A) MCA and ipsilateral (B) ICA show 135 cm/s MBFV in the right (B) MCA and 23 cm/s MBFV in the right ICA. Lindegaard ratio was 5.8, indicative of mild to moderate vasospasm.

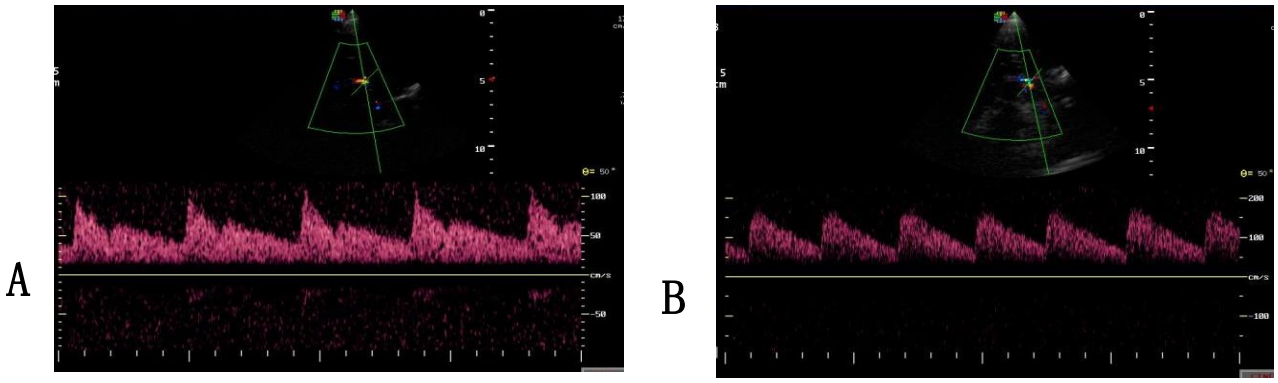



**Figure (3):** (A) Transcranial Doppler US image showed normal MBFV of 66 cm/s in the right MCA immediately after aneurysm rupture. (C) Follow-up transcranial duplex Doppler US image obtained after 10 days showed an increased MBFV of 116 cm/s in the right MCA. The MFV is normal (66 cm/s) but has also increased since baseline (66 cm/s).

**Nimodipine therapy protocol:**

The recommended dosage for adults was 60 mg (Nimotop 30 mg; Bayer AG, Leverkusen, Germany), which can be taken as two 30 mg tablets every four hours for a period of 7 consecutive days. In specific patients with poor grades, the administration of oral nimodipine was switched to intravenous nimodipine immediately after the establishment of a central line.

Nimodipine (Nimotop 10 mg; Bayer AG Leverkusen, Germany) is administered intravenously via a central venous catheter using an electronic syringe pump to ensure accurate dosing. If hemodynamic stability was preserved, the dosage began at 1 mg/h and was later increased to 2 mg/h. The intended daily dose was 48 mg. Hypotension was addressed with fluid boluses or vasopressor agents, primarily noradrenaline [10].

**The methods of SGB:** Once the patients were in the recumbent position with their vertex gently inclined to the other side, the anatomical structure was determined using a linear transducer (10 MHz, Toshiba Medical Systems, Tokyo, Japan). A 22-gauge needle was used to introduce 1% lidocaine (Hikma Pharmaceuticals, London, UK) subcutaneously to anesthetize the skin. After locating the transverse process at C6, the probe was shifted to verify that SG was situated above the longus coli muscle. As shown in figure 4 (adapted from **Goel *et al.*** [11]), Complications associated with stellate ganglion nerve block: a systematic review.

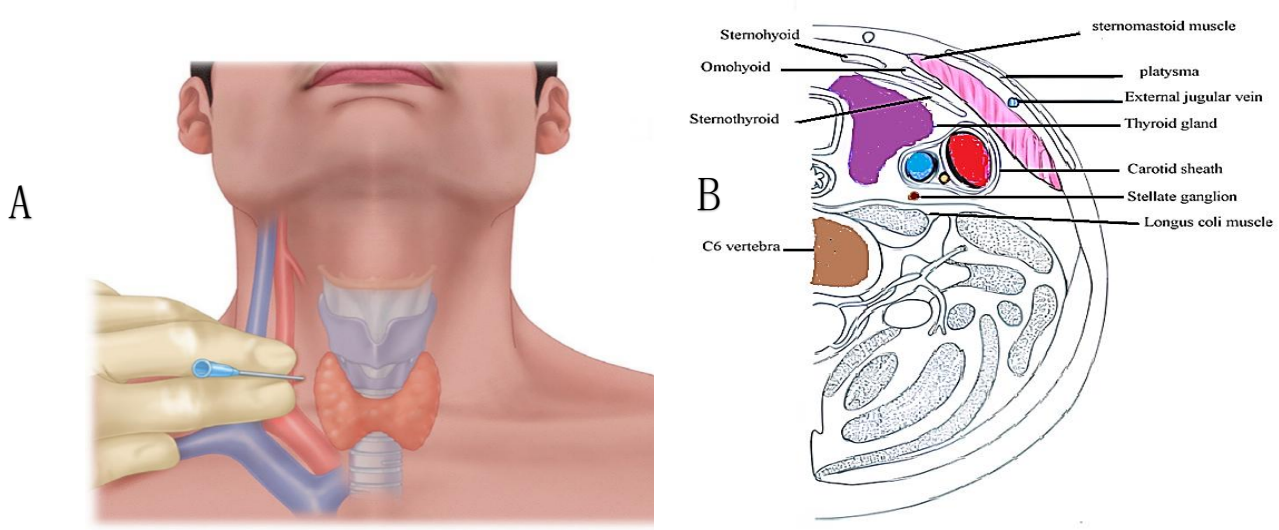



**Figure (4):** A provided a visual representation detailing the critical surface anatomical landmarks utilized for the **SGB** when performed via the classic, non-image-guided technique. The procedure commences with the identification and careful palpation of the **cricoid cartilage**, a structure that anatomically corresponds to the C6 vertebral level. Following this, the prominent carotid neurovascular bundle is meticulously displaced in a lateral direction to prevent accidental puncture. The needle tip is subsequently inserted at the skin insertion point, advancing in a plane precisely **perpendicular** to the skin surface, typically aiming for the anterior tubercle of the C6 transverse process. This methodological step is crucial for accurate anesthetic delivery to the stellate ganglion. **Figure 4 B** further complements this by illustrating the corresponding cross-sectional anatomy at the level of the **C6 vertebra**, highlighting the spatial relationship between the target and adjacent vital structures. This visualization was adapted from the established work of **Goel *et al.*** [11].

A 22-gauge, 2-inch needle was inserted into the SG under real-time ultrasound guidance. Subsequently, 0.5 ml of 0.5% bupivacaine was administered, and the needle was repositioned to ensure even distribution of the solution (Figures 5 and 6). The patient was then transported to the resuscitation area to assess any potential complications. Monitoring will continue for an additional three hours prior to discharge. In order to confirm that the procedure was successful, we checked the patients at 5-minute intervals for the onset of ipsilateral Horner's syndrome after SGB. This included contracted pupils, ptosis, enophthalmos, conjunctival hyperemia, and facial redness without perspiration

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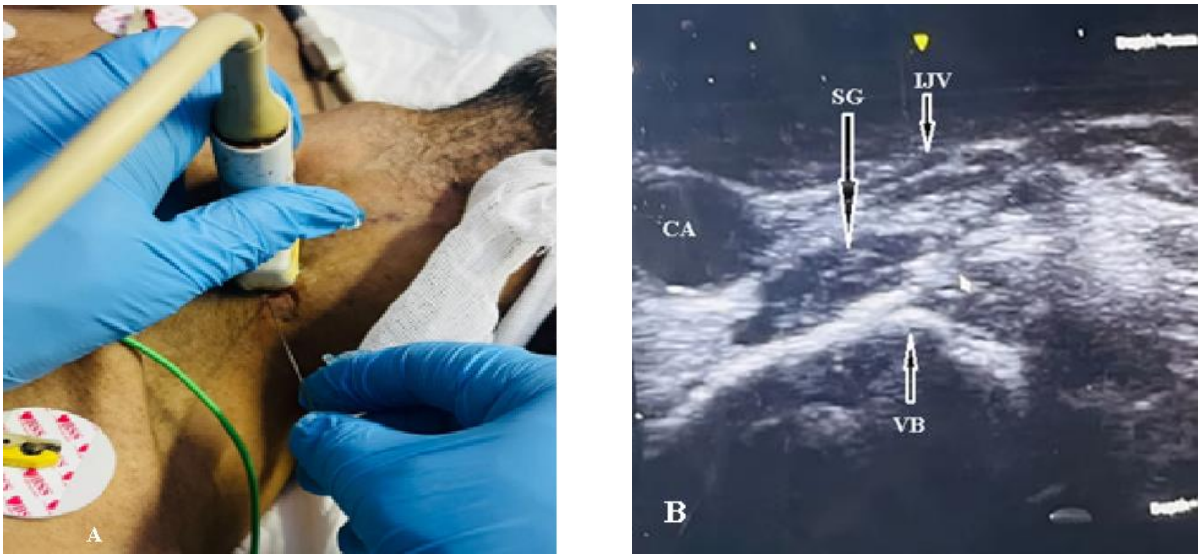


**Figure (5):** Transducer position and corresponding ultrasound image of the stellate ganglion block (SGB). **A** Performance of the SGB using transverse scanning with needle in-plane approach. **B;** Ultrasound images for the SGB. The internal jugular vein was compressed (IJV); CA carotid artery, VB C6 vertebral body.

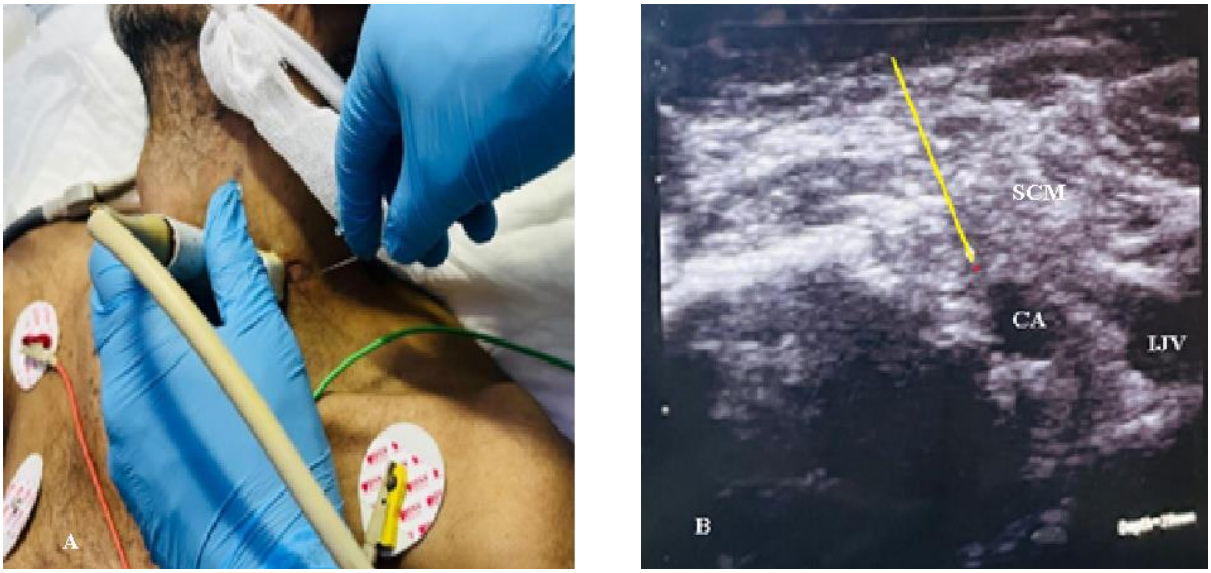


**Figure (6):** Transducer position and corresponding ultrasound image of the stellate ganglion block (SGB) at inferior C6 transverse process level. SCM, sternocleidomastoid muscle; ⋆Indicated stellate ganglion block area; Indicated simulated puncture path; IJV, internal jugular vein; CA, carotid artery.

When neurological deficits persisted despite medical treatment and the CBFV in TCD remained above 120 cm/s, SGB was performed on the affected side. In patients with elevated MBFV on both sides, SGB injections were performed on both sides.

**Post-procedural evaluation and outcomes:** Following the procedures, all patients were returned to the ICU, where safety was ensured through the continuous monitoring of vital signs, complications, and laboratory measurements until their clinical and TCD criteria were stabilized or they achieved complete recovery during the entire study period.

Drug-related complications were characterized as abrupt alterations in neurological signs that manifested during the procedure or shortly after the cessation of the medication: Mild complications are characterized by their brief duration, minimal requirement for intervention, and absence of long-term effects on the outcome. Moderate complications can be managed and alleviated through appropriate medical treatment. Severe complications are those that persist and significantly affect the outcome.

The endpoints were the onset of ipsilateral Horner's syndrome following SGB and the reductions in blood pressure (>10% of median systolic or diastolic) within 1 hour following NT in comparison with median baseline blood pressures [(12)].

**The primary efficacy outcome** was the occurrence of CV within 7 days following treatment, clinically defined as new focal or global neurological dysfunction and assessed by TCD where the average MBFV of MCA is ≤ 120 cm/s.

**The secondary outcomes** of the investigation, focusing on long-term neurological recovery and functional status, were systematically assessed at the one-month post-intervention timepoint. Two validated, globally recognized instruments were employed for this evaluation: the **Glasgow Outcome Scale (GOS)** [(13)] and the **modified Rankin Scale (mRS)** [(14)]. The GOS was utilized to categorize overall functional recovery into two distinct endpoints. A **favorable outcome** was strictly defined as a GOS score indicative of either **good recovery or moderate disability**, corresponding to grades 1 and 2 respectively. Conversely, an **unfavorable outcome** was designated by the presence of **severe disability, a persistent vegetative state or mortality** encompassing GOS grades 3, 4 and 5.

The mRS provided a more granular assessment of neurological disability and dependence with scores ranging from 0 to 6. A score of 0 indicated the complete absence of symptoms, while a score of 1 signified only minor symptoms that did not impact lifestyle. A score of 2 represented some restriction in lifestyle, with a score of 3 denoting significant restriction. A score of 4 indicated partial dependence for daily needs and a score of 5 represented full dependence. The maximum score of 6 was assigned in the event of death. This detailed stratification allowed for a comprehensive understanding of patient functional status. The detailed distribution of the outcomes based on these scales is presented in tables (2 and 3).

**Table (2):** Glasgow outcome scale

| Score | Rating | Definition |
|---|---|---|
| 5 | Good recovery | Characterized by the patient's capacity to resume a normal, independent life, notwithstanding the presence of minor residual neurological deficits. |
| 4 | Moderate disability | Indicating that the individual is disabled yet remains independent, often capable of working within a sheltered setting |
| 3 | Severe disability | The patient is conscious but functionally disabled, necessitating dependence on external assistance for essential daily activities. |
| 2 | Persistence vegetative | Minimal responsiveness |
| 1 | Death | Non survival |

This hierarchical framework provides a clear clinical methodology for categorizing the continuum of post-injury outcomes.

**Table (3):** Modified Rankin Scale

| Points | Grade of disability |
|---|---|
| 0 | No symptoms |
| 1 | No significant disability despite symptoms; able to carry out all usual duties and activities |
| 2 | Slight disability; patient retains the capacity to look after their own affairs without external assistance, although they are unable to perform all previous activities |
| 3 | Moderate disability; some degree of help but maintains the crucial ability to walk without physical assistance |
| 4 | Moderately severe disability; patient is unable to ambulate without assistance and is unable to attend to their own bodily needs without external help. |
| 5 | Severe disability; patient is completely bedridden, incontinent, and requires constant specialized nursing care and attention. |
| 6 | Death |

**Ethical approval:**

The entire research protocol was meticulously proposed and designed in full accordance with the ethical principles for medical research involving human subjects as outlined in The Declaration of Helsinki.

Furthermore, the protocol was officially registered under the identifier (NCT05182619) and formally gained the necessary ethical approval from the Ethical Committee for Research of Sohag Faculty of Medicine. This essential compliance and registration ensure the study's ethical integrity and adherence to international standards for transparent clinical investigation.

***Statistical analysis***

All data collected during the course of the investigation were subjected to rigorous statistical analysis utilizing the Statistical Package for the Social Sciences (SPSS), specifically version 17.0 (SPSS Inc., Chicago, Illinois, USA). Descriptive statistics for quantitative continuous variables were consistently expressed as the mean ± standard deviation (SD), whereas qualitative categorical variables were summarized using percentages to represent frequencies. The comparison of quantitative variables between groups necessitated an initial assessment of their distribution. Variables confirmed to be normally distributed were compared using the independent samples Student's t-test. Conversely, quantitative variables found to be non-normally distributed were analyzed using the Mann–Whitney U test, the appropriate non-parametric alternative. For the assessment of association and difference between qualitative (categorical) variables, the Chi-squared test was primarily performed. However, the Fisher's exact test was performed as a methodological alternative when the assumptions for the Chi-squared test were violated, such as in cases involving small expected cell counts. For all statistical tests performed across the data set, the level of significance was prospectively established at a P-value of $\leq 0.05$.

**RESULTS**

The initial screening cohort consisted of 120 patients admitted and treated within the Intensive Care Unit. From this population, 78 patients were definitively identified as fulfilling the established clinical and radiological criteria for **CV** following **TBI** and subsequently received either **SGB** or **NT** during the investigation period. To maintain the internal validity and ensure the homogeneity of the comparison groups, a total of 18 patients were systematically excluded from the study because they received other interventional treatments that would have introduced significant confounding variables.

The remaining eligible participants were then allocated to two distinct and balanced intervention groups: 30 patients were included in the SGB group, and an equal number of 30 patients were allocated to the NT group, resulting in a final study population of 60 subjects. The duration of the post-intervention follow-up period for all participants was established as one month, with the comprehensive participant flow diagram being visually represented in figure (7).

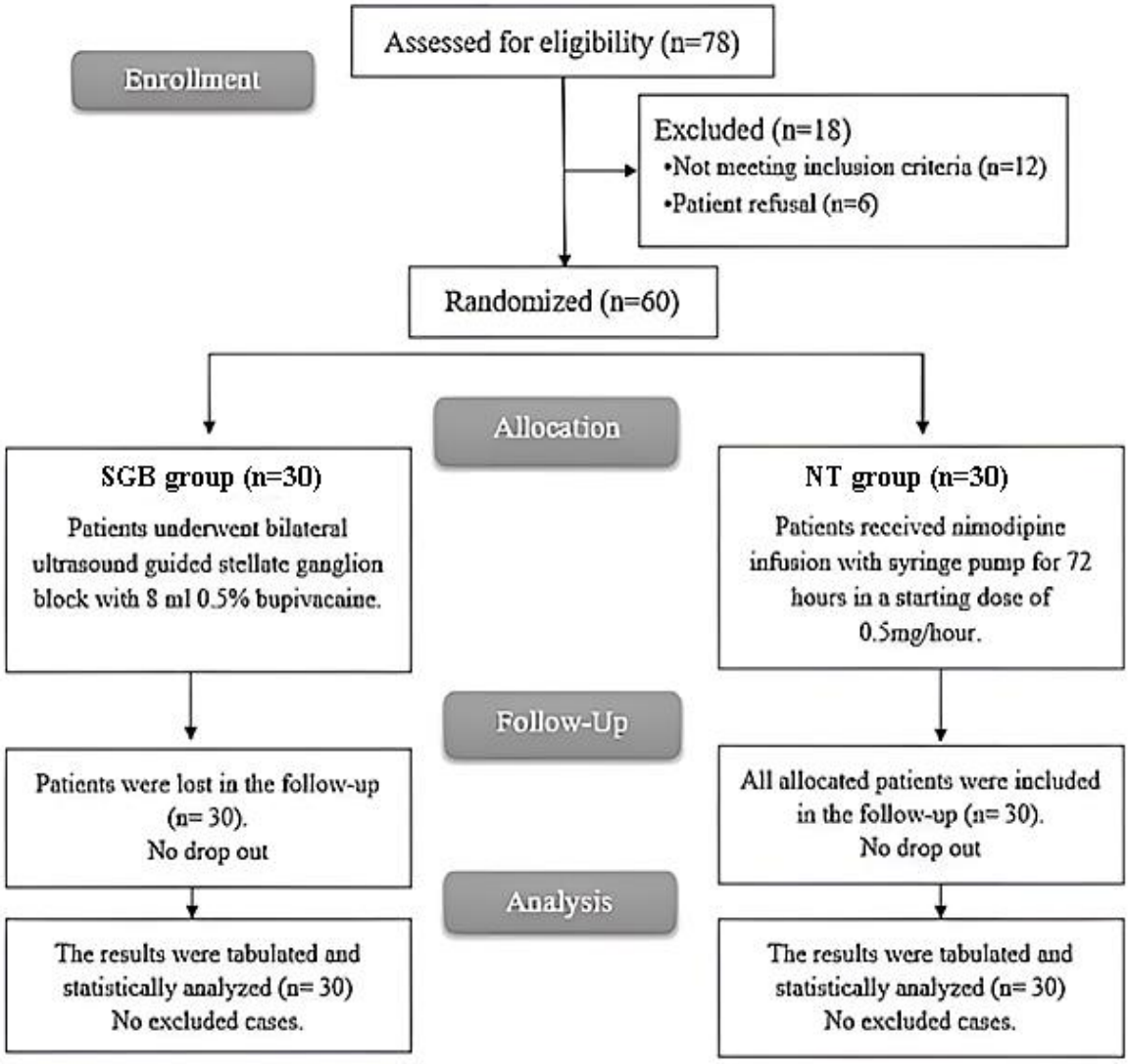


**Figure (7):** Flow chart of the process of study and extraction of patients meeting the inclusion criteria. SGB, stellate ganglion nerve block; NT, nimodipine therapy.

The baseline characteristics were illustrated in table (4). The median ages for the SGB and NT groups were 45.4 and 45 years respectively (P = 0.6). Both groups exhibited a greater proportion of males; the SGB group comprised 24 males (80%), while the NT group included 21 males (70%).

Table 4. Baseline characteristics of nimodipine therapy and SGB groups

| Variable | | Groups | | P value |
|---|---|---|---|---|
| | | Group SGB (n=30) | Group N (n=30) | |
| Age (years) | Mean ± SD<br>Range | 45.4 ± 10.61<br>26 - 70 | 46.83 ± 9.65<br>30 – 65 | 0.586 |
| Gender n (%) | Male<br>Female | 24 (80%)<br>6 (20%) | 21 (70%)<br>9 (30%) | 0.371 |
| Neurological Complaint n (%) | Headache<br>Memory problem<br>irritability | 12 (40%)<br>6 (20%)<br>12 (53.33%) | 10 (33.3%)<br>5 (16%)<br>15 (50%) | 0.3 |
| Brain pathology n (%) | SAH<br>SDH<br>Brain contusions<br>Axonal damage | 25 (83.33%)<br>1 (3.33%)<br>2 (6.67%)<br>1 (3.33%) | 26 (86.67%)<br>0 (0%)<br>0 (0%)<br>1 (3.33%) | 0.499 |
| Heart rate (beats/min) (n) | Mean ± SD<br>Range | 98.57 ± 9.91<br>85 - 118 | 94.37 ± 6.54<br>80 - 105 | **0.058*** |
| MAP (mmHg) (n) | Mean ± SD<br>Range | 90.4 ± 12.62<br>60 - 110 | 93.57 ± 6.15<br>80 - 110 | 0.222 |
| $Spo_2$ (%) (n) | Mean ± SD<br>Range | 92.73 ± 2.13<br>88 - 96 | 93.23 ± 1.65<br>90 - 97 | 0.314 |
| RBS (mg/dL) (n) | Mean ± SD<br>Range | 147.5 ± 28.55<br>120 - 210 | 132.5 ± 15.63<br>100 - 165 | **0.014*** |

Abbreviations; N (nimodipine): SGB (stellate ganglion block): MAP (mean arterial pressure): RBS (random blood sugar): GCS (Glasgow coma scale): CBVF (cerebral blood flow velocity): MCA (middle cerebral artery): $SpO_2$ (blood oxygen saturation) Subarachnoid hemorrhage (SAH): Subdural hemorrhage (SDH).

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The majority of patients demonstrated subarachnoid hemorrhage, with rates that were similar between the two groups (83.33% for SGB and 86.67% for NT). Only the SGB group presented with subdural hemorrhage (3.33%) and brain contusion (6.67%). Axonal damage was found to be similar across both groups (3.33%).

The most common neurological complaint was headache, which was noted in 12 (40%) of patients in the SGB group and in 10 (33.3%) in the NT group. Memory problem was identified in 6 (20%) of patients in the SGB group and 5 (16%) in the NT group. The percentage of patients experiencing irritability was 53.3% in the SGB group and 50% in the NT group. The overall baseline characteristics of both groups, including age, gender, neurological complaints, and brain pathology, did not show significant differences from each other. Concerning hemodynamic parameters, we observed that the heart rate and RBS in the NT group were significantly lower compared to the SGB group (p= 0.058 and 0.014, respectively).

No statistically significant difference was found in the MBFV of the MCA between the two treatment types regarding the TCD parameters recorded before treatment (P = 0.563). Following the SGB, the MBFV experienced a significant decrease of over 20% ($p < 0.001$), and a significant reduction of approximately 24.6% (p = 0.001) was observed after NT. A significant reduction in MBFV was identified in the NT group when compared to the SGB group, with a p-value of 0.047 (Table 5 and figures 8 and 9).

**Table (5):** Evaluation of the MBFV of MCA at baseline and at the conclusion of treatment across different groups

| Follow-up period | | Group SGB (n=30) | Group NT (n=30) | P value |
|---|---|---|---|---|
| **Baseline** | Mean ± SD<br>Range | 155.33±9.55<br>149.3- 160.2 | 153.83±10.4<br>147.2-158.3 | 0.563 |
| **At end of treatment** | Mean ± SD<br>Range | 123.97±14.83 | 116±11.57 | **0.047*** |
| **P value within group** | | P1<**0.001*** | P1<**0.001*** | |

Abbreviations; NT (nimodipine treatment): SGB (stellate ganglion block): MCA (middle cerebral artery)

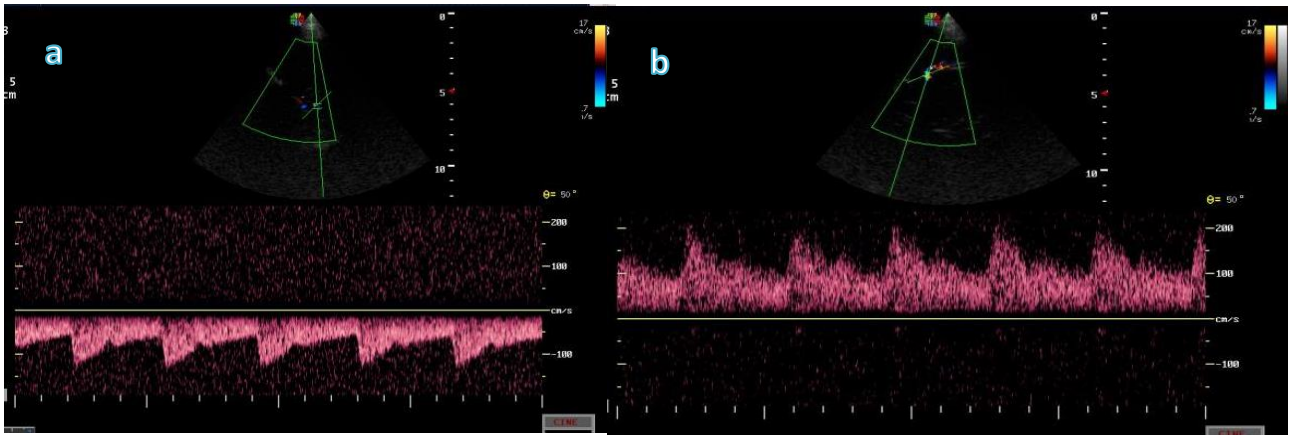


**Figure (8):** Illustrated the TCD assessment of MBFV in the MCA both prior to and subsequent to NT. a; TCD assessment of right MCA flows shows an MBFV of 133 cm/s, indicating mild vasospasm. b; The measurement of right MCA flows after treatment is 83 cm/s, confirming an improvement in vasospasm.

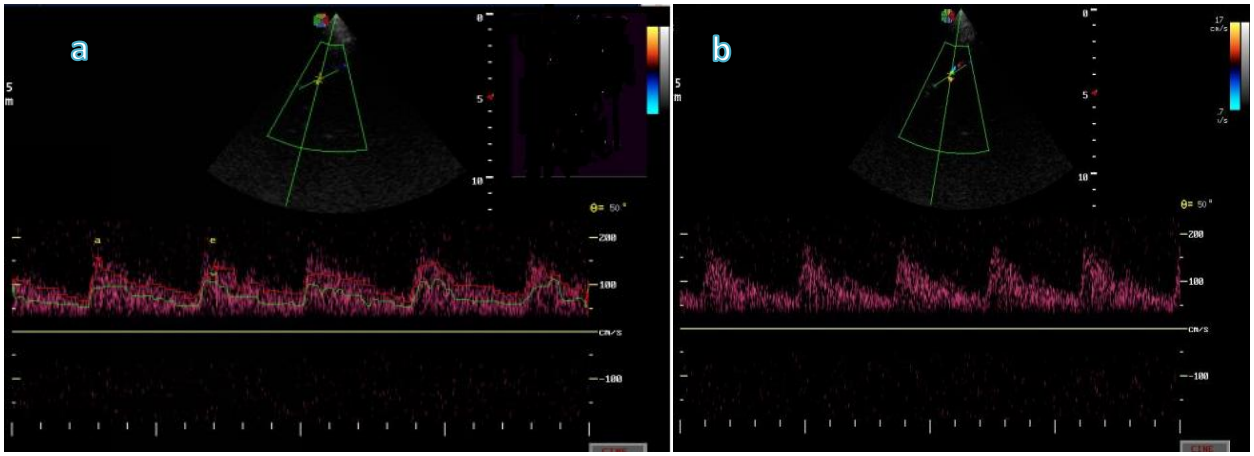


**Figure (9):** illustrated the Transcranial Doppler (TCD) assessment of mean blood flow velocity (MBFV) in the middle cerebral artery (MCA) both prior to and subsequent to stellate ganglion block (SGB). a; The TCD assessment of right MCA flows shows an MBFV of 127 cm/s. b; The measurement of right MCA flows after treatment is 101 cm/s, confirming an improvement in vasospasm.

The treatment proved to be successful for all of our patients. Throughout the duration of the study, there were no recorded deaths related to the procedures or medications. Regarding patient outcomes, the analysis of predicted complications indicated that there was no significant difference in the incidence of complications between the two regimens, yielding a p-value of 0.4. The overall complication rate linked to the SGB therapy was 6.7%. Among the complications, there were one local hematoma that resolved spontaneously after three days and another patient requiring analgesics for pain relief. Nevertheless, there were no adverse effects from the NT. Our research did not encounter any severe complications (Figure 10).

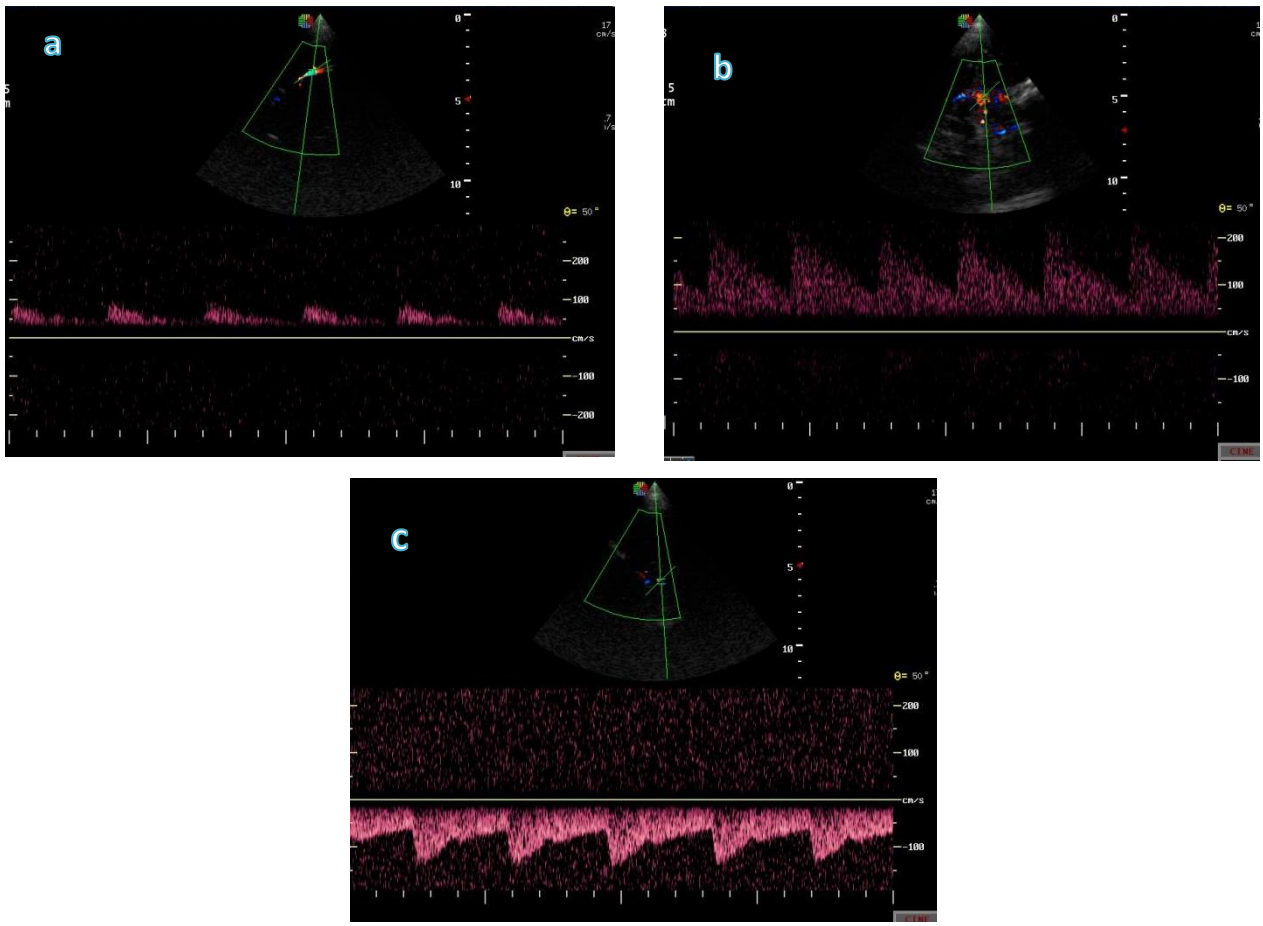


**Figure (10):** a) Spectral Doppler of MCA post trauma, demonstrated diastolic blunting secondary to raised intra-cranial pressure (pulsatility index = 3.14, PSV = 85 cm/sec). b) Further progression with change in baseline mean velocity of > 40 cm/s per 24 h demonstrating a mean MCA velocity of 125 cm/s (in-keeping with mild vasospasm). c) Following SGB interventions to reduce vasospasm, there was normalization of diastolic flow in the MCA, and resolution of high intracranial pressure (pulsatility index = 0.2, PSV = 97 cm/sec).

A favorable clinical outcome (GOS ≥ 12) was observed to be elevated at discharge in both groups, with 100% of the patients demonstrating this result. In terms of the modified Rankin score, there were highly significant differences between the groups studied, with the SGB group showing a significantly higher score compared to the NT group (P < 0.01), whereas moderate and moderately severe disabilities were observed in 9 and 3 patients, accounting for 30% and 10% respectively (Table 6).

**Table (6):** Assessment of outcome parameters following treatment between groups

| Variables | | Groups | | P value |
|---|---|---|---|---|
| | | Group SGB (n=30) | Group NT (n=30) | |
| Complication (n) | Mild<br>Moderate<br>Severe | 2 (6.7 %)<br>0<br>0 | 0<br>0<br>0 | 0.4 |
| Modified Rankin score | Median<br>Range | 3<br>2-4 | 1<br>0-2 | **<0.01*** |
| GCS<br>Median<br>Range | Baseline | 12<br>11.- 13 | 11<br>10-11 | 0.2 |
| | At end of treatment | 13<br>12-15 | 13<br>12-15 | |

Abbreviations; NT (nimodipine treatment): SGB (stellate ganglion block): GCS (Glasgow Coma Scale).

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## DISCUSSION

CV is closely linked to subarachnoid hemorrhage (SAH), which is seen in approximately 40% of TBI patients. It can also manifest in the absence of SAH, with reported occurrences ranging from 19% to 68%. However, only 3.9% to 16.6% of these patients show noticeable clinical impairments [(15)].

There are particular challenges involved in managing CV in patients with TBI, especially due to the onset of hypertension and hypervolemia that arise from the application of Triple-H therapy [(16)]. This therapy, which is fundamental for treating aneurysmal SAH, may not be appropriate for those who are suffering from CV after TBI where cerebral edema and/or increased intracranial pressure are predominant signs [(17)].

SGB and nimodipine have distinct mechanisms and evidence bases. SGB induces sympathetic denervation in the head and neck, leading to dilation of intracerebral vessels and enhanced blood flow, thereby alleviating vasospasm and preventing neurological deficits [(6)]. In contrast, nimodipine, as an L-type calcium channel blocker, inhibits calcium entry into neurons and smooth muscles, demonstrating neuroprotective effects and reducing risks of subsequent ischemia and adverse outcomes [(4)].

In order to control post-TBI CV, we decided to compare the safety and efficacy of SGB and NT in the current study. Our results showed that both ultrasound-guided SGB and NT have shown comparable effectiveness in reducing the risk of CV in patients with TBI. However, SGB did not reach statistical significance regarding the modified Rankin scores, including the degree of disability or dependence. We utilized TCD as our approach for monitoring CV. Digital subtraction angiography (DSA) is recognized as the gold standard for assessing CV, however its invasive characteristics and reliance on specialized neuroradiological facilities limit its application **[(17)]**. TCD has been used to monitor the progression of CV, following the guidelines set forth by the American Heart Association and the American Stroke Association for managing subarachnoid hemorrhage (SAH) [(18)]. This non-invasive technique allowed for dynamic monitoring and can be employed on a regular basis. It is consistent with previous research regarding TCD's sensitivity and specificity in comparison with DSA findings, demonstrating a TCD sensitivity range of 80% to 99% and a specificity range of 69% to 89% in identifying MCA spasm [(19, 20)].

The impact of SGB and NT on MBFV remained evident in our study cohort, where we observed a significant reduction in MBFV following SGB treatment, with an average decline of 20.5% from the baseline. In contrast, NT led to a significant 25.4% reduction in MCA MBFV. Our findings surpass those of **Jain *et al.* [(21)]** who reported a mean 14% decrease in MBFV following SGB treatment and are consistent with several other studies that have demonstrated the positive effects of NT on traumatic CV [(22, 23)]. We considered the substantial reduction in MBFV attributed to NT to be objective when compared to SGB.

In the pooled safety analysis, the overall incidences of adverse events were comparable among both groups.

SGB was associated with a higher incidence of minor complication (6.7%) compared to NT (0%). However, most patients who developed adverse events post-SGB were adequately managed and recovered during hospitalization, with no fatal cases reported. These adverse events are consistent with the known class effects of SGB, with similar or lower incidence rates. A prior systematic review found 260 adverse events related to SGB, with image guidance used in 134 cases (51.5%). Of these complications, 64 (24.6%) were related to ultrasound. Additionally, 178 patients (68.4%) had systemic or medication-related side effects, while 82 patients (31.5%) faced local or procedure-related issues [(24)].

Upon discharge, all patients in our cohort exhibited a favorable clinical outcome across both groups. Significant differences in modified Rankin scores were noted between the SGB group and the NT group ($P < 0.01$), with the SGB group demonstrating elevated scores. Furthermore, 30% of patients displayed moderate disabilities, while 10% showed moderately severe disabilities. Leriche and Fontaine discovered that SGB significantly lessened postoperative hemiplegia symptoms. The effectiveness of SGB in reducing CV associated with cerebral thrombosis and embolism has been validated by further research. However, little is known about how SGB affects cerebral hemodynamics, particularly when it comes to CV after subarachnoid hemorrhage [(25)].

Several studies have investigated the clinical application and efficacy of SGB in CV after SAH. Some studies have indicated a decrease in the severity of vasospasm, and improved patient outcomes [(6, 25, 26)]. A comprehensive review and meta-analysis of eight studies involving 182 patients demonstrated favorable outcomes in 52% of the patients, with the overall complication rate being 2%, and a mortality rate associated with vasospasm of 11% [(27)]. **Jain *et al.* [(21)]** evaluated the efficacy of stellate ganglion block (SGB) in patients who developed refractory cerebral vasospasm following surgical clipping of an aneurysm. This intervention resulted in an improved Glasgow Coma Score observed 30 minutes after SGB, and a reduction in neurological deficits in 11 patients. Similarly, **Samagh *et al.* [(28)]** assessed the efficacy and safety of SGB in 200 patients presenting with both clinical and angiographic evidence of vasospasm after aneurysmal clipping. Their study reported a statistically significant reduction in the Lindegaard ratio of the middle cerebral artery (MCV) and observed neurological improvement in five patients (25%).

The enhancement in cerebral blood flow linked to SGB is believed to be objective. This takes place as the intracerebral vessels, especially the pial vessels that are abundantly innervated by noradrenergic sympathetic nerve fibers, experience vasodilation of blood vessels due to the disruption of these fibers, consequently improving cerebral blood flow. This process enhances brain microcirculation, thereby providing a protective effect on the brain [(21)]. A previous study indicated that SGB may enhance patient outcomes by mitigating the inflammatory response associated with TBI, alleviating endothelial dysfunction and potentially aiding in the prevention of CV. This is likely attributable to the extensive influence of sympathetic innervation on the immune system [(29)].

Multiple imaging guidance are in use to perform SGB [(30)]. The possible advantages of ultrasound-guided for the management of CV following trauma include avoiding vital vascular and allowing for a more precise sympathetic blockade using a smaller volume of local anesthetic. Furthermore, the ability to perform the procedure at the bedside makes it even more attractive particularly while dealing with a critically-ill patients [(31)].

The current findings provided compelling evidence that ultrasound-guided Stellate Ganglion Block (SGB) holds considerable promise as a novel therapeutic methodology for the prophylactic prevention of cerebral vasospasm (CV) and its associated detrimental neurological sequelae. However, to definitively establish the superior clinical efficacy and optimal position of SGB within the neurocritical care treatment algorithm, additional extensive and well-controlled comparative investigations are imperative. Specifically, these future studies must include rigorous, head-to-head comparisons against the established pharmacological standard of care, nimodipine therapy, to fully delineate the relative merits and demerits of each intervention.

## LIMITATIONS

The most relevant limitation of our study surely was its retrospective nature, obtained from a single center, and it has to be further validated by a prospective multi-center study. The limited size of the cohort and its inability to evaluate the long-term effects of the treatment indicate that additional research is essential to investigate the long-term outcomes and to incorporate a larger group of participants in future studies.

## CONCLUSION

Both ultrasound-guided SGB and NT showed comparable effectiveness in reducing the risk of CV in patients with TBI. However, given the relatively high level of disability linked to SGB, it may be employed as an adjunctive strategy for managing CV, particularly in cases where traditional methods are inappropriate or have not yielded successful results.

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