

## Post-traumatic cerebral infarction (PTCI), an institutional experience

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### Abstract

**Aim:** In this study, we aimed to assess effect of less studied risk factors associated with the development of post-traumatic cerebral infarction (PTCI) as severe systemic injury apart from head injury, length of stay on mechanical ventilation in ICU, sepsis, coagulopathy, DVT together with common risk factors as age, GCS severity, etc. and significance of bedside tool as TCD to detect it earlier and its implication in final clinical outcome in such cases.

**Material and Methods:** 400 patients of head injury treated in our department between July 2018 to July 2019 were included in the study. 28 patients were diagnosed with PTCI. Risk factors for development of PTCI were analysed. Utility of TCD in early detection and clinical management of PTCI was assessed.

**Results:** PTCI was observed in 7 % (28/400) of patients. Among risk factor analysis age ( $p=0.029$ ), initial Glasgow coma scale score  $\leq 8$  ( $p<0.028$ ), decompressive craniotomy ( $p=0.023$ ) and prolonged ICU stay (0.031) and TCD parameters as MCA velocity  $\geq 200$ cm/s ( $p\leq 0.028$ ), PCA velocity  $\geq 70$ cm/s ( $p=0.025$ ) were significantly associated with PTCI than without it on multivariate regression analysis. Mortality was lower in PTCI cases detected earlier ( $p<0.09$ ) together with earlier clinical improvement ( $p<0.009$ ) in neurological features in comparison to cases detected late.

**Discussion:** Prolonged length of stay on a mechanical ventilator with poor GCS is a significant risk factor for the development of PTCI and early detection with a bedside tool as TCD helps in planning management better with a significant improvement in short-term mortality and clinical improvement in such cases.

### Keywords

Head injury; PTCI; Risk factors; TCD; Institutional experience

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## Introduction

Preventions and risk factors of one of the uncommon secondary brain injuries whose incidence is 1.9–40 % are not well known. Few risk factors as old age, GCS <8 are commonly reported in the literature but other risk factors as a severe systemic injury associated with head injury, length of stay in ICU, sepsis, MODS (multiple organ dysfunction), coagulopathy, DVT, etc. should also be considered especially in the cases having polytrauma because these factors may be contributory in development of PTCI. We have tried to analyze the effect of the above-mentioned risk factors on the development of PTCI and the significance of such a bedside tool as TCD to detect it and also tried to evaluate the effect of aggressive management in clinical outcomes in such cases in the present study.

## Material and Methods

We reviewed 400 head injury cases admitted to our department from July 2018 to December 2019 at our institute. We included cases of all age groups and all grades of head injury and well-demarcated area of hypodensity on NCCT head subsequent to admission, but not the cases with a documented history of the past cerebrovascular accident with brain infarct.

Detailed written informed consent was taken at the time of enrolment for this study from the patient/next of kin/guardian / to use the information and their data for teaching and clinical research purposes. Institutional ethical clearance number IEC/2020/1250.

The diagnosis of cerebral infarction was made on non-enhanced CT based on a well-demarcated region of low attenuation conforming to an arterial vascular territory. Cases were further evaluated for PTCI on magnetic resonance imaging (MRI) by visualization of a well-demarcated region, in an arterial vascular distribution, of low signal on T1-weighted images, high signal on conventional T2-weighted or fluid attenuation inversion recovery sequences, and of high signal on diffusion-weighted images, indicating diminished perfusion, associated with a corresponding decrease in signal on the apparent diffusion coefficient map. Cerebral infarctions were classified as watershed infarctions when they were located between the long penetrating cortical and striatocapsular branches (deep watershed infarction), in the supply area terminating in periventricular white matter (terminal zone infarction), or in border zones between the anterior and middle cerebral artery (MCA) or between the posterior and MCA (anterior or posterior border zone infarction, respectively). We reviewed all CT scans, MRIs, and cerebral angiograms identified as positive for infarction. A diagnosis of cerebral infarction was revised if follow-up studies indicated that the findings were actually related to evolving contusions, artifact, or were inconsistently visualized. Patient demographics (age, sex), injury specifics and clinical data (Abbreviated injury score (AISS>3)), length of stay (LOS) in the intensive care unit (ICU), and the hospital were analyzed. Cases with AISS>3 were included in it as it indicates severe injury requiring surgical management which could have aggravated secondary brain injury by causing hypovolemia, shock, etc. and there was less chance of interobserver variation in score >3. The incidence of PTCI, mortality rates, and site(s) of infarctions were ascertained. The following risk factors were

evaluated: age, abbreviated injury score (AIS), admission GCS, the performance of decompressive craniectomy, coagulopathy, abbreviated injury score (AIS), length of stay (LOS) in intensive care unit (ICU) and hospital. The difference in mortality and clinical benefit was compared in PTCI cases that were detected earlier than late detected.

PTCI detected within 10 days was labeled early and detected thereafter as late, since the cumulative frequency graph revealed 70% of cases detected within 10 days, and in the majority of the studies, these cases were detected within 3 weeks.

Improvement in clinical features, measured on GCS on follow up, was labeled as an improvement.

Continuous variables were expressed as mean±standard deviation (SD) and were compared using independent t- tests. Categorical variables were expressed as number (percentage) and were compared with the Chi-square test or the Fisher exact test, as appropriate. Multiple logistic regressions were used to identify PTCI-related factors that are helpful in recovery. Factors with a P- value <0,5 in the univariate analysis, were entered into the multivariate analysis. SPSS (Version 20.0; IBM Corp, Armonk, NY) was used for statistical analysis.

## Results

In this study, 6.5 % (26/400) of cases were detected to have PTCI after sustaining a head injury. Cases of less than 60 years were detected to be less prone to develop PTCI as compared with cases ≥60 years, and there was a significant difference in incidence in these two groups ( $p=0.029$ ) (Tables 1 and 3). Male cases were slightly more common as compared to female cases to develop it (m:f, 1:3). Severe head injury  $GCS\leq 8$  (Figure 1) was significantly more prone to develop it ( $P=0.028$ ) but cases with PTCI were also found with moderate head injury 34.61 % (9/26) (Figure 2) as well as mild head injury 7.6 % (2/26). The mass effect in the form of midline shift  $\geq 0.5\text{mm}$  was significantly associated with the development of PTCI, and cases with a herniating brain who had undergone decompressive craniotomy were detected to have more chance of developing it, as 42.5% of head injury cases (17/40) who had undergone this procedure developed PTCI ( $p\leq 0.012$ ), compared to only 3.33% of head injury cases (12/360) who were managed conservatively. Other associated systemic injuries, as measured on abbreviated injury score ( $AIS\geq 3$ ), were contributing to the development of PTCI, a positive correlation coefficient (0.126) was found with the development of PTCI but its association was not significant ( $p\leq 0.536$ ). Among other factors, the length of stay on a mechanical ventilator (MV) was significantly associated with the development of PTCI (Tables 1 and 3) that caused prolongation of stay in ICU and hospital.

The cases who developed PTCI were repeatedly screened with a bedside tool such as transcranial Doppler, which revealed significant association of detecting it with middle cerebral artery (MCA) blood flow velocity  $\geq 150\text{cm/s}$  ( $p\text{-value}\leq 0.014$ ) and posterior cerebral artery (PCA) blood flow velocity  $\geq 70\text{cm/s}$  ( $p\text{-value}\leq 0.012$ ) (Table-2). These entire features correlate with stenosis in the path of vessels compressed by the herniating brain, dissection or thromboembolic phenomenon. These findings on Doppler were labeled as positive findings on TCD

**Table 1.** Demography and clinical profile of cases

Sl.no	Factors		Cases without PTCI (n=372)	Case with PTCI (n=28)
1	Sex	male	64.51%(240/372)	57.14%(16/28)
		female	35.48%(132/372)	42.85%(12/28)
2	Age	≤60years	60.21%(224/372)	35.71%(10/28)
		≥60 years	39.78%(148/372)	64.28%(18/28)
3	Initial GCS(at admission)	<8	13.44%(50/372)	57.14%(16/28)
		8-12	53.22%(198/372)	35.71%(10/28)
		13-15	33.33%(124/372)	7.14%(2/28)
4	Midline shift	≥5mm	6.98%(26/372)	4%(16/28)
		≤5mm	93.02%(346/372)	3%(12/28)
5	Decompressive craniotomy	yes	2.15%(8/372)	57.14%(16/28)
		no	97.84%(364/372)	42.85%(12/28)
6	AIS score	abdomen≥	4.56%(17/372)	7.14%(2/28)
		chest≥	5.37%(20/372)	7.14%(2/28)
		pelvis≥	3.22%(12/372)	3.57%(1/28)
		limbs≥	4.30%(16/372)	7.14(2/28)
11	Hospital LOS in days		21.23±18.42(SD)	32.4±26.6(SD)
13	Mechanical ventilation LOS in days		9.48±12.89(SD)	20.0±17.8(SD)
14	Sepsis		5%(20/400)	21.42%(6/28)
15	coagulopathy		1.5%(6/400)	7.14%(2/28)
17	mortality		12.36%(46/372)	42.85%(12/28)
18	Follow up in months		12±9.87(SD)	14.24±16.567(SD)

**Table 2.** TCD& findings in Head injury cases with PTCI and cases without it

TCD parameters		Non PTCI cases N=372	PTCI cases N=28
MCA velocity	<120	75%(300/400)	-
	120-200	23.5%(94/400)	17.85%(5/28)
	>200	1.5%(6/400)	50%(14/28)
VA velocity>70cm/s		1.25%(5/400)	14.28%(4/28)
Basilar artery velocity >70cm/s		2.50%(10/400)	14.28%(4/28)
PCA artery velocity>70cm/s		5%(20/400)	42.85%(12/28)
Lindgard ratio	<1.5	52.50%(210/400)	32.14(9/28)
	1.5-2.5	15.5%(62/400)	14.28%(4/28)
	>2.5	7%(28/400)	53.57%(15/28)
Pulsatility index	<0.5	1.25%(5/400)	7.14%(2/28)
	0.5-1.2	76.25%(305/400)	50%(14/28)
	>1.2	22.5%(90/400)	75%(21/28)
RI	>0.75	1.25%(5/400)	7.14%(2/28)

**Table 3.** Factors affecting development of PTCI after multivariate regression analysis

Sl.no.	Factors affecting outcome	Univariate analysis	P value ≤0.05	Multivariate analysis	P value ≤0.05
		OR(95% CI)	P value	OR(95% CI)	P value
1	age≥60years	0.923(0.789-1.234)	0.037	0.876(0.784-1.216)	0.129
2	GCS≤8	0.823(0.675-1.219)	0.001	0.765(0.674-1.014)	0.028
3	Midline shift≥0.5mm	1.235(0.675-1.967)	0.044	1.265(1.118-1.423)	0.214
4	Hospital LOS in days	1.345(1.023-1.467)	0.001	1.457(1.237-1.674)	0.218
5	ICU LOS in days	3.476(3.234-3.567)	0.001	3.674(3.346-3.542)	0.031
6	Mechanical ventilation LOS in days	4.568(4.346-4.654)	0.001	4.721(4.246-5.124)	0.028
7	Decompressive craniotomy	2.428(2.213-2.623)	0.012	2.342(2.124-2.528)	0.123
8	Mca velocity≥200cm/s	3.462(3.124-3.678)	0.014	3.213(3.012-3.401)	0.028
9	Lindguard ratio≥2.5	1.248(1.121-1.312)	0.011	1.316(1.136-1.524)	0.021
10	PCA arthey velocity≥70cm/s	4.265(4.126-4.263)	0.012	4.324(4.236-4.412)	0.025
11	Pulsatility index≥0.5	3.236(3.102-3.348)	0.001	3.324(3.126-3.676)	0.123

and its sensitivity and specificity to detect PTCI were found to be 97% and 84% with area under the curve (AUC) 0.859 and p-value of 0.016, suggesting good predictive value to detect PTCI (Figure 3).

All the factors which were found to have a significant association with the development of PTCI on univariate regression analysis were subjected to multivariate regression analysis. Only age ≥60 years (p=0.029), GCS ≤8 (p=0.028), length of stay on a mechanical ventilator (p=0.028) were significantly associated with the development of PTCI (Table 3).

Clinical improvement in 72.7% of cases (8/11) detected early by a bedside screening tool (TCD) and confirmed on CT of the head, showed a significantly good recovery compared to 23.5% of cases (4/17) in which it was detected late (p=0.011). We found a significant difference in short-term mortality in 18.18% of cases (2/11) detected earlier as compared to 82.35% of cases (13/17) detected late (p=0.003). Cases with PTCI had follow-up of 12±9.87 (SD) months and cases without it had follow-up of 14.24±16.567 (SD) months (Table-1).

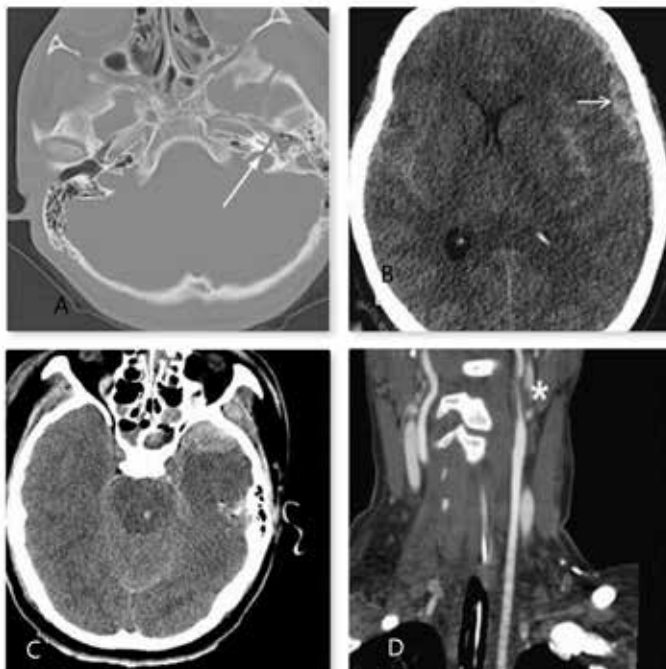
**Discussion**

Posttraumatic cerebral infarction is a known secondary entity after traumatic brain injury and often associated with poor prognosis. Its incidence in the present study is similar to other studies which reported its incidence in the range between 1.9 and 10.4% [1-3]. The reason of the observed variation may be due to different groups of case selection, as few studies have included cases of both severe and moderate head injuries, and few had only cases of severe head injury. Different studies have suggested a significantly higher incidence of severe head injuries compared to moderate head injury cases. In this study, we have noticed PTCI in all severity grades of head injury, as it was also detected in cases of moderate and mild head injury. Inclusion of moderate and mild head injuries is necessary to detect more cases for its better management.

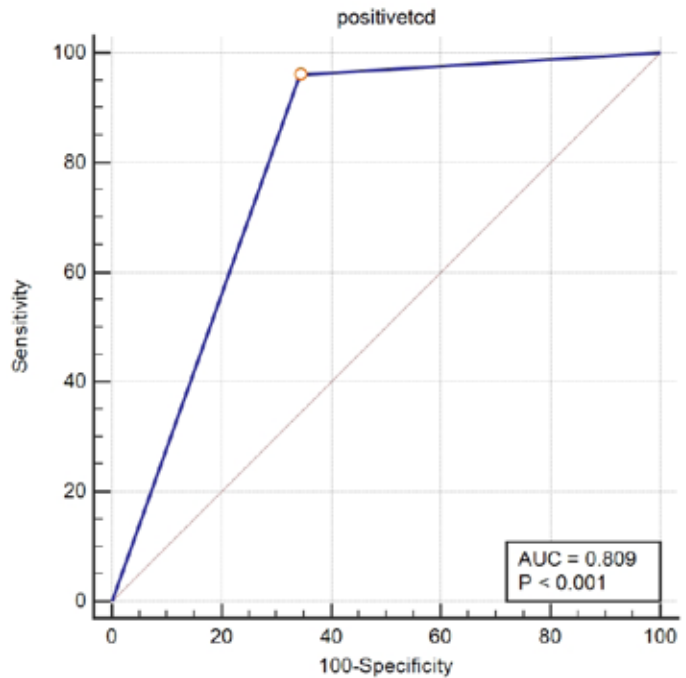
Cases having head injury were common in age group < 60 years compared to > 60 age group, incidence of PTCI was significantly higher in aged>60 years with increased mortality, as observed in this study and other studies, probably because this age group is more predisposed for the presence of atherosclerosis of vessels, brain atrophic changes leading to more shearing stress and to severe cerebral vascular injury together with other



**Figure 1.** NCCT of severe head injury case suggestive of postoperative changes with Posterior Cerebral artery territory infarct with infarct in Middle Cerebral Artery and Anterior Cerebral artery territory.



**Figure 2.** A) NCCT (non-contrast computed tomography). Head of a case with skull base fracture (left temporal bone). B) NCCT of head, suggestive of thin SDH (Subdural hematoma) with SAH (subarachnoid hemorrhage) in the left temporal-parietal region. C) NCCT of the same case revealing left temporal infarct after 3 days of admission. D) CT angiography of the neck vessel, which was suggestive of left ICA (internal carotid artery) dissection with left middle cerebral artery (MCA) infarct.



**Figure 3.** Response operative curve revealing area under curve 0.859 with p-value 0.016, sensitivity 96%, and specificity 90% and positive predictive value 0.84.

associated comorbid diseases.

PTCI cases were detected as early as 3rd day in this study and as late as 1.5 months, but the majority of cases were detected within 2 weeks, which is on the lower side compared to other studies, where the majority of the cases had been found within 3 weeks and above [2-4]. The appearance of well-demarcated area of hypodensity in particular vascular distribution suggests PTCI, which is commonly caused by mass effect with herniation of brain and compression of vessels underlying the tentorial margin, falx cerebri, and compression against bony margin. In our study, there are cases with no features of brain herniation and mass effect on CT and clinically belong to moderate and mild head injury [5-7]. These findings can be explained by pathological mechanisms such as vasospasm, vascular dissection with a thromboembolic phenomenon, etc. for which repeated examination with bedside tools like transcranial Doppler helps detecting PTCI early as in this study, compared to other studies [8-10]. Clinically, in cases with secondary deterioration in sensorium and other diminishing clinical features, we should be suspicious of PTCI together with post-traumatic hydrocephalus, post-traumatic seizures, brain edema, etc.

In cases admitted in the emergency ward, there are cases with polytrauma, in which venous embolism as DVT, PE, peripheral arterial embolism are less common entity, but its contribution to the development of PTCI should not be ignored [11, 12]. This is one of the first articles taking into consideration the severity injury score of other systems as abdomen, chest, limbs, and pelvis. On analysis of associated injuries of other systems which may be as risk factor for the development of

PTCI but its significance could not be proved as they were found in less number in the present study. Few studies suggested significantly higher ICU LOS, hospital LOS, and days on a mechanical ventilator compared to the case without the above-mentioned risk factors in thromboembolic phenomena [11-12]. Cases that require prolonged mechanical ventilator support have itself many factors associated with it which can contribute to the development of brain infarct as a thromboembolic phenomenon, impaired perfusion pressure, cerebral compliance change due to frequent change in intracompartmental pressure change of chest and cranium, etc. In this study significant association between development of PTCI and prolonged mechanical ventilation was found which creates a vicious cycle of prolonged ICU stay and hospital stay so the effort to minimize this cycle is imperative in such cases.

There is variability in detecting PTCI using CT of the head but a high index of suspicion keeps PTCI as one of the factors for no improvement or deterioration neurologically, and using bedside tools like transcranial Doppler and frequent CT of the head at regular intervals can help us detect this pathology earlier as in the present study [8-10]. Increased MCA blood flow velocity, higher PCA blood flow velocity, higher Lindegaard ratio, and pulsatile index raise the suspicion of vascular compression either due to raised intracranial pressure (ICP), brain herniation, arterial dissection, thromboembolic phenomenon, etc. TCD may play a role in monitoring the early development of cerebral vasospasm after traumatic subarachnoid hemorrhage (SAH) and cases with impending vascular occlusion due to brain herniation and the early or delayed development of increased ICP following traumatic brain injury (TBI). This is achieved by frequent monitoring with TCD which is a relatively low-cost, risk-free, bedside available, and high temporal resolution device, and suitable for the emergency setting. Significant limitations to the clinical utility of TCD in TBI include limited spatial resolution, assumptions made regarding the vessel diameter on TCD, operator dependence, and in patients who do not have an adequate acoustic temporal window for insonation [9-11]. Despite all these limitations, it is quite useful as in our study with good predictive value. Administering Calcium channel blockers in such cases with clinical suspicion of vaso occlusive phenomena had been reported in cases with head injury admitted in the emergency ward and found helpful in few studies, but cerebral protectant as nimodipine is not proven in posttraumatic infarct cases with vasospasm, but we have administered in cases with PTCI, and good clinical improvement in early detected cases were found [13-18].

In studies, the timing of decompressive craniotomy was a significant risk factor for the development of PTCI. These studies suggested that brain tissues and vessels passing through the bony defect are compressed by the dural and bony margin, leading to further cascade of congestion and edema. Larger craniectomy may influence the development of PTCI. The optimal timing for decompressive craniectomy is crucial to prevent the occurrence of PTCI or decrease its severity [18-22]. Although decompressive craniotomy is done for impending brain herniation and to prevent a cascade of cycle causing vascular compression as of PCA territory in uncal herniation

leading to PCA territory infarct, its association with PTCI has been documented in several studies [19-21]. Decompressive craniectomy is mainly done for significant mass effect with raised ICP and it has been found to decrease mortality in many studies. The size of craniotomy and timing of decompressive craniectomy is always a matter of discussion and different authors have different opinions. Even few authors have reported increased association of larger decompressive craniotomy with PTCI proposing dura and bone to produce a constrictive effect on bulging brain but the majority of studies have differing opinions as wider decompressive craniotomy lowers ICP better and can help in reducing the development of PTCI. These varying opinions may indicate towards decompression of lesion, contusion, and hematoma to minimize the constricting effect of bone and dura around the bulging brain at the site of decompression, but early decompression as in the present study, was found beneficial after being detected on TCD and impending infarct was found on CT of the head [20-22].

Factors responsible for the development of post-traumatic cerebral infarction, such as raised intracranial pressure, decreased cerebral perfusion pressure, adequate size of decompressive craniotomy, as early as possible, with adequate lesionectomy, cerebral protectant together with calcium channel blockers, have been uniformly reported in the majority of studies, to be beneficial to prevent the development of PTCI. The treatment of PTCI lies in prevention. Avoidance of hypotension and hypoxemia is crucial to prevent the occurrence of PTCI. The availability of bedside tools as transcranial Doppler is very helpful as described in the literature, to identify features of raised ICP, spasm, etc. The use of cerebral protectants together with nimodipine and timely decompressive craniotomy helped in improving clinical outcome. These findings are also motivating factors for increasing the use of DSA in cerebral injury case and judicious use of intraarterial cerebral protectants, such as Nimodipine, as done in spontaneous cerebral vasospasm causing delayed ischemic neurological deficits, the role of which can be further explored after early detection of PTCI.

#### **Conclusion**

Prolonged length of stay on a mechanical ventilator with poor GCS is a significant risk factor for the development of PTCI and early detection using bedside tool such as TCD help in planning management better with significant improvement in short-term mortality and clinical improvement in such cases.

#### **Scientific Responsibility Statement**

*The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.*

#### **Animal and human rights statement**

*All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.*

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#### **Conflict of interest**

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## References

1. Server A, Dullerud R, Haakonsen M, Nakstad PH, Johnsen UL, Magnaes B. Post-traumatic cerebral infarction. Neuroimaging findings, etiology and outcome. *Acta Radiol.* 2001; 42(3):254-60.
2. Tawil I, Stein DM, Mirvis SE, Scalea TM. Posttraumatic cerebral infarction: Incidence, outcome, and risk factors. *J Trauma.* 2008; 64(4):849-53.
3. Tian HL, Geng Z, Cui YH, Hu J, Xu T, Cao HL, et al. Risk factors for posttraumatic cerebral infarction in patients with moderate or severe head trauma. *Neurosurg Rev.* 2008; 31(4):431-7.
4. Sato M, Tanaka S, Kohama A, Fujii C. Occipital lobe infarction caused by tentorial herniation. *Neurosurgery.* 1986;18(3):300-5.
5. Rothfus WE, Goldberg AL, Tabas JH, Deeb ZL. Callosomarginal infarction secondary to transfalcial herniation. *AJNR Am J Neu-roradiol.* 1987; 8(6):1073-6.
6. Robertson SC, Lennarson P, Hasan DM, Traynelis VC. Clinical course and surgical management of massive cerebral infarction. *Neurosurgery.* 2004; 55(1):55-61; discussion 61-62.
7. Schievink WI, Mokri B, O'Fallon WM. Recurrent spontaneous cervical-artery dissection. *N Engl J Med.* 1994; 330(6):393-7.
8. Zubkov AY, Pilkington AS, Bernanke DH, Parent AD, Zhang J. Posttraumatic cerebral vasospasm: clinical and morphological pre-sentations. *J Neurotrauma;* 1999; 16(9):763-70.
9. Fatima N, Shuaib A, Chughtai TS, Ayyad A, Saqqur M. The role of Transcranial Doppler in Traumatic Brain Injury: A Systemic Review and Meta-Analysis. *Asian J Neurosurg;* 2019; 14(3):626-33.
10. Fisher CM, Kistler JP, Davis JM. Relation of cerebral vasospasm to subarachnoid hemorrhage visualized by computerized tomographic scanning. *Neurosurgery.* 1980; 6(1):1-9.
11. Lichte P, Kobbe P, Almahmoud K, Pfeijfer R, Andruszkow H, Hildebrand F, et al. Trauma Register DGU. Post-traumatic thrombo-embolic complications in polytrauma patients. *Int Orthop.* 2015;39(5):947-54.
12. Franchini M, Mannucci PM. Association between venous and arterial thrombosis: clinical implications. *Eur J Intern Med.* 2012; 23(4): 333-7.
13. Martin NA, Doberstein C, Zane C, Caron MJ, Thomas K, Becker DP. Posttraumatic cerebral arterial spasm: transcranial Doppler ultrasound, cerebral blood flow, and angiographic findings. *J Neurosurg.* 1992;77(4):575-83.
14. Oertel M, Boscardin WJ, Obrist WD, Glenn TC, McArthur DL, Gravori T, et al. Posttraumatic vasospasm: the epidemiology, severity, and time course of an underestimated phenomenon: a prospective study performed in 299 patients. *J Neurosurg.* 2005;103(5):812-24.
15. Sahuquillo J, Robles A, Poca A, Ballabriga A, Mercadal J, Secades JJ. A controlled, double-blind, randomized pilot clinical trial of nicardipine as compared with a placebo in patients with moderate or severe head injury. *Rev Neurol.* 2000;30:401-8.
16. Compton JS, Lee T, Jones NR, Waddell G, Teddy PJ. A double blind placebo controlled trial of the calcium entry blocking drug, nicardipine, in the treatment of vasospasm following severe head injury. *Br J Neurosurg.* 1990;4:9-15.
17. The European Study Group on Nimodipine in Severe Head Injury. A multicenter trial of the efficacy of nimodipine on outcome after severe head injury. *J Neurosurg.* 1994; 80(5):797-804.
18. Eberle BM, Schnuriger B, Inaba K, Gruen JP, Demetriades D, Belzberg H. Decompressive craniectomy: Surgical control of traumatic intracranial hypertension may improve outcome. *Injury.* 2010;41(9):894-8.
19. Su TM, Lan CM, Lee TH, Shih FY, Hsu SW, Lu CH. Posttraumatic Cerebral Infarction After Decompressive Craniectomy for Traumatic Brain Injury: Incidence, Risk Factors and Outcome; *Turk Neurosurg.* 2017; DOI: 10.5137/1019-5149.JTN.20761-17.1.
20. Tian HL, Geng Z, Cui YH, Hu J, Xu T, Cao H, et al. Risk factors for posttraumatic cerebral infarction in patients with moderate or severe head trauma. *Neurosurg Rev.* 2008; 31(4):431- 7.
21. Ham HY, Lee JK, Jang JW, Seo BR, Kim JH, Choi JW. Post- traumatic cerebral infarction: Outcome after decompressive hemicraniectomy for the treatment of traumatic brain injury. *J Korean Neurosurg Soc.* 2011; 50(4):370-6.
22. Honeybul S. Complications of decompressive craniectomy for head injury. *J Clin Neurosci.* 2010; 17(4):430-5.

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