



Acute traumatic brain injury induced endotheliopathy and its impact on clinical outcome

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Abstract

Background: Injury & hypoperfusion induced stimulation of the neurohumoral system leads to endothelial cell activation, glycocalyx degradation, upregulation of procoagulant/ profibrinolytic factors causing acute coagulopathy. TBI associated coagulopathy (TBI-AC) is linked with progression of haemorrhagic lesions, with an overall prevalence of 32.7% and correlates with high risks of mortality. **Objective:** We designed a prospective study to assess difference in markers of endotheliopathy [glycocalyx shedding (Syndecan-1) and endothelial disruption (Thrombomodulin)] among severe isolated TBI patients with/without early coagulopathy and determine its effect on 48hours and 30-day mortality. **Materials/methods:** We screened for and recruited iSTBI patients (GCS ≤ 8) at emergency department. Sampling was done, ≤ 12 hrs. of injury after informed consent, prior to transfusion. Patients with H/O anticoagulants, liver disease, hypotension, extracranial injuries were excluded. TBI coagulopathy was defined based on conventional coagulation tests as $INR \geq 1.27$ &/or $PT \geq$

McArthur D., Boscardin W.J., Glenn T.C., Lee J.H., Tooraj Gravori M.P.H., Obukhov D., McBride D.Q., Martin N.A. Progressive hemorrhage after head trauma: Predictors and consequences of the evolving injury. *J. Neurosurg.* 2002;96:109–116. doi: 10.3171/jns.2002.96.1.0109. 16.7sec &/or aPTT ≥ 28.8 sec. 20 healthy controls were included. Plasma levels of Syd-1 & TM were estimated by ELISA. 48 hours and 30 days mortality were defined outcomes. Results: 120 cases met the inclusion criteria, aged 35.7 ± 12.12 years, 96% males. TBI-AC was had an incident of 41.6% (50). TBI-AC occurred independently of age, gender, GCS. Conclusion: TBI associated coagulopathy was prevalent in 41.6% (50) and was an independent predictor of mortality (OR = 4.73; 95% CI 1.68 to 13.3). Significant elevations in plasma syndecan associated with TBI-AC, whilst thrombomodulin levels did not vary significantly. “High” syndecan-1 levels (≥ 30.50 ng/mL) were significantly associated with elevated risk of early mortality. We recommend clinically, timed interventions aimed at protecting and repairing the endothelium, to attenuate traumatic endotheliopathy and potentially improve outcomes in isolated TBI patients.

Biography

Arulsevi done her MBBS from Thanjavur Medical college, Tamilnadu, India and MD (Pathology) in Kasturba Medical College, Manipal, Karnataka, India. She is currently working as professor in Department of Lab Medicine, JPNA Trauma Centre of All India Institute of Medical Sciences (AIIMS), New Delhi, India. she joined this institute since 2005 and have 95 publications in various international and national indexed journals. She has completed and is currently holding various projects funded by ICMR, DST, DBT, DHR and AIIMS institute. She has been awarded the “Indo US academician and best researcher award” in 2012 and won 12 awards from various papers and posters in various conferences. Been reviewer to various journals and associate editor for Journal of Laboratory Physicians.

Publications

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