Traumatic brain injury case series

2023

Shafiei et al. performed a retrospective cross-sectional study using the files of patients with mild traumatic brain injury who had cerebral contusions from 21 March 2021 to 20 March 2022. The severity of brain injury was determined using the Glasgow Coma Score. Furthermore, we used a cut-off value of a 30% increase in contusion size in the secondary CT scans (up to 72 hours) compared to the first one to define the significant progression of the contusions. For the patients with multiple contusions, we measured the biggest contusion.

705 patients with traumatic brain injury were found, the severity of the injury was mild in 498 of them, and 218 had cerebral contusions. 131 (60.1%) patients were injured in vehicle accidents. 111 (50.9%) had significant contusion progression. Most patients were conservatively managed, but 21 out of them (10%) required delayed surgical intervention.

They found that the presence of subdural hematoma, subarachnoid hemorrhage, and epidural hematoma were predictors of radiological contusion progression, and the patients with subdural hematoma and epidural hematoma were more likely to undergo surgery. In addition to providing prognostic information, predicting risk factors for the progression of the contusions is crucial for identifying patients who might benefit from surgical and critical care therapies [1)].

Palabiyik et al. retrospectively analyzed intensive care unit patients with traumatic brain injury. They recorded patients' ages; genders; diagnoses; Glasgow Coma Scale scores; length of intensive care unit stay (in days); mean platelet volume, platelet distribution width, platelet count-to-total lymphocyte count ratio, and red cell distribution width values upon hospital admission; and health on the 7th and 30th days of their stays.

They analyzed data from 110 patients. Of these, 84 (76.4%) were male and 26 (23.6%) were female. On the 7- and 30-day mortality evaluations, compared to the living patients, the deceased patients had a significantly higher median age and a significantly lower median Glasgow Coma Scale. Thus, increased age and lower Glasgow Coma Scale scores were associated with increased 7- and 30-day mortality rates. mean platelet volume and platelet distribution width values were similar in living and deceased patients. platelet count-to-total lymphocyte count ratio values were lower in deceased patients, but this difference was not statistically significant. Within 30 days after traumatic brain injury, deceased patients' red cell distribution width values were significantly elevated in deceased patients compared to those of living patients.

Mean platelet volume, platelet distribution width, and platelet count-to-total lymphocyte count ratio values were not associated with 7- and 30-day mortality, whereas only elevated red cell distribution width was associated with 30-day mortality [2)].

Bei et al. analyzed the clinical data of 244 TBI patients who underwent craniotomy or decompressive craniectomy. The generalized additive mixed model (GAMM) was used to analyze the effects of propofol and sevoflurane on the Glasgow Coma Scale (GCS) on postoperative days 1, 3, and 7.

Multivariate regression analysis was used to analyze the effects of the two anesthetics on the Glasgow Outcome Scale (GOS) at discharge.

It showed no significant difference in GCS at admission between the propofol and the sevoflurane groups among craniotomy patients (β = 0.75, 95%CI: -0.55 to 2.05, P = 0.260). However, the elevation in GCS from baseline was 1.73 points (95%CI: -2.81 to -0.66, P = 0.002) less in the sevoflurane group than in the propofol group on postoperative day 1, 2.03 points (95%CI: -3.14 to -0.91, P < 0.001) less on day 3, and 1.31 points (95%CI: -2.43 to -0.19, P = 0.022) less on day 7. The risk of unfavorable GOS (GOS 1, 2, and 3) at discharge was higher in the sevoflurane group (OR = 4.93, 95%CI: 1.05 to 23.03, P = 0.043). No significant difference was observed among decompressive craniectomy patients in GCS and GOS.

Compared to propofol, sevoflurane was associated with worse neurological recovery during the hospital stay in TBI patients undergoing craniotomy. This difference was not detected in TBI patients undergoing decompressive craniectomy [3)]

2022

One-hundred sixty-nine adult Traumatic brain injury patients, treated at the neurointensive care (NIC) unit, at Uppsala University Hospital, 2008-2020, with ICP and cerebral microdialysis (MD) monitoring, were included. Of the 169 TBI patients, 131 (78%) were male and 38 (22%) female. Male patients were more often injured by motor vehicle accidents and less often by bicycle accidents ($p < 0.05$). There was otherwise no difference in age, neurological status at admission, and types of intracranial hemorrhages between the sexes. The percent of monitoring time with ICP above 20 mmHg and CPP below 60 mmHg were similar for both sexes. Males exhibited more disturbed cerebral pressure autoregulation (PRx55-15 (mean ± SD); 0.28 ± 0.18 vs. 0.17 ± 0.23, $p < 0.05$) day 1, worse cerebral energy metabolism (MD-lactate-/pyruvate-ratio (median (IQR)); 25 (19-31) vs. 20 (17-25), $p < 0.01$) and mitochondrial dysfunction (higher burden of MD-LPR > 25 and MD-pyruvate > 120 µM (median (IQR)); 13 (0-58) % vs. 3 (0-17) %, $p < 0.05$) day 2 to 5, increased excitotoxicity (MD-glutamate median (IQR); 9 (4-32) µM vs. 5 (3-10) µM, $p < 0.05$) day 2 to 5, and higher biomarker levels of cellular injury (MD-glycerol median (IQR); 103 (66-193) µM vs. 68 (49-106) µM, $p < 0.01$) most pronounced day 6 to 10. There was no difference in mortality or the degree of favorable outcome between the sexes. Altogether, females exhibited more favorable cerebral physiology post-TBI, particularly better mitochondrial function, and reduced excitotoxicity, but this did not translate into better clinical outcomes compared to males. Future studies need to further explore potential sex differences in secondary injury mechanisms in TBI [4)].

2021

A total of 46 TBI participants across all levels of injury and 23 healthy controls were enrolled in a case-control study. Wechsler Memory Scale-Chinese Revision (WMS-CR) picture, recognition, associative learning, comprehension memory, and digit span were administered to evaluate several categories of memory capacity. The Hospital Anxiety and Depression Scale (HADS) was employed to evaluate the anxiety and depressive symptoms. Stepwise multiple linear regressions were conducted.

Compared to healthy controls, the participants with TBI reported more anxiety and depressive symptoms. In the meanwhile, they performed more poorly on memory tests, showing 1.84 SDs, 1.07

SDs, and 0.68 SDs below healthy participants on visuospatial memory, working memory, and verbal memory, respectively. A variety of variables, including HADS depression, HADS anxiety, age, GCS, and education were associated with posttraumatic memory function in the bivariate models. The stepwise multiple linear regressions demonstrated a negative association between HADS depression and posttraumatic memory function, especially performance on visuospatial and verbal memory and a positive association between education and posttraumatic memory function.

More depressive symptoms rather than anxiety symptoms and less years of education are significant predictors for posttraumatic memory dysfunction [5].

Nineteen patients with diffuse, blunt, non-severe TBI (mean age 32.7 ± 11.4 years; 4 women and 15 men; Glasgow Coma Scale before transcranial alternating current stimulation (tACS) 14.1 ± 0.5) were treated by 10 Hz in-phase tACS applied for 30 minutes to the left and right lateral prefrontal cortex at 21 days after TBI. Regional cerebral oxygen saturation (SctO2) in the frontal lobes was measured simultaneously by the cerebral oximeter. Significance was preset to $P < 0.05$. The SctO2 values before tACS were not different between hemispheres ~65%. After 15 minutes of tACS, a significant ($p < 0.05$) decrease in regional SctO2 was observed with the minimum at the eighth minute of 53.4 ± 3.2% and 53.4 ± 3.2% in the left and right hemispheres, respectively. At the end of the stimulation (30 minutes), the hemispheric differences in cerebral oxygen saturation became statistically insignificant again ($p > 0.05$). Therefore, tACS causes a significant decrease in SctO2, probably, due to neuronal activation. This data indicate that tACS may need to be supplemented with oxygen therapy. Further research is required [6].

2020

A sample of 109 people with a new TBI was recruited from three hospitals in Mexico City, Mexico, and in Cali and Neiva, Colombia. Participants completed measures of cognitive dysfunction and social disadaptation before hospital discharge and measures of depression at baseline, 2 months, and 4 months.

Results suggested that depression scores were found to decrease over time in a quadratic (or U-shaped) fashion, and more significant cognitive dysfunction at hospital discharge was associated with higher longitudinal depression trajectories. Social disadaptation did not exert a unique effect on depression trajectories after controlling for cognitive dysfunction. Depression trajectories changed differentially over time as a function of baseline cognitive dysfunction, such that for those with high cognitive impairment, depression scores started high and then dropped to a moderated range and plateaued, but for individuals with low cognitive dysfunction, depression scores started lower and decreased linearly but moderately.

The results suggest a strong need for neuropsychological assessments and evidence-based cognitive rehabilitation strategies to be implemented immediately after TBI in Latin America, which could exert salubrious effects on depression trajectories over time [7].

2019

A study of Eom included 540 men and 364 women. The age distributions in the male and female

groups were statistically significantly different. The most common cause of trauma was a fall and diagnosis was acute subdural hematoma. The incidence was the highest in men aged 80-84 years and in women aged 75-79 years. The most common time of arrival to hospital after TBI was within 1 hour and 119 rescue team provided first aid earliest to patients with TBI. The mortality rate stratified according to the cause of trauma was significantly different, with mortality rates of 3.77% in fall and 11.65% in traffic accident. The mortality rates according the severity of brain injury, Glasgow Coma Scale score, and treatment were statistically significant.

This study is the first to focus on elderly patients with TBI in Korea and particularly investigate mortality and characteristics related to TBI-related death based on data from the Korean Neuro-Trauma Data Bank System (KNTDBS). Although the study has some limitations, the results may be used to obtain useful information to study targeted prevention and more effective treatment options for older TBI patients and establish novel treatment guidelines and health polish for the geriatric population [8].

From a retrospective cohort of 97 TBI patients with arterial blood pressure (ABP), ICP and bilateral TCD monitoring, 24 presented unilateral lesion and midline shift confirmed by computer tomography. nICP and non-invasive cerebral perfusion pressure (nCPP) on the left and right brain hemispheres were retrospectively calculated using a mathematical model associating TCD-derived cerebral blood flow velocity and ABP.

The nCPP difference was correlated with midline shift (R=-0.34, p<.01) showing a tendency to record higher CPP at the side of expansion. Accordingly, nICP at the side of expansion was significantly lower in comparison to the compressed side (18.86 [±5.71] mmHg (mean ± standard deviation) versus 20.30 [±6.78] mmHg for expansion and compressed sides, respectively). Subsequently, nCPP was greater on the side of brain expansion (79.48±7.84, 78.03±8.93 mmHg [p<.01], for expansion and compressed sides, respectively).

TCD-based interhemispheric nCPP difference showed significant correlation with midline shift. Cerebral perfusion pressure was greater on the side of brain expansion, acting as the driving force to shift brain structures. [9].

2017

Cicuendez et al. retrospectively analyzed 264 TBI patients to whom a MR had been performed in the first 60 days after trauma. All clinical variables related to prognosis were registered, as well as the data from the initial computed tomography. The MR imaging protocol consisted of a 3-plane localizer sequence T1-weighted and T2-weighted fast spin-echo, FLAIR and gradient-echo images (GRET2*). Traumatic axonal injury (TAI) lesions were classified according to Gentry and Firsching classifications. They calculated weighted kappa coefficients and the area under the ROC curve for each MR sequence. A multivariable analyses was performed to correlate MR findings in each sequence with the final outcome of the patients.

TAI lesions were adequately visualized on T2, FLAIR and GRET2* sequences in more than 80% of the studies. Subcortical TAI lesions were well on FLAIR and GRET2* sequences visualized hemorrhagic TAI lesions. We saw that these MR sequences had a high inter-rater agreement for TAI diagnosis (0.8). T2 sequence presented the highest value on ROC curve in Gentry (0.68, 95%CI: 0.61-0.76, p<0.001, Nagerlkerke-R2 0.26) and Firsching classifications (0.64, 95%CI 0.57-0.72, p<0.001, Nagerlkerke-R2

0.19), followed by FLAIR and GRET2* sequences. Both classifications determined by each of these sequences were associated with poor outcome after performing a multivariable analyses adjusted for prognostic factors (p<0.02).

They recommend to perform conventional MR study in subacute phase including T2, FLAIR and GRET2* sequences for visualize TAI lesions. These MR findings added prognostic information in TBI patients [10].

2016

634 consecutive neurosurgical trauma patients, who presented with mild-to-severe traumatic brain injury (TBI) from January 2013 to April 2014 at a tertiary care center in rural Nepal. All pertinent medical records (including all available imaging studies) were reviewed by the neurosurgical consultant and the radiologist on call. Patients' worst CT image scores and their outcome at 30 days were assessed and recorded. They then assessed their independent performance in predicting the mortality and also tried to seek the individual variables that had significant interplay for determining the same.

Both imaging score Marshall computed tomography classification and Rotterdam CT score can be used to reliably predict mortality in patients with acute TBI with high prognostic accuracy. Other specific CT characteristics that can be used to predict early mortality are traumatic subarachnoid hemorrhage, midline shift, and status of the peri-mesencephalic cisterns.

They demonstrated in this cohort that though the Marshall computed tomography classification has the high predictive power to determine the mortality, better discrimination could be sought through the application of the Rotterdam CT score that encompasses various individual CT parameters. They thereby recommend the use of such comprehensive prognostic model so as to augment the predictive power for properly dichotomizing the prognosis of the patients with TBI. In the future, it will therefore be important to develop prognostic models that are applicable for the majority of patients in the world they live in, and not just a privileged few who can use resources not necessarily representative of their societal environment [11].

Of 66 patients with head injuries who had talked at some time after injury, 25% did not have intracranial hematoma at necropsy. Most of these had intracranial hypertension, and the commonest finding was local swelling related to contusions. Almost half of the non-haematoma cases had ischaemic or hypoxic brain damage, usually without contusions; 3 were children who had had status epilepticus. Fatality without raised I.C.P. was most often due to meningitis. In deteriorating patients without haematoma mortality and morbidity might be reduced by more diagnosis and treatment, particularly of raised ICP [12].

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