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Original Article

Extracranial pressure (ECP) monitoring in severe traumatic brain injury (TBI): A prospective study validating intra-abdominal pressure (IAP) measurement for predicting intracranial pressure (ICP)

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ABSTRACT

Background: Intracranial pressure (ICP)--guided therapy is the standard of care in the management of severe traumatic brain injury (TBI). Ideal ICP monitoring technique is not yet available, based on its risks associated with bleeding, infection, or its unavailability at major centers. Authors propose that ICP can be gauged based on measuring pressures of other anatomical cavities, for example, the abdominal cavity. Researchers explored the possibility of monitoring intra-abdominal pressure (IAP) to predict ICP in severe TBI patients.

Methods: We measured ICP and IAP in severe TBI patients. ICP was measured using standard right frontal external ventricular drain (EVD) insertion and connecting it to the transducer. IAP was measured using a well-established technique of vesical pressure measurement through a manometer.

Results: A total of 28 patients ($n = 28$) with an age range of 18–65 years (mean of 32.36 years ± 13.52 years [Standard deviation]) and the median age of 28.00 years with an interquartile range (21.00–42.00 years) were recruited in this prospective study. About 57.1% ($n = 16$) of these patients were in the age range of 18–30 years. About 92.9% ($n = 26$) of the patients were male. The most common mode of injury (78.6%) was road traffic accidents ($n = 22$) and the mean Glasgow Coma Scale at presentation was 4.04 (range 3–9). The mean ICP measured at the presentation of this patient cohort was 20.04 mmHg. This mean ICP (mmHg) decreased from a maximum of 20.04 at the 0 h' time point (at the time of insertion of EVD) to a minimum of 12.09 at the 96 hr time point. This change in mean ICP (from 0 h to 96 h) was found to be statistically significant (Friedman Test: $\chi^2 = 87.6$, $P \leq 0.001$). The mean IAP (cmH2O) decreased from a maximum of 16.71 at the 0 h' time point to a minimum of 9.68 at the 96 h' time point. This change was statistically significant (Friedman Test: $\chi^2 = 71.8$, $P \leq 0.001$). The per unit percentage change in IAP on per unit percentage change in ICP we observed was correlated to each other. The correlation coefficient between these variables varied from 0.71 to 0.89 at different time frames. It followed a trend in a directly proportional manner and was found to be statistically significant ($P < 0.001$) in each time frame of the study. The rise in one parameter followed the rise in another parameter and vice versa.

Conclusion: In this study, we established that the ICP of severe TBI patients correlates well with IAP at presentation. This correlation was strong and constant, irrespective of the timeframe during the treatment and monitoring. This study also established that draining cerebrospinal fluid to decrease ICP in severe TBI patients is reflected in IAP. The study validates that IAP is a strong proxy of ICP in severe TBI patients.

Keywords: Extracranial pressure, Intra-abdominal pressure, Intracranial pressure, Monitoring, Severe traumatic brain injury

INTRODUCTION

There are invasive and noninvasive cerebral monitoring techniques to prevent/halt secondary brain damage. Intracranial pressure (ICP) monitoring is the most commonly used invasive procedure to prevent, early detect, and treat secondary insults to the brain. ICP-directed therapy shows at least more salvageability in traumatic brain injury (TBI) patients. Further, ICP monitoring became an established practice following recommendations for its use in Brain Trauma Foundation (BTF) guidelines,[7] first published in 1995 and subsequently modified, latest in 2016.

The fundamental question of whether we can gauze ICP without inserting anything inside the brain was investigated in our study. To address this, we postulated that elevated ICP in TBI patients is also transmitted to other anatomical compartments, such as the abdominal cavity. We extrapolated this fact to measure and correlate extracranial pressure (ECP), that is, intra-abdominal pressure (IAP) with ICP in severe TBI patients. Suppose ICP can be replaced with ECP (e.g., IAP). In that case, the benefits of ICP-guided therapy can be given to severe TBI patients without subjecting these patients to more invasive, expensive, and technically demanding ICP monitors.

MATERIALS AND METHODS

All patients who presented to the Neurotrauma Centre at All India Institute of Medical Sciences (AIIMS), Rishikesh, with a diagnosis of severe TBI underwent (in addition to the standard of care of severe TBI, including ICP monitoring) IAP monitoring by the international standard. In this prospective study, patients were recruited from July 2021 to February 2023. Age, gender, mode of injury, and presence/absence of extracranial injuries, such as abdominal-thoracic trauma, pelvic injuries, or long bone fractures, were noted. Brain imaging parameters for TBI, such as midline shift, contusion site, contusion volume, or basal cistern effacement, were recorded according to Marshal computed tomography (CT) grading. Glasgow Coma Scale (GCS) and pupillary response were recorded at presentation, immediately following resuscitation, and continuously every 4th h.

An external ventricular drain (EVD) catheter was used to monitor ICP. An indwelling urinary catheter was inserted to monitor urinary output and to measure IAP. ICP and IAP were continuously monitored and recorded until the removal of the ICP monitor was deemed appropriate (stable ICP of < 15 mmHg for 48 hours) or for a maximum monitoring duration of one week. One week is chosen as the decision for surgical decompression or continuation of best medical management is made by this time.

Inclusion criteria

We included all patients who sustained TBI with a GCS score of 10 or below after resuscitation and an abnormal CT scan. The patient group with GCS 9 and 10 was added to the severe TBI (GCS 3–8) group to improve the sample size and salvageability. Patients with severe TBI and a normal head CT were only included if two or more features were present at admission: age >40 years, unilateral or bilateral motor posturing, or a systolic blood pressure <90 mmHg. We only included patients that were followed for at-least 3 months post-injury.

Exclusion criteria

Patients suffering from severe coagulopathy, the only major contraindication to ICP monitoring by EVD insertion, were excluded from the study. We excluded all patients with severe TBI who had concomitant abdominal injuries requiring laparotomy or serial observations by general surgeons. We excluded pregnant women and children <3 years of age. We also excluded patients who had undergone abdominal, urinary bladder, or urethral surgery, as these conditions make IAP measurement difficult or unreliable. Patients with urethral strictures/injuries (inability to pass an indwelling catheter) were also excluded from the study. Patients with concomitant spine trauma were omitted as dysautonomia after spinal shock influences abdominal pressure on its own.

Ethical approval

The study was approved by the Institutional Ethics Committee (IEC), AIIMS/IEC/21/610.

IAP measurement

We used vesical pressure measurement as a low-cost, upfront, and reproducible method for calculating the IAP using a 50 mL syringe, an intravenous (IV) infusion set, an appropriate-sized Foley catheter, a measuring scale, and a hemostat.

The IV infusion set connection was disconnected from the infusion tube and attached to a syringe containing 25 mL of saline. Saline was injected into the empty bladder after being attached to the main drainage channel of the Foley catheter. The connector was then secured with the aid of a rubber-shod hemostat. The connector connected to the Foley catheter was removed along with the empty syringe. Next, the connector was attached to the IV set tubing, held vertically above the symphysis. After releasing the hemostat, saline flows out of the catheter drainage tubing until it reaches the IAP in saline height (measured in cm). The measurement is done supine at end-expiration, zeroed at the iliac crest in the mid-axillary line, and taken 30–60 s after injecting 25 mL of saline to give the bladder detrusor muscle time to relax. Values can be expressed in millimeters of mercury (1 mmHg = 1.36 cmH_2O).

ICP measurement

ICP was measured by placing a standard EVD catheter into one of the lateral ventricles (preferably the right frontal horn), which was connected to a transducer through a three-way tap. The transducer was zeroed to atmospheric pressure at the level of tragus. The transducer was then connected to monitor for ICP values, 10ml/hr of CSF was drained if deemed necessary by the treating physician to reduce the ICP. Readings were taken 10 min after stopping the CSF drainage through a three-way tap, and pressure readings were taken on the ICP monitor.

Zeroing in Philips Monitor EVD transducer placed at the level of ear (i.e., Foramen of Monro) and 3-way tap in EVD system was turned off. The cap was removed to open the transducer to the atmosphere, and "zero" was pressed on the monitor while the transducer was still open. The button was pressed twice, and the machine beeped once completed. The transducer connected to the monitor when the screen displayed "ICP zeroed." CSF samples were sent for culture, and non-contrast CT was performed to verify the catheter position. 10 mL CSF was drained every time the ICP threshold raised to 22 mmHg or above, as per the protocol.

RESULTS

Demographic Data

Demographic data are shown in Table 1. A total of 28 patients (*n* = 28) with an age range of 18–65 years (mean of 32.36 years ± 13.52 years standard deviation [SD]). 57.1% (*n* = 16) of these patients were in the age range of 18–30 years, thereby, the median age was 28 years. 92.9% (*n* = 26) of the patients were male, while 7.1% (*n* = 2) were female in this study. The most common mode of injury (78.6%) was found to be road traffic accidents (RTA; *n* = 22), and the mean GCS at presentation was 4.04 (range 2–9). Exactly half (*n* = 14) of these patients presented to our center within 24 hours of sustaining injuries, and the rest half (*n* = 14) after elapsing the crucial 24-h postinjury period. About 64% of the patients (*n* = 18) had sustained no extracranial injuries, while the rest of the patients, that is, 36% (*n* = 10), had associated injuries; however, these injuries did not fall under the exclusion criteria of our study. Out of 28 patients enrolled in the study, 71.4% of patients (*n* = 20) had received primary treatment elsewhere before reaching our center. Only 28.6% (*n* = 8) of the patients presented at our trauma center without receiving any primary treatment.

Table 1: Demographic features of the study group.

Parameter	Value
Age	
Mean	32.36±13.52
Median	28.00
Range	18.00–65.00
Gender (%)	
Male	26 (92.90)
Female	2 (7.10)
GCS (at presentation)	
Mean	4.04
Median	4.00
Range	3.00–9.00
Associated injuries (%)	
Present	10 (35.72)
Absent	18 (64.28)
Mode of injury (%)	
RTA	22 (78.6)
FFH	6 (21.4)
Marshall CT (%)	
Class 2	2 (7.1)
Class 3	17 (60.7)
Class 4	3 (10.7)
Class 5	6 (21.4)

GCS: Glasgow Coma Scale, CT: Computed tomography, RTA: Road traffic accident, FFH: Fall from height

A maximum number of patients enrolled in the study presented with Marshall CT class 3, i.e., 60.7% (*n* = 17) and class 5, i.e., 21.4% (*n* = 6). 10.7% (*n* = 3) of the patients had Marshall CT Class 4, and only two patients presented with Marshall CT Class 2.

ICP

All patients receive standardized intensive care unit (ICU) care consisting of anti-edema, anti-epileptics, and other conservative management protocols. The overall trend of change in ICP can be seen in Table 2 for mean/median ICP and in Figures 1 and 2 for bar/line diagram. As mentioned in the methods, 10 mL CSF was drained every time the ICP threshold raised to 22 mmHg or above, as per the protocol. The mean ICP measured at the presentation of this patient cohort was 20.04 mmHg. This means ICP (mmHg) decreased

from a maximum of 20.04 at the 0 hr time point (at the time of insertion of EVD) to a minimum of 12.09 at the 96 hr time point. This change in mean ICP (from 0 h to 96 h) was found to be statistically significant (Friedman Test: $\chi^2 = 87.6$, $P \leq 0.001$).

As shown in Table 3, *post hoc* pairwise analysis was performed. This was done to explore the significant difference in ICP (mmHg) values in comparison to the ICP values at 0 h' time point, that is, at the time of EVD insertion after resuscitation. This revealed that ICP (mmHg) differed significantly from the 0 h time point at the following time frame: 36 h, 54 h, 60 h, 72 h, 78 h, 84 h, 90 h, 96 h, and the maximum change from the 0 h' time point was observed at the 96 h' time point. This may reflect the effect of ICP control measures received by the patient.

IAP

The mean IAP (cmH2O) decreased from a maximum of 16.71 at the 0 h' time point to a minimum of 9.68 at the 96 h' time point. This change was statistically significant (Friedman test: $\chi^2 = 71.8$, $P \leq 0.001$). The overall trend of change in IAP can be seen in Table 4 for mean/median IAP and in Figures 3 and 4 for bar/line diagram.

As shown in Table 5, *post hoc* pairwise analysis was performed to explore at which time points the IAP (mmHg) differed significantly from the 0 h' time point. We found that like ICP, the mean IAP (cmH2O) decreased from a maximum of 16.71 at the 0 h' time point to a minimum of 9.68 at the 96 h' time point. It was observed that following the trend of ICP, the IAP (cmH_2O) also differed significantly from the 0 h time point at 36 h, 60 h, 72 h, 78 h, 84 h, and 90 h with maximum change from the 0 h' time point was observed at the 96 h' time point.

Relationship between ICP and IAP

To further scrutinize our notion, we followed and compared the percentage change in ICP with the percentage change in IAP at different time frames. The correlogram shown in Figure 5 discloses the linear relationship between these two variables. As seen in Figures 6–24, these changes start from 6 h and extend up to 96 h.

As shown in Table 6, in summarizing the per unit percentage change in IAP on per unit percentage change in ICP, we observed that both were correlated to each other. The correlation coefficient between these variables varied from a minimum of 0.71 to 0.89 at different time frames. It followed a trend in a directly proportional manner and was found to be statistically significant ($P < 0.001$) in each time frame of the study. The rise in one parameter followed the rise in another parameter and vice versa. These observations prove our notion that the IAP follows the trend of ICP.

Table 2: Summary of ICP pressure (mmHg) of all patients (n=28) at various time points.

ICP (mmHg)	Mean±SD	Median (IQR)	Min–Max
0 h	20.04±6.47	19.50 (14.75–26.00)	7.0–30.0
6 h	18.64±7.00	19.50 (12.00–26.00)	6.0–28.0
12 h	18.39±6.10	17.50 (14.00–24.00)	8.0–29.0
18 h	17.33±5.66	18.00 (12.50–20.00)	9.0–30.0
24 h	16.41±5.18	16.00 (11.50–20.00)	9.0–26.0
30 h	16.54±5.59	15.50 (12.50–19.50)	6.0–28.0
36 h	16.00±5.26	14.00 (12.50–18.50)	7.0–26.0
42 h	15.81±5.05	14.00 (12.00–18.00)	8.0–26.0
48 h	15.93±4.71	15.00 (12.50–18.00)	9.0–26.0
54 h	14.50±4.51	14.00 (11.00–16.75)	9.0–28.0
60 h	13.81±4.91	14.00 (11.25–16.00)	6.0–30.0
66 h	14.24±3.85	13.00 (12.00–16.00)	9.0–25.0
72 h	17.84±22.48	13.00 (12.00–14.00)	8.0–124.0
78 h	13.21±4.84	13.00 (10.00–14.25)	4.0–28.0
84 h	12.74±4.81	12.00 (10.50–14.00)	3.0–29.0
90 h	12.73±4.81	13.50 (10.00–14.00)	1.0–26.0
96 h	12.09±3.87	13.00 (10.25–14.00)	3.0–18.0

IQR: Interquartile range, SD: Standard deviation, ICP: Intracranial pressure

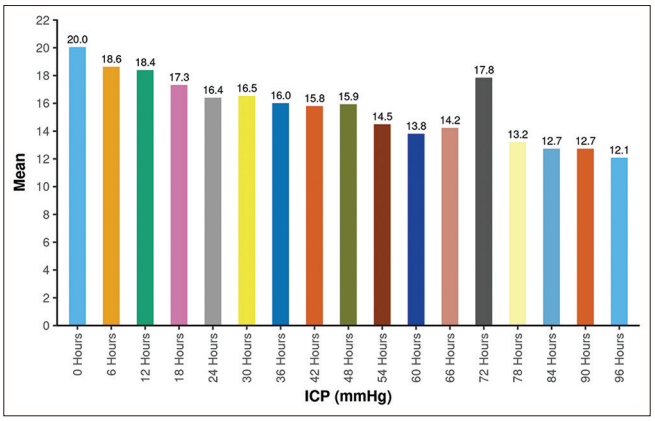

Figure 1: Bar diagram showing the change in ICP (Intracranial Pressure, mmHg) over time.

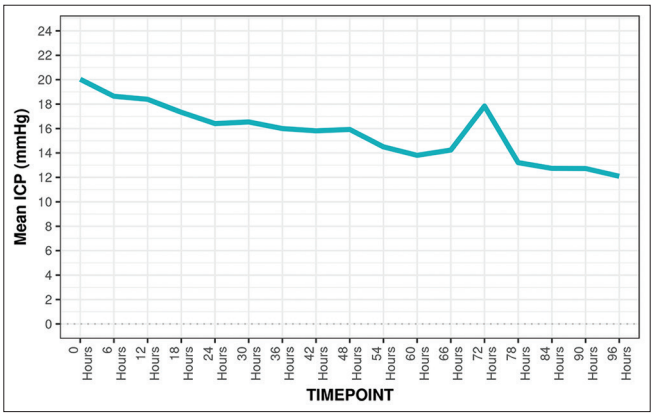

Figure 2: Line diagram showing the trend of ICP (Intracranial Pressure, mmHg) over different time frames.

Table 3: *Post hoc* pairwise analysis of 0 h ICP with different time frames.

Comparison of ICP (mmHg) at various time points versus 0 h	Mean (SD) of difference	Median (IQR) of difference	Range of difference	*P*-value
6 h–0 h	−1.39 (3.30)	−2.00 (5.00)	−8.00–6.00	0.994
12 h–0 h	−1.64 (3.58)	−2.00 (4.25)	−11.00–7.00	0.999
18 h–0 h	−2.93 (4.14)	−2.00 (3.50)	−12.00–6.00	0.774
24 h–0 h	−3.85 (4.70)	−3.00 (4.50)	−16.00–7.00	0.288
30 h–0 h	−4.04 (4.94)	−3.50 (5.00)	−14.00–5.00	0.201
36 h–0 h	−4.33 (5.43)	−3.00 (8.00)	−15.00–4.00	0.036
42 h–0 h	−4.52 (5.12)	−3.00 (7.50)	−14.00–4.00	0.063
48 h–0 h	−4.41 (5.09)	−3.00 (6.50)	−15.00–3.00	0.149
54 h–0 h	−5.62 (5.31)	−4.00 (6.00)	−17.00–3.00	0.020
60 h–0 h	−6.31 (5.75)	−5.00 (7.50)	−20.00–2.00	0.008
66 h–0 h	−5.80 (5.17)	−4.00 (8.00)	−16.00–2.00	0.074
72 h–0 h	−2.20 (23.06)	−5.00 (7.00)	−17.00–106.00	0.008
78 h–0 h	−6.71 (5.35)	−6.00 (6.50)	−18.00–1.00	0.003
84 h–0 h	−7.00 (5.77)	−6.00 (7.00)	−19.00–2.00	0.003
90 h–0 h	−7.23 (5.30)	−6.00 (7.00)	−18.00–2.00	0.010
96 h–0 h	−7.86 (6.60)	−8.50 (6.75)	−23.00–3.00	0.014

Post hoc pairwise tests for Friedman test performed using Nemenyi test method for *P*-value correction. Green background denotes a statistically significant difference at $P<0.05$. IQR: Interquartile range, SD: Standard deviation, ICP: Intracranial pressure

Table 4: Summary of IAP (cmH2O) values at different time frames.

IAP (cmH_2O)	Mean±SD	Median (IQR)	Min–max
0 h	16.71±6.23	16.50 (10.00–24.00)	5.0–25.0
6 h	15.61±6.75	17.50 (9.00–22.00)	5.0–26.0
12 h	15.21±5.92	15.00 (10.00–21.00)	5.0–25.0
18 h	14.07±5.74	13.00 (9.00–19.00)	6.0–24.0
24 h	13.22±5.55	12.00 (9.00–19.00)	6.0–22.0
30 h	13.50±6.14	12.00 (9.00–17.50)	5.0–25.0
36 h	12.74±5.16	10.00 (9.00–16.50)	6.0–24.0
42 h	12.67±5.22	10.00 (9.00–14.50)	6.0–25.0
48 h	12.89±5.07	11.00 (9.00–18.00)	6.0–23.0
54 h	11.85±5.04	10.00 (8.25–14.25)	6.0–24.0
60 h	10.77±4.83	9.50 (8.25–12.75)	5.0–26.0
66 h	11.36±4.37	10.00 (9.00–12.00)	6.0–22.0
72 h	15.24±17.76	10.00 (8.00–12.00)	5.0–78.0
78 h	10.29±4.69	9.50 (7.50–12.00)	4.0–24.0
84 h	9.87±4.31	9.00 (8.00–10.50)	4.0–24.0
90 h	10.05±3.92	9.50 (8.25–12.00)	4.0–22.0
96 h	9.68±3.05	10.00 (9.00–10.75)	4.0–19.0

IQR: Interquartile range, SD: Standard deviation, IAP: Intraabdominal pressure

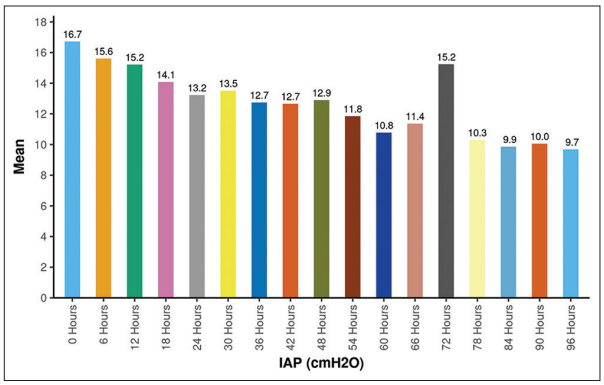

Figure 3: Bar diagram showing the change in IAP (Intra-abdominal Pressure, cmH_20) over time.

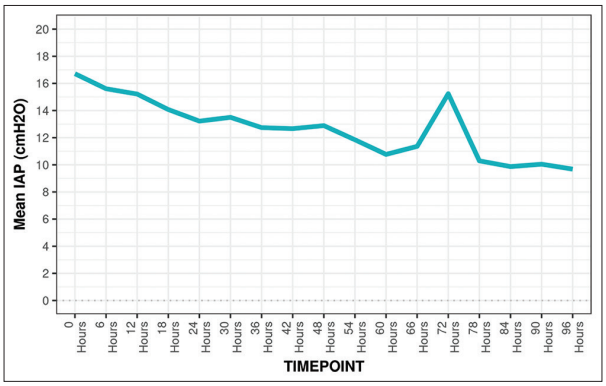

Figure 4: Line diagram showing the IAP (Intra-abdominal Pressure, cmH_20) trend over different time frames.

GCS

Monitoring GCS as shown in Table 7, we found the mean GCS: total increased from a minimum of 4.04 at the 0 h time point to a maximum of 5.30 at the 96 hr time point, and this improvement in GCS was statistically significant over time. Figures 6 and 7, in the line diagram and the box-and-whisker plot depicts the distribution of GCS: total over different time points. Out of 28 patients, a single patient underwent bifrontal decompressive craniectomy for refractory raised ICP (more than 22 mmHg). Out of

Table 5: *Post hoc* pairwise analysis of 0 h IAP with different time frames.

Comparison of IAP (cmH_2O) at various time points versus 0 h	Mean (SD) of Difference	Median (IQR) of difference	Range of difference	*P*-value
6 h–0 h	−1.11 (2.92)	−1.00 (4.00)	−6.00–4.00	0.995
12 h–0 h	−1.50 (2.78)	−1.50 (3.00)	−7.00–5.00	0.998
18 h–0 h	−2.70 (3.65)	−3.00 (4.50)	−10.00–5.00	0.774
24 h–0 h	−3.56 (3.97)	−3.00 (3.00)	−13.00–3.00	0.347
30 h–0 h	−3.58 (4.44)	−3.00 (5.75)	−14.00–3.00	0.383
36 h–0 h	−4.26 (4.71)	−4.00 (6.50)	−14.00–4.00	0.017
42 h–0 h	−4.33 (4.62)	−4.00 (7.00)	−15.00–4.00	0.177
48 h–0 h	−4.11 (4.78)	−3.00 (5.50)	−15.00–2.00	0.338
54 h–0 h	−4.88 (4.55)	−3.50 (5.00)	−14.00–1.00	0.080
60 h–0 h	−5.96 (5.39)	−5.00 (5.75)	−16.00–1.00	0.012
66 h–0 h	−5.08 (5.01)	−4.00 (7.00)	−16.00–2.00	0.356
72 h–0 h	−1.20 (17.98)	−4.00 (7.00)	−16.00–61.00	0.024
78 h–0 h	−5.96 (5.15)	−6.00 (5.25)	−16.00–5.00	0.003
84 h–0 h	−6.35 (5.47)	−6.00 (7.00)	−16.00–5.00	0.019
90 h–0 h	−6.45 (5.10)	−6.50 (7.50)	−15.00–3.00	0.031
96 h–0 h	−6.82 (5.59)	−6.50 (10.25)	−15.00–2.00	0.068

Post hoc pairwise tests for the Friedman test were performed using the Nemenyi test method for *P*-value correction. A green background denotes a statistically significant difference at $P<0.05$. IQR: Interquartile range, SD: Standard deviation, IAP: Intraabdominal pressure

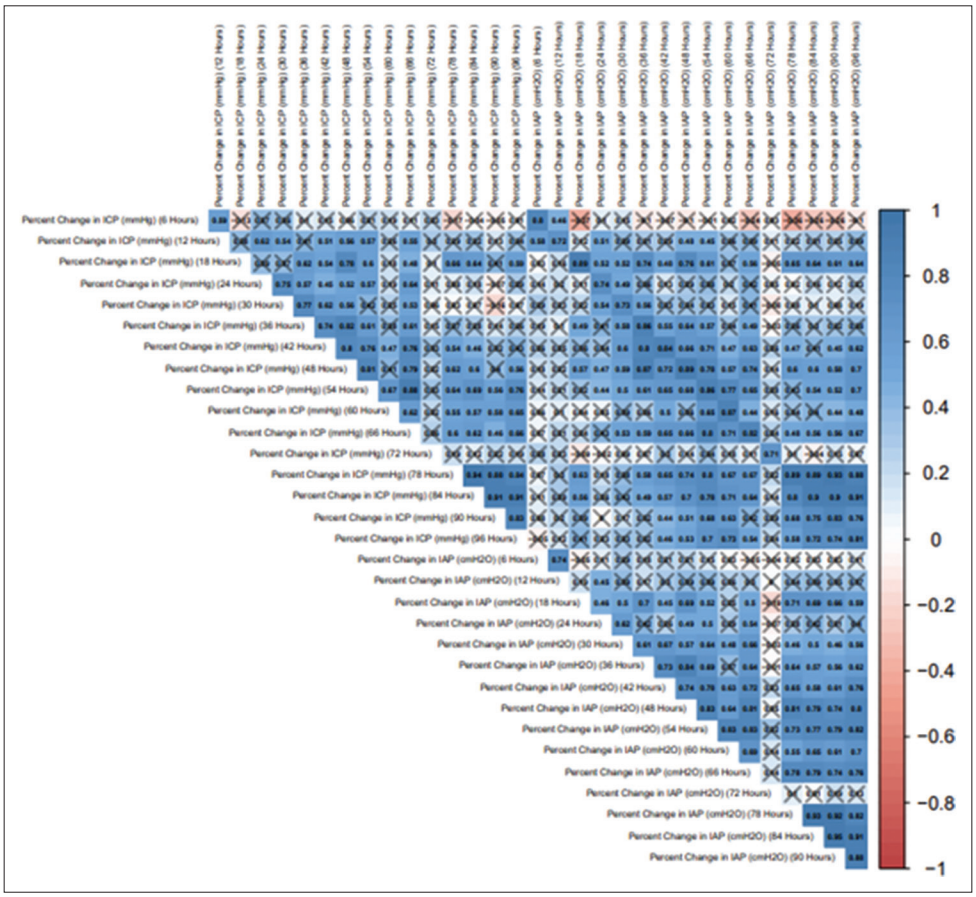

Figure 5: The correlogram shows the correlation between percentage change in intracranial pressure and intra-abdominal pressure at different time frames.

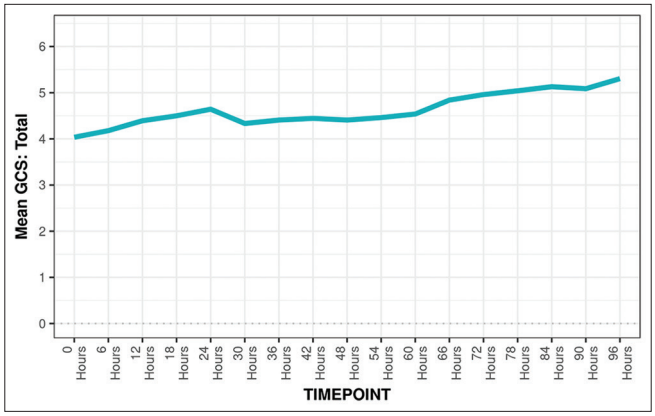

Figure 6: Line diagram depicting the trend of the Glasgow Coma Scale (GCS) over different time frames.

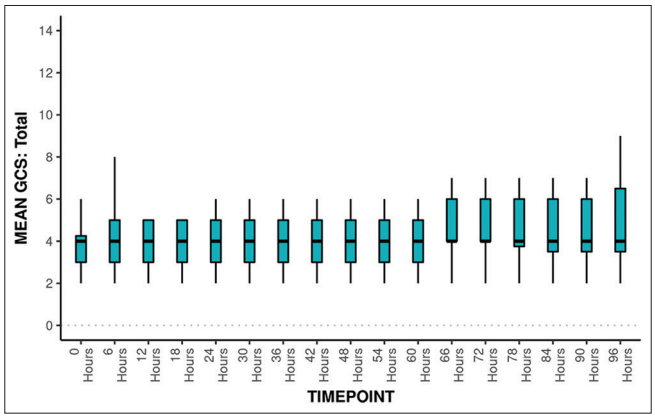

Figure 7: Box and Whisker plot showing the distribution of the Glasgow Coma Scale (GCS) over the different periods.

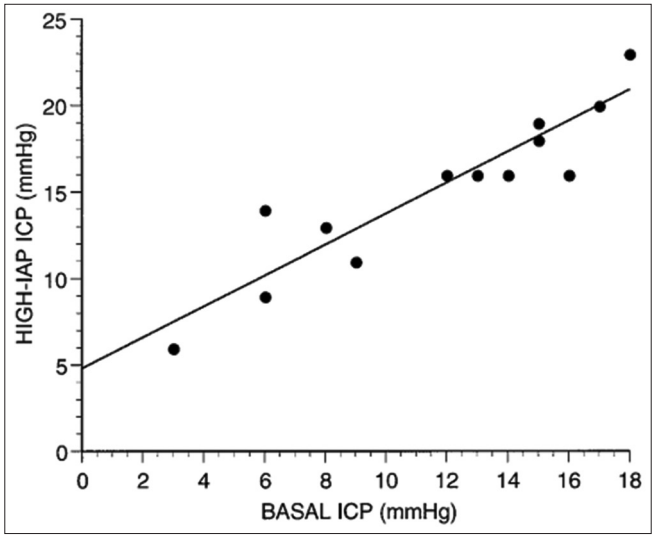

Figure 8: Baseline intracranial pressure (ICP) values correlate with the ICP levels reached during weight positioning (high-intra-abdominal pressure, IAP), depicted in the linear regression model[5] that best fits the relationship.

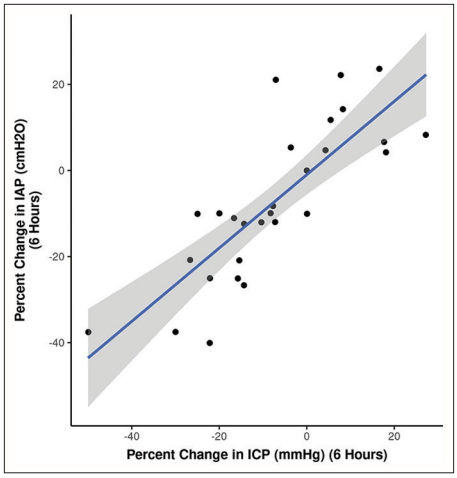

Figure 9: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 6 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH_20, 6 h)."

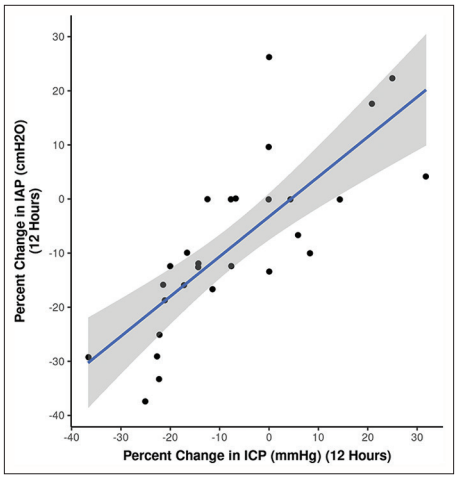

Figure 10: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 12 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH_20, 12 h)."

28 patients, 15 patients died and 13 patients were followed up for 6 months, giving us a mortality rate of 53.6%. The mean Glasgow Outcome Scale Extended at 3 months and 6 months was 1.85 (range 1–5) and 2.96 (range 1–8), respectively.

DISCUSSION

Raised ICP is undisputedly the most significant contributor to poor outcomes after TBI. However, barring a few Level I trauma centers in LMIC (Low and Middle Income

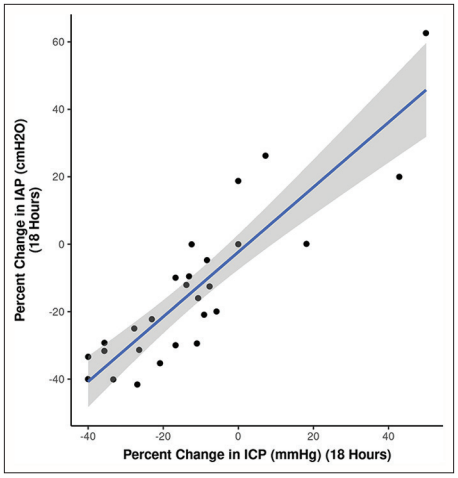

Figure 11: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 18 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 18 h)."

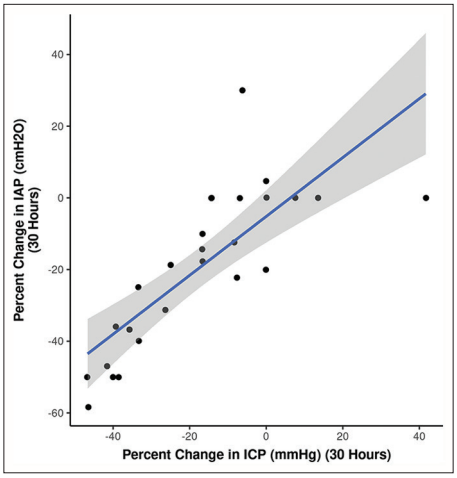

Figure 13: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 30 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 30 h)."

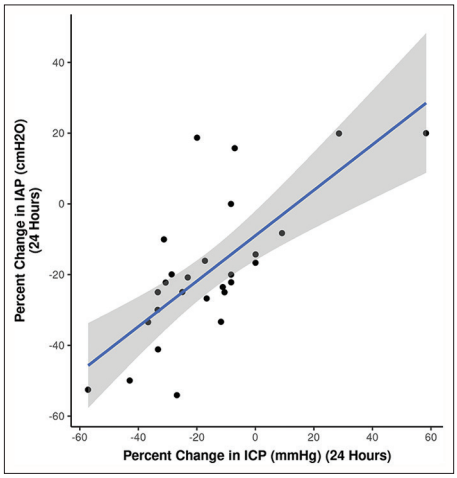

Figure 12: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 24 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 24 h)."

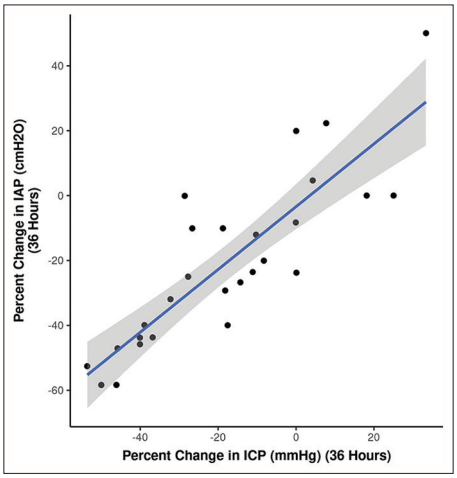

Figure 14: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 36 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 36 h)."

Countries), the clinical management of patients with TBI rarely includes ICP monitoring.

The conventional approach, which calls for the invasive insertion of an ICP sensor into brain parenchymal tissue or ventricles, might be one of the factors for the infrequent ICP monitoring at most centers. Additional limiting factors include the expense of an ICU set-up required for implanting the ICP sensors and the scarcity of skilled personnel.

Direct and indirect additional investments are included in the cost of ICP monitoring in managing severe TBI patients. The stated benefit, treatment recommendations, and potential prognostic value that ICP monitoring carries must all be weighed against the cost of purchasing and maintaining ICP sensor probes, monitoring parameters, ICU and nursing care following insertion, and infection risk (its treatment included).

Hence, finding other methods to gauge ICP becomes essential in such resource limited settings. Such an alternative must effectively replace ICP monitoring by being accurate, cost-effective, easy to monitor, and less invasive to minimize complications. IAP is one such proxy meter in patients suffering from TBI.

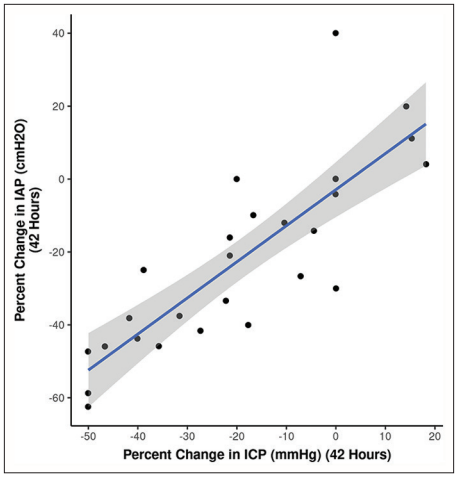

Figure 15: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 42 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 42 h)."

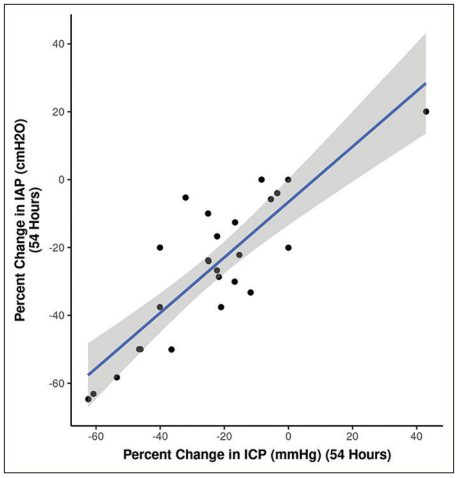

Figure 17: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 54 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 54 h)."

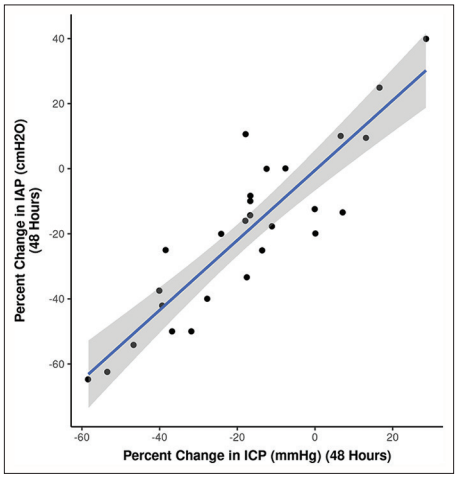

Figure 16: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 48 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 48 h)."

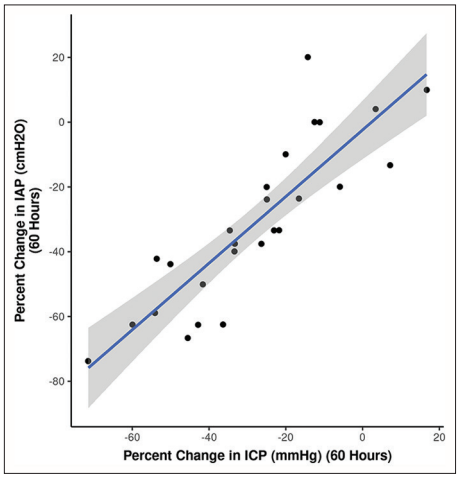

Figure 18: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 60 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 60 h)."

In India, 1 out of every six trauma victims dies, compared to 1 out of every 200 in the US. This disparity, which appears impenetrable, speaks much about the refined prehospital practices in the US (or other developed nations) and their almost thorough absence in India. Within the first 2 h of damage, there is almost a 50% mortality rate. It is now well recognized that secondary injury, which develops over hours and days after the primary impact, contributes more significantly to the deterioration than the primary insult. Increased mortality and disability may be the result of uncontrolled inflammation and edema, the hallmark of secondary injury. As a result, outcomes following TBI depends on the prompt and appropriate therapy directed towards mitigating secondary effects of TBI. Decompressive craniectomy, followed by cranioplasty (and its associated complications) in survivors, is the most common surgical response after sustaining TBI.[4]

TBI is a significant cause of disability among trauma victims in India. The age range 20–29 years is the most affected, followed by 30–39 years, and thus, TBI can also be considered an epidemic of the young population.[9]

In the current study, the mean (SD) of age (years) was 32.36 (± 13.52) with an age range from 18 to 65 years, and the

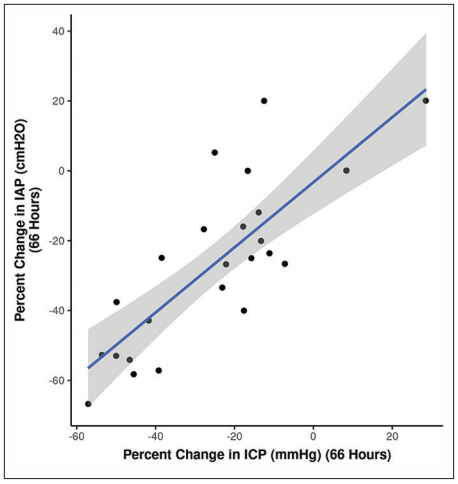

Figure 19: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 66 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 66 h)."

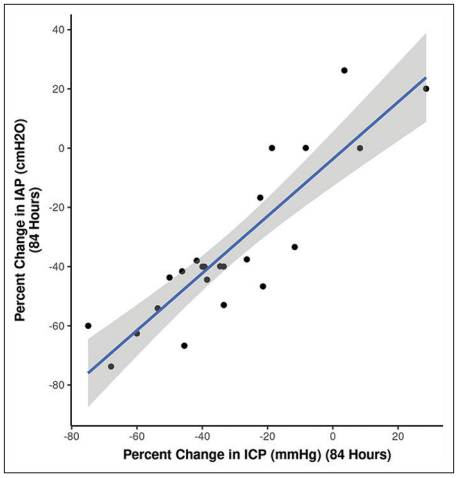

Figure 21: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 78 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 78 h)."

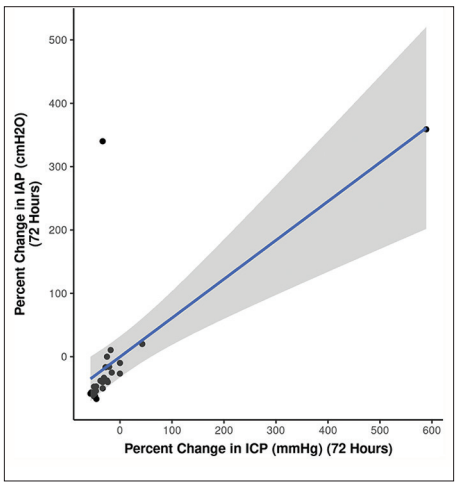

Figure 20: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 72 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 72 h)."

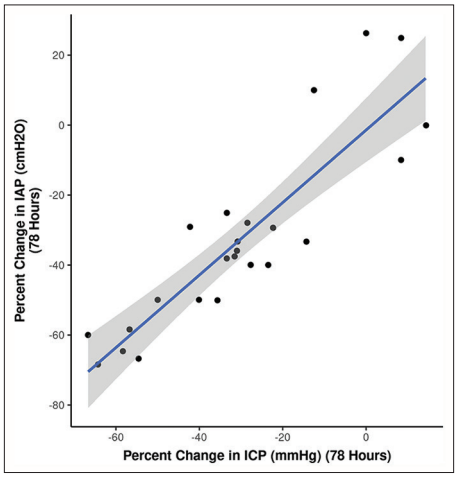

Figure 22: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 84 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 84 h)."

median (IQR) of age (years) was 28.00 (21–42). This large proportion of the young population holding the majority share among a cohort of TBI patients is well explained by the increased risk of exposure to contributory factors of TBI as they are the most traveling group of the population to earn a livelihood, to study, and are also the primary workforce of society.

Age has been extensively studied as a predictor of mortality in TBI patients. In many studies, old age has been identified as a predictor of poor outcomes.[1,8] In the current study, the mean age of patients who died despite receiving essential critical care was 35.25 years, whereas the mean age of patients who survived was 24.35 years.

In India, road traffic injuries are the primary cause of TBIs (60%), followed by falls (20–25%) and violence (10%).[2] We also found that out of 28 patients enrolled in our study, 78.6% (n = 22) of the patients suffered TBI due to RTA, while 21.4% (n = 6) of the patients presented after fall from height as the cause of TBI.

Alali *et al.*, in their study,[1] developed a clinical rule for the prediction of intracranial hypertension in severe TBI and included Marshall Grades III and IV as major criteria.

Table 6: Correlation of per unit percentage change ICP for 1 unit percentage change in IAP and vice versa.

Time frame (h after insertion of EVD)	% Change in ICP (for 1 unit % change in IAP)	% Change in IAP (for 1 unit % change in ICP)	Correlation coefficient (r)	*P*-value
6	0.77	0.85	0.81	<0.001
12	0.80	0.74	0.84	<0.001
18	0.81	0.96	0.85	<0.001
24	0.81	0.64	0.71	<0.001
30	0.76	0.82	0.79	<0.001
36	0.76	0.97	0.85	<0.001
42	0.70	0.99	0.83	<0.001
48	0.74	1.07	0.89	<0.001
54	0.89	0.82	0.85	<0.001
60	0.72	1.03	0.86	<0.001
66	0.72	0.93	0.82	<0.001
72	0.82	0.61	0.82	<0.001
78	0.75	1.04	0.84	<0.001
84	0.81	0.96	0.84	<0.001
90	0.79	0.83	0.84	<0.001
96	0.82	0.74	0.76	<0.001

ICP: Intracranial pressure, IAP: Intra-abdominal pressure, EVD: External ventricular drain

Table 7: Mean, median, and range of GCS (total) at different time frames.

Time point	GCS: Total			Friedman test	
	Mean (SD)	Median (IQR)	Range	Chi-square	*P*-value
0 h	4.04 (1.40)	4.00 (1.25)	2.00–9.00	79.4	<0.001
6 h	4.18 (1.33)	4.00 (2.00)	2.00–8.00		
12 h	4.39 (1.95)	4.00 (2.00)	2.00–10.00		
18 h	4.50 (2.38)	4.00 (2.00)	2.00–12.00		
24 h	4.64 (2.74)	4.00 (2.00)	2.00–14.00		
30 h	4.33 (2.15)	4.00 (2.00)	2.00–12.00		
36 h	4.41 (2.26)	4.00 (2.00)	2.00–12.00		
42 h	4.44 (2.36)	4.00 (2.00)	2.00–12.00		
48 h	4.41 (2.27)	4.00 (2.00)	2.00–12.00		
54 h	4.46 (2.37)	4.00 (2.00)	2.00–12.00		
60 h	4.54 (2.50)	4.00 (2.00)	2.00–12.00		
66 h	4.84 (2.56)	4.00 (2.00)	2.00–12.00		
72 h	4.96 (2.59)	4.00 (2.00)	2.00–12.00		
78 h	5.04 (2.56)	4.00 (2.25)	2.00–12.00		
84 h	5.13 (2.60)	4.00 (2.50)	2.00–12.00		
90 h	5.09 (2.61)	4.00 (2.50)	2.00–12.00		
96 h	5.30 (2.80)	4.00 (3.00)	2.00–12.00		

GCS: Glasgow Coma Scale, SD: Standard deviation, IQR: Interquartile range

In contrast, Grade II was a minor criterion. They observed that the rule was highly sensitive, that is, 93.9%. However, they had a very low specificity, that is, 42.3%, in predicting intracranial hypertension and thus can be a helpful guide to select patients who require ICP monitoring.

In the current study, the average Marshall score of patients who expired was 3.9, which was much higher than the average Marshall score of patients who survived, which was 2.85.

In patients with head injuries, ICP monitoring is critical[11] if the clinical assessment is inadequate or if there is a high risk of elevated ICP, as recommended by BTF guidelines.

Farahvar *et al*., in their study [8] monitored the ICP in 1202 individuals, while 244 patients were not monitored at all. They concluded that severe TBI patients should be treated with ICP-directed therapy with ICP monitoring as it is associated with lower mortality. Saul and Ducker, in their study[12] on severe head injury patients who were managed with mannitol therapy and CSF drainage, found that

mortality reduced to 28% in which ICP was 15 mmHg while it was 46% in patients with ICP between 20 and 25 mmHg. In the current study, patients with mortality had a mean ICP of 19.5 and a mean IAP of 16.8 at 0 h, while the mean ICP was 17.71 and mean IAP was 14.35 in patients who survived. The mean ICP (mmHg) decreased from a maximum of 20.04 at the 0-h time point to a minimum of 12.09 at the 96-h time point.

ICP monitoring can be done either by invasive or noninvasive methods. Among the invasive methods, EVD is the gold standard and has the benefits of being therapeutic apart from its diagnostic role. However, in patients with brain swelling with small ventricles or distorted normal anatomy due to mass effect, EVD insertion can be difficult.[10] In such cases, intraparenchymal sensors are more helpful, which may be why they are more commonly used than EVDs.

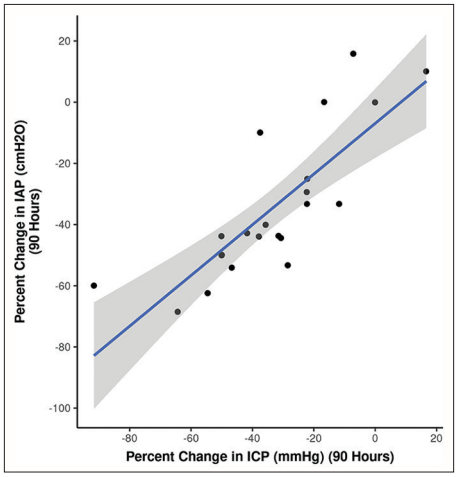

Figure 23: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 90 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 90 h)."

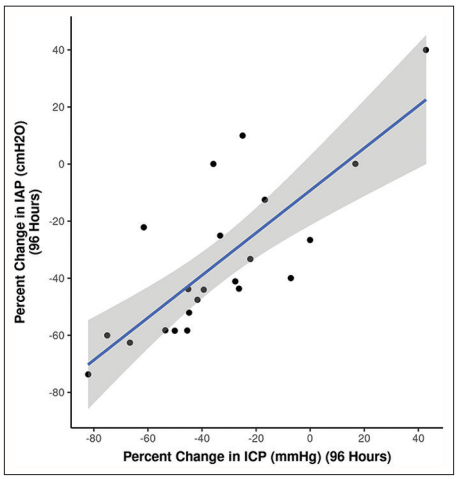

Figure 24: Scatter plot with trend line diagram showing an association between "percent change in ICP (Intracranial Pressure, mmHg, 96 h)" and "percent change in IAP (Intra-abdominal Pressure, cmH20, 96 h)."

Bloomfield *et al*. tried to study[3] the effects of elevated IAP on intracranial pressure (ICP) (7.6 ± 1.2 vs. 21.4 ± 1.0) and cerebral perfusion pressure (CPP) before and after intravascular volume resuscitation and found elevated IAP significantly increased ICP, pleural pressure, and central venous pressure; whereas cardiac index and CPP decreased significantly and then abdominal decompression was done which returned ICP (11.2 ± 1.8) toward baseline and further increased CPP.

Bloomfield *et al*. extended their study[2] and inserted an inflated balloon in the peritoneal cavity of anesthetized swine and found that an increase of IAP to 25 mmHg above baseline caused significant increases in ICP (7.3 ± 0.6 [standard error of the mean] to 16.4 ± 1.9 mmHg) and further observed that sternotomy and pleuropericardotomy negate all effects of increased IAP except the decreased cardiac index.

Citerio *et al*. in their study[5] evaluated the effect of a stepwise increase in IAP on ICP in 15 sedated ventilated patients by positioning a soft, 15-L water bag on the patient's abdomen and continuously monitoring IAP and ICP before and 20 min after IAP was increased. Over the basal ICP values examined in this study, the baseline ICP values were correlated with the ICP levels reached during weight positioning (high-IAP ICP) as shown in Figure 8. A linear regression model best fitted this relationship.

Deeren *et al*. were the first to examine with a large number of measurements whether increases in IAP are associated with increases in ICP and decreases in CPP in ventilated patients with non-TBI.[6] The study included 11 critically ill patients (7 men, four women; mean age 63 ± 16 years) with a primary diagnosis of ischemic (n = 4), hemorrhagic (n = 5), and metabolic encephalopathy (n = 2) and received ICP monitoring due to clinical and radiological signs of intracranial hypertension. ICP and IAP were continuously monitored, and 214 averaged consecutive measurements were compared. It was found that increases in IAP were associated with increases in ICP and decreases in CPP. They also observed a significant decrease in IAP after administering 10 mg cis-atracurium IV bolus, which was associated with a concomitant fall in ICP in 12 simultaneous IAP and ICP measurements. These authors concluded that increases in IAP are associated with increases in ICP and decreases in CPP.

To the best of our knowledge, no study has monitored continuous IAP and ICP in severe TBI patients and establishing relationships, if any. Our study is the first ever done on humans (no such study on animal models either)

to establish a trend between IAP and ICP in TBI patients and test its validity for using IAP as a proxy for ICP. Our study monitored 17 continuous readings 6 hours apart and established that the IAP trend follows the ICP trend. We also drain 10 mL CSF every time the ICP reading goes above 22 mmHg and noticed that IAP also follows the fall trend in ICP on draining CSF. Both IAP and ICP differed significantly from the 0 h' time point compared to other time points, and IAP followed the trend of ICP with varying magnitudes on each unit change. Our study also observed that the 0 h IAP and ICP differed significantly from 36 h IAP and ICP, respectively.

In neurointensive care, monitoring is increasingly utilized to detect brain ischemia before it occurs and to direct focused therapy to improve cerebral perfusion and oxygenation.[13-15]

This study has its strengths and limitations. As the CSF was drained to control ICP, the waveform did not follow a usual trend, which it might have followed with other ICP monitoring techniques where CSF is not drained, for example, the parenchymal probe. Furthermore, in retrospect, authors tend to consider a long tunnel EVD to keep it longer and thus follow the trend beyond 96 h. Despite this, the study has the largest sample size in the literature for continuous ICP and concomitant IAP readings in patients with severe TBI. It is the first study that recorded the effect of change in ICP on IAP based on continuous ICP monitoring. In addition, our study established that free-hand EVD placement could be done even in thin ventricles with diffuse brain edema, as in cases of severe TBI.

Despite its limitations, ICP monitoring remains central to the monitoring and managing severe TBI. Multiple studies in the past have done interventions to change IAP and see its effect on change in ICP. However, this study was the first to do an intervention to change ICP (EVD and CSF drainage) and recorded a concomitant change in IAP.

CONCLUSION

In this study, authors established that the ICP of severe TBI patients correlate well with IAP at presentation. This correlation was intense and constant, irrespective of the timeframe during the treatment and monitoring. This study also established that draining CSF to decrease ICP in severe TBI patients is reflected in IAP. The study further validates that IAP is a proxy of ICP in severe TBI patients.

Ethical approval

The research/study approved by the Institutional Review Board at AIIMS Rishikesh India, number AIIMS/IEC/21/610, dated November 26, 2021.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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