

Accepted Manuscript

Does Base Deficit predict mortality in patients with severe traumatic brain injury?

Hussain Shallwani, Muhammad Waqas, Shahan Waheed, Mubbashira Siddiqui,
Asher Froz, MSc Statistics, Muhammad Ehsan Bari, Assistant Professor



PII: S1743-9191(15)00288-5

DOI: [10.1016/j.ijisu.2015.05.054](https://doi.org/10.1016/j.ijisu.2015.05.054)

Reference: IJSU 1934

To appear in: *International Journal of Surgery*

Received Date: 11 April 2015

Accepted Date: 4 May 2015

Please cite this article as: Shallwani H, Waqas M, Waheed S, Siddiqui M, Froz A, Bari ME, Does Base Deficit predict mortality in patients with severe traumatic brain injury?, *International Journal of Surgery* (2015), doi: 10.1016/j.ijisu.2015.05.054.

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Does Base Deficit predict mortality in patients with severe traumatic brain injury?

Hussain Shallwani

hussainshallwani@hotmail.com

Section of Neurosurgery,
The Aga Khan University Hospital Karachi.

Muhammad Waqas

shaiq_waqas@hotmail.com

Section of Neurosurgery,
The Aga Khan University Hospital Karachi.

Shahan Waheed

docshahan83@hotmail.com

Department of Emergency Medicine
The Aga Khan University Hospital Karachi.

Mubbashira Siddiqui

mubbashira_siddiqui@hotmail.com

Section of Neurosurgery,
The Aga Khan University Hospital Karachi.

Asher Froz

asher.froz@aku.edu

MSc Statistics,
The Aga Khan University Hospital Karachi.

Muhammad Ehsan Bari

ehsan.bari@aku.edu

Contact # +922134864764

Assistant Professor,
Section of Neurosurgery,
The Aga Khan University Hospital Karachi.

Does Base Deficit predict mortality in patients with severe traumatic brain injury?

Abstract:

Objective: Base Deficit (BD) is a marker of tissue hypoxia in polytrauma patients. It guides resuscitative measures, and predicts outcomes, complications and mortality. The aim of this study was to examine the presence of BD in patients with isolated severe traumatic brain injury (TBI), and to assess if it correlates with the outcomes in these patients.

Method: This was a retrospective observational study. All patients over the age of 16 years presenting to Aga Khan University Hospital from 2009-2013 with isolated TBI, were included. Data was extracted from 2009-2013. Glasgow Outcome Scale (GOS) of 4 and 5 at last follow up was categorized as favorable outcome. Data was analyzed using SPSS version 19 and receiver operative curve (ROC) was generated for BD as a predictor of mortality and unfavorable outcome.

Results: One hundred and eight patients were analyzed. Ninety-eight (90.7%) were males. Mean age was 36.69 ± 17.65 . Eighty-eight (81.5%) patients had BD, while 20 (18.5%) patients had base excess. 62 (58.5%) of the patients had unfavorable outcomes. BD on admission had a statistically significant negative correlation with Glasgow Coma Scale (GCS) on presentation ($r = -0.239$, $p = 0.025$) and Revised Trauma Score (RTS) ($r = -0.214$, $p = 0.046$). However, there was no statistically significant difference in means of BD between survivors and non survivors. Area under receiver operator curve (ROC) for BD as a predictor of mortality statistically non-significant.

Conclusion: Although BD is correlated with GCS at presentation and RTS, it is not a reliable prognostic marker for outcome and mortality in patients with isolated TBI.

Introduction:

Base excess and Base Deficit are measures of acid-base abnormality, described as the amount of acid or base required to bring 1 L of whole blood to a pH of 7.4, given that arterial partial pressure of CO₂ and temperature remain constant at 40mmHg and 37degrees respectively (1). In trauma patients, BD has been long established as an important marker of tissue perfusion and hypoxia (2-5). Apart from the usual markers including heart rate, blood pressure, hemoglobin and hematocrit, etc., BD at admission is an important index to guide resuscitative measures (5-9). Several studies suggest that BD also has a notable role in predicting outcomes, risk of complications, and mortality (8-20), and is an important addition to prognostic scores such as Revised Injury Severity Classification (RISC) and RISC II (15, 20).

Despite its importance as a predictive marker in polytrauma patients, BD has not been evaluated as a prognostic indicator in patients with isolated head injury. A study by Siegel et al. showed that arterial BD was associated with poor outcome when patients had other extracranial injuries along with blunt TBI (21). Presence of TBI itself has been described as a marker of poor prognosis in trauma patients with

high BD (9, 10, 15). A recent study identified BD of greater than 4 as one of the greatest predictors of neurosurgical intervention in patients with mild TBI or intracranial injury (22). However, data regarding BD at admission and its effect on outcome and mortality in patients with isolated TBI is scarce.

Isolated head injury patients may not experience significant blood loss and hypovolemia given the nature of restricted space in the cranial cavity. Consequently, the body may not experience hypoperfusion and hypoxia, and the patients may have normal values of BD. The aim of this study was to examine the trend in BD values in patients with isolated severe TBI, and to assess if it correlates with the outcomes in these patients. As a secondary objective, we aim to analyze the predictive value of BD as an indicator of mortality in patients with isolated severe TBI.

Methods:

This was a retrospective observational study, conducted at the section of neurosurgery, department of surgery at The Aga Khan University Hospital (AKUH) Karachi. AKUH is a JCIA accredited, ISO certified tertiary care hospital with over 43 specialties, including a well-established section of neurosurgery and emergency medicine. Chart review was done over a 4-month period from March 2014 to June 2014. Data was extracted from the previous 5 years (2009-2013).

Inclusion Criteria:

We included all the patients over the age of 16 years, either male or female, presenting to AKUH with isolated TBI.

Exclusion Criteria:

We excluded patients with pre-existing central nervous system (CNS) pathology like stroke, seizures or any congenital CNS pathologies, polytrauma, major systemic injuries and patients with pre-existing congestive heart failure, endocrine disorders or renal disease before presentation.

Data Collection:

A pro forma was used as a data-collecting tool, which was filled by the investigator after identifying the patients fulfilling the inclusion criteria. The data collection tool was tested before the start of the study and the investigator was trained on how to fill the questionnaire. The data collection pro forma consisted of following five components:

- I. Demographics;
- II. Information of events and presentation;
- III. Laboratory and radiological findings;
- IV. Hospital course;
- V. Outcome

Data Collection Instrument:

The following data was recorded in the data collection instrument:

1. Demographic data (age, gender and co-morbidities) that was later coded for confidentiality and analysis;
2. Mechanism of injury;
3. Time of the event, and pre-hospital delays, and hospitalization information for the event;
4. Severity of injury according to the Glasgow Coma Scale (GCS), Revised Trauma Score (RTS), other systemic injuries, and pupil reaction;
5. Laboratory data including pH, pCO₂, pO₂, O₂sat, bicarbonate, base excess and BD
6. Radiological data including characteristic of basal cistern, midline shift, presence/absence of epidural hematoma and/or intraventricular/subarachnoid hemorrhage;
7. Length of ICU stay and length of hospital stay
8. Follow-up and outcome, measured with the Glasgow Outcome Scale (GOS); scores of 4 and 5 were categorized as favorable outcome.

Data analysis:

Data analysis was done using SPSS version 19. Continuous variables with normal and non-normal distributions were reported as mean \pm SD and median [inter-quartile range (IQR)], respectively. Categorical variables were presented as frequencies and percentages. Data was further analyzed for correlation of BD at admission with presenting GCS and outcome. A receiver operator curve (ROC) was generated for BD as a predictor of mortality or unfavorable outcome, and area under the curves was calculated.

Results:

A review of hospital medical record database yielded a total of 164 patients with head injury during the study period. Files of these 164 patients were examined for eligibility, and 108 patients were found eligible for final inclusion and analysis; thirty patients were below the age of 16, ten patients were excluded due to incomplete records, fifteen patients due to their major systemic injuries, and 1 patient had a history of chronic renal failure. Demographic and clinical characteristics of the study population are provided in the Table 1. The median follow-up was 4.0 months (IQR: 2.0-12.0 months); two patients were lost to follow up.

Of the 108 patients included in the study, BD was present in 88 patients, while 20 patients had base excess. Other arterial blood gas parameters are summarized in Table 2.

Forty-four (41.5%) of the patients had favorable outcome of GOS 4 or 5, while 62 (58.5%) of the patients had unfavorable outcomes.

Correlation analysis showed that age of the patient was negatively correlated to the GOS of patient on follow-up ($r=-0.458$, $p<0.001$). Similarly Rotterdam CT scores also had a negative correlation with the GOS ($r=-0.238$, $p=0.02$). BD on admission had a statistically significant negative correlation with GCS on presentation ($r=-0.239$, $p=0.025$) and RTS ($r=-0.214$, $p=0.046$). However, BD did not reach statistically significant correlation with Rotterdam CT score, length of ICU stay, length of hospital stay, or GOS ($p>0.05$). Base excess had a statistically significant positive correlation with the delay to arrival ($r=0.745$, $p<0.001$), GCS ($r=0.545$, $p=0.016$), and RTS ($r=0.552$, $p=0.012$), but did not correlate with Rotterdam CT score, length of ICU stay, length of hospital stay, or GOS ($p>0.05$). Moreover, GCS on presentation and RTS did not reach statistically significant correlation with length of stay or GOS ($p>0.05$).

Further analysis showed there was statistically significant difference in mean GCS on arrival and Revised Trauma score in patients presenting with bilaterally equal and reactive pupils, anisocoric pupils, or fixed and dilated pupils ($p<0.001$) (Table 3). Similarly, there was statistically significant difference in the mean GOS on follow-up in patients with normal, compressed or absent cisterns ($p=0.032$), and in patients with and without IVH/SAH ($p=0.048$). There was also statistically significant difference in the mean BD in patients with and without epidural hematoma ($p=0.016$). However, there was no statistically significant difference in means of BD in patients with favorable outcome and those with unfavorable outcome, and in those who lived and those who expired ($p>0.05$). The summary of the results is given in Table 3.

Receiver operator curve (ROC) analysis for BD as a predictor of mortality showed an area under the curve of 0.479 (95% CI 0.352-0.606) (Figure 1). Similarly, ROC analysis for BD as a predictor of outcome showed an area under the curve of 0.515 (95% CI 0.389-0.641) (Figure 2). Both curves failed to reach statistically significant difference from the area under unity line (0.5).

Discussion:

Measurement of BD for polytrauma patients has become a common practice in the emergency room because it is crucial in guiding resuscitation (5-9) and its significance as a prognostic marker (8-20). Nevertheless, the importance of BD in patients with isolated TBI is unclear.

The brain is situated in a closed cranial cavity, sharing its space with cerebrospinal fluid and blood. Isolated TBI may result in hemorrhage in the cranial cavity (epidural, subdural, subarachnoid, or intraparenchymal), along with swelling of the brain parenchyma. However, it may not be associated with excessive blood loss, hypovolemia, and systemic hypoperfusion. Zehtabchi et al. noted that, even though CSF lactate is correlated with severity of TBI, increased CSF lactate may not affect serum concentration of lactate; therefore serum BD and lactate levels are poor indicators of TBI in patients with isolated head trauma (23). In our study, we found that 88 (81.5%) patients with TBI had a BD and 20 (18.5%) patients had base excess on arterial blood gases evaluation.

In a study by Cohen et al., TBI patients with hypoperfusion (defined as BD >6) were observed to develop early coagulopathy with higher prothrombin time (PT) and partial thromboplastin time (PTT) (24). These patients also needed more blood transfusions, had lower ventilator-free days, and higher mortality (66% vs. 11%) (24). Cohen et al. also identified that higher BD in TBI patients is associated with lower GCS at presentation, lower mean arterial pressure, and higher injury severity score (24). We found similar

results with negative correlation of BD with GCS on presentation and RTS. We also found that base excess had a positive correlation with delay to arrival, GCS and RTS.

Only a few studies have evaluated the association of BD with outcomes and mortality in patients with isolated TBI. Stewart et al. studied the correlation of BD with mortality in pediatric patients with abusive head trauma (25). In their experience, survivors had a mean BD of 5.3 ± 0.6 mEq/L, whereas non-survivors had a mean BD of 12.6 ± 1.6 mEq/L (25). They concluded that higher BD was associated with increased risk of mortality; however they also noted that this might be due to secondary brain injury from hypoxia since non-survivors were more likely to receive CPR or be intubated (25).

In a recent study, Mutschler et al. described the role of BD as an indicator of hypovolemic shock in trauma patients with presence of TBI (26). They classified hypovolemic shock into 4 classes based on BD at admission, and showed that higher BD was associated with worse outcomes in terms of mortality, mean hospital length of stay, ICU days, ventilator days, multiple organ failure and sepsis (26). However, their sample of patients with TBI also included those with other systemic injuries, and was not restricted to isolated head trauma; mortality rates were higher in TBI patients with multiple injuries (26).

We found that in patients with isolated TBI, without major systemic injuries, BD and base excess were not correlated to length of ICU stay, length of hospital stay, or GOS at follow-up. Moreover, mean BD is not statistically different in survivors and non-survivors, and in favorable/unfavorable outcome groups. Based on ROC analysis, BD is not a reliable predictor of mortality or unfavorable outcome in such patients.

In our study, we also found a statistically significant difference in the mean BD in patients with and those without epidural hematoma. Mean BD was higher in patients with epidural hematoma than those without. Although, epidural hematoma is within the cranial vault and hemostasis may occur secondary to increasing intracranial pressure, there may be modest hypovolemia and systemic hypoperfusion, leading to a higher BD observed in such patients. Further studies may be warranted to evaluate this phenomenon in the future.

Furthermore, our results identify that there is a statistically significant difference in the mean length of stay between patients who survived and those who did not. Survivors had a higher mean length of stay than non-survivors. This is not unusual, as it is commonly noted that patients with TBI have a higher mortality in early hours after trauma.

This is one of the first studies evaluating the level of BD in patients with isolated TBI. However, there are a few limitations to this study. Firstly, the retrospective nature limits the information that could be assessed and evaluated. A prospective design would better establish a temporal association, and eliminate the biases associated with retrospective studies. Secondly, the power of this study is limited due to small sample size.

In addition, our study lacks a detailed account of pre-hospital resuscitation. In the developing country of Pakistan, there is a significant shortfall in pre-hospital para-medical and emergency medical services, and there is no institutionalized pre-hospital resuscitation system in place. Hence, most of the patients receive little or no pre-hospital resuscitation. Moreover, there was considerable variation in the time from injury to presentation ($SD=147.58$ hours), which can also have an effect on the level of BD.

Conclusion:

In summary, although BD is correlated with GCS at presentation and RTS, it is not a reliable prognostic marker for outcome and mortality in patients with isolated TBI. These patients may have BD or base excess, and presence of BD may indicate secondary extracranial/systemic injury (23, 26).

Conflict of interest: none

Funding: not applicable

Ethical Approval: not obtained

Research Registration Unique Identifying Number (UIN): researchregistry124

Author contribution:

Hussain Shallwani – Analysis and writing of the draft

Muhammad Waqas – Design, Methodology and Writing

Shahan Waheed – Conception of the idea, methodology

Asher Froz – Statistics and analysis

Mubbashira Siddiqui – Data Collection

Muhammad Ehsan Bari – Supervision, critical analysis of the design, methodology and review of final draft

Guarantor: Muhammad Ehsan Bari

References:

1. Hindy-Francois C, Meyer P, Blanot S, Marque S, Sabourdin N, Carli P, Orliaguet G. Admission BD as a long-term prognostic factor in severe pediatric trauma patients. *The Journal of trauma*. 2009;67(6):1272-7.
2. Kincaid EH, Miller PR, Meredith JW, Rahman N, Chang MC. Elevated arterial BD in trauma patients: a marker of impaired oxygen utilization. *Journal of the American College of Surgeons*. 1998;187(4):384-92.
3. Paydar S, Fazlzadeh A, Abbasi H, Bolandparvaz S. BD: a better indicator for diagnosis and treatment of shock in trauma patients. *The Journal of trauma*. 2011;70(6):1580-1.
4. Arnold TD, Miller M, van Wessem KP, Evans JA, Balogh ZJ. BD from the first peripheral venous sample: a surrogate for arterial BD in the trauma bay. *The Journal of trauma*. 2011;71(4):793-7; discussion 7.
5. Mutschler M, Nienaber U, Brockamp T, Wafaisade A, Fabian T, Paffrath T, Bouillon B, et al. Renaissance of BD for the initial assessment of trauma patients: a BD-based classification for hypovolemic shock developed on data from 16,305 patients derived from the TraumaRegister DGU(R). *Critical care*. 2013;17(2):R42.
6. Davis JW, Shackford SR, Mackersie RC, Hoyt DB. BD as a guide to volume resuscitation. *The Journal of trauma*. 1988;28(10):1464-7.
7. Rainer TH, Ho AM, Yeung JH, Cheung NK, Wong RS, Tang N, Ng SK, et al. Early risk stratification of patients with major trauma requiring massive blood transfusion. *Resuscitation*. 2011;82(6):724-9.
8. Davis JW, Parks SN, Kaups KL, Gladen HE, O'Donnell-Nicol S. Admission BD predicts transfusion requirements and risk of complications. *The Journal of trauma*. 1996;41(5):769-74.
9. Rutherford EJ, Morris JA, Jr., Reed GW, Hall KS. BD stratifies mortality and determines therapy. *The Journal of trauma*. 1992;33(3):417-23.
10. Tremblay LN, Feliciano DV, Rozycki GS. Assessment of initial BD as a predictor of outcome: mechanism of injury does make a difference. *The American surgeon*. 2002;68(8):689-93; discussion 93-4.
11. Smith I, Kumar P, Molloy S, Rhodes A, Newman PJ, Grounds RM, Bennett ED. Base excess and lactate as prognostic indicators for patients admitted to intensive care. *Intensive care medicine*. 2001;27(1):74-83.
12. Siegel JH, Rivkind AI, Dalal S, Goodarzi S. Early physiologic predictors of injury severity and death in blunt multiple trauma. *Archives of surgery*. 1990;125(4):498-508.
13. Rixen D, Raum M, Bouillon B, Lefering R, Neugebauer E, Arbeitsgemeinschaft "Polytrauma" of the Deutsche Gesellschaft fur U. BD development and its prognostic significance in posttrauma critical illness: an analysis by the trauma registry of the Deutsche Gesellschaft fur unfallchirurgie. *Shock*. 2001;15(2):83-9.
14. Ouellet JF, Roberts DJ, Tiruta C, Kirkpatrick AW, Mercado M, Trottier V, Dixon E, et al. Admission BD and lactate levels in Canadian patients with blunt trauma: are they useful markers of mortality? *The journal of trauma and acute care surgery*. 2012;72(6):1532-5.
15. Lefering R, Huber-Wagner S, Nienaber U, Maegele M, Bouillon B. Update of the trauma risk adjustment model of the TraumaRegister DGU: the Revised Injury Severity Classification, version II. *Critical care*. 2014;18(5):476.
16. Husain FA, Martin MJ, Mullenix PS, Steele SR, Elliott DC. Serum lactate and BD as predictors of mortality and morbidity. *American journal of surgery*. 2003;185(5):485-91.
17. Hodgman EI, Morse BC, Dente CJ, Mina MJ, Shaz BH, Nicholas JM, Wyrzykowski AD, et al. BD as a marker of survival after traumatic injury: consistent across changing patient populations and resuscitation paradigms. *The journal of trauma and acute care surgery*. 2012;72(4):844-51.
18. Callaway DW, Shapiro NI, Donnino MW, Baker C, Rosen CL. Serum lactate and BD as predictors of mortality in normotensive elderly blunt trauma patients. *The Journal of trauma*. 2009;66(4):1040-4.

19. Jung J, Eo E, Ahn K, Noh H, Cheon Y. Initial BD as predictors for mortality and transfusion requirement in the severe pediatric trauma except brain injury. *Pediatric emergency care*. 2009;25(9):579-81.
20. Lefering R. Development and validation of the revised injury severity classification score for severely injured patients. *European Journal of Trauma and Emergency Surgery*. 2009;35(5):437-47.
21. Siegel JH. The effect of associated injuries, blood loss, and oxygen debt on death and disability in blunt traumatic brain injury: the need for early physiologic predictors of severity. *Journal of neurotrauma*. 1995;12(4):579-90.
22. Joseph B, Pandit V, Aziz H, Kulvatunyou N, Zangbar B, Green DJ, Haider A, et al. Mild traumatic brain injury defined by Glasgow Coma Scale: Is it really mild? *Brain injury*. 2015;29(1):11-6.
23. Zehtabchi S, Sinert R, Soghoian S, Liu Y, Carmody K, Shah L, Kumar M, et al. Identifying traumatic brain injury in patients with isolated head trauma: are arterial lactate and BD as helpful as in polytrauma? *Emergency medicine journal : EMJ*. 2007;24(5):333-5.
24. Cohen MJ, Brohi K, Ganter MT, Manley GT, Mackersie RC, Pittet JF. Early coagulopathy after traumatic brain injury: the role of hypoperfusion and the protein C pathway. *The Journal of trauma*. 2007;63(6):1254-61; discussion 61-2.
25. Stewart CL, Holscher CM, Moore EE, Bronsert M, Moulton SL, Partrick DA, Bensard DD. BD correlates with mortality in pediatric abusive head trauma. *Journal of pediatric surgery*. 2013;48(10):2106-11.
26. Mutschler M, Nienaber U, Wafaisade A, Brockamp T, Probst C, Paffrath T, Bouillon B, et al. The impact of severe traumatic brain injury on a novel BD- based classification of hypovolemic shock. *Scandinavian journal of trauma, resuscitation and emergency medicine*. 2014;22:28.

Table 1: Demographic and clinical characteristics of the study population	
Variables (n)	Numbers (%) / Means (\pm SD) / Median (IQR)
Gender (108)	
Male	98 (90.7%)
Female	10 (9.3%)
Mean Age (108)	36.69 (\pm 17.65)
Median Delay to presentation (hours) (105)	2.50 (1.27-6.00)
Comorbids (108)	
IHD	12 (11.1%)
DM	15 (13.9%)
HTN	11 (10.2%)
Mechanism of injury (108)	
RTA (Pedestrian)	18 (16.7%)
RTA (Motor biker)	51 (47.2%)
RTA (Other)	8 (7.4%)
Fall	15 (13.9%)
Gunshot Injuries	11 (10.2%)
Others	5 (4.6%)
Pupils on arrival (100)	
BERL	52 (48.1%)
Anisocoric	44 (40.7%)
Fixed and dilated	4 (3.7%)
Mean GCS at presentation (107)	7.51 (\pm 3.05)
Mean RTS (107)	9.89 (\pm 1.22)
Epidural hematoma (96)	28 (25.9%)
IVH/SAH (96)	55 (50.9%)
Rotterdam CT Score (96)	3.95 (\pm 1.27)
Outcome	
Mean length of stay (days) (108)	17.72 (\pm 11.78)
Mean GOS on last follow-up (106)	2.90 (\pm 1.67)
Mortality (106)	35 (32.4%)

Table 2: Arterial blood gas parameters of the study population

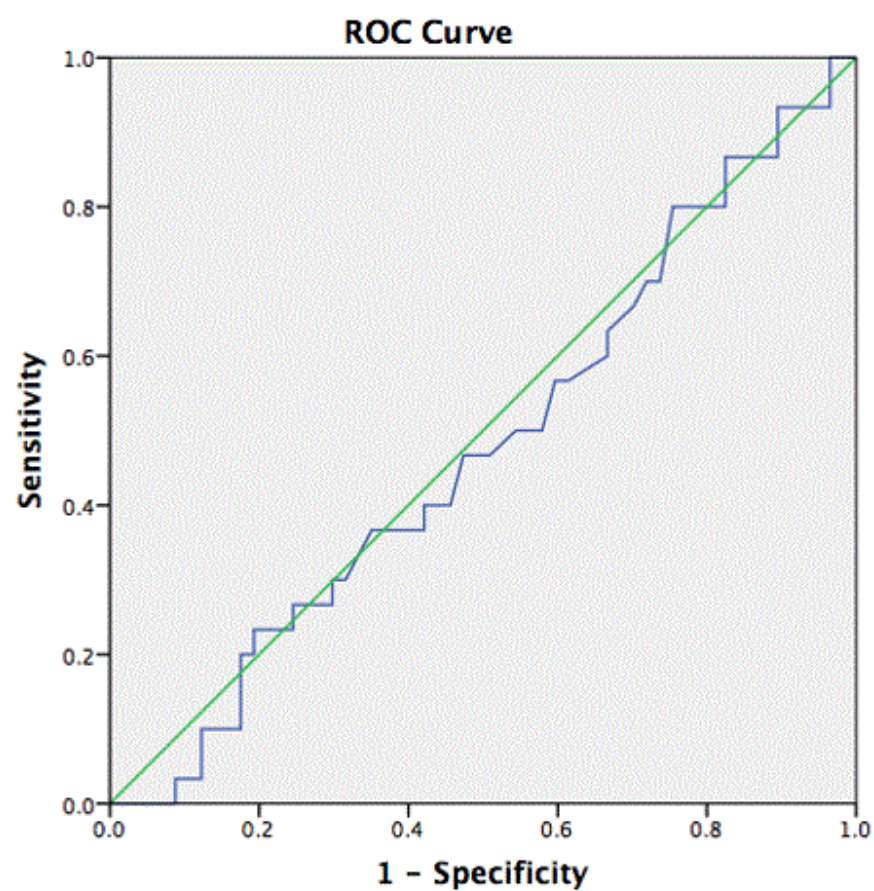
ABG Parameter	Mean (\pm SD)
Base Deficit (88)	5.69 (\pm 4.45)
Base Excess (20)	3.92 (\pm 4.33)
pH (108)	7.37 (\pm 0.13)
PCO ₂ (108)	35.23 (\pm 7.39)
pO ₂ (108)	237.97 (\pm 110.84)
O ₂ Sat (108)	98.22 (\pm 3.74)
Bicarbonate (108)	20.39 (\pm 5.01)

Table 3: Summary of results (one-way ANOVA)				
Variable (n)	Mean \pm SD (n)			p-value
	Pupils			
	BERL	Anisocoric	Fixed and Dilated	
Mean GCS on arrival (100)	9.1 \pm 3.03 (52)	5.57 \pm 1.82 (44)	7.75 \pm 2.87 (4)	p<0.001
Revised Trauma Score (99)	10.52 \pm 0.96 (52)	9.21 \pm 0.97(43)	8.75 \pm 1.89 (4)	p<0.001
Rotterdam CT Score (88)	3.75 \pm 1.22 (44)	4.20 \pm 1.35 (41)	4.33 \pm 1.15 (3)	p=0.252
GOS on last follow-up (98)	3.14 \pm 1.76 (50)	2.57 \pm 1.47 (44)	3.0 \pm 2.31 (4)	p=0.250
Base deficit (82)	6.60 \pm 5.63 (41)	5.54 \pm 3.17 (37)	6.15 \pm 3.43 (4)	p=0.878
Base excess (18)	5.48 \pm 5.29 (11)	1.57 \pm 1.37 (7)	n=0	p=0.076
	Cistern			
	Normal	Compressed	Absent	
Length of stay (96)	24.67 \pm 16.21 (18)	16.81 \pm 11.32 (53)	16.44 \pm 8.78 (25)	p=0.04
Mean GCS on arrival (95)	7.11 \pm 2.06 (18)	7.5 \pm 3.07 (52)	7.12 \pm 3.47 (25)	p=0.827
Revised Trauma Score (95)	9.89 \pm 0.9 (18)	9.94 \pm 1.20 (52)	9.56 \pm 1.45 (25)	p=0.429
GOS on last follow-up (95)	2.5 \pm 1.54 (18)	3.17 \pm 1.68 (52)	2.2 \pm 1.38 (25)	p=0.032
Base deficit (79)	4.38 \pm 2.99 (16)	5.65 \pm 4.44 (41)	6.80 \pm 5.60 (22)	p=0.277
Base excess (17)	0.1 \pm 0.14 (2)	3.12 \pm 2.21 (12)	4.06 \pm 5.83 (3)	p=0.340
	Midline shift			
	≤ 5	> 5		
Length of stay (96)	19.67 \pm 13.24 (39)	17.18 \pm 11.26 (57)		p=0.324
Mean GCS on arrival (95)	8 \pm 2.73 (39)	6.86 \pm 3.10 (56)		p=0.067
Revised Trauma score (95)	10 \pm 1.12 (39)	9.71 \pm 1.28 (56)		p=0.263
GOS on last follow-up (95)	3 \pm 1.71 (38)	2.65 \pm 1.56 (57)		p=0.305
Base deficit (79)	5.36 \pm 6.15 (32)	5.96 \pm 3.14 (47)		p=0.570
Base excess (17)	4.11 \pm 4.12 (7)	2.1 \pm 1.62 (10)		p=0.178
	Epidural			
	Present	Absent		
Length of stay (96)	18.29 \pm 11.60 (28)	18.15 \pm 12.38 (68)		p=0.960
Mean GCS on arrival (95)	6.78 \pm 2.74 (27)	7.54 \pm 3.08 (68)		p=0.263
Revised Trauma score (95)	9.54 \pm 1.17 (28)	9.96 \pm 1.22 (67)		p=0.126
GOS on last follow-up (95)	3.26 \pm 1.68 (27)	2.6 \pm 1.58 (68)		p=0.075
Base deficit (79)	7.57 \pm 6.70 (24)	4.90 \pm 2.99 (55)		p=0.016
Base excess (17)	1.58 \pm 1.41 (4)	3.35 \pm 3.25 (13)		p=0.314

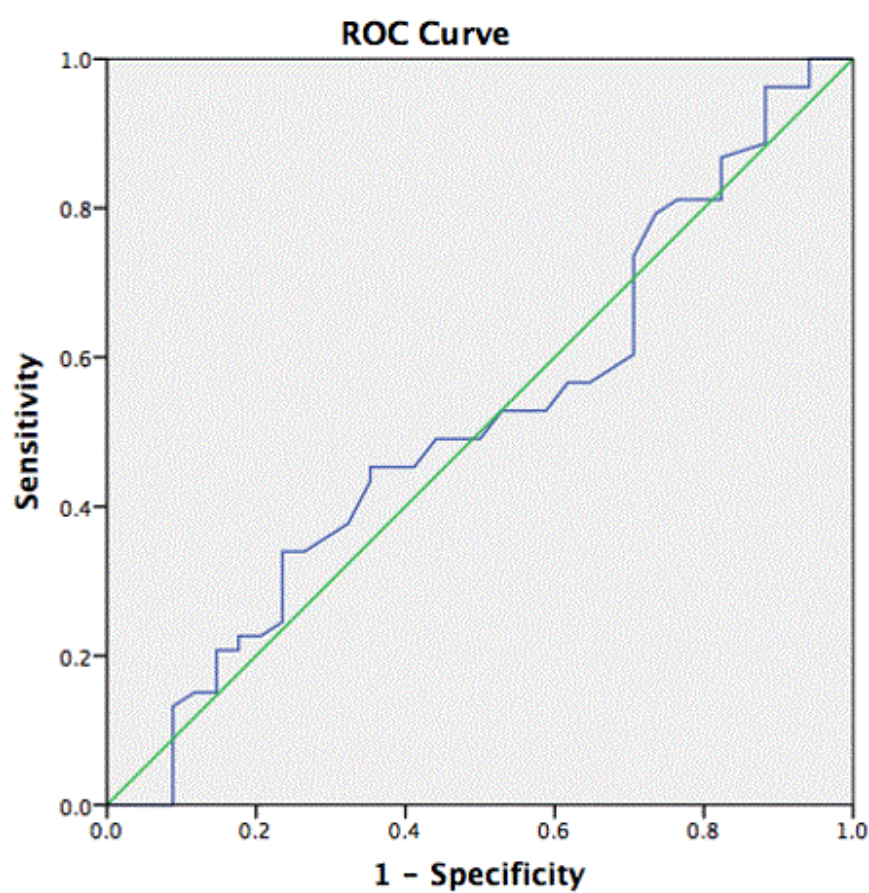
	IVH/SAH		
	Absent	Present	
Length of stay (96)	18.95 ± 11.91 (41)	17.62 ± 12.32 (55)	p=0.596
Mean GCS on arrival (95)	7.1 ± 2.38 (40)	7.49 ± 3.38 (55)	p=0.533
Revised Trauma score (95)	9.88 ± 0.87 (41)	9.8 ± 1.43 (54)	p=0.748
GOS on last follow-up (95)	3.18 ± 1.58 (40)	2.51 ± 1.61 (55)	p=0.048
Base deficit (79)	5.55 ± 4.78 (35)	5.85 ± 4.46 (44)	p=0.773
Base excess (17)	1.85 ± 1.36 (6)	3.52 ± 3.49 (11)	p=0.284
	Mortality		
	Alive	Expired	
Length of stay (106)	20.63 ± 10.00 (71)	12.37 ± 13.35 (35)	p=0.001
Mean GCS on arrival (105)	7.41 ± 2.86 (70)	7.37 ± 3.2 (35)	p=0.945
Revised trauma score (105)	9.84 ± 1.07 (70)	9.86 ± 1.42 (35)	p=0.954
ICU stay (106)	4.59 ± 3.78 (71)	6.09 ± 6.52 (35)	p=0.139
Rotterdam CT score (95)	3.83 ± 1.20 (63)	4.25 ± 1.34 (32)	p=0.121
Base deficit (87)	5.99 ± 5.00 (57)	5.26 ± 3.17 (30)	p=0.468
Base excess (19)	2.89 ± 3.21 (14)	4.12 ± 2.55 (5)	p=0.450
	Outcome		
	Favorable	Unfavorable	
Length of stay (106)	18.64 ± 9.24 (44)	17.39 ± 13.40 (62)	p=0.594
GCS on arrival (105)	7.79 ± 2.88 (43)	7.13 ± 3.01 (62)	p=0.262
Revised Trauma score (105)	9.93 ± 1.02 (44)	9.79 ± 1.31 (61)	p=0.541
ICU stay (106)	3.61 ± 3.17 (44)	6.13 ± 5.59 (62)	p=0.008
Rotterdam CT score (95)	3.64 ± 0.96 (36)	4.17 ± 1.38 (59)	p=0.046
Base deficit (87)	6.16 ± 5.86 (34)	5.47 ± 3.29 (53)	p=0.486
Base excess (19)	2.81 ± 3.41 (10)	3.66 ± 2.68 (9)	p=0.559

Figure 1. This figure is showing receiver operator curve (ROC) analysis for Base Deficit as a predictor of mortality with an area under the curve of 0.479 (95% CI 0.352-0.606)

Figure 2. Receiver operator curve (ROC) analysis for Base Deficit as a predictor of unfavorable outcomes is shown here. Area under the curve is 0.515 (95% CI 0.389.641)



Diagonal segments are produced by ties.



Diagonal segments are produced by ties.

Highlights of the Study

1. Despite its importance as a predictive marker in polytrauma patients, BD has not been evaluated as a prognostic indicator in patients with isolated head injury
2. Study is a critical analysis of 108 patients with severe head injury and base deficit
3. BD on admission had a statistically significant negative correlation with Glasgow Coma Scale (GCS) on presentation ($r=-0.239$, $p=0.025$) and Revised Trauma Score (RTS) ($r=-0.214$, $p=0.046$)
4. There was no statistically significant difference in means of BD between survivors and non survivors.
5. Area under receiver operator curve (ROC) for BD as a predictor of mortality statistically non-significant.