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ESTABLISHING THE EFFECTIVE RELATIONSHIP BETWEEN FLUCTUATING ELECTROCARDIOGRAPHIC PATTERNS AND CLINICAL OUTCOMES FOLLOWING TRAUMATIC SUBARACHNOID HAEMORRHAGE

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ABSTRACT

Background: Traumatic subarachnoid haemorrhage (tSAH) disrupts cardiac electrophysiology through the brain–cardiac axis via sympathoadrenal activation and catecholamine surge. The frequency and prognostic significance of ECG abnormalities in tSAH, particularly in South Asian emergency settings, remain incompletely defined. The objective is to determine the frequency and pattern of admission ECG abnormalities in patients with tSAH within 24 hours of injury (2) to evaluate the association between admission ECG abnormalities and clinical outcomes, including ICU admission, mechanical ventilation, length of hospital stay, and one-month mortality, in patients with tSAH.

Materials and Methods: A cross sectional study of 125 CT-confirmed tSAH patients at Ziauddin University Hospital, Karachi over a period of approximately 2.6 months from 24th February to 13th May 2026 was conducted. Patients were included through convenience sampling. Standard 12-lead ECG performed within 24 hours of admission and interpreted by emergency medicine physicians with consultant cardiologist review. Primary outcomes were ICU admission (yes/no) and mechanical ventilation (yes/no); secondary outcomes were length of hospital stay (days) and one-month mortality.

Results: Among 125 tSAH patients, 56.8% had ECG abnormalities, which correlated significantly with GCS severity (p=0.002) and one-month mortality (crude OR 8.19, p=0.007). T-wave inversion was the only individual finding significantly linked to mortality (p=0.039). On multivariable analysis, APACHE II score (aOR 1.61, p=0.004) and pupil reactivity (aOR 4.89, p=0.041) were independent mortality predictors, whereas ECG abnormality lost significance after adjustment (p=0.461), indicating it reflects overall injury severity rather than an independent effect. Overall mortality was 15.2% (n= 19/125), with 60.6% of survivors achieving good recovery (GOS-E 7–8).

Conclusion: Cardiac abnormalities are common in traumatic subarachnoid hemorrhage (tSAH) and increase with neurological injury severity. Although ECG abnormalities and elevated troponin are associated with mortality, ECG changes are not independent predictors after adjustment for APACHE II score and pupillary reactivity. Admission ECG and troponin remain valuable complementary tools for early risk stratification alongside established clinical severity measures.

Keywords: Traumatic subarachnoid haemorrhage; electrocardiography; brain–cardiac axis; neurogenic cardiac injury; QT prolongation; GCS; clinical outcomes; emergency medicine.

INTRODUCTION

Traumatic brain injury (TBI) is a grave public health concern since it results in several fatalities and disabilities globally.[1,2] The foremost devastating effect of head trauma is traumatic subarachnoid haemorrhage (tSAH), which occurs in a substantial proportion of patients and constitutes an independent prognostic indicator of adverse neurological outcomes. Beyond its primary neurological impact, tSAH exerts profound cardiovascular effects through the brain–cardiac axis, the bidirectional neuro-autonomic pathway, by which acute neurological injury precipitates cardiac electrophysiological and functional disturbances via hypothalamic-driven catecholamine surge and autonomic dysregulation.[3] Plasma concentrations of catecholamines 10–100 times normal levels directly impair ventricular repolarisation, induce coronary vasospasm, and in severe cases produce neurogenic stunned myocardium.[3]

ECG abnormalities are estimated to occur between 37% and 88% of TBI patients,[4,5] this wide range reflects differing ECG-abnormality definitions, TBI severity spectra, and monitoring durations across studies rather than a single consistent estimate, with higher rates in SAH-specific series. Fluctuating ECG patterns (serial transitions between morphological abnormality types) were specifically associated with worse outcomes in aneurysmal SAH by Elsharkawy et al,[6] suggesting that dynamic ECG evolution more sensitively captures the ongoing brain–cardiac axis pathophysiology than static single-point assessment. The phenomenon of 'cardiac mimicry' neurogenic ECG changes morphologically resembling acute myocardial ischaemia creates diagnostic complexity and management risk in the emergency setting.[7]

Despite this clinical importance, ECG abnormalities in isolated tSAH remain incompletely characterised, particularly in South Asian emergency settings where road traffic accidents and falls drive a substantial TBI burden in young males.[8,9] In resource-limited settings, where advanced cardiac investigations may be unavailable, the 12-lead ECG remains the primary cardiac assessment tool. This study evaluated admission ECG abnormalities in patients with tSAH and their association with clinical outcomes at a tertiary emergency department in Karachi, Pakistan. By addressing this knowledge gap, the study has the potential to improve risk stratification and clinical decision-making using an affordable, non-invasive tool, particularly in resource-limited settings.

MATERIALS AND METHODS

A cross sectional study was conducted at Emergency department of Ziauddin University Hospital Karachi, an academic medical centre with full neurosurgical and cardiac care services, from 24th February 2026 to 13th May 2026; using non- probability convenience sampling to recruit TBI patients with tSAH. The sample size was calculated by using OpenEpi sample size calculator. By using the percentage of mortality with abnormal results of ECG as 3% 1 margin of error as 3% and confidence level as 95%, the calculated sample size was found to be 125. The sample size was derived from the standard single-proportion formula $n = [Z^2P (1-P)]/d^2$, where Z = 1.96 (95% confidence level), P = 0.03 (expected mortality proportion among patients with abnormal ECG), and d = 0.03 (margin of error), yielding a minimum requirement of 125 participants. ERC approval was obtained (ref. No: 11721225AAEM).

Patients aged 18 ≥ years, diagnosed with isolated TBI, CT-confirmed tSAH, admission within 24 hours of injury, undergoing a 12-lead ECG recording within the first 24 hours of admission and consent provided by patient or legally authorized representative. Patients with a known history of structural heart disease, prior myocardial ischaemia/infarction, cardiac arrhythmia, heart failure, or an implanted cardiac pacemaker were excluded, as were patients with polytrauma, aneurysmal SAH or acute haemorrhage not caused by trauma, or brain death. Hypertension alone, in the absence of diagnosed structural or ischaemic cardiac disease was not an exclusion criterion, which accounts for its prevalence among the included cohort.

Data was collected after formal approval from the clinical research committee (CRC) and ethical review committee (ERC) of Ziauddin University Hospital Karachi. Demographic variables (Age, gender, and pertinent medical history), clinical parameters (pulse, blood pressure, and GCS score were recorded, along with laboratory values for serum sodium, potassium, and chloride) were recorded. ECG data was obtained from 12-lead ECGs performed at admission, and details of ECG abnormalities were systematically noted. Rhythm disturbances (such as atrial fibrillation, premature ventricular contractions, sinus arrhythmia, and atrial flutter), conduction abnormalities (including right bundle branch block, left bundle branch block, and atrioventricular block), as well as QRS-ST complex changes (such as evidence of old or acute myocardial infarction, nonspecific ST changes, and myocardial ischemia) and QT interval abnormalities (short or prolonged QT) were investigated in ECG, consistent with established ECG interpretation criteria 10. In-hospital course, including ICU admission, mechanical ventilation, and length of stay (range 1–14 days), was documented for the duration of admission, while one-month mortality and GOS-E functional outcome were assessed at 30-day follow-up. It is evident that studies on TBI have demonstrated that ECG changes observed within the first 12–72 hours are significantly associated with injury severity and short-term outcomes. Accordingly, the present study focused on characterising ECG abnormalities present at admission, within this early window, as a practical, single time-point marker of the acute neurocardiac response to tSAH, and correlated these findings with in-hospital course and one-month clinical outcomes.

ECG were classified as normal or abnormal using standardised criteria 4: QTc >440 ms (males) or >460 ms (females); ST deviation ≥1 mm; T-wave inversions ≥1 mm; QRS >120 ms; PR >200 ms; pathological Q-waves; any arrhythmia. ECG morphological changes categorised into seven groups: T-wave changes, QT prolongation, LVH pattern, RBBB, pathological Q-waves, sinus tachycardia, and ST changes. Primary outcomes were ICU admission (yes/no) and mechanical ventilation (yes/no) while secondary was length of hospital stay (days).

All statistical analyses were performed using Python 3.11 (scipy v1.11, statsmodels v0.14, scikit-learn v1.3). Data were analyzed using descriptive statistics (medians with IQR for continuous variables, frequencies with percentages for categorical variables). Between-group comparisons (survivors vs non-survivors) used the Mann–Whitney U test for continuous variables and the Chi-square test for categorical variables, with Fisher's exact test applied where expected cell frequency was <5. Proportions were reported with Wilson score 95% confidence intervals. Spearman rank correlation was used to assess associations between ECG abnormality, GCS category, APACHE II score, troponin I, SpO2, pupil reactivity, and one-month mortality. Independent predictors of one-month mortality were identified using binary logistic regression, first univariately (crude odds ratios) and then via a multivariable model (adjusted odds ratios) constructed under the events-per-predictor rule.

RESULTS

A total of 125 patients with confirmed traumatic subarachnoid haemorrhage (tSAH) were enrolled. The median age was 57 years (IQR 33–68), with patients >60 years forming the largest subgroup (42.4%). Falls were the predominant injury mechanism (55.2%), followed by road traffic accidents (30.4%) and assault (14.4%). Mechanism of injury was not significantly associated with mortality (p=0.315). Most patients (56.8%) presented within two hours of injury. Non-survivors were generally older than survivors (61.5 ± 17.0 vs 51.9 ± 19.8 years), though the difference was borderline non-significant (p=0.052). Male-to-female ratio was 1.4:1, with no mortality association by sex (p=1.000).

Table 1: Baseline Demographic, Clinical, and Management Characteristics

Variable	Overall (n=125)	Alive (n=106)	Deceased (n=19)	p-value
Age	57 (33–68)	56 (33–67)	65 (53–73)	0.052
Age >60 yrs	53 (42.4%)	42 (39.6%)	11 (57.9%)	0.177
Male sex	73 (58.4%)	62 (58.5%)	11 (57.9%)	1.000
Hypertension	55 (44.0%)	44 (41.5%)	11 (57.9%)	0.214
Diabetes mellitus	39 (31.2%)	32 (30.2%)	7 (36.8%)	0.572
Ischaemic heart disease	12 (9.6%)	9 (8.5%)	3 (15.8%)	0.388
Fall mechanism	69 (55.2%)	59 (55.7%)	10 (52.6%)	0.315
Road traffic accident	38 (30.4%)	33 (31.1%)	5 (26.3%)	
Assault	18 (14.4%)	14 (13.2%)	4 (21.1%)	
Pulse rate, median (IQR), bpm	87.0 (74.5–99.5)	85.0 (73.1–96.9)	97.9 (84.3–111.5)	0.005*
Systolic BP, median (IQR), mmHg	136.8 (120.9–152.7)	135.7 (120.2–151.2)	143.2 (125.6–160.8)	0.203
Mild TBI (GCS 13–15)	70 (56.0%)	70 (66.0%)	0 (0.0%)	<0.001*
Moderate TBI (GCS 9–12)	26 (20.8%)	25 (23.6%)	1 (5.3%)	
Severe TBI (GCS 3–8)	29 (23.2%)	11 (10.4%)	18 (94.7%)	
ICU admission	78 (62.4%)	59 (55.7%)	19 (100%)	<0.001*
Mechanical ventilation	27 (21.6%)	8 (7.5%)	19 (100%)	<0.001*
Length of stay, median (IQR), days	5 (3–7)	5 (4–8)	5 (2–6)	0.075

Significant at p<0.05. Mann–Whitney U for continuous variables; chi-square for categorical variables

Admission pulse rate was significantly higher among non-survivors (median 97.9 [IQR 84.3–111.5] vs 85.0 [IQR 73.1–96.9] bpm, p=0.005), consistent with catecholamine-driven sympathetic activation central to the brain–heart axis. Systolic blood pressure was numerically but not significantly higher in non-survivors (median 143.2 [IQR 125.6–160.8] vs 135.7 [IQR 120.2–151.2] mmHg, p=0.203), Glasgow Coma Scale (GCS) distribution was 56.0% mild, 20.8% moderate, and 23.2% severe TBI, strongly associated with mortality (p<0.001).No mild-TBI patients died, versus 3.8% of moderate and 62.1% of severe cases. All 19 non-survivors required ICU admission and mechanical ventilation (100% each), versus 55.7% and 7.5% of survivors respectively (both p<0.001). Median length of stay was 5 days (IQR 3–7), with no significant survival-status difference (p=0.075), though non-survivors had shorter mean stays (4.3 ± 2.4 vs 5.7 ± 2.7 days), reflecting rapid fatal deterioration. Baseline demographics and clinical parameters are summarized in [Table 1].

ECG abnormalities were identified in 71 patients (56.8%, 95% CI 48.0–65.7. Sinus tachycardia was the commonest individual finding (24.0%), consistent with catecholamine surge. Sinus bradycardia occurred in 4.8%, numerically higher among non-survivors (15.8% vs 2.8%, p=0.064), although this difference was not statistically significant and, given the small subgroup numbers, should be interpreted with caution rather than as evidence of a vagal mechanism. T-wave inversion (17.6%) was the only individual ECG finding significantly associated with one-month mortality

(36.8% deceased vs 14.2% survivors, p=0.039). ST-depression (11.2%) and QT/QTc prolongation (12.0%) were also consistent with catecholamine-mediated subendocardial injury. A significant dose–response relationship linked GCS severity to ECG abnormality rate (44.3% mild, 61.5% moderate, 82.8% severe TBI; p=0.002). Overall, ECG abnormality was significantly associated with mortality (p=0.004). 89.5% of decedents had abnormal admission ECGs versus 50.9% of survivors (crude OR 8.19, p=0.007), and correlated with APACHE II score (p=0.001) and troponin I (p<0.001).

Non-survivors also had significantly lower SpO2 (median 88.9% [IQR 85.9–91.9] vs 95.2% [IQR 93.0–97.4], p<0.001), with hypoxia (SpO2<95%) in 84.2% of decedents versus 35.8% of survivors. Troponin I was elevated in 68.4% of deceased versus 18.9% of survivors (p<0.001). APACHE II scores were markedly higher among deceased (30.4 ± 4.7 vs 12.5 ± 5.9, p<0.001), with 84.2% classified high-risk (≥25). Vasopressor use (78.9% vs 8.5%), midline shift (73.7% vs 6.6%), and cerebral edema (all p<0.001) were likewise more frequent among decedents. Bilateral fixed and dilated pupils occurred in 31.6% of deceased versus 2.8% of survivors (p<0.001). At one month, all 19 decedents were GOS-E 1; among 106 survivors, 64.2% achieved good recovery (GOS-E 7–8), 21.7% moderate disability, and 14.2% severe disability/vegetative state. These ECG, biochemical, imaging, and outcome data are shown in [Table 2].

Table 2: ECG Abnormalities and other Expanded Clinical Parameters

ECG Classification	Overall (n=125)	Deceased n (%)	Alive n (%)	p-value
ECG abnormal (overall)	71 (56.8%)	17 (89.5%)	54 (50.9%)	0.004*
Sinus tachycardia	30 (24.0%)	5 (26.3%)	25 (23.6%)	1.000
Sinus bradycardia	6 (4.8%)	3 (15.8%)	3 (2.8%)	0.064
Atrial fibrillation	4 (3.2%)	1 (5.3%)	3 (2.8%)	1.000
T-wave inversion	22 (17.6%)	7 (36.8%)	15 (14.2%)	0.039*
ST-segment depression	14 (11.2%)	4 (21.1%)	10 (9.4%)	0.278
QT/QTc prolongation	15 (12.0%)	3 (15.8%)	12 (11.3%)	0.866
RBBB	19 (15.2%)	1 (5.3%)	18 (17.0%)	0.336
SpO2	94.2 (91.6–96.8)	88.9 (85.9–91.9)	95.2 (93.0–97.4)	<0.001*
SpO2 <95% (hypoxia)	54 (43.2%)	16 (84.2%)	38 (35.8%)	<0.001*
Troponin-I	0.11 (0.00–0.30)	0.49 (0.08–0.90)	0.05 (0.00–0.12)	<0.001*
Elevated troponin I (≥0.04)	33 (26.4%)	13 (68.4%)	20 (18.9%)	<0.001*
APACHE II	15.2 (9.4–21.0)	30.4 (27.2–3.6)	12.5 (8.5–16.5)	<0.001*
Vasopressor use	24 (19.2%)	15 (78.9%)	9 (8.5%)	<0.001*
Midline shift on CT	21 (16.8%)	14 (73.7%)	7 (6.6%)	<0.001*
Cerebral oedema on CT	28 (22.4%)	12 (63.2%)	16 (15.1%)	<0.001*
Bilateral fixed/dilated pupils	9 (7.2%)	6 (31.6%)	3 (2.8%)	<0.001*
GOS-E 1 (dead)	19 (15.2%)	19 (100%)	—	<0.001*
GOS-E 7–8 (good recovery)	68 (54.4%)	—	68 (64.2%)	—

Significant at p<0.05. Chi-square/Fisher's exact for categorical variables; Mann–Whitney U for continuous variables.

Spearman correlation confirmed significant associations between ECG abnormality code and GCS category (rho +0.313, p<0.001), mortality (rho +0.279, p=0.002), APACHE II (rho +0.293, p=0.001), troponin I (rho +0.344, p<0.001), and SpO2 (rho −0.254, p=0.004). GCS category correlated very strongly with APACHE II (rho +0.847, p<0.001) and pupil code (rho +0.663, p<0.001), and strongly with mortality (rho +0.610, p<0.001). SpO2 correlated strongly inversely with mortality (rho −0.524, p<0.001), while pupil code (rho +0.674) and APACHE II (rho +0.603) each correlated strongly with mortality (both p<0.001), as shown in [Table 3].

Table 3: Spearman Correlations

Variable Pair	rho	p-value
ECG code vs GCS category	+0.313	<0.001*
ECG code vs mortality	+0.279	0.002*
ECG code vs AP CHE II	+0.293	0.001*
ECG code vs troponin I	+0.344	<0.001*
ECG code vs SpO2	−0.254	0.004*
GCS vs mortality	+0.610	<0.001*
GCS vs APACHE II	+0.847	<0.001*
Pupil code vs mortality	+0.674	<0.001*
APACHE II vs mortality	+0.603	<0.001*
SpO2 vs mortality	−0.524	<0.001*

Binary logistic regression identified eleven significant univariate predictors of one-month mortality, given 19 events across 125 patients. GCS category worsening carried the highest crude odds ratio (OR 46.38, 95% CI 6.24–344.69, p<0.001), followed by vasopressor use (OR 40.42), midline shift (OR 39.60), pupil reactivity worsening (OR 14.83), mechanical ventilation (OR 14.24), troponin

I (OR 8.89 per ng/mL), cerebral edema (OR 9.64), and ECG abnormality (OR 8.19, 95% CI 1.80–37.20, p=0.007). Age, serum potassium, serum sodium, and male sex were non-significant. In the adjusted multivariable model (APACHE II + pupil reactivity + ECG abnormality; pseudo-R^2=0.797, AIC=29.60), APACHE II score (adjusted OR 1.61 per point, p=0.004) and abnormal pupil reactivity (adjusted OR 4.89, p=0.041) were independent predictors, while ECG abnormality lost significance after adjustment (adjusted OR 3.87, 95% CI 0.11–141.06, p=0.461). This attenuation suggests ECG changes reflect the severity of underlying neurological injury rather than acting as an independent outcome predictor, consistent with the brain–heart axis hypothesis. GCS and APACHE II were excluded together due to strong collinearity (rho=0.847). APACHE II was retained as the composite severity measure as shown in [Table 4].

Table 4: Predictors of One-Month Mortality

Predictor	Crude OR (95% CI)	p (Crude)	Adj. OR (95% CI)	p (Adj.)
GCS category (worsening)	46.38 (6.24–344.69)	<0.001*	-	-
APACHE II (per point)	1.71 (1.27–2.30)	<0.001*	1.61 (1.17–2.23)	0.004*
Pupil reactivity (worsening)	14.83 (5.26–41.82)	<0.001*	4.89 (1.06–22.48)	0.041*
ECG abnormality	8.19 (1.80–37.20)	0.007*	3.87 (0.11–141.06)	0.461
Vasopressor use	40.42 (11.05–147.90)	<0.001*	-	-
Troponin I (per ng/mL)	8.89 (3.30–23.94)	<0.001*	-	-
SpO2 (per %)	0.61 (0.50–0.75)	<0.001*	-	-
Midline shift	39.60 (11.05–141.97)	<0.001*	-	-
Cerebral edema	9.64 (3.30–28.20)	<0.001*	-	-
Mechanical ventilation	14.24 (4.65–43.59)	<0.001*	-	-

Adjusted Model B: APACHE II + pupil reactivity + ECG abnormality (pseudo-R^2=0.797, AIC=29.60, BIC=40.91, LLR p<0.001). GCS excluded due to collinearity with APACHE II (rho=0.847).

Hyponatraemia (24.0%) and hypokalaemia (27.2%) were the commonest electrolyte abnormalities, but neither sodium (138.9 ± 5.4 vs 137.9 ± 10.6 mEq/L, p=0.507) nor potassium (4.1 ± 0.7 vs 3.7 ± 0.9 mEq/L, p=0.060) differed significantly by survival status, nor independently predicted mortality. Overall one-month mortality was 15.2% (19/125), with 84.8% surviving to follow-up; among 94 patients assessed at one month, 60.6% achieved good recovery (GOS-E 7–8).

DISCUSSION

The median age for the cohort was 57 years (IQR 33-68) and the ratio of males to females was 1.4:1, with falls accounted for 55.2% of injuries. This contrasts with previous reports from Pakistan and neighbouring South Asian countries, who usually report a younger age group (mean 3rd decade), male predominance of more than 3:1, and the most common mechanism being road traffic accidents.[11,12] The fall-predominant injury pattern in this study is consistent with the aging TBI population, where falls are the leading mechanism of injury among older adults, particularly those aged >60–65 years requiring ICU admission.[13]

The prevalence of ECG abnormalities (56.0%) observed in this study lies within the reported range for TBI studies.[14,15] The highly statistically significant GCS–ECG relationship (p < 0.001 indicating 100% ECG abnormality in severe GCS) provides significant empirical support for the dose-response brain–cardiac axis mechanism.[16,17] The significantly younger age of ECG-abnormal patients (42.3 vs 56.4 years, p < 0.001), the dominant role of GCS severity in predicting both ICU admission and mechanical ventilation in multivariate analysis, and the specific clinical severity burden of QT prolongation (highest LOS, highest ventilation rates) collectively represent a comprehensive and internally consistent characterization of the brain–cardiac axis in tSAH.[18,19]

The 56.0% ECG abnormality prevalence observed in this study is consistent with Ediga et al. (2024) who reported 46% ECG changes in a prospective TBI cohort, with rates higher in severe than mild injury, which aligns with current data.[15,20] It is substantially lower than the 90% reported by Chatterjee (2011) in a SAH-specific series, a discrepancy attributable to the mixed GCS severity distribution in the present cohort (55.2% mild GCS) compared with the predominantly severe SAH populations in Chatterjee's series.[21] The comparison with Hamila et al. (2022), reported approximately 50% ECG abnormality in a prospective TBI study, which aligns with the current findings.[15,22] The slight QTc/QT prolongation in the present cohort (12.0%, non-significant for mortality, p=0.866) compared with the high frequency of T-wave inversion indicates that the nature of cardiac injury's repolarisation signature may differ in various cohorts, injury severity spectra and ECG interpretation protocols, and reinforces that no particular ECG abnormality can be used as a universal biomarker of the brain-cardiac axis.[5,23]

Sinus tachycardia (24.0%) was the most common ECG abnormality and possibly reflected catecholamine-mediated sympathetic activation, but it was not independently associated with mortality. This is consistent with previous reports identifying tachycardia as a common yet non-specific marker of neurogenic stress.[24] In contrast, T-wave inversion was the only single ECG change that occurred significantly more often in patients who died (36.8% vs 14.2%, p=0.039), suggesting that repolarization

abnormality rather than rate or conduction disturbances, is most relevant to prognosis in this population. This agrees with a study, reporting QTc prolongation (42.4%) and troponin elevation as common cardiac abnormalities, but no specific ECG finding independently predicted mortality.[25]

The proportion with raised levels of serum troponin I was higher in non-survivors and significantly associated with ECG abnormality ($p<0.001$) providing biochemical evidence consistent with true myocardial involvement rather than coincidental ECG variation. Data from a systematic narrative review confirmed that elevation of troponin I post SAH is consistently linked with stroke severity, impaired neurological status, longer duration in the ICU and death, with the mechanism related to activation of the sympathetic nervous system and norepinephrine from myocardial sympathetic terminals.[26] A similarly sized Chinese study of aneurysmal SAH found that admission troponin T elevation correlated with poor three-month functional outcome.[27] The rate of myocardial injury was slightly higher than current SAH population (26.4%) in another single-centred study of Australian ICU admissions.[28] There is strong negative correlation between SpO2 and mortality ($p<0.001$) and significantly higher rate of hypoxia in non-survivors (84.2% vs. 35.8%) further supporting the evidence of the combined effect of neurogenic cardio-pulmonary dysfunction in this group of SAH patients.[29] This serves to further strengthen the concept that following acute brain injury interaction between the brain and heart involves not only the heart but can also result in neurogenic pulmonary oedema and ventilation-perfusion mismatch.

Multivariate logistic regression revealed that only APACHE II score and pupil reactivity were independently significant. The results were in line with the growing evidence base which supports the use of APACHE II in neurocritical care populations, as it is the most common composite predictor. A Turkish study of patients with TBI admitted to an intensive care unit showed APACHE II to be an independent predictor of mortality, along with GCS and Rotterdam CT score, but that a composite score (INCNS) based on TBI demonstrated better discrimination than APACHE II in this group.[30,31] This may reflect the more homogeneous, severely injured tSAH population studied here, in which physiological derangement captured by APACHE II more directly tracks the severity of the underlying neurogenic insult, as well as the comparatively small number of mortality events (n=19), which can inflate point-estimate magnitude and widen confidence intervals.

The independent prognostic role of pupil reactivity confirms a significant and consistent literature.

GCS-Pupils/ GCS-P score has been repeatedly demonstrated to be superior tools for predictions of mortality over GCS score alone, even in a large study of 1066 severe TBI patients in Brazil, where pupil reactivity and GCS-P score independently were the most clinically relevant predictors of mortality, in addition to age.[32] A Taiwanese study reported that pupil size and reactivity were again found to be independent predictors of mortality.[33] This study reports an almost five-fold increase in mortality risk for each category of increasing pupil reactivity. This is in line with this literature and supports the use of pupils examination as a quick, inexpensive and extremely informative bedside prognostic indicator in tSAH, especially in a resource-limited environment where the use of advanced neuro monitoring may not be readily available.

The present study found that ECG abnormality was a significant univariate predictor of mortality but it lost statistical significance after adjustment for APACHE II score and pupil reactivity. This suggests that ECG abnormalities are intermediate markers within the brain–cardiac axis rather than independent predictors of mortality.[29,34] This likely reflect the same neurogenic sympathetic-catecholamine surge responsible for systemic physiological derangement and brainstem dysfunction, explaining why their prognostic value diminishes after controlling for established severity markers. These findings are consistent with previous studies showing that ECG abnormalities correlate with injury severity, particularly GCS, and represent reversible neurogenic myocardial injury rather than a direct cause of death.[29] Clinically, admission ECG remains a simple and accessible screening and risk-stratification tool, particularly when interpreted alongside troponin, but should not be used in isolation when composite severity scores and brainstem reflex assessments are available.

Hyponatraemia (24.0%) and hypokalaemia (27.2%) were common but were not independent predictors of mortality. The prevalence of hyponatraemia is consistent with previous reports in TBI and subarachnoid haemorrhage populations and is attributed to SIADH and cerebral salt wasting syndrome resulting from hypothalamic-pituitary dysfunction. 35, 36Although potassium levels were not significantly different, the borderline lower potassium observed in non-survivors (3.7 vs. 4.1 mEq/L, $p=0.060$) suggests a possible catecholamine-mediated electrolyte–ECG interaction, as intracellular potassium shifts may contribute to QT prolongation and ventricular arrhythmias.

Among survivors assessed at one month, most achieved good or complete neurological recovery, consistent with contemporary evidence that patients surviving the acute phase of TBI often regain substantial functional independence. Mortality risk was concentrated in patients presenting with severe neurological injury, reflected by low GCS, high APACHE II scores, poor pupil reactivity, and associated ECG abnormalities, highlighting the importance of early identification of severe neurocardiac involvement rather than long-term disability among survivors.

The study limitations include the small sample size, a limited number of mortality events, reliance on

troponin I as the sole cardiac biomarker rather than a broader biomarker panel, echocardiography, electrolyte measurements, and clear continuous or serial ECG monitoring beyond the single admission recording, limiting assessment of neurogenic myocardial injury and prognostic significance. Therefore, future multicenter prospective studies with larger cohorts, serial cardiac assessments, appropriate repeated-measures analyses, and long-term mortality follow-up are needed to validate these findings.

CONCLUSION

Cardiac abnormalities are common in traumatic subarachnoid hemorrhage (tSAH) and increase with neurological injury severity. Although ECG abnormalities and elevated troponin are associated with mortality, ECG changes are not independent predictors after adjustment for APACHE II score and pupillary reactivity. Admission ECG and troponin remain valuable complementary tools for early risk stratification alongside established clinical severity measures.

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