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ORIGINAL ARTICLE

# Spontaneous intracranial haemorrhage in children—intensive care needs and predictors of in-hospital mortality: a 10-year single-centre experience

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## Abstract

**Purpose** Spontaneous intracranial haemorrhage (SICH) in children, although uncommon, is associated with significant mortality and morbidity. Paediatric data is however limited.

**Material and methods** Case records of 105 children with SICH, > 1 month to 12 years, admitted to a tertiary level PICU of a teaching and referral hospital between January 2009 and May 2018 were analysed retrospectively. In-hospital mortality was the primary outcome. Variables between survivors and non-survivors were compared to determine predictors of mortality.

**Results** The median (IQR) age of subjects was 6 (2.25, 70) months. Common clinical features were altered sensorium ($n = 87$, 82.9%), seizures ($n = 73$, 69.5%), pallor ($n = 66$, 62.9%) and bulging anterior fontanelle ($n = 52$, 49.5%). Median (IQR) Glasgow Coma Scale (GCS) at admission was 10 (6, 13) with herniation noted in 27 (25.7%) children. Vitamin K deficiency bleeding (VKDB) and arteriovenous malformation (AVM) were the most common etiology for bleeding among infants and older children respectively. The most common site of bleeding was intracerebral ($n = 47$, 44.8%) followed by subdural ($n = 26$; 24.8%). Sixteen (15.2%) children died during hospital stay. On univariate analysis, GCS < 8, Pediatric Risk of Mortality score (PRISM III) > 20, need for intubation, thiopentone coma for refractory intracranial pressure (ICP) and progression to shock and acute kidney injury (AKI) predicted mortality. Seizures were favourably associated with survival. Age, site of bleeding, etiology or type of management for raised ICP (conservative versus decompressive craniectomy) did not affect the outcome. On multivariable analysis, progression to AKI (OR 5.86; 95% CI, 1.53–22.4; $p$ 0.01) predicted poor outcome. Seizures, however, were associated with better odds for survival (OR 0.12; 95% CI, 0.03–0.47; $p$ 0.002).

**Conclusions** VKDB and AVM were the common etiologies among infants and older children respectively. Age, site, etiology of bleeding and type of management did not affect outcome. Severe decompensation at presentation, thiopentone for refractory ICP and progression to multiorgan dysfunction determined mortality.

**Keywords** Spontaneous intracranial bleeding · Intracerebral haemorrhage · Paediatric · Non-traumatic coma · Decompressive craniectomy · Vitamin K deficiency bleeding

## Introduction

Spontaneous intracranial haemorrhage (SICH) is defined as spontaneous non-traumatic pathological extravasation and accumulation of blood inside the cranial cavity. It is a common cause of acute brain injury in children leading to significant mortality and morbidity [1]. Incidence of SICH has been estimated to be 12–15 per 100,000 persons annually. Incidence in children is about 1.1–4.5 per 100,000 and associated with more than 50% mortality [1–4]. In children, SICH is seen more often in the foetal and neonatal brain than at other time points of development owing to the structural, physiological

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and hemodynamic differences in brain circulation. The etiopathogenesis, clinical course and interventions needed across various age groups are different from adults. About 50% of paediatric strokes are haemorrhagic, compared with 10–30% of adult strokes which are haemorrhagic [2, 5]. Risk factors such as diabetes, hypertension and smoking predisposing adults to cerebrovascular accidents are uncommon in children. Data on SICH, secondary to specific entities such as arteriovenous malformations (AVM), aneurysms, inherited coagulation disorders, Moyamoya disease, cortical sinus venous thrombosis (CSVT), cavernomas, vasculitis and brain tumours are available [6–9]. Similarly bleeding due to acquired coagulopathy secondary to Vitamin K deficiency or other medications are also reported in literature [10–12]. However, comprehensive and composite clinical data and structured management guidelines related to paediatric SICH as a whole are lacking [13, 14]. Over the last few years, adult data has shown that better neurological monitoring, protocol-based approach for raised intracranial pressure (ICP) and early surgical interventions have resulted in decreased mortality [5].

Given the high mortality (33%) and morbidity (permanent neurodeficits 40%) of paediatric SICH [15–17], a protocol-based approach with focus on early diagnosis and management is necessary. The first step in this direction is to generate data regarding clinical profile, risk factors, critical care needs, neuroradiology, outcome and predictors of outcome in children with SICH. This data will help us outline a structured approach to diagnosis and management of these children when they present to an emergency room.

## Materials and methods

This was a retrospective case series of children with SICH admitted consecutively to the pediatric intensive care unit (PICU) of a tertiary care teaching and referral hospital in North India over a 10-year period from January 1, 2009 through May 31, 2018. Medical records of subjects aged more than 1 month to 12 years, coded as per International Classification of Diseases (ICD) for a diagnosis of non-traumatic ICH and further confirmed by radiology, were retrieved. Neonatal ICH, silent or asymptomatic haemorrhage, posttraumatic, neurosurgical procedure-related haemorrhage and tumour apoplexy were excluded. Data with respect to demography, clinical characteristics, etiology of bleeding, severity of illness scoring [Pediatric Risk of Mortality (PRISM III) score] [18], haematological and biochemical parameters at admission, treatment, categorisation of acute kidney injury (AKI) based on pRIFLE criteria [19] (Supplementary Table 1), complications and outcome at discharge were accessed from the PICU Electronic Database and recorded in a predesigned case record form for analysis. Ethical clearance from our institute was obtained for the study.

All subjects underwent computed tomography (CT) of the brain, followed by MRI in those where the diagnosis was unclear. The site of bleeding was classified into intraparenchymal, subdural, extradural, subarachnoid, intraventricular, cerebellar and multisite bleed. All children with raised ICP were managed as per the standard unit protocol [20]. Surgical decompression (burr hole drainage or decompressive craniectomy) was undertaken in those with radiological evidence of focal space occupying hematoma with midline shift (> 5 mm), persistent clinical signs of raised ICP or GCS < 8 despite medical measures. ICP monitoring was not done in our children. Postprocedure, all children were monitored in PICU. The primary outcome was defined as in-hospital mortality. Variables among survivors and non-survivors were compared to identify the predictors of mortality.

## Statistical analysis

Continuous variables are expressed as mean ± standard deviation (SD) for normal and as a median and interquartile range (IQR) for skewed distribution. Categorical data were compared using Chi-square or Fisher's exact tests while continuous data were compared using paired and unpaired *t* tests wherever appropriate. Regression analysis was done to identify independent predictors of mortality. A $p$ value < 0.05 was considered statistically significant. Analysis was done in SPSS version 21.

## Results

### Demographic characteristics

During this study period, a total of 7575 children were admitted to PICU of which 1348 (17.8%) had non-traumatic coma (Fig. 1). Of these, SICH comprised 7.8% ($n = 105$) cases of non-traumatic coma. The trend over 10 years has shown an increased incidence of SICH (Fig. 2). The median (IQR) age at presentation was 6 (2.25,70) months; with nearly two-thirds ($n = 69$, 65.7%) being infants and three-fourth ($n = 76$, 72.4%) were boys. The median (IQR) duration of symptoms prior to presentation was 3 (1.5,5) days. The clinical presentation varied with age of the child; infants often presented with non-specific features such as irritability, poor feeding, lethargy, sudden onset pallor, seizures and vomiting while in older children headache and focal deficits were common. Seizure semiology was focal in 43 (58.9%) and generalized tonic clonic seizures (GTCS) in the rest. Thirty-six (34.3%) children had low GCS (< 8) at admission. The median (IQR) Pediatric Risk of Mortality score (PRISM III) was 17 (13,23). Features of herniation such as unequal pupils with decorticate/ decerebrate posturing at presentation were seen in one-fourth of the children ($n = 27$, 25.7%) (Table 1).

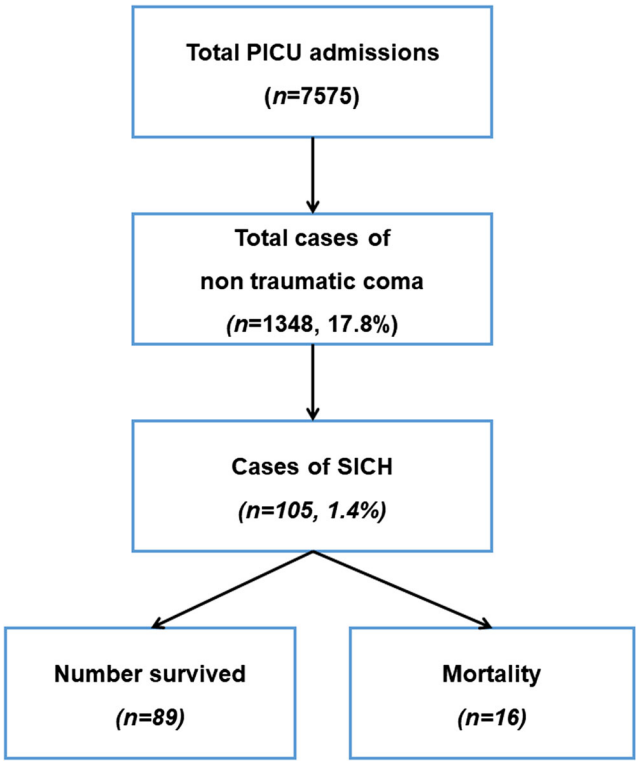


**Fig. 1** Study flow diagram

## Site and location of SICH

Most common site of bleeding was intracerebral ($n = 47$; 44.8%) followed by subarachnoid ($n = 26$; 24.8%) and multisite in 22 (21%) children (Table 2). Two children (1.9%) had intraventricular haemorrhage while one had cerebellar bleeding. Midline shift >5 mm was evident on CT in 58 (55.2%) children.

## Etiology of SICH

Arteriovenous malformations (AVM) was the most common etiology beyond 5 years of age contributing to 50% of cases followed by inherited clotting factor deficiencies ($n = 5/28$ (17.8%)) and hypertensive emergency ($n = 5/28$ (17.8%)). On the other hand, Vitamin K deficiency bleeding (VKDB) was found to be the most common etiology among infants ($n = 31$, 44.9%) followed by inherited factor deficiency ($n = 8$, 11.6%) (Table 2). Four children had non-accidental trauma; 3 with SDH and 1 with intraparenchymal bleeding. Among them 2 had associated retinal bleeding, 1 had multiple rib fractures and 1 presented with inconsistent history, significant cutaneous bleeding without coagulopathy.

## Treatment

All children were initiated on medical measures to control raised ICP. Seventy-four (70.5%) children responded to medical measures alone (Table 3), while the rest required surgical intervention; decompressive craniectomy in 26 (24.8%) and burr hole evacuation in 5 (4.8%). Packed red blood cell (PRBC) transfusion was needed in 72 (68.6%) children. Seventy-six children were mechanically ventilated (69.5%) for a median (IQR) duration of 108 (72, 144) hours. Two children needed tracheostomy. Twenty-three children (21.9%) had acute kidney injury of which 15 were in 'risk', 5 in 'injury' and 3 in 'failure' category. Five children (4.7%) had refractory raised ICP requiring thiopentone coma. Thirty-one children (29.5%) developed shock requiring fluid bolus or vasopressor support. Median (IQR) length of PICU stay was 5.5 (2.25,9.25) days.

## Outcome

Of the 105 children, 89 (84.8%) survived and 16 (15.2%) died. Univariate analysis revealed admission GCS < 8, higher PRISM III (> 20), need for intubation, and refractory raised ICP requiring thiopentone coma and progression to multiorgan dysfunction (shock and AKI) were significant predictors of mortality. Seizures were favourably associated with survival (Table 4). Age, site of bleeding, etiology or type of

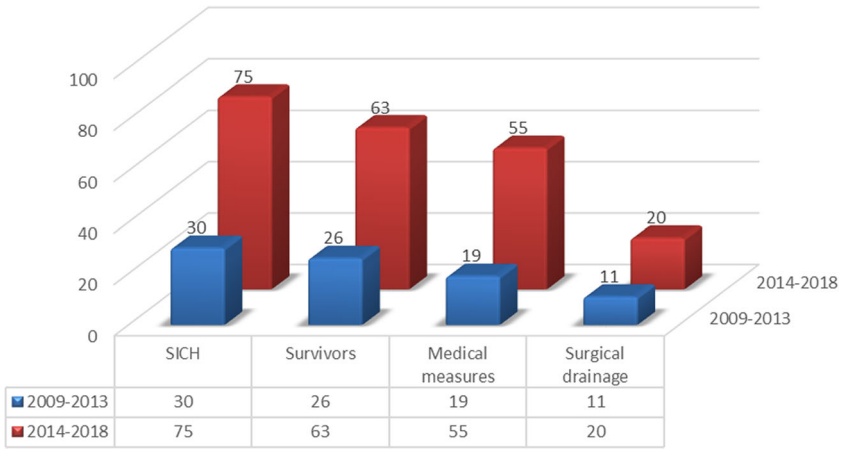


**Fig. 2** Comparison of incidence, outcome and management of SICH over the decade

**Table 1** Baseline characteristics of study cohort

| Variable | Total ($n = 105$) | Age group | | |
|---|---|---|---|---|
| | | 1–12 months ($n = 69$) | 12–60 months ($n = 8$) | > 60 months ($n = 28$) |
| Age in months (median (IQR)) | 6 (2.25,70) | 3 (2,6) | 26 (15.25,50.75) | 105.5 (95,123.25) |
| Male (*n* (%)) | 76 (72.4%) | 50 (72.5) | 6 (75) | 20 (71.4) |
| Duration of symptoms in days (median (IQR)) | 3 (1.5,5) | 3 (2,5) | 3 (1,5) | 4 (1,6) |
| PRISM III (median (IQR)) | 17 (13,23) | 17 (13,23) | 20.5 (15.25,26) | 14.5 (12,22) |
| Symptoms (*n* (%)) | | | | |
| Altered sensorium | 87 (82.9) | 60 (87) | 7 (87.5) | 20 (71.4) |
| Seizures | 73 (69.5) | 57 (82.6) | 6 (75) | 10 (35.7) |
| Fever | 38 (36.2) | 34 (49.3) | 2 (25) | 2 (7.1) |
| Vomiting | 57 (54.3) | 30 (43.5) | 5 (62.5) | 22 (78.6) |
| Headache | 28 (26.7) | 3 (4.3) | 3 (37.5) | 22 (78.6) |
| Excessive cry | 31 (29.5) | 29 (42) | 0 | 2 (7.1) |
| Poor feeding | 24 (22.9) | 21 (30.4) | 1 (12.5) | 2 (7.1) |
| Focal deficit | 21 (20) | 10 (14.5) | 1 (12.5) | 10 (35.7) |
| Signs | | | | |
| GCS at admission (median (IQR)) | 10 (6,13) | 8 (6,12) | 13 (10,15) | 13 (9.25,15) |
| Low GCS (*n* (%)) | 36 (34.3) | 32 (46.4) | 1 (12.5) | 3 (10.7) |
| Pallor (*n* (%)) | 66 (62.9) | 53 (76.8) | 5 (62.5) | 8 (28.6) |
| Anisocoria (*n* (%)) | 27 (25.7) | 22 (31.9) | 1 (12.5) | 4 (14.3) |
| Non-reactive pupils (*n* (%)) | 15 (14.3) | 12 (17.4) | 1 (12.5) | 2 (7.1) |
| Bulging anterior fontanelle (*n* (%)) | 52 (49.5) | 51 (73.9) | 1 (12.5) | 0 |
| Skin bleeds (*n* (%)) | 20 (19) | 17 (24.6) | 1 (12.5) | 2 (7.1) |
| Shock during hospital stay (*n* (%)) | 31 (29.5) | 24 (34.8) | 3 (37.5) | 4 (14.3) |

*GCS* Glasgow Coma Scale, *PRISM* Pediatric Risk of Mortality, *IQR* interquartile range, *SD* standard deviation, *ICH* intracranial hypertension

management for raised ICP (conservative versus decompressive craniectomy) did not affect the outcome. On binomial logistic regression analysis, AKI (odds ratio 5.86; 95% CI, 1.53–22.4; $p$ 0.01] was an independent predictor of mortality while seizure (odds ratio 0.12; 95% CI, 0.03–0.47; $p$ 0.002) was favourably associated with survival (Nagelkerke $R^2$– 0.43). Two epochs (2009–2013 and 2014–2018) were arbitrarily chosen as separate 5 years' periods to analyse for differences in outcome over the years. We found that proportion of children who were conservatively managed were more in the latter epoch. The mortality rate however remained the same despite improvement in intensive care facilities and surgical management (Fig. 2).

## Discussion

Our study revealed that clinical presentation and etiology of SICH varied from infancy to older children. GCS < 8, higher PRISM III, need for intubation, and thiopentone coma for refractory raised ICP and progression to shock and AKI were associated with poor outcome. Seizures at presentation were however associated with a favourable outcome.

SICH is not uncommon; it accounted for 7.8% of non-traumatic coma presentings to an emergency room, similar to previous published reports [21]. Studies from the USA have described an incidence rate of 1.1 to 4.5 per 100,000 person-years for haemorrhagic stroke [2–4]. However, comparison of our hospital-based incidence to population-based incidence reported from other studies may not be appropriate.

**Clinical presentation** The median age of presentation in our children was 6 months. Our cohort predominantly comprised of infants unlike the mean age reported in other studies of 7 to 14 years [17, 22–24]. The reason for this skewing towards infancy was possibly twofold: VKDB a disease of infancy was the most common etiology and

**Table 2** Etiology and site of SICH

| Variable | Total ($n = 105$) | Age group | | |
|---|---|---|---|---|
| | | 1–12 months ($n = 69$) | 12–60 months ($n = 8$) | > 60 months ($n = 28$) |
| Etiology of SICH | | | | |
| Coagulopathy | | | | |
| Vitamin K deficiency bleeding | 32 (30.5) | 31 (44.9) | 1 (12.5) | – |
| Inherited factor deficiencies | 15 (14.3) | 8 (11.6) | 2 (25) | 5 (17.8) |
| Cholestatic liver disease | 5 (4.8) | 5 (7.2) | – | – |
| Drug induced | 1 (1.0) | – | – | 1 (3.6) |
| Vascular malformations | 21 (20) | 5 (7.2) | 2 (25) | 14 (50) |
| Arteriovenous malformations | 18 | 4 | 1 | 13 |
| Cavernoma | 1 | 1 | – | – |
| Moyamoya disease | 2 | – | 1 | 1 |
| Thrombocytopenia | | | | |
| Leukaemia | 3 (2.9) | 2 (2.9) | – | 1 (3.6) |
| Aplastic anaemia | 1 (1.0) | – | – | 1 (3.6) |
| Thalassemia with hypersplenism | 1 (1.0) | – | 1 (12.5) | – |
| Hypertension | 5 (4.8) | – | – | 5 (17.8) |
| Haemorrhagic transformation of stroke | 4 (3.8) | 4 (5.8) | – | – |
| Complicated meningitis | 5 (4.8) | 4 (5.8) | – | 1 (3.6) |
| Non-accidental trauma | 4 (3.8) | 3 (4.4) | 1 (12.5) | – |
| Idiopathic | 8 (7.6) | 7 (10.1) | 1 (12.5) | – |
| Site of SICH on CT | | | | |
| Parenchymal | 47 (44.8) | 26 (37.7) | 2 (25) | 19 (67.9) |
| Subdural | 26 (24.8) | 18 (26.1) | 3 (37.5) | 5 (17.9) |
| Extradural | 2 (1.9) | 1 (1.4) | 1 (12.5) | – |
| Subarachnoid | 2 (1.9) | 1 (1.4) | 1 (12.5) | – |
| Cerebellar | 1 (1.0) | – | – | 1 (3.6) |
| Ventricular | 5 (4.8) | 2 (2.9) | 1 (12.5) | 2 (7.1) |
| Multiple sites | 22 (21) | 21 (30.4) | – | 1 (3.6) |
| Presence of midline shift | 58 (55.2) | 42 (60.9) | 5 (62.5) | 11 (39.3) |

Data expressed as *n* (%); *CT* computed tomography

exclusion of the adolescent age group (> 12 to 18 years). There was male predominance with 3:1 gender ratio similar to other studies [22–24].

Our finding revealed that clinical presentation and etiology of SICH differed among different age groups. The symptoms tended to be more non-specific in infancy such as altered sensorium, seizures, poor feeding, irritability as compared with headache and focal deficits in older children. These findings were consistent with previously published studies [17, 22].

**Etiology** VKDB was the most common etiology in infancy. The high incidence in developing countries has been attributed to missing of birth dose of vitamin K leading to its deficiency [25]. Although the awareness of VKDB and prophylactic vitamin K coverage at birth has improved, VKDB still persists pointing to an alternative mechanism that could contribute to this deficiency. This also warrants a relook at the existing prophylactic vitamin K administration practice and dosing, along with better modalities for early detection of Protein Induced in Vitamin K absence (PIVKA) [26, 27].

Vascular malformation-related bleeding is common in older children, similar to our observation. In our series, 50% of children with SICH beyond 5 years were secondary to vascular malformation [9, 23, 24, 28, 29]. Paediatric vascular malformations differ from adults in male predominance, more symptomatic bleeding with high rate of recurrence and posterior circulation aneurysms [29]. Additionally, unlike adults, the risks factors are predominantly connective tissue disorders, fibromuscular dysplasia and rarely infectious and inflammatory vasculitis. In our series, nearly one-fifth were inherited coagulopathic bleeding, most common being Haemophilia A,

**Table 3** Laboratory parameters and treatment details

| Variable | Total ($n = 105$) | Age group | | |
|---|---|---|---|---|
| | | 1–12 months ($n = 69$) | 12–60 months ($n = 8$) | > 60 months ($n = 28$) |
| Lab parameters | | | | |
| Haemoglobin gm/dl (median (IQR)) | 8 (5.85,9.95) | 6.9 (5.5,9.2) | 5.95 (3.55,5.95) | 11 (9.4,12.325) |
| Thrombocytopenia ($n$ (%)) (< 150,000/mm$^3$) | 16 (15.2) | 10 (14.5) | 2 (25) | 4 (14.3) |
| Coagulopathy ($n$ (%)) | 50 (47.6) | 40 (58) | 4 (50) | 6 (21.4) |
| Elevated transaminases ($n$ (%)) | 19 (18.1) | 14 (20.3) | 3 (37.5) | 2 (7.1) |
| AKI ($n$ (%)) | 23 (21.9) | 14 (20.3) | 3 (37.5) | 6 (21.4) |
| Hyponatremia at admission ($n$ (%)) | 28 (26.7) | 19 (27.5) | 3 (37.5) | 6 (21.4) |
| Treatment details | | | | |
| Osmotherapy ($n$ (%)) | 79 (75.2) | 52 (75.4) | 6 (75) | 21 (75) |
| Packed red cell transfusion ($n$ (%)) | 72 (68.6) | 59 (85.5) | 7 (87.5) | 6 (21.4) |
| FFP transfusion ($n$ (%)) | 43 (41.0) | 36 (52.2) | 3 (37.5) | 4 (14.3) |
| Platelet transfusion ($n$ (%)) | 14 (13.3) | 9 (13.0) | 2 (25.0) | 3 (10.7) |
| Thiopentone coma ($n$ (%)) | 5 (4.7) | 2 (2.9) | 1(12.5) | 2 (7.1) |
| Need for ventilation ($n$ (%)) | 73 (69.5) | 54 (78.3) | 7 (87.5) | 12 (42.9) |
| Duration of ventilation; hours (median (IQR)) | 108 (72,144) | 120 (72,144) | 96 (96,120) | 72 (38,204) |
| Day of extubation (median (IQR)) | 5 (3,7) | 6 (4,7.5) | 5 (4,6) | 2 (0,3) |
| Intervention | | | | |
| Conservative ($n$ (%)) | 74 (70.5) | 46 (66.7) | 5 (62.5) | 23 (82.1) |
| Decompressive craniectomy ($n$ (%)) | 26 (24.8) | 19 (27.5) | 3 (37.5) | 4 (14.3) |
| Burr hole ($n$ (%)) | 5 (4.7) | 4 (5.8) | 0 | 1 (3.6) |
| Time to DC; hours (median (IQR)) | 24 (24,40.5) | 24 (24,30) | 24 (12,24) | 48(24,108) |
| Outcome | | | | |
| Survival ($n$ (%)) | 89 (84.8) | 60 (87) | 7 (87.5) | 22 (78.6) |
| Death ($n$ (%)) | 16 (15.2) | 9 (13) | 1 (12.5) | 6 (21.4) |
| Length of PICU stay, days (median (IQR)) | 5 (3,9) | 6 (3,10) | 6 (5,10.75) | 3 (2,4.75) |
| Length of hospital stay, days (median (IQR)) | 14 (9,14) | 15 (9.5,22) | 13 (10.75,14) | 12 (8.25,15) |

*AKI* acute kidney injury, *FFP* fresh frozen plasma, *DC* decompressive craniectomy, *IQR* interquartile range, *SD* standard deviation

both in infancy and older children. Cause of bleeding could not be ascertained in 8 patients of our series despite the best possible investigations. The possibility of a small AVM or inherited bleeding diathesis in them cannot be ruled out.

**Site of bleeding** In the present study, the most common site of bleeding was intracerebral followed by SDH consistent with other studies [15, 17, 22, 23, 30]. However, haemorrhage in the putamen or thalamus was rare in contrast to adults where these bleeds are usually related to hypertension [31]. Few studies have described SDH as the most common site especially in non-accidental trauma in children less than 2 years of age [32, 33]. In our study, four children had non-accidental trauma; 3 SDH and 1 intraparenchymal bleeding.

Initial stabilisation and treatment in our cohort were based on a multimodal approach with emphasis on adequate management of raised ICP. However, it is also important to identify children who will benefit from early surgical intervention either for raised ICP or as a therapeutic option. Although there is lack of clear consensus on the role of surgery for ICP or for definitive therapy, focal lesions with midline shift on neuroimaging may benefit from early surgery. Controversies still exist over decompressive craniectomy (DC) for SICH with several studies favouring and negating its role [34–37]. Surgery however still remains the treatment of choice for AVM, aim being the complete removal of the malformation, thus guaranteeing cure [38, 39]. It has been shown that 70–90% of AVM in children can be removed surgically with no neurological deficits at postoperative follow-up in 52–75% cases. We could not demonstrate any difference with respect to mortality between SICH managed conservatively and surgically.

Mortality in the present series was 15.2% well within the reported range of 6–38% [22, 28]. The outcome for patients depends upon several factors. Studies in adults have reported

**Table 4** Predictors of mortality on univariate analysis

| Variable | Outcome | | *p* value | OR (95% CI) |
|---|---|---|---|---|
| | Non-survivor ($n = 16$) | Survivor ($n = 89$) | | |
| Demographic | | | | |
| Age in months (median (IQR)) | 10 (3.5111) | 5 (2,56.5) | 0.12 | |
| Boys (*n* (%)) | 9 (56.3) | 67(75.3) | 0.86 | |
| Clinical features and severity | | | | |
| Seizures (*n* (%)) | 7 (43.8) | 66 (74.2) | *0.01* | *0.27 (0.09,0.81)* |
| Vomiting (*n* (%)) | 9 (56.3) | 48 (54.3) | 0.86 | |
| Fever (*n* (%)) | 6 (37.5) | 32 (36) | 0.91 | |
| Focal deficit (*n* (%)) | 4 (25) | 17 (19.1) | 0.4 | |
| Poor feeding (*n* (%)) | 1 (6.3) | 23 (25.8) | 0.07 | |
| GCS < 8 (*n* (%)) | 10 (62.5) | 26 (29.2) | *0.01* | *4.03 (1.33,12.26)* |
| Pallor (*n* (%)) | 9 (56.3) | 57 (64) | 0.55 | |
| Anisocoria (*n* (%)) | 4 (25) | 23 (25.8) | 0.61 | |
| Bulging anterior fontanelle (*n* (%)) | 5 (31.3) | 47 (52.8) | 0.09 | |
| PRISM III (median (IQR)) | 25 (18,32) | 15(12,22) | *0.001* | *0.98(0.91,1.07)* |
| Site of bleed | | | | |
| Intraparenchymal bleed | 9 (56.3) | 38 (42.7) | 0.32 | |
| Subdural bleed | 1 (6.2) | 25 (28.1) | 0.06 | |
| Midline shift (*n* (%)) | 9 (56.3) | 49 (55.1) | 0.93 | |
| Laboratory parameters | | | | |
| Haemoglobin gm/dl (median (IQR)) | 9.3 (5,11) | 7.6 (5.9,9.8) | 0.382 | |
| Thrombocytopenia (*n* (%)) (< 150,000/mm$^3$) | 5 (31.3) | 11 (12.8) | 0.053 | |
| Coagulopathy (*n* (%)) | 8 (50) | 42 (47.2) | 0.83 | |
| Management | | | | |
| Intubation at admission | 13 (81.3) | 46 (51.7) | *0.03* | *4.05 (1.07,15.2)* |
| Shock (*n* (%)) | 9 (56.3) | 22 (24.7) | *0.01* | *3.91 (1.30,11.75)* |
| AKI (*n* (%)) | 9 (56.3) | 14 (15.7) | *0.001* | *6.89 (2.22,21.55)* |
| Thiopentone coma (*n* (%)) | 3 (18.8) | 2 (2.2) | *0.02* | *10 (1.53, 65.9)* |
| Osmotherapy (*n* (%)) | 14 (87.5) | 65 (73) | 0.217 | |
| Definitive therapy | | | | |
| Medical measures only (*n* (%)) | 12 (75) | 62 (69.7) | 0.77 | |
| Burr hole (*n* (%)) | 1 (6.3) | 4 (4.5) | 0.57 | |
| Decompressive craniectomy (*n* (%)) | 3 (18.8) | 23 (25.8) | 0.75 | |

*GCS* Glasgow Coma Scale, *PRISM* Pediatric Risk of Mortality score, *IQR* interquartile range, *OR* odd ratio, *CI* confidence interval, *SD* standard deviation, *DC* decompressive craniectomy, *ICH* intracranial hypertension

$p < 0.05$ was considered as significant

better outcome in patients with small haemorrhage and minimal neurodeficits compared with severe disability or death in those with GCS < 4 at admission [40]. Over the last decade, improvement in diagnosis and early surgical management has improved outcomes [5].

In the present study, GCS < 8, higher PRISM III, need for intubation and thiopentone coma for refractory raised ICP and progression to shock and AKI during hospital stay were predictors of poor outcome. The non-specific symptoms especially in infants, possibly lead to a delayed diagnosis and more severe presentation in our patients. Seizures at presentation were associated with better odds of survival probably due to three factors. First, onset of seizures could have hastened the need for seeking medical help and hospitalisation. Seizures increase the specificity for a CNS etiology thus drawing early attention towards an intracranial pathology. Also, focal seizures may indicate a focal pathology amenable to therapy. Age, site or etiology of bleeding did not correlate with outcome. On the contrary, other studies have shown that age < 3 years, GCS < 7 at admission, intracranial bleeding due to haematological disorders or aneurysm, infratentorial localisation, cerebellar bleed, intraventricular haemorrhage

and delayed presentation to hospital were associated with poor outcome [17, 24, 41, 42]. Adult literature reports, GCS < 9, at the time of presentation and volume of intraparenchymal haemorrhage to be strong predictors of 30-day mortality [43]. Adult studies have also described SAH to be associated with worse outcome; however, we could not demonstrate any such association [16].

## Strengths and limitations

Our study has a good sample size with adequate number in different age groups throwing light on the different etiologies. This 10 years' review has enabled us to show a trend for the hospital-based incidence and mortality of SICH in the background of improved intensive care treatment. This review opens door for more detailed analysis of VKDB subgroup as it contributed to a higher proportion of infantile SICH. Future prospective studies especially in infants evaluating vitamin K prophylaxis coverage and Protein Induced in Vitamin K Absence (PIVKA) levels to qualify VKDB better are warranted. This study has its inherent limitations of a single-centre retrospective analysis. ICP monitoring was not done. Only children aged less than 12 years are admitted in our PICU; hence, adolescents, who could have had a different etiology, could not be analysed. Also, assessment of long-term neurological and functional disability could not be done.

## Conclusions

VKDB was the most common etiology of SICH among infants whereas AVM was the most common among older children. Age, site of bleeding or type of management for raised ICP (conservative versus decompressive craniectomy) did not have any bearing on mortality. GCS < 8, need for intubation and thiopentone coma for refractory ICP, progression to shock and AKI were associated with worse outcome. Presence of seizures was associated with better odds for survival.

### Compliance with ethical standards

**Conflict of interest** None.

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