Research Article

A study of clinical profile of intracranial bleed

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Abstract

Introduction: Primary intracerebral hemorrhage (ICH), i.e. spontaneous extravasation of blood into the brain parenchyma, begins very suddenly and is a medical catastrophe. Despite a large amount of clinical interest and numerous prior studies, it remains unclear how often patients with clinical acute stroke upon initial evaluation have intracranial hemorrhage on their screening head CT scan1. **Aims and Objectives:** The present study was conducted to find clinical parameters, laboratory findings, C.T. scan findings and outcome in patients presenting with intracranial bleed. And also to correlate presence of hypertension and intracranial bleed in these patients. **Material and Methods**: The present observational study was conducted in the Department of Medicine of Government Medical College, Nanded from January 2009 and June 2010. Patients admitted in Medicine wards and ICU were studied. .All the patients presenting with acute stroke were evaluated clinically and with C.T scan brain. **Observations and Results:** A total 110 patients presenting with acute stroke, 79% of patients had hypertension. Intraparenchymal bleed in 63% and SAH in 23% of patients. Basal ganglia bleed in 36%, lobar bleed in 27% of patients. In hospital mortality was high in patients having intraventricular extension and midline shift > or equal to 3mm. **Conclusions:** GCS score < or equal to 8 was found to be a strong predictor of poor outcome. Hypertension was found to be most common etiological factor. The most common site of intraparenchymal bleed was internal capsule and basal ganglia.
Keywords: Acute stroke, Haemorrhage, Hypertension.

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INTRODUCTION

Approximately 15% of all strokes are due to intracranial hemorrhage. Hemorrhagic stroke can be diffuse i.e. bleeding into the subarachnoid or intraventricular spaces or focal i.e. intraparenchymal hemorrhage. About two thirds of intracranial bleeding are predominantly subarachnoid hemorrhages, whereas about a third are intracerebral hemorrhages. Subarachnoid hemorrhage is usually caused by rupture of vessels on or near the surface of the brain or ventricles e.g., aneurysms, vascular malformations, with bleeding mainly into the cerebrospinal fluid (CSF) spaces. Intracerebral hemorrhage is most frequently caused by the rupture of arteries that are within the brain substance e.g., hypertensive hemorrhage, vascular malformations but do not extend to the CSF spaces. Both types of hemorrhagic stroke have high mortality rates, depending on subtype and location. Prevention is the mainstay of management because there are no sufficiently efficacious therapies for hemorrhage-induced cerebral injury.[2] Intracranial hemorrhage is caused by bleeding directly into or around the brain, it produces neurologic symptoms by producing a mass effect on neural structures, from the toxic effects of blood itself or by increasing intracranial pressure[3]. The classic presentation of ICH is sudden onset of a focal neurological deficit that progresses over minutes to hours with accompanying headache, nausea, vomiting, decreased consciousness, and elevated blood pressure. In the Harvard Stroke Registry and the Michael Reese Stroke Registry, 51% to 63% of patients with ICH had a smooth progression of neurological symptoms, whereas 34% to 38% of patients had maximal symptoms at onset.

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By comparison, only 5% to 20% of the various ischemic stroke subtypes and 14% to 18% of patients with SAH had gradual progression of symptoms. The early progression of neurological deficit in many patients with an ICH is frequently due to ongoing bleeding and enlargement of the hematoma during the first few hours[4]. Hemorrhages are classified by their location and the underlying vascular pathology. Bleeding into subdural and epidural spaces is principally produced by trauma. SAHs are produced by trauma and rupture of intracranial aneurysms. Intracranial hemorrhage is often discovered on noncontrast CT imaging of the brain during the acute evaluation of stroke. Since CT is more sensitive than routine MRI for acute blood, CT imaging is the preferred method for acute stroke evaluation. Intraparenchymal hemorrhage is the most common type of intracranial hemorrhage. It accounts for ~10% of all strokes and is associated with a 50% case fatality rate. Incidence rates are particularly high in Asians and African Americans. Hypertension, trauma and cerebral amyloid angiopathy cause the majority of these hemorrhages. Advanced age and heavy alcohol consumption increase the risk, and cocaine use is one of the most important causes in the young. Other useful diagnostic tools include a complete blood count, prothrombin time, activated partial thromboplastin time, electrolytes, electrocardiography, and chest x-ray. The white blood cell count can detect underlying infection such as hemorrhages associated with endocarditis. Hemoglobin analysis may also provide clues to diagnosis and is an indicator of blood loss. Prothrombin time and activated partial thromboplastin time may offer clues to coagulation problems, either iatrogenic or acquired. Electrolytes can reveal evidence of primary renal failure as an associated cause of ICH or disturbances in sodium that may accompany brain hemorrhage. Electrocardiography may reveal underlying dysarrhythmia or myocardial ischemia associated with brain hemorrhage. Chest x-ray may show underlying aspiration or another pulmonic process that may complicate treatment. Hypertensive intraparenchymal hemorrhage (hypertensive hemorrhage or hypertensive intracerebral hemorrhage) usually results from spontaneous rupture of small penetrating arteries deep in the brain. The most common sites are the basal ganglia (especially the putamen), thalamus, cerebellum, and pons. When hemorrhages occur in other brain areas or in nonhypertensive patients, greater consideration should be given to hemorrhagic disorders, neoplasm's, vascular malformations, and other causes. The small arteries in these areas seem most prone to hypertension-induced vascular injury. The hemorrhage may be small or a large clot may form and compress adjacent tissue, causing herniation and death. Blood may dissect into the ventricular space, which substantially increases morbidity and may cause hydrocephalus. Despite a large amount of clinical interest and numerous prior studies, it remains unclear how often patients with clinical acute stroke upon initial evaluation have intracranial hemorrhage on their screening head CT scan[1]. In this study, patients admitted to our hospital with suspected acute stroke were identified, and their initial (screening) NCCT results were evaluated for intracranial hemorrhage.

METHODS

The present cross-sectional observational study was conducted in the department of Medicine of Government Medical College, Nanded from January 2009 and June 2010. Patients admitted in medicine wards and intensive care unit were studied. All the patients presenting with acute stroke were evaluated clinically and with C.T scan brain. Patients more than 13 years of age having intraparenchymal bleed, patients having subarachnoid bleed were included in the study. Patients diagnosed to have transient ischemic attack, ischemic stroke, subdural hematoma, extra Dural hematoma, traumatic intracranial bleed and venous hemorrhagic infarct were not included in the study. An institutional ethical committee approved this study on October 20th 2009. All the Selected Patients were evaluated in detail and data was collected in the proforma which was regularly entered in the MS Excel spreadsheet 2007. Data was then analyzed and following observations and results were obtained.

OBSERVATIONS AND RESULTS

In the present study 44/110 (40%) patients were in the age group of 64-73yrs and 75 (68%) were in between 54-73 yrs. Prevalence of I.C.H. was higher 61% in males as compared to 43% in females. 65 [59%] patients had right or left hemiplegia or hemiparesis (focal neurological deficit) and 59 (54%) patient presented with altered sensorium on admission, followed by headache in 44 (40%) and vomiting in 40 (36%) patients. 13 (12%) patients had history of convulsion as presenting symptom. 37/110 (34%) patients had history of smoking and 31/110 (28%) had history of alcohol intake. Percentage of smokers was greater than that of alcoholics. 4(4%) patients had history of cocaine addiction and 10 (9%) were on anticoagulants for some reasons. Most common presenting sign was limb Weakness (rt or lt) seen in 65 (59%) patients followed by altered higher mental function seen in 59 (54%) of patients, followed by cranial nerve involvement in 26 (24%) patients. Cerebellar sign and sign of meningial irritation were observed in 7 (6%) and 11 (10%) of patients respectively. 35 (32%) patients had GCS < or equal to 8 and 75 (68%) patients had GCS >8 on admission. 28 out of 35 (80%) patients having GCS <

or equal to 8 on admission and 14 out of 75 (19%) patients having G.C.S>8 on admission, died in hospital. Patients having GCS score< or equal to 8 had higher mortality as compared to that of >8.The difference is statistically significant.

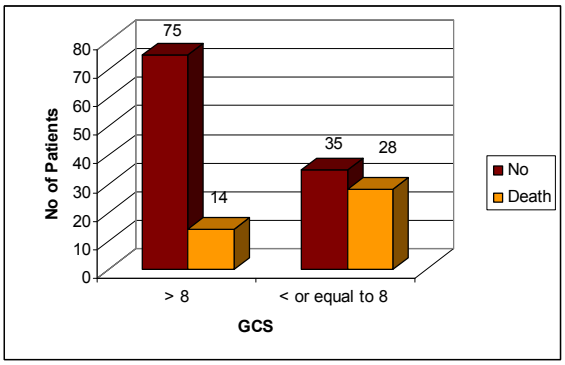

Figure 1: Showing outcome of patient of I.C.H in relation to GCS

X^2=38.4, p<0.001, DF=1 highly significant

39/110 (35%) patients had Leukocytosis, 24/110 (22%) had deranged BSL's. Abnormal kidney and liver function test were observed in 22 (20%) and 17 (15%) patients respectively, 1 (1%) patients had coagulation defect. In the present study most common electrolyte abnormalities were hyponatremia in 20 (18%) patients followed by hypernatremia 9 (8%) patients. Hypokalema was observed in 8 (7%) patients and hyperkalemia in 3 (3%) patients. In the present study 15 (14%) patients had high level of cholesterol >250mg% followed by 23 (21%) having levels between 200-250mg%. 72 (65%) patients had levels< 200mg%. Most common ECG changes associated with ICH were prolonged QTc seen in 30% of patients, STandT changes in 28% and LVH or Left ventricular strain pattern in 28%. LBBB was found in 12 %, U wave in 8% of patients Brady and Tachyarrhythmia's in 2% and no abnormality was detected in 10% of patients. Hypertensive retinopathy was seen in 40 (36%) patients, was the most common finding on fundus examination followed by papiloedema in 24% and diabetic retinopathy in 10% of patients. 87 (79%) patients had hypertension which was found to have statistically significant association with presence of I.C. bleed (p<0.05). 24 (22%) patients had diabetes, 10 (9%) were on chronic anticoagulation treatment. 4 (4%) patients had history of cocaine abuse, 2 (2%) patients had bleed in brain tumor and 1 (1%) had A-V malformations and coagulation defect each. Intraparanchymal bleed which was observed in 70 (63%) patients was the most common type of bleed followed by Subarachnoid bleed in25 (23%) patients and intraparechymal with subarachnoid extension in 15 (14%) patients. Fatal outcome was seen in patients of intraparanchymal with subarachnoid extension (53%) followed by SAH (36%) then intraparanchymal bleed (35%) of patients. Mortality was highest in patients having brain stem bleed 5 out of 6 (83%) patients, followed by in lobar bleed (39%), followed by cerebellum (37%) and then basal ganglia (35%) and thalamus (27%). 41 (38%) patients had intraventricular extension and it was absent in 69 (62%) patients. Patients of intracranial bleed having intraventricular extension had more mortality 27 (66%) patients as compared to that without intraventricular extension15 (22%) patients. The difference was found to be statistically highly significant. X^2=21.20, p<0.001, DF=1 highly significant. 34 (32%) patients had midline shift > or equal to 3mm, and 76 (68%) patients had midline shift <3mm or absent. Patients having I.C. bleed and midline shift on C.T. brain of > or equal to 3mm had high mortality as compared to that of <3mm the difference was statistically significant. X^2=35.44, p<0.001, DF=1 highly significant. Mortality was highest (47%) in patients having grade 2 hypertension and it was 38% in grade 1, 23% in pre hypertensives and 10% in the patient who had normal BP on presentation.

DISCUSSION

Even though intracranial bleed accounts for 20% of cerebrovascular accidents mortality and morbidity resulting from it is higher compared to thrombotic stroke. In various studies mortality in patients with I.C.H.is 40-60%. Even after 45 yrs of invention of C.T. scan treatment of I.C.H. remains a challenge to medical fraternity. Even though effective treatment like factor VII available, it is far from reach of the poor. Therefore efforts must be directed towards understanding modification of risk factors of I.C.H. On interpretation of data and results observed we compared our findings with that of similar Indian and international studies and found that age, sex of the patients affected, presenting symptoms, ECG findings results were compasrable. In this study 79% of patients had hypertension which was the most common risk factor for intracranial bleed. Matti hillborn, Mansooreh togha *et al*, Hemphill *et al*, Adanan quereshi *et al*, Feldmann *et al* all observed in their study that most common etiological factor was hypertension with the incidence between 50%-80%[5,6,7,8,9.] Out of total 110 patients 85 had intraparenchymal bleed. It was observed that internal capsule and basal ganglia was the most common site of intrapranchymal bleed. Followed by lobar, thalamus, cerebellar, brain stem and primary intraventricular. Intraparenchymal bleed was observed in 63% and sub arachnoid haemorrhage in 23% of patients. Broderick JP, Brott T *et al* [10], K ueda y hauso *et al* [11], Mumtaz Ali Marwat, *et al* [12] and Bamford, P Sandercock [13] had found intrapaenchymal bleed in 68%, 65%, 72%

and 63% respectively and subarachnoid haemorrhage in 32%, 35%, 28%, 27% of patients respectively which was comparable to our finding. 27 patients out of 41 (38%) had I.C. bleed with intraventricular extension as observed in table no. 18, had high mortality (66%) and in 69 (62%) patients did not have intraventricular extension and mortality was seen only in 22% of the patients this difference on correlation by chi square test was found to be statistically significant. i.e. p<0.001. Observations of the present study were comparable with different studies as mentioned in the table no. 1 below. [5,7,14,15.]

Table 1: N%=number of patients in percentage, M%=mortality in percentage in N number of patients

Intraventricular ext	Mansooreh *et al*		J.Claud Hemphil *et al*		P.Daverat *et al*		Daniel Godoy *et al*		Present study	
	N%	M%	N%	M%	N%	M%	N%	M%	N%	M%
Present	43%	71%	55%	66%	44%	69%	48%	48%	38%	66%
Absent	57%	29%	45%	19%	56%	23%	52%	22%	62%	22%
Total	**100%**		**100%**		**100%**		**100%**		**100%**	

34 (32%) patients had midline shift > or equal to 3mm with I.C. bleed as it is observed in table no. 19, had high mortality (79%), and in 76 (68%) patients had midline shift <3mm or absent and mortality was seen in only 20% of patients, this difference on correlation by chi square test was found to be statistically significant i.e. p<0.001. Mortality was highest in patients having brain stem bleed 5/6 (83%) of patients, followed by in lobar bleed (39%).followed by cerebellum (37%) and then basal ganglia (35%) and thalamus (27%). Observations of the present study were comparable with different studies. J. Claud Hemphil *et al*[7.] Chronic hypertension produces a small vessel vasculopathy characterized by lypohyalinosis, fibrinoid necrosis and development of charcot-Bouchard aneurysms, affecting penetrating arteries throughout brain. Hypertension has been traditionally regarded as the most powerful risk factor for primary intracranial bleed. Several investigators have examined relation between elevated blood pressure levels and the risk of intracranial bleed and they consistently reported a graded relation between systolic and diastolic blood pressure and the risk of intracranial hemorrhage. In the present study, 87 (79%) patients had hypertension and 53 (48%) patients presented with grade two hypertension i.e. [B.P.>160/110mmHg]. 34 (31%) had grade 1 hypertension, 13 (12%) were pre hypertensive and 10 (9%) had normal blood pressure on presentation. Mortality was highest (47%) in patients having grade 2 hypertension and it was 38% in grade 1, 23% in pre hypertensive's and 10% in the patient who had normal BP on presentation So, it was observed that as grade of hypertension on admission increases both incidence and mortality of intracranial bleed increases. In the present study patients of I.C. bleed were divided in two groups hypertensive and non hypertensive, chi square value calculated (5.32) for associating hypertension with intracranial bleed was found to be statistically significant i.e. p<0.05. Same finding was observed in the study done by Mansooreh *et al*, Yasuo Terayama *et al* and Gebel *et al*. [5, 16, 17.] As discussed above outcome of patients after I.C. bleed depends upon the location of hemorrhage, size of hematoma, level of consciousness, presence of hypertension and preceding history of oral anticoagulation therapy.

CONCLUSIONS

- This study comprised 110 patients of intracranial hemorrhage of which 85 patients had C.T. proved intraparenchymal bleed and 25 patients had subarachnoid bleed.
- GCS score < or equal to 8 was found to be a strong predictor of poor outcome. in I.C.H (p<0.001).
- Hypertension was found to be most common etiological factor, which was found to have statistically significant association with I.C.H (p<0.05).
- Intraparenchymal bleed was found to be the most common type of bleed followed by SAH.
- The most common site of intraprenchymal bleed was internal capsule and basal ganglia .Mortality was found to be highest in patients having brain stem bleed. I.C. bleeding with intraventicular extension was found to be a strong predictor of poor outcome in I.C.H (p=<0.001). I.C.
- bleeding with midline shift > or equal to 3 was found to be a strong predictor of poor outcome in I.C.H (p=<0.001).

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