

Original Research Article

Clinicoetiological and imaging profile of intracerebral haemorrhage in a tertiary care hospital of Western Odisha

Purna Chandra Karua*, Shiny Joy

Department of General Medicine, VSS Institute of Medical Science and Research, Burla, Odisha, India

Received: 03 November 2020

Revised: 16 November 2020

Accepted: 18 November 2020

***Correspondence:**

Dr. Purna Chandra Karua,

E-mail: karuapcvss@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Intracerebral hemorrhage has an annual incidence of 10-30/100,000 population, accounting for 2 million (10-15%) of about 15 million strokes worldwide each year. The outcome of ICH depends on the clinical presentation and radiological parameters. The objective of current study was to study the etiology, clinical patterns and imaging profile in patients of intracerebral hemorrhage.

Methods: All patients admitted in department of medicine, VIMSAR, Burla, with a diagnosis of ICH during a period of November 2017 to October 2019 were evaluated for their clinical presentation, etiology and radiological parameters.

Results: A total of 105 patients with a diagnosis of ICH were included in the study. The sites of ICH were basal ganglia (49%) followed by lobar (19%), thalamus (14%), cerebellum (11%) and brain stem (8%). A pre-diagnosis of hypertension was present in 33% of the cases. Headache was the most common presenting symptom, present in 38.2% of patients followed by paralysis in 29.5%, vomiting in 27.6% and seizures in 20.9% of cases. Overall mortality rate was 39%. The mean GCS of patients who expired was 8.8 when compared to 11.28 of those patients who survived ($p=0.00009$). The mean hematoma size of patients who expired was 20.98 while that of those who survived was 17.41 ($p=0.047$). The presence of IVC was associated with mortality ($p=0.006$).

Conclusions: A lower GCS at presentation and a mean hematoma volume >20 ml with intraventricular extension at presentation are associated with increased mortality in ICH.

Keywords: Glasgow coma scale, Mean hematoma volume, Intraventricular extension, Mortality

INTRODUCTION

Cerebrovascular diseases are the third leading cause of death after heart disease and cancer in developed countries. Intracerebral hemorrhage (ICH) is a common devastating neurologic event that causes high morbidity and mortality with profound economic implication. ICH will seem to continue to be an important problem in both India and other developed countries. Nontraumatic ICH occurs due to bleeding from a vascular source directly into the brain substance. It is a major public health

problem with an annual incidence of 10-30/100,000 population, accounting for 2 million (10-15%) of about 15 million strokes worldwide each year.¹⁻⁴

Primary or spontaneous intracerebral hemorrhage, which is defined as spontaneous rupture of the intracerebral small vessels following cerebral vessel wall degeneration due to frequent chronic hypertension or rarely to cerebral amyloid angiopathy. The risk factors for ICH are identified as hypertension, advancing age, male sex, excessive alcohol intake, anticoagulation therapy,

smoking, and diabetes. To determine these risk factors is very important in terms of developing preventative measures.⁵⁻⁸

Hospital admissions for ICH have increased by 18% in the past 10 years, probably because of increases in the number of elderly people, many of whom lack adequate blood pressure (BP) control, and the increasing use of anticoagulants, thrombolytics, and antiplatelet agents. Incidence might have decreased in some populations with improved access to medical care and BP control.⁹⁻¹²

Hence, we aimed to study, the clinical, radiological profile in patients of ICH. In clinical profile risk factors, symptomatology and physical findings were studied. In radiological profile, size, and location of hematomas, correlation of the size of hematomas with size were also studied. We also studied mortality, morbidity, and predictive factors of clinical outcome of ICH.

METHODS

Presented hospital based observational study was carried out on the patients admitted to department of general medicine, VIMSAR, Burla during the period from November 2017 to October 2019.

Inclusion criteria

All patients admitted to department of general medicine, VIMSAR with a confirmed diagnosis of intracerebral haemorrhage based on neuroimaging techniques were included in the study.

Exclusion criteria

Patients presenting with traumatic intracerebral haemorrhage. Patients with primary subarachnoid, extradural, intradural haemorrhage. Patients who do not give consent for the same were excluded from the study.

The following parameters were recorded with the help of a structured proforma. Demographics, co morbidities, addictions, presenting complaints at admission, vital signs; pulse rate, blood pressure, respiratory rate and glasgow coma scale at the time of admission, investigations, biochemical; plasma glucose, serum electrolytes, blood urea, serum creatinine, liver function tests, urine analysis, lipid profile, complete blood count, serology for HIV, HBV and HCV, CT scan of brain, non contrast CT scan, MRI scanning if available, special investigations that might be required in selected patients, coagulation profile; BT, CT, PT with INR, aPTT, events and treatments during course of hospital stay.

Data analysis

All recorded data were analysed calculating percentages, mean values, standard deviation and Chi square test and through standard statistical methods including standard diagrams and graphs and findings were discussed in detail to draw appropriate conclusions.

RESULTS

Out of 105 patients of IC bleed who were prospectively studied from 2017 to 2019, 64 (61%) were males and 41(39%) were females and 15.23% patients were in the 30-40 age group, 37.14% in the 41-50 years age group, 28.57% in the 51-60 years age group, 17.14% in the 61-70 years age group and 1.9% in the 71-80 years age group (Figure 1).

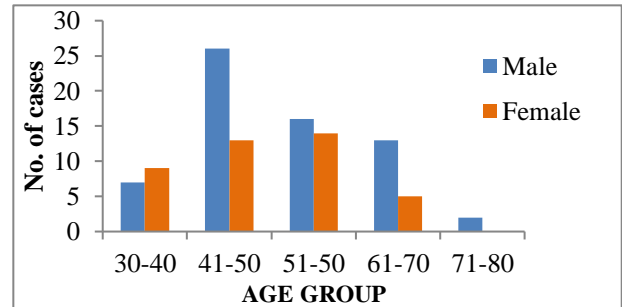


Figure 1: Age distribution.

Out of 105 patients, 35 patients (33%) had hypertension and 72 patients (67%) didn't have pre-diagnosed hypertension. Of all the age groups, only 17.6% of patients with age >60 years had pre-diagnosed hypertension, while 56% patients with age <40 years had pre-diagnosed hypertension (Figure 2).

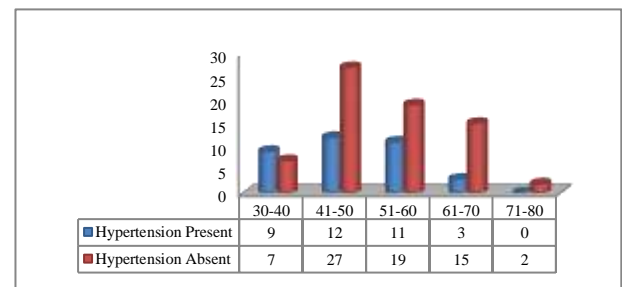


Figure 2: Diagnosed hypertension cases based on age group.

Out of 105 patients, 22.85% had a diagnosis of diabetes made prior to the IC bleed episode. Out of all the age groups, the extremes of age group had more percentage of diabetes 37.5% of patients with age <40 years and 100% patients with age >70 years had a diagnosis of diabetes mellitus. Only 9.5% had a past history of coronary artery disease. Of the age groups, 25% patients of age >60 years had a history of CAD prior to this IC bleed episode. 10 (9.5%) had a prior history of CVA. Out of these 10 patients, 3 had hemorrhagic CVA in the past. 25% patients in age group <40 years had a prior history of CVA, which was statistically significant. 48% had bleeding in the basal ganglia, 19% had bleeding in the lobar area, 14% had bleeding in the thalamus, 11% had bleeding in the cerebellum and 8% had bleeding in the brain stem.

Out of 105 patients, 14 patients were using antiplatelets. 3 out of 8 patients (37.5%) who had brain stem bleed were using anti-platelets when compared to only 4 out of 50 patients (8%) with basal ganglia bleed, but this was statistically insignificant (p=0.245) (Table 1).

Table 1: Anti-platelet use and bleeding site.

Site	N	(%)	P value
Basal ganglia	4	8	0.245
Lobar	3	15	
Thalamus	2	13.33	
Cerebellum	2	16.67	
Brain stem	3	37.5	
Total	14		

Out of 105 patients with IC bleed, headache was the most common symptom, present in 38.2% of patients followed by paralysis, vomiting and seizures in 29.5%, 27.6% and 20.9% respectively. 66.67% of cerebellar bleeds had history of persistent headaches while only 10% of lobar hemorrhage had the same history which was statistically significant. 76% of patients with brain stem bleed had a GCS <8, when compared to only 8% of patients with cerebellar bleeds, but this was not statistically significant. Mean GCS was 6.37 for patients who had a brain stem bleed whereas it was 11.16 for those with cerebellar bleed and all other sites with values in between. There was statistically significant difference in the mean volume of hematoma in the different sites. The maximum mean of hematoma size was in the lobar (28.9 ml) with least in the brain stem (12.4 ml). 37 patients (35.2%) had intraventricular extension. 50% of lobar bleed patients had IV extension, while only 28% of basal ganglia bleed had it. This difference was not statistically significant. 41 patients (39%) expired in the first 4 weeks. The maximum mortality rate was for brain stem bleed (75%) followed by cerebellar bleed (50%), lobar bleed (35%), basal ganglia bleed (34%) and thalamic bleed (33%). The difference in mortality rates between all the sites were not statistically significant (Table 2). The mean hematoma size of the patients who expired was statistically significantly higher (by 3.5 ml) than those patients who survived.

Table 2: Mortality rate.

Site	Mortality rate (%)	P value
Basal ganglia	34	0.212
Lobar	35	
Thalamus	33	
Cerebellum	50	
Brain Stem	75	
Total	41	

DISCUSSION

105 cases of intracerebral bleed admitted in our hospital from November 2017 to October 2019 were evaluated for this present study. There were 64 males (61%) and 41 females (39%). Hypertension was the most common risk factor being present in 33% of the population. It was present in 40% of cases of basal ganglia bleed, 25% of cases of lobar bleed, 40% of cases of thalamic bleed, 41% of cases of cerebellar bleed and 37% of cases of brain stem bleed. This data was similar to those published by Ojha et al in which 29.5% had pre-existing hypertension.¹³ But some other studies had published much higher rates of hypertension in patients who presented with IC bleed. The study by Woo et al found out that 63% of patients with intracranial bleed had hypertension.¹⁴ But in our present study the prevalence of hypertension was lower, which may be due to in many of the prior studies hypertension was said to be present when the BP at presentation was high and other reason may be the presence of undiagnosed hypertension in this community in which contact with a healthcare worker is at a premium.

In this study 9.5% of the patients who presented with intracerebral hemorrhage had prior history of CVA. Out of this 70% were ischemic CVA and 30% hemorrhagic. This is similar to the reported 1.5%-2% risk of recurrence for intracerebral hemorrhage.¹⁵

Out of 105 patients with IC bleed, 14 patients (13.3%) had history of intake of anti-platelets. 37.5% of patients with brain stem bleed had history of intake of anti-platelets as compared to only 8% of patients with basal ganglia bleed. But this difference was not statistically significant (p=0.245). The use of anti-platelet was also not associated with increased mortality (p=0.75).

The mean GCS at presentation was 10.31. When the mean GCS at presentation was sub classified based on the site of bleed, brain stem bleed had a mean GCS of 6.37 while cerebellar bleed had a mean GCS of 11.16. This difference was statistically significant (p=0.009). 76% of patients with brain stem bleed had a GCS <8 at presentation while only 8% of patients with cerebellar bleed had a GCS <8 at presentation. The mean GCS of patients who expired was 8.8 when compared to 11.28 of those patients who survived (p=0.00009).

The mean hematoma size of 105 patients with IC bleed was 18.8 ml. When the mean hematoma size was sub classified depending on the site of bleed, lobar hemorrhage was associated with a mean hematoma size of 28.9 ml whereas brain stem hematomas were only about 12.4 ml in average (p<0.0001). The mean hematoma size of patients who expired was 20.98 while the mean of those who survived was 17.41. This difference in mean hematoma size was also statistically significant (p=0.047).

IVC extension was present in 35.2% of patients. The presence of IVC was associated with mortality ($p=0.006$).

The overall mortality rate was 39%. This was similar to the mortality rates reported in studies by Gobindram and Woo.^{14,16} The maximum mortality rate was for brain stem bleed with 75% and least was for thalamic bleed with 33%, but this difference was not statistically significant ($p=0.212$).

Limitations

Limitations of the current study were; the sample size was smaller, it could have affected our observations and conclusions. Due to limited resources in our setting, we could not do CT angiography, MR angiography to localise the vessels. As the study was done only in our centre, there may be regional variations in etiology and also management strategies for which we cannot generalise to the whole population.

CONCLUSION

Cerebrovascular accidents are one of the leading cause of mortality and morbidity in developed as well as developing countries with intracerebral haemorrhage causing the highest overall mortality. Patients had hemorrhagic CVA in the past indicating lack of proper follow up and risk factor management leading to increased rate of restroke. The most common site of bleeding was found in basal ganglia followed by lobar bleed, thalamus, cerebellum brain stem. In patients with antiplatelet associated intracranial haemorrhage most common site was found to be basal ganglia. In patients with IC bleed, the maximum mean of hematoma size was in the lobar with least in the brain stem. The mean hematoma size of the patients who expired was statistically significantly higher than those patients who survived. When compared mortality during hospital admission, the maximum mortality rate was for brain stem bleed followed by cerebellar bleed, lobar bleed, basal ganglia bleed and thalamic bleed. From this study it was found that brainstem bleed was associated with low GCS at presentation and have high mortality and morbidity while comparing with other sites. Hematoma size had a prognostic role as patient who had a higher volume by 3.5 ml were expired compared with those who survived. Seizure was associated with 20.9% cases of intracerebral haemorrhage as clinical presentation. Early management of intracranial hypertension and other complications prolonged survival. Further studies are required to delineate various aetiologies like cerebral amyloid angiopathy and imaging modalities like CT angiography, MR angiography to find out the exact vessel involvement. Also for determining optimal treatment strategies so that it will help in reduction of morbidity and mortality.

ACKNOWLEDGEMENTS

Authors would like to thank patients for their adherence and kind cooperation in current study and the staff and technicians of the central laboratory for performing the different tests required for the study.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Qureshi AI, Tuhim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. *N Engl J Med.* 2001;344:1450-60.
2. Labovitz DL, Halim A, Boden-Albala B, Hauser WA, Sacco RL. The incidence of deep and lobar intracerebral hemorrhage in whites, blacks, and Hispanics. *Neurology.* 2005;65:518-22.
3. Sudlow CL, Warlow CP. Comparable studies of the incidence of stroke and its pathological types: Results from an international collaboration. International stroke incidence collaboration. *Stroke.* 1997;28:491-9.
4. Qureshi AI, Mendelow AD, Hanley DF. Intracerebral haemorrhage. *Lancet.* 2009;373:1632-44.
5. Ariesen MJ, Claus SP, Rinkel GJ, Algra A. Risk factors for intracerebral hemorrhage in the general population: A systematic review. *Stroke.* 2003;34:2060-5.
6. Schwarz S, Häfner K, Aschoff A, Schwab S. Incidence and prognostic significance of fever following intracerebral hemorrhage. *Neurology.* 2000;54:354-61.
7. Cheung RT, Zou LY. Use of the original, modified, or new intracerebral hemorrhage score to predict mortality and morbidity after intracerebral hemorrhage. *Stroke.* 2003;34:1717-22.
8. Woo D, Sauerbeck LR, Kissela BM, Khoury JC, Szaflarski JP, Gebel J, et al. Genetic and environmental risk factors for intracerebral hemorrhage: Preliminary results of a population-based study. *Stroke.* 2002;33:1190-5.
9. Qureshi AI, Suri MF, Nasar A, Kirmani JF, Ezzeddine MA, Divani AA, et al. Changes in cost and outcome among US patients with stroke hospitalized in 1990 to 1991 and those hospitalized in 2000 to 2001. *Stroke.* 2007;38:2180-4.
10. Feigin VL, Lawes CM, Bennett DA, Anderson CS. Stroke epidemiology: A review of population-based studies of incidence, prevalence, and case-fatality in the late 20th century. *Lancet Neurol.* 2003;2:43-53.
11. Narayan SK, Sivaprasad P, Sushma S, Sahoo RK, Dutta TK. Etiology and outcome determinants of intracerebral hemorrhage in a south Indian population, A hospital-based study. *Ann Indian Acad Neurol.* 2012;15(4):263-6.

12. Omkar P, Baidya, Sunita Tiwari, Kauser Usman. Clinical profile of acute hemorrhagic stroke patients: a study in tertiary care hospital in Northern India. *Int J Res Med Sci.* 2014;2(4):1507-10.
13. Ojha P, Sardana V, Maheshwari D, Bhushan B, Kamble S. Clinical profile of patients with acute intracerebral hemorrhage and ich score as an outcome predictor on discharge, 30 Days and 60 days follow-up. *J Assoc Physicians India.* 2019; 67(8):14-8.
14. Woo D, Sauerbeck LR, Kissela BM, Khoury JC, Szaflarski JP, Gebel J, et al. Genetic and environmental risk factors for intracerebral hemorrhage: preliminary results of a population-based study. *Stroke.* 2002;33:1190-6.
15. Rymer MM. Hemorrhagic stroke: intracerebral hemorrhage. *Mo Med.* 2011;108(1):50-4.
16. Gobindram A. Stroke care: Experiences and clinical research in stroke units in Chennai. *Ann Indian Acad Neurol.* 2006;9:193-8.

Cite this article as: Karua PC, Joy S. Clinicoetiological and imaging profile of intracerebral haemorrhage in a tertiary care hospital of Western Odisha. *Int J Res Med Sci* 2020;8:4369-73.