DOI: 10.7860/JCDR/2022/55362.16736

Original Article

Internal Medicine Section

Clinical and Radiological Profile in Non Hypertensive Intracerebral Haemorrhage- A Prospective Observational Study

SIDDAGANGA[1], BHARAT L KONIN[2], SWATHI BHAT[3]

ABSTRACT

Introduction: Intracerebral Haemorrhage (ICH) is less frequent than ischaemic stroke, but has higher mortality and morbidity, it being one of the first causes of severe disability. Hypertension is the most common cause of ICH but Non Hypertensive Intracerebral Haemorrhages (NHICH) are not rare. Hence, the importance of recognising these conditions and need for urgent specific therapy which may play a vital role in therapeutic planning and prevention of ICH. Hence this study was performed to study.

Aim: To study the clinical and radiological profile in NHICH to identify risk factors, and to determine whether clinical/neuroradiological parameters would predict the prognosis of ICH.

Materials and Methods: The present prospective observational study was conducted in the Department of General Medicine Mahadevappa Rampure Medical College Gulbarga, Karnataka, India, from January 2016 to January 2017. It involved 50 subjects with NHICH. Demographic details, clinical and radiological data was collected in patients presenting with signs and symptoms of stroke and confirmed by Computed Tomography (CT) scan/Magnetic Resonance Imaging (MRI) brain as ICH, who were non hypertensive and with age more than 18 years. Clinical outcome of the patients were measured based on Glasgow Coma Scale (GCS), site and volume of haemorrhage. Descriptive statistics of the explanatory and outcome variables were calculated by mean, Standard Deviation (SD) and Chi-square test was applied for qualitative variables.

Results: In the present study 31 (62%) were males, and 19 (38%) were females. The age of patients ranged from 18-85 years. The most common risk factor associated with intracerebral haemorrhage was alcohol consumption 16 (32.0%), followed by smoking 13 (26%), and anticoagulant intake 13 (26%). Most common clinical presentation were hemiplegia/hemiparesis, speech defect, vomiting, convulsion, pupillary defect and cranial nerve involvement, in decreasing frequency. High ICH score and low GCS were poor prognostic factors for outcome of intracerebral haemorrhage patient in the present study. In hospital mortality rate was 28%. During 30 days follow-up, there was 22.2% mortality among the discharged patient.

Conclusion: Although hypertension remains a most common risk factor for intracerebral bleed, other risk factors such as significant alcohol consumption, coagulopathy should also be kept in mind especially in young individual. Non hypertensive haemorrhage usually occurs at sites not typical for hypertensive bleed. All efforts should be directed to establish the aetiological factors for intracerebral bleed, so that appropriate timely therapy can be provided to prevent further morbidity and mortality.

Keywords: Aetiology, Glasgow coma scale, Mortality, Outcome

INTRODUCTION

Intracerebral Haemorrhage (ICH) accounts for 10-15% of total cerebral vascular accident. ICH is less frequent than ischaemic stroke, but has higher mortality and morbidity, it being one of the first causes of severe disability [1]. Non traumatic intracerebral haemorrhage results from rupture of blood vessels in the brain parenchyma. It accounts as a major public health problem with an annual incidence of 10-30 per 100 000 population, accounting for 2 million (10-15%) of about 15 million strokes worldwide each year [2-4]. There are many non hypertensive causes of ICH including Cerebral Amyloid Antipathy (CAA), vacuities, vascular malformations, and the use of anticoagulants, fibrinolysis, antiplatelet agents, underlying co-morbidities like diabetes mellitus, alcohol intake, smoking, drug abuse like cocaine, and finally genetic and ethnicity also play an important role in causation of intracerebral bleed [5-9]. Primary and secondary (anticoagulant induced) intracerebral haemorrhages have similar underlying pathological changes [10]. Hence, the importance of recognising these conditions and need for urgent specific therapy which may play a vital role in therapeutic planning and prevention of ICH [11].

Spontaneous ICH has still remained a serious disease despite attempts at improving outcome by medical and neurosurgical treatment. There are many clinical/neuroradiological parameters like GCS, severity of neurological deficit, site, size, volume of haemorrhage, presence of intraventricular extension, hydrocephalous and others that would predict the outcome of ICH [12,13]. Hence this study was performed to study clinical and radiological findings in non hypertensive intracerebral haemorrhage with an aim to find out the other non hypertensive risk factors and causes for intracerebral haemorrhage, and to determine whether clinical/neuroradiological parameters would predict the outcome of ICH.

MATERIALS AND METHODS

The prospective observational cohort study was conducted in the Department of General Medicine, at Mahadevappa Rampure Medical College Gulbarga, Karnataka, India; from January 2016 to January 2017. The Institutional Ethical Committee (HKES/MRMCK/IEC/17/11/24) clearance was obtained. The study involved 50 subjects with non hypertensive intracerebral haemorrhage.

Inclusion criteria: Patients presenting with signs and symptoms of stroke and confirmed by CT scan/MRI brain as ICH, who were non hypertensive and were more than 18 years of age were included in the study.

Exclusion criteria: Known case of chronic hypertension on antihypertensive medication, patient with ischaemic stroke and traumatic origin of intracerebral haemorrhage were excluded from the study.

Study Procedure

Demographic details, clinical and radiological data were collected. Clinical outcome of the patients were measured based on GCS, site, and volume of haemorrhage.

Initial workup of patients included:

- CT scan head (plain)
- Complete haemogram
- Coagulation profile
- Random Blood Sugar (RBS), Blood urea, Serum creatinine
- Lipid profile
- Other investigations like chest radiograph, MRI brain with Magnetic Resonance Angiography (MRA), echocardiography, liver function tests, were done whenever needed. The following data were extracted by a radiologist from the patient's CT scan, obtained at the time of admission
- Site of haematoma-infratentorial.
- Volume of haematoma by ABC/2 formula. Haematoma volume was calculated by using ABC/2 formula of Kothari RU et al., by plan metric methods, and expressed either as >30 cm^3 or <30 cm^3 [14].

Elements which comprise of ICH score: age, Glasgow Coma Scale (GCS) score at the time of hospital admission, haematoma volume on the initial CT scan (as measured manually using the ABC/2 method by a single examiner, presence of Intraventricular Haemorrhage (IVH) on the initial CT scan, and haematoma origin (either infratentorial or supratentorial). The ICH score was determined by creating a sumscore of points assigned for individual components: GCS (3-4=2, 5-12=1, 13-15=0), haematoma volume (≥30 mL=1, <30 mL=0), presence of IVH (yes=1, no=0), infratentorial origin (yes=1, no=0), and patient age ≥80 (yes=1, no=0). Patient ICH scores ranged from 0 to 5. Increasing points on the ICH Score with increased risk of mortality or decreased likelihood of favorable functional outcome [15].

STATISTICAL ANALYSIS

Descriptive statistics of the explanatory and outcome variables were calculated by mean, Standard Deviation (SD), and Chi-square test was applied for qualitative variables. Data was entered in Microsoft excel and analysed using Statistical Package for the Social Sciences (SPSS) statistical software (SPSS, version 20.0). A p-value <0.05 was considered statistically significant.

RESULTS

The study was conducted on 50 non hypertensive cases of CT scan/MRI brain proven intracerebral haemorrhage. The male to female ratio was 1.6:1. The mean age of study population was 51.51±17.9 years. Maximum number of cases 22 (44%) belonged to the age group of 51-70 years, followed by 31-50 years 14 (28%). Youngest age was 18 years and oldest was 85 years in the study. [Table/Fig-1]. The most common presentation was hemiplegia/hemiparesis 39 (78%), followed by vomiting 29 (58%) and speech defect 29 (58%) [Table/Fig-2].

Age (years)	No. of males n (%)	No. of females n (%)	Total n (%)
18-30	5 (16.1%)	4 (21%)	9 (18%)
31-50	10 (32.2%)	4 (21%)	14 (28%)
51-70	13 (41.9%)	9 (47.4%)	22 (44%)
71-85	3 (9.8%)	2 (10.6%)	5 (10%)
Total	31 (62.0%)	19 (38.0%)	50 (100.0%)
Mean±SD	52.06±16.21	50.52±20.75	51.51±17.9
t-test value and p-value	t=0.292		
	p=0.771		

[Table/Fig-1]: Age and gender wise distribution of cases.

Clinical presentation	No. of males n (%)	No. of females n (%)	Total n (%)
Headache	18 (36.0%)	10 (20.0%)	28 (56.0%)
Vomiting	16 (32.0%)	13 (26.0%)	29 (58.0%)
Convulsions	12 (24.0%)	6 (12.0%)	18 (36.0%)
Speech defect	19 (38.0%)	10 (20.0%)	29 (58.0%)
Pupillary abnormality	11 (22.0%)	5 (10.0%)	16 (32.0%)
Cranial nerve palsy	9 (18.0%)	6 (12.0%)	15 (30.0%)
Hemiparesis/hemiplegia	27 (54.0%)	12 (24.0%)	39 (78.0%)

[Table/Fig-2]: Clinical presentations of cases.

Alcohol consumption 16 (32%) of about 30 to 40 gram for 10 to 15 years followed by smoking a pack year were >20 packs/day were associated with intracerebral bleed. Other factors like intake of anticoagulant were seven cases all were on warfarin (3-4 mg/day) for Cerebral Venous Thrombosis (CVT) and Atrial Fibrillation (AF), antiplatelet intake were six patient for Cerebrovascular Accident (CVA), clotting disorder like factor XIII deficiency was present in one case, other two cases had liver failure due to HbsAg positive and cirrhosis of liver, three patient had immune thrombocytopenic purpura all were female patient, four patients had aneurysmal bleed all were in anterior circulation confirmed by MRA, two had microbleed, one patient had Arteriovenous Malformation (AVM) [Table/Fig-3,4]. Lower the GCS patients had a higher mortality [Table/Fig-5]. RBS level was significantly higher among the patients who died [Table/Fig-6]. The most common site for intracerebral haemorrhage was basal ganglia followed by lobar and least common was brain stem. A total of 100% mortality was seen in brain stem lesion, followed by thalamus [Table/Fig-7].

Risk factors	No. of males n (%)	No. of females n (%)	Total n (%)
Alcohol consumption	16 (100.0%)	0	16 (32.0%)
Smoking	13 (100.0%)	0	13 (26.0%)
Diabetes mellitus	2 (66.7%)	1 (33.3%)	3 (6.0%)
Anticoagulant/antiplatelet	7 (54.0%)	6 (46.0%)	13 (26.0%)
Clotting/bleeding disorder	1 (16.7%)	5 (73.3%)	6 (12.0%)
Aneurysm/MB/AVM	3 (42.8%)	4 (57.1%)	7 (14%)

[Table/Fig-3]: Risk factors according to gender distribution.
AVM: Ateriovenous malformation; MB: Microbleed
χ^2=19.17 p>0.001

Age group (years)	Number of cases	Alcohol n (%)	Smoking n (%)	Anticoagulant/ Antiplatelets n (%)	Clotting/ Bleeding disorder n (%)
18-30	9	2 (12.5%)	1 (7.7%)	2 (15.3%)	2 (33.3%)
31-50	14	4 (25.0%)	6 (46.2%)	5 (38.5%)	3 (50.0%)
51-70	22	9 (56.3%)	5 (38.4%)	5 (38.5%)	1 (16.7%)
71-85	5	1 (6.2%)	1 (7.7%)	1 (7.7%)	0
Total	50	16	13	13	6

[Table/Fig-4]: Risk factors according to age distribution.
χ^2=2.03 p>0.05

GCS	Survived n (%)	Died n (%)	Total n (%)
≤ 8	2 (15.4%)	11 (84.6%)	13 (26.0%)
>8	34 (91.9%)	3 (8.1%)	37 (74.0%)
Total	36 (72.0%)	14 (28.0%)	50 (100.0%)
χ^2-test value	27.93		
p-value	0.001		

[Table/Fig-5]: Distribution of cases based on GCS.
p-value <0.05 considered significant

A total of 4 (8.0%) patients had aneurysmal bleed, and all were seen in anterior circulation- two patients underwent aneurysm clip procedure, 2 (4.0%) had microbleed and 1 (2.0%) patient had

Factors	Survived (n=36) Mean±SD	Died (n=14) Mean±SD	Total cases (N=50) Mean±SD	t-test value	p-value
RBS (mg/dL)	161.44±53.2	233.3±94.8	181.6±73.9	3.41	0.001
Blood urea (mg/dL)	30.58±24.25	38.64±29.4	32.84±25.7	0.99	0.326
Serum creatinine (mg/dL)	1.23±0.93	1.63±1.35	1.34±1.06	1.18	0.242
Serum cholesterol (mg/dL)	172.1±53.1	184.5±71.1	175.56±58.1	0.67	0.543

[Table/Fig-6]: Comparison of biochemical parameters with outcome.
p-value <0.05 considered significant

Site	Survived n (%)	Died n (%)	Total n (%)
Basal ganglia	15 (41.6%)	6 (42.6%)	21 (42.0%)
Lobar	15 (41.6%)	4 (28.6%)	19 (28%)
Thalamus	2 (5.5%)	2 (14.2%)	4 (8%)
Cerebellum	4 (11.1%)	0	4 (8%)
Brain stem	0	2 (7.14%)	2 (4%)
Multiple bleed	2 (5.5%)	0	2 (4%)
Total	38 (72.0%)	14 (28.0%)	52 (100.0%)

[Table/Fig-7]: Comparison of site of bleed with outcome.

AVM. All these patients survived [Table/Fig-8]. Around 3 (6.0%) patients had low platelet count, and were diagnosed with immune thrombocytopenic purpura. They were on steroid and intermittent platelet transfusion, and all of them survived [Table/Fig-9]. A total of 11 (22.0%) cases had intracerebral bleed because of increased Prothrombin Time/International Normalised Ratio (PT/INR) (INR was >3.5) due to warfarin overdose [Table/Fig-9]. As the Intracerebral Haemorrhage (IHC) score increased the mortality also significantly increased. Mortality was seen in those with an ICH score of 4 and 5 [Table/Fig-10]. Telephonic follow-up on 30 days revealed that six patients died-six patients were males and two patients were females. So, the 30 days mortality was 22.2% (all had more than 1 ICH score) [Table/Fig-11].

AVM/microbleed/aneurysm	Survived n (%)	Died n (%)	Total n (%)
Aneurysm	4 (100%)	0	4 (8.0%)
MB	2 (100%)	0	2 (4.0%)
AVM	1 (100%)	0	1 (2.0%)
χ^2-test value	4.86		
p-value	0.041		

[Table/Fig-8]: Comparison AVM/microbleed/aneurysm with outcome.
AVM: Arteriovenous malformation; MB: Microbleed

PT/INR	Survived n (%)	Died n (%)	Total n (%)	Platelet count	Survived n (%)	Died n (%)	Total n (%)
Normal (12.14 sec/1.1)	27 (69.3%)	12 (30.7%)	42 (84.0%)	Normal (1.5-4.5 lakh/ microliter)	33 (70.2%)	14 (29.8)	47 (94%)
Increased (25 seconds/5)	9 (81.8%)	2 (18.2%)	11 (22.0%)	Decreased (less than 1.5 lakhs/ microliter)	3 (100%)	0	3 (6%)
Total	36 (72.0%)	14 (28.0%)	50 (100.0%)	Total	36 (72.0%)	14 (28.0%)	50 (100.0%)
χ^2-test value	0.67			χ^2-test value	0.3		
p-value	0.893			p-value	0.900		

[Table/Fig-9]: Comparison of platelet count and PT/INR ratio with outcome.

ICH score	Survived n (%)	Died n (%)	Total n (%)
1	23 (100.0%)	0	23 (46.0%)
2	8 (100.0%)	0	8 (16.0%)
3	4 (100.0%)	0	4 (8.0%)
4	1 (16.7%)	5 (83.3%)	6 (12.0%)
5	0	9 (100.0%)	9 (18.0%)
Total	36 (72.0%)	14 (28.0%)	50 (100.0%)
χ^2-test value	χ^2=47.26		
p-value and significance	p=0.001		

[Table/Fig-10]: ICH score and mortality of cases.

Age (years)	Survived n (%)	Died n (%)	Total n (%)		No. of males n (%)	No. of females n (%)	Total n (%)
18-30	8 (100%)	0	8 (100%)	Survived	13 (68.4%)	15 (88.2%)	28 (77.7%)
31-50	12 (100%)	0	12 (100%)	Died	6 (31.5%)	2 (11.7%)	8 (22.2%)
51-70	7 (53.8%)	6 (46.1%)	13 (100%)	Total	19 (100%)	17 (100%)	36 (100%)
71-85	1 (33.3%)	2 (66.6%)	3 (100%)	χ^2-test value, p-value	2.03, p=0.06		
t-test, p-value	t=0.286, p<0.05						

[Table/Fig-11]: 30 days follow-up after intracerebral bleed.
p-value <0.05 considered significant

DISCUSSION

In the present study, ICH was highest in the age group 51-70 years (44% of the patients). There was significant gender difference also. The distribution was similar to several other studies [16,17]. The most common presentation was hemiplegia/hemiparesis, followed by vomiting and speech defect. The occurrence of headache (56%), vomiting (58%), and seizures (36%) were comparable with other similar studies [18,19]. Most common factors like alcohol intake, cigarette smoking, anticoagulant/antiplatelet, coagulation disorder, aneurysm, arteriovenous malformation and diabetic's mellitus were associated with intracerebral bleed. This is comparable to other studies [20-23]. It is proposed that high blood glucose at admission contributes to poor outcome, due to exacerbation of cerebral oedema and cerebral damage. A recent meta-analysis by Zheng J et al., in 2018, concluded that hyperglycaemia was associated with poor outcome in patients with ICH as seen present study [24]. The most common site of haematoma was the basal ganglia (42%) followed by, cerebral lobes, thalamus,cerebellum and brain stem (28%, 8%,8% and 4%, respectively), which is in agreement with the pattern described for the non white races in the United States of America (USA) [25].

In present study, based on the GCS, 84.6% of the patients had poor outcome. Poor outcome was associated with lower baseline GCS larger baseline ICH volume, and the presence of IVH at baseline, conforming to a pattern described previously [26-30]. A study from Cincinnati, USA, reported a high sensitivity (96%) and specificity (98%) for the combined ICH volume and baseline GCS score to predict the 30 day mortality [28].

The most important factors predicting the final outcome was the size of haematoma, intraventricular extension, midline shift, GCS score and ICH score. These results indicated that high ICH score, low GCS score at the time of admission, presence of IVH, and midline shift were significantly associated with poor clinical outcome. These results are similar to other studies [15,31].

The ICH still remains a grave medical emergency with high mortality and morbidity. In spite of improvement in outcomes in ischaemic stroke, outcomes in patients with ICH still remain poor with no specific medical treatment and poor consensus and controversial outcomes of surgical intervention [32,33]. The mortality rate in hospital and 30 days in the present study was 28.7% and 22.2% repectively. It was lesser than other studies, where the mortality ranged from 35 to 52%. Variations in mortality could be because of early intervension in the present study, and that it excluded hypertension patients which is an independent risk factor [34-36].

Limitation(s)

This hospital based study from a single center did not aim to provide true prevalence or burden of ICH in the community. The sample size was limited, and the follow-up period of one month allowed only short-term outcome assessment.

CONCLUSION(S)

The Intracerebral Haemorrhage (ICH) has remained a serious disease despite recent improvements in management. So, efforts must be directed towards better understanding and modification of risk factors. ICH had slight affinity towards male sex and mostly occurred after 5th decade of life hemiplegia/hemiparesis, speech disturbances and vomiting were frequent presenting features found in this study. All efforts should be directed to establish the aetiological factors for intracerebral bleed, so that appropriate timely therapy can be provided to prevent further morbidity and mortality.

REFERENCES

[1] Escudero Augusto D, Marqués Alvarez L, Taboada Costa F. Actualización en hemorragia cerebral espontánea [Up-date in spontaneous cerebral hemorrhage]. Med Intensiva. 2008;32(6):282-95.
[2] Qureshi AI, Tuhrim S, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. N Engl J Med. 2001;344:1450-60.
[3] 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.
[4] Sudlow CL, Warlow CP. Comparable studies of the incidence of stroke and its pathological types: Results from an international collaboration. Stroke. 1997;28:491-99.
[5] Feigin VL, Lawes CMM, 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.
[6] Ikram MA, Wieberdink RG, Koudstaal PJ. International epidemiology of intracerebral hemorrhage. Curr Atheroscler Rep. 2012;14(4):300-06.
[7] O'Donnell MJ, Xavier D, Liu L, Zhang H, Chin SL, Rao-Melacini P, et al. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): A case-control study. Lancet. 2010;376(9735):112-23.
[8] Sarwar N, Gao P, Seshasai SR, Gobin R, Kaptoge S, Di Angelantonio E, et al. The Emerging Risk Factors Collaboration. Diabetes mellitus, fasting blood glucose concentration, and risk of vascular disease: A collaborative meta-analysis of 102 prospective studies. Lancet. 2010;375(9733):2215-22.
[9] Martin-Schild S, Albright KC, Hallevi H, Barreto AD, Philip M, Misra V, et al. Intracerebral hemorrhage in cocaine users. Stroke. 2010;41(4):680-84.
[10] Steiner T, Rosand J, Diringer M. Intracerebral hemorrhage associated with oral anticoagulant therapy: Current practices and unresolved questions. Stroke. 2006;37:256-62.
[11] Jha S, Jose M. Non-hypertensive intracerebral haemorrhage: Some interesting observations. J Assoc Physicians India. 2006;54:485-87.
[12] Doifode DV, Ghuge DM, Zanwar SD, Kate SK. Prognostic factors in intracerebral hemorrhages. JAPI. 1998;46:36.
[13] Mitra D, Das SK, Ganguly PK, Roy TN, Maity B, Dutta Munshi AK, et al. Prognostic factors in intracerebral haemorrhage. JAPI. 1995;43:603-04.
[14] Kothari RU, Brott T, Broderick JP, Barsan WG, Sauerbeck LR, Zuccarello M, et al. The ABCs of measuring intracerebral hemorrhage volumes. Stroke. 1996;27(8):1304-05. Doi: 10.1161/01.str.27.8.1304. PMID: 8711791.
[15] Nag C, Das K, Ghosh M, Khandakar MR. Prediction of clinical outcome in acute hemorrhagic stroke from a single CT scan on admission. N Am J Med Sci. 2012;4:463-67.
[16] Das SK, Banerjee TK, Biswas A, Roy T, Raut DK, Mukherjee CS, et al. A prospective community-based study of stroke in Kolkata India. Stroke. 2007;38(3):906-10.
[17] Suthar NN, Patel KL, Saparia C, Parikh AP. Study of clinical and radiological profile and outcome in patients of intracranial hemorrhage. Ann Afr Med. 2016;15(2):69-77.
[18] Mohr JP, Caplan LR, Melski JW, Goldstein RJ, Duncan GW, Kistler JP, et al. The harvard cooperative stroke registry a prospective registry. Neurology. 1978;28:754-62.
[19] Pillai AM. Experience with spontaneous intracerebral haematomas. Journal of the Indian Medical Association. 1988;86:233-36.
[20] Chen CY, Lin PT, Wang YH, Syu RW, Hsu SL, Chang LH, et al. Etiology and risk factors of intracranial hemorrhage and ischemic stroke in young adults. J Chin Med Assoc. 2021;84(10):930-36.
[21] Lioutas VA, Beiser AS, Aparicio HJ, Himali JJ, Selim MH, Romero JR, et al. Assessment of incidence and risk factors of intracerebral hemorrhage among participants in the framingham heart study between 1948 and 2016. JAMA Neurol. 2020;77(10):1252-60.
[22] Senadim S, Cabalar M, Yayla V, Bulut A. The evaluation of the relationship between risk factors and prognosis in intracerebral hemorrhage patients. Ideggyogy Sz. 2017;70(1-2):33-41.
[23] 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):263-66.
[24] Zheng J, Yu Z, Ma L, Guo R, Lin S, You C, et al. Association between blood glucose and functional outcome in intracerebral hemorrhage: A systematic review and meta-analysis. World Neurosurgery. 2018;114:e756-65.
[25] Flaherty ML, Woo D, Haverbusch M, Sekar P, Khoury J, Sauerbeck L, et al. Racial variations in location and risk of intracerebral haemorrhage. Stroke. 2005;36:934-37.
[26] Qureshi AI, Tuhrim ST, Broderick JP, Batjer HH, Hondo H, Hanley DF. Spontaneous intracerebral hemorrhage. N Engl J Med. 2001;344:1450-60.
[27] Wong KS. Risk factors for early death in acute ischemic stroke and intracerebral hemorrhage: A prospective hospital-based study in Asia. Asian acute stroke advisory panel. Stroke. 1999;30:2326-30.
[28] Broderick JP, Brott TG, Duldner JE, Tomsick T, Huster G. Volume of intracerebral hemorrhage: A powerful and easy-to-use predictor of 30-day mortality. Stroke. 1993;24:987-93.
[29] Steiner T, Diringer MN, Schneider D, Mayer SA, Begtrup K, Broderick J, et al. Dynamics of intraventricular hemorrhage in patients with spontaneous intracerebral hemorrhage: Risk factors, clinical impact, and effect of haemostatic therapy with recombinant activated factor VII. Neurosurgery. 2006;59:767-73.
[30] Wang CW, Liu YJ, Lee YH, Hueng DY, Fan HC, Yang FC, et al. Hematoma shape, hematoma size, Glasgow coma scale score and ICH score: Which predicts the 30-day mortality better for intracerebral hematoma? PLoS One. 2014;9(7):e102326.
[31] Hemphill JC 3rd, Farrant M, Neill TA Jr. Prospective validation of the ICH score for 12-month functional outcome. Neurology. 2009;73:1088-94.
[32] Gregson BA, Broderick JP, Auer LM, Batjer H, Chen XC, Juvela S, et al. Individual patient data subgroup meta-analysis of surgery for spontaneous supratentorial intracerebral hemorrhage. Stroke. 2012;43:1496-04.
[33] Sacco S, Marini C, Toni D, Olivieri L, Carolei A. Incidence and 10-year survival of intracerebral hemorrhage in a population-based registry. Stroke. 2009;40:394-99.
[34] Zia E, Engström G, Svensson PJ, Norrving B, Pessah-Rasmussen H. Three-year survival and stroke recurrence rates in patients with primary intracerebral haemorrhage. Stroke 2009;40:3567-73.
[35] Flaherty ML, Haverbusch M, Sekar P, Kissela B, Kleindorfer D, Moomaw CJ, et al. Long-term mortality after intracerebral hemorrhage. Neurology. 2006;66:1182-86.
[36] Bhatia R, Singh H, Singh S, Padma MV, Prasad K, Tripathi M, et al. A prospective study of in-hospital mortality and discharge outcome in spontaneous intracerebral hemorrhage. Neurol India. 2013;61(3):244-48.

PARTICULARS OF CONTRIBUTORS:
1. Assistant Professor, Department of General Medicine, SDM Medical College, Sattur, Dharwad, Karnataka, India.
2. Professor, Department of General Medicine, MRMC Medical College, Gulbarga, Karnataka, India.
3. Postgraduate, Department of General Medicine, SDM Medical College, Sattur, Dharwad, Karnataka, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Siddaganga,
Hallikeri Farm, Vinayak Nagar, Kelgeri Raod, Dharwad-58000, Karnataka, India.
E-mail: drsiddaganga@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Feb 06, 2022
- Manual Googling: May 23, 2022
- iThenticate Software: Jul 09, 2022 (24%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was Ethics Committee Approval obtained for this study? Yes
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. NA

Date of Submission: **Feb 02, 2022**
Date of Peer Review: **Apr 04, 2022**
Date of Acceptance: **May 31, 2022**
Date of Publishing: **Aug 01, 2022**