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Original Article

Study of Hypertensive Intracerebral Hemorrhage

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Abstract

Introduction: Cerebrovascular accident is one of the most common leading causes of morbidity and mortality in the world. Arterial occlusion with subsequent near brain infarction and arterial rupture with subsequent hemorrhage is the two important types of cerebrovascular accidents.

Aim: The aim of the study was to correlate the clinically presumed hypertensive intracerebral hemorrhage site with C.T. scan findings and prognostic factors.

Materials and Methods: The study was conducted at the medical wards of Mount Zion Medical College Hospital, Chayalode, Adoor, Pathanamthitta District, Kerala. All the patients admitted in medical wards with a clinical diagnosis of hypertensive intracerebral hemorrhage were taken for study.

Results: Of 50 patients, 30 patients were male, and 20 patients were female. In 50 patients, 15 patients were below 50 years, 13 patients were aged between 51 and 60 years, based on the site and size of hemorrhage, 43 patients had a large hemorrhage, and 7 patients had a small hemorrhage. Based on hemorrhage, 24 patients had hemorrhage site in basal ganglia, 2 patients in the brain stem, 7 patients in the thalamus, 1 patient in the cerebellum, and 8 patients in the cortex, 6 patients in multiple areas, and 2 patients in intraventricular areas. Based on complications, 23 patients had intraventricular extension/subarachnoid extension, 12 patients had a midline shift, and 4 patients had hydrocephalus. Thirty-four died among these patients 24 patients died due to intracranial pressure, 5 patients due to aspiration pneumonia, 3 patients because of septicemia 1 patient had atrial fibrillation, and 1 patient had aspiration pneumonia and septicemia.

Conclusion: From this study, we concluded that the death rate was more common in the higher age group, and patients had other medical problems. The correct sites of hypertensive intracerebral hemorrhage were made clinically in 52% of the patients. Small hemorrhages were associated with better prognosis, whereas large hemorrhages were associated with poor prognosis.

Key words: Intracerebral, Hemorrhage, Hypertension, Computed tomography scan

INTRODUCTION

Cerebrovascular accident is one of the most common leading causes of morbidity and mortality in the world. Arterial occlusion with subsequent near brain infarction and arterial rupture with subsequent hemorrhage is the two important types of cerebrovascular accidents.[1,2]

Intracerebral hemorrhage is one of the most common neurological emergencies encountered in medical wards apart from cerebrovascular infarction. Many etiological factors such as hypertension, aneurysms, arteriovenous malformations, and arteritis, which lead to intracerebral hemorrhage.[3-5]

Hemorrhage may be described as massive, small, slit, and petechial. Massive refers to hemorrhage several centimeters in diameter; small applies to those 1–2 cm in diameter; and slit that lies subcortically at the junction of white and gray matter. The most common sites of hypertensive hemorrhage are (1) the putamen and adjacent internal capsule (50%), (2) various parts of central white matter (lobar hemorrhage), (3) thalamus, (4) cerebellar hemisphere, and (5) pons. The vessels involved are usually a penetrating artery.[6]

The hemorrhages at these various sites produce specific clinical features, by identification of which, a fairly good

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idea about the site of hemorrhage can be made. This study is aimed at such a clinical localization of hemorrhage site with the available signs and to correlate the presumed clinical site of hemorrhage with the site of hemorrhage in the computed tomography (C.T.) scan picture.

Increase in intracranial pressure with consequent brainstem compression, secondary medical complication such as aspiration pneumonia, septicemia, and deep venous thrombosis and worsening of underlying other medical disorders such as diabetes mellitus, cardiac failure, and renal failure are some of the reasons for mortality in hypertensive intracerebral hemorrhage.

Aim

The aim of the study was to correlate the clinically presumed hypertensive intracerebral hemorrhage with C.T. scan findings and prognostic factors.

MATERIALS AND METHODS

The study was conducted at the medical wards of Mount Zion Medical College Hospital, Chayalode, Adoor, Pathanamthitta District, Kerala. All the patients admitted in medical wards with a clinical diagnosis of hypertensive intracerebral hemorrhage were taken for study. Inclusion criteria include patients who had a history of hypertension in the past whether treated or untreated, patients who did not have a history of hypertension in the past but who were found to be hypertensive at the time of admission and hypertensive end-organ changes like retinal vascular changes, all forms of hypertension with intracerebral hemorrhage such as essential hypertension and renal hypertension were taken for study. Exclusion criteria include patients who suffered from some form of cerebrovascular accidents in the past, patients who were not found to be hypertensive at the time of admission and patients who did not have a history of hypertension.

A detailed history was taken from all selected patients with particular emphasis on hypertension's duration, nature of the treatment taken, and whether they were taking it regularly or not. The presence of other major diseases such as diabetes mellitus, ischemic heart disease, renal disease, and left ventricular failure were also enquired. A detailed history of the present episode of intracerebral hemorrhage was taken with emphasis on the following factors, time of onset, what the patient was doing at the time of stroke, loss of consciousness, occurrence of convulsions, paucity of limb movements, and history of vomiting and headache. A clinical diagnosis of intracerebral hemorrhage was made, when the cerebrovascular accident had occurred during day time or work, with immediate, profound loss of consciousness associated with or without vomiting or convulsions with clinical features fitting into hemorrhage at well-defined sites such as putamen, thalamus, pons, cerebellum, and cortex.

A clinical diagnosis of site of bleed and possible complications was made. Other findings such as loud A2, heaving apical impulse, electrocardiogram criteria for left ventricular hypertrophy (LVH), X-ray findings for LVH, and fundus changes were noted. Complete basic investigations were done in all the patients. After confirmation of hemorrhage by C-T scan, all the patients were treated with a standard regimen.

While these patients were being treated on these lines, they were examined daily for the following – state of consciousness, improvement or further deterioration of neurological deficit, control of hypertension, futures of increased intracranial tension, complications, particularly (a) bedsores, (b) pneumonitis, (c) urinary tract infection, (d) deep vein thrombosis, (e) metabolic and electrolytic disturbances, and others.

The following factors which are known to influence the outcome of hypertensive intracerebral hemorrhage were studied. These are age, sex, hypertension – duration, treated or untreated, admission blood pressure, admission blood sugar level, and presence of other medical diseases. The size and other hemorrhage consequences such as intraventricular extension, midline shift, and hydrocephalus in CT scan picture and complications.

RESULTS

Of 50 patients, 30 patients were male, and 20 patients were female [Table 1].

Of the 50 patients, 15 patients were below 50 years, 13 patients were aged between 51 and 60 years, 17 patients were aged between 61 and 70 years, and 5 patients were aged above 70 years [Table 2].

Of 50 patients, 43 patients had a large hemorrhage and 7 patients had a small hemorrhage. Based on hemorrhage, 24 patients had hemorrhage site in basal ganglia, 2 patients in the brain stem, 7 patients in the thalamus, 1 patient in the cerebellum, 8 patients in the cortex, 6 patients in multiple areas, and 2 patients in intraventricular areas [Table 3].

Based on complications, 23 patients had intraventricular extension/subarachnoid extension, 12 patients had a midline shift, and 4 patients had hydrocephalus [Table 4].

Out of 50 patients, 34 were died among these patients, 24 patients died due to intracranial pressure, 5 patients due to aspiration pneumonia, 3 patients because of septicemia, 1 patient had atrial fibrillation, and 1 patient had aspiration pneumonia septicemia [Table 5].

DISCUSSION

In our study, more patients died in the age group of 50–70 years (75%), whereas survival was better in patients aged less than 50 years. Similar observations were made by Whisnant *et al.* in 2009, Douglas; Hearer in 2010, Kwak *et al.* in 2010, and Banet; 1994.[7-10] Death rate is equal in both male and female sexes. Hence, sex alone is not a prognostic indicator.

In our study, the majority had severe hypertension and patients' higher death was among the severe hypertension patients. Suzuki, Kelley, Duncan in 2014 observed that persistent inadequate blood pressure control adversely affects the prognosis in hypertensive intracerebral hemorrhage.[11] Proper control of hypertension with regular medical treatment will reduce hypertension related vascular damage. This is also observed by authors such as Douglas and Hearer in 1982 and Feschi *et al.* in 1977.[8,12]

The presence of hyperglycemia alone is not a bad prognostic factor. Diabetes mellitus may be a contributory factor in causing death by predisposing the patient to infection and consequent septicemia. Diabetes exerting bad prognosis was earlier observed in their studies by Kadoya, Susuki in 1983, Caplan *et al.* in 2018.[8,9,13]

The presence of underlying cardiac disease can be taken as a bad prognostic factor as the cardiac disease itself can lead to death irrespective of the cerebral complications. A similar observation was made by Caplan in 2005, Feldman in 2011, and Furlan *et al.* in 1979.[7,13-15]

If the size of the bleeding is small chances of survival is more. Only two out of seven died, and death was not due to cerebral cause but due to aspiration pneumonia, so prevention of complication is one of the main principles in managing intracerebral hemorrhage. Ojemann Heros observed large hemorrhages and multiple hemorrhages exerting bad prognosis. R.C. in 2005, Feldman E in 1991, and Susuki, Kelley in 1995 observed that chances of death from brain stem hemorrhage are more with any hemorrhage than 50 ml in volume.[11,14,16]

In CT scan, midline shift alone cannot be taken independent prognostic indicator because midline shift can occur in the following ways: Large cortical or subcortical hemorrhages may increase the intracranial pressure very much to produce massive cerebral edema which can shift the midline structures and hemorrhages small or large near the midline can also directly shift the midline to the opposite side. Hence, in C.T. scan, the prognostication of the midline shift's presence should not be considered alone and independently. It has to be considered along with other features in C.T. such as the size of the hemorrhage, site of hemorrhage, and associated cerebral edema.

As a consequence of intraventricular hemorrhage, adhesion may form in the cerebrospinal fluid (CSF) pathway, obstructing the CSF flow causing stagnation of CSF and dilatation of ventricle, increasing the intracranial pressure which will have an adverse effect on the outcome. Hence, the

Table 1: Sex distribution

S. No.	Sex	No. of. patients
1	Male	30
2	Female	20

Table 2: Age distribution

S. No.	Age (years)	No. of. patients
1	Below 50	15
2	51–60	13
3	61–70	17
4	Above 70	5

Table 3: Site and size of hemorrhage distribution

S. No.	Large hemorrhage		Small hemorrhage	
	Site	No. of patients	Site	No. of patients
1	Basal ganglia	21	Basal Ganglia	3
2	Brian stem	0	Brain stem	2
3	Thalamus	6	Thalamus	1
4	Cerebellum	0	Cerebellum	1
5	Cortex	8	Cortex	0
6	Multiple areas	6		
7	Intraventricular	2		

Table 4: Complications

S. No.	Complications	No. of patients
1	Intraventricular extension/subarachnoid extension	23
2	Midline shift	12
3	Hydrocephalus	4

Table 5: Causes of death

S. No.	Causes	No. of Patients
1	Intracranial pressure	24
2	Aspiration pneumonia	5
3	Septicemia	3
4	Aspiration pneumonia+Septicemia	1
5	Others (atrial fibrillation)	1
6	Total	34

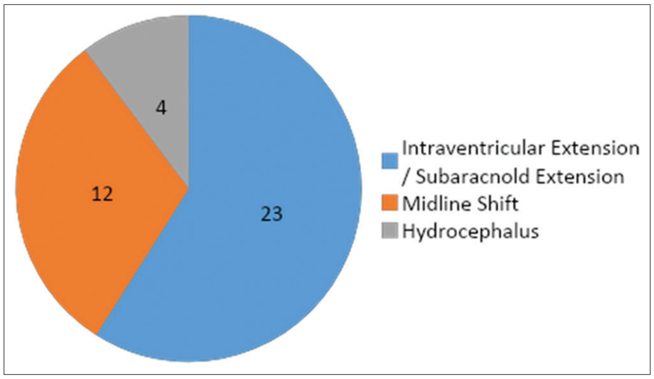

Figure 1: Complications

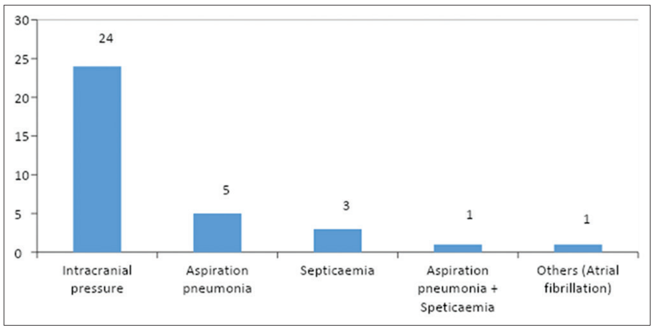

Figure 2: Causes of death

presence of dilated ventricles in C.T. scan can be taken as a bad prognostic indicator. Broderick JP opined from his studies that displacement of ventricles and or early hydrocephalus were associated with poor prognosis [Figure 1].[17]

Aspiration pneumonia, septicemia, urinary tract infection, bedsores, and cardiac arrhythmias were some of the extracerebral complications observed from the study. Few patients died exclusively of these complications. These complications usually occur in the 2nd week, whereas death due to increased intracerebral pressure occurred earlier in the 1st week. These complications were usually associated with other underlying medical problems such as diabetes mellitus, renal disease, and cardiac disorders. Thus, the occurrence of complications like the above may be taken as harmful prognostic factors. These complications can influence prognosis adversely. Those were also described earlier by Caplan L, 2005, Doug; Hearer, 2016, Feschi, Carolet, Fiorelli in 2000, Kwak, Kadoya, Susuki in 2005, and Whisnant *et al*. in 2005 [Figure 2].[7-9,12,13]

CONCLUSION

This study concluded that the death rate was more common in higher age groups and patients had other medical problems. The correct sites of hypertensive intracerebral hemorrhage were made clinically in 52% of the patients. Small hemorrhages were associated with better prognosis, whereas extensive hemorrhages were associated with poor prognosis.

REFERENCES

1. Krishnamurthi RV, Moran AE, Feigin VL, Barker-Collo S, Norrving B, Mensah GA, *et al*. Stroke prevalence, mortality and disability-adjusted life years in adults aged 20-64 years in 1990-2013: Data from the global burden of disease 2013 study. Neuroepidemiology 2015;45:190-202.
2. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, *et al*. Heart disease and stroke statistics-2017 update: A report from the American heart association. Circulation 2017;135:e146-603.
3. Broderick JP, Adams HP Jr., Barsan W, Feinberg W, Feldmann E, Grotta J, *et al*. Guidelines for the management of spontaneous intracerebral haemorrhage: A statement for healthcare professionals from a special writing group of the stroke council, American heart association. Stroke 1999;30:905-15.
4. Fang MC, Chang Y, Hylek EM, Rosand J, Greenberg SM, Go AS, *et al*. Advanced age, anticoagulation intensity, and risk for intracranial haemorrhage among patients taking warfarin for atrial fibrillation. Ann Intern Med 2004;141:745-52.
5. Del Zoppo GJ, Mori E. Hematologic causes of intracerebral haemorrhage and their treatment. Neurosurg Clin North Am 1992;3:637-58.
6. 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;36:934-7.
7. Whisnant JP, Basford JR, Bernstein EF, Cooper ES, Dyken ML, Easton JD, *et al*. Classification of cerebrovascular diseases III. Stroke 1990;21:637-76.
8. Douglas MA, Haerer AF. Long-term prognosis of hypertensive intracerebral haemorrhage. Stroke 1982;13:488-91.
9. Kwak R, Kadoya SA, Susuki T. Factors affecting the prognosis in thalamic haemorrhage. Stroke 1983;14:493-500.
10. Banet GA. Stroke in young adults: A retrospective analysis.. J Vasc Neurosurg 1994;12:101-5.
11. Dandapani BK, Suzuki S, Kelley RE, Reyes-Iglesias Y, Duncan RC. Relation between blood pressure and outcome in intracerebral haemorrhage. Stroke 1995;26:21-4.
12. Feschi C, Carolei A, Fiorelli M. Changing prognosis of primary intracerebral hemorrhage: Results of a clinical and computed tomographic follow-up study of 104 patients. Stroke 1988;19:192-5.
13. Caplan L. Intracerebral haemorrhage revisited. Neurology 1988;38:624-7.
14. Feldman E. Intracerebral haemorrhage. Stroke 1991;22:684-91.
15. Furlan AJ, Whisnant JP, Elveback LR. The decreasing incidence of a primary intracerebral haemorrhage population study. Ann Newrol 1979;5:367-73.
16. Ofemann RG, Heros RC. Spontaneous brain haemorrhage. Stroke 1983;14:468-75.
17. Broderick JP, Brott TG, Tomsick T, Barsan W, Spliker J. Ultra early evaluation of intracerebral haemorrhage. J Neurosurg 1990;72:195-9.

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