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RESEARCH ARTICLE

Computed Tomography Evaluation of Spontaneous Intracranial Hemorrhages

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ABSTRACT

Introduction: To describe the etiology and spectrum of findings on computed tomography in patients with spontaneous intracranial hemorrhages and study the correlation of volume and site of intracranial hemorrhages with clinical outcome at the time of discharge. Neuroimaging is essential for the treating physician to identify the cause of hemorrhage and to understand the location and severity of hemorrhage, the risk of impending cerebral injury, and to guide often emergent patient treatment. CT evaluation of intracranial hemorrhage was done with the goal of providing a broad overview of the diverse causes and varied appearances of intracranial hemorrhage.

Material and Methods: It is a prospective observational study conducted on 100 cases of spontaneous intracranial hemorrhages referred to the department of Radiodiagnosis, Shri Ram Murti Smark Institute of Medical Sciences (SRMS IMS) in between November 2019 to April, 2021. Using 32 slice computed tomography (CT) scan and 128 dual source CT scan, helical CT sections of brain (1-mm thickness) were obtained with volume acquisition from base of skull upto vertex, parallel to orbito-meatel line (30°angulation) and volume and site of intracranial hemorrhage, Intraventricular and Subarachanoid extension of intracerebral hemorrhage, Perilesional oedema and mass effect like midline shift was evaluated. The risk factors associated with intracranial hemorrhages, symptomatology and clinical outcomes in terms of Glasgow coma scale on discharge, need for Intensive care unit admission and mortality were also studied.

Results: Majority of the cases ie. 35.16% of patients, had basal ganglia bleed, followed by 24.17% cases of lobar bleed, whereas 6.59% and 7.69% of patients had cerebellum and brainstem bleed, respectively. 12.08% cases had a bleed in the thalamus. No patient in the study group had Intracranial hemorrhage Score 6. Only 5.55% cases with Score 0 deteriorated against 94.44% of those who improved and this difference was observed to be statistically significant (p-value 0.00039). There was a statistically significant observed for GCS Score < 8, IVH with p-value < 0.0001 and 0.0072, respectively.

Conclusion: A diagnostic flowchart that incorporates neuroimaging, size, location, volume, clinical risk factors, presenting complaints along with a careful assessment of age, and pathogenesis, when considered together, can lead to a more accurate diagnosis and, thus, a better management of intracranial hemorrhages. Regarding neuroimaging, CT provides an excellent imaging modality for early detection and proper management of intracranial hemorrhage which definitely improves patients' functional outcome, life expectancy, as well as well-being of the patient.

Keywords: Computed Tomography Scan, Spontaneous Intracranial hemorrhage, Intracerebral hemorrhage, Subarachnoid hemorrhage, Intraventricular hemorrhage.

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INTRODUCTION

Spontaneous intracranial hemorrhage refers to non-traumatic bleeding within the cranial vault including the brain parenchyma and surrounding meningeal spaces.[1] Spontaneous intracranial hemorrhage can affect the different intracranial compartments; the epidural space (epidural hematoma), the subdural space (subdural hematoma), the subarachnoid space (subarachnoid hemorrhage), as well as the brain parenchyma (intracerebral hemorrhage).

Intracerebral hemorrhages refers to non-traumatic extravasation of blood into the brain parenchyma, resulting from rupture of blood vessels, with potential to extend into the ventricles and the subarachnoid space.[2] It has been identified as the subtype of stroke that accounts for 10–50% of all cases and associated with poorest prognosis3 and is being identified as a major public health problem, with an annual incidence of 10-30 per 100,000 population and comprising 18% of the hospital admission.[2]

More than 85% of Intracerebral hemorrhages occurs

as a primary event.[1] Around 60% - 70% of primary intracerebral hemorrhage is hypertension related likely to involve deep structures.[1] However, in the elderly population, amyloid angiopathy accounts for up to one-third of the cases of primary hemorrhage and usually affects lobar regions. Secondary Intracerebral hemorrhages can be related to multiple causes like trauma, coagulopathy, arteriovenous malformation Intracranial aneurysm, dural venous sinus thrombosis and vasculitis.[4]

It is a devastating disease associated with high rates of morbidity and mortality.[4] The most common sites of a cerebral hemorrhage are the Putamen and the adjacent internal capsule (50%), Central White Matter of temporal, parietal and frontal lobes, thalamus, cerebellar hemisphere and the Pons.[5]

Neuroimaging has emerged as an essential tool for the treating physician to understand the location and volume of hemorrhages, assess the risk of impending cerebral injury, and formulate a plan for emergent patient treatment.

Enzmann et al., classified the evolution of intracerebral hemorrhages into four stages: acute (days 1 -3), subacute (days 4-8), capsule (days 9-13), and organization (days 13 and after).[6] The sequence of CT changes closely correlates with the neuropathologic findings.

Before the advent of the computed tomography, the main stay of diagnosis of intracerebral hemorrhages was mainly based upon clinical expertise, plain skull roentgenograms, cerebral angiography and isotope brain scans. With the advent of computed tomography in 1972, accurate and early diagnosis of hemorrhages has been achieved.

A cranial computed tomographic scan confirms the diagnosis, estimates hematoma volume and identifies mass effect and intraventricular extension. The bleeding pattern indicates the potential cause of the intracerebral hemorrhage, and the extravasation of contrast into the hematoma predicts hematoma expansion.[4] Within 6 hours of symptom onset, the sensitivity of computed tomographic scan is observed to be 100% with a negative predictive value of 100%.[1]

MRI is as sensitive as computed tomography for acute detection of ICH and superior in identifying perihematomal edema but is most frequently used as a follow-up investigation not as initial assessment, because of limited availability and logistical barriers. Also, blood is not easily detectable in both T1- weighted and T2-weighted MRI sequences in the acute setting probably because the generation of de-oxy-hemoglobin with paramagnetic properties is delayed.[1]

Here we reviewed the imaging of intracerebral hemorrhages with the goal of providing a broad overview of the diverse causes and varied appearances of intracranial hemorrhages. The use of computed tomography is emphasized as it is the most commonly performed technique in the emergency evaluation of patients with suspected or known intracranial hemorrhages. To describe the etiology and spectrum of findings on Computed Tomography in patients with Spontaneous Intracranial Hemorrhages and study the correlation of volume and site of Intracranial Hemorrhages with clinical outcome at the time of discharge.

MATERIAL AND METHODS

It is a prospective observational study conducted on 100 cases of spontaneous intracranial Hemorrhages referred to the department of Radiodiagnosis, Shri Ram Murti Smark Institute of Medical Sciences (SRMS IMS) in, between November 2019 to April, 2021.

Inclusion Criteria

- Patients above 18 years of age with intracranial hemorrhages that are spontaneous in origin.

Exclusion Criteria

- Trauma-induced intracranial hemorrhages.
- Previous Neurosurgery patients.
- Patients with a previous history of stroke.

Using Somatom Scope 32 slice CT scan (Siemens Medical Solution) and Somatom definition flash 128 dual source CT scan (Siemens Medical Solutions), Helical CT Sections of brain (1 mm thickness) were obtained with volume acquisition from base of skull upto vertex, parallel to orbito-meatel line (30°angulation) and volume and site of intracranial hemorrhages, Intraventricular and subarachanoid extension of intracerebral hemorrhages, Perilesional edema and mass effect like midline shift was evaluated.

The risk factors associated with intracranial hemorrhages, symptomatology and clinical outcomes in terms of GCS Score7 on discharge, ICH Score8, need for ICU admission and mortality were also studied and the

Table 1: Baseline Characteristics among the Study Population

Age (years)	55.62 ± 15.0
Body Mass Index (kg/m^2)	24.65 ± 3.29
Sex	Male (64%)
Socio-economic Status	Lower Class (60%)
Common Risk Factor	Hypertension (85%)
Common Symptoms	Loss of Consciousness, and Limb Weakness (55%)

Table 2: Distribution of Patients based on Location of Intraparenchymal bleed

Location of ICH	No of Patients (n=91)	Percent (%)
Basal Ganglia	32	35.16%
Thalamus	11	12.08%
Thalamo – Ganglia	13	14.29%
Lobar	22	24.17%
Cerebellum	6	6.59%
Brainstem	7	7.69%
Total	91	100.0%

results were analyzed on software IBM SPSS version 20.0.

RESULTS

Among the study population, the majority of cases belonged to the male population (64%), with the mean age 55.62 ± 15.0 years, and BMI 24.65 ± 3.29 kg/m2, belonging to lower socioeconomic status.

The common risk factors associated with spontaneous Intracerebral Hemorrhages were hypertension (85%), alcohol dependence (51%), smoking (39%), hyperlipidemia (36%) and intracranial aneurysm (13%).

55% of the patients had history of loss of consciousness and limb weakness while 49% cases had history of nausea/vomiting and 47% cases complained of headache. Only 11% cases had episodes of seizures.

Of the total patients, 91% cases had spontaneous intraparenchymal hemorrhages and the rest 9% had spontaneous subarachnoid hemorrhages. None had epidural or subdural hemorrhages or isolated intraventricular hemorrhages.

Of the 91 cases of intraparenchymal bleed, 75.83% cases were of non-lobar bleed against 24.17% cases of lobar bleed. 85.71% had supratentorial location of bleed against 14.29% cases with infratentorial bleed.

Majority of the cases ie. 35.16% of patients had basal ganglia bleed, followed by 24.17% cases of lobar bleed, whereas 6.59% and 7.69% of patients had cerebellum and brainstem bleed, respectively. 12.08% cases had bleed in thalamus.

The maximum volume of bleed observed in our study population was 190 cc with the mean volume of 60.12 ± 48.95 cc while the minimum volume of bleed was seen in thalamic bleed ie. 0.35 cc with the mean of 8.68 ± 6.84 cc.

A total of 17 cases among the 91 cases of intraparenchymal bleed had subarachnoid extension, with maximum number of cases among cerebellar bleed (66.67%) and no case of subarachnoid hemorrhages was seen in subjects with brainstem bleed and 35 cases had intraventricular bleed, among which majority of cases (61.53%) were from thalamo-gangliar bleed while 18.75% cases from basal ganglia bleed had intraventricular extension. Maximum number of cases (72.72%) from lobar bleed had midline shift followed by thalamo-gangliar bleed (69.23%). No case of brainstem bleed had any midline shift.

Perilesional edema was most commonly seen in cases with thalamus bleed ie. (81.25%) cases and least common among cases with brainstem bleed ie. only 57.14% cases.

Among 91 patients of intraparenchymal bleed, 15 patients had volume < 5 cm3, out of which 6.67% patients had subarachnoid extension and midline shift each and 26.67% had ventricular extension while 32 patients had volume > 30 cm3, among which 21.88% and 56.25% cases had subarachnoid and ventricular extension respectively while 84.37% cases had midline shift. However, this difference was not statistically significant (p-value 0.4733).

Among the 3 patients with Intraparenchymal bleed who expired, the mean volume of hematoma was 47.53 ± 11.06 cm3 against 17.63 ± 11.98 cm3 among those who survived, and this difference was found to be statistically significant (p-value 0.036).

The volume of bleed among patients with GCS between 4-8 was found to be 45.72 ± 34.56 cm3 against 18.83 ± 10.16 cm3 among patients with GCS between 13-15. 90.41% cases among those with intraparenchymal bleed were managed conservatively while 9.52% underwent surgical procedures. 87.53% cases of subarachnoid

Table 3: Distribution of Patients based on Volume of Intraparenchymal bleed

Location of ICH	Volume of bleed (n=91)		Mean ± SD (cc)
	Minimum (cc)	Maximum (cc)	
Basal Ganglia (32)	3.08	87	23.04 ± 21.82
Thalamus (11)	0.35	22	8.68 ± 6.84
Thalamo – Ganglia (13)	6.3	116	31.06 ± 30.38
Lobar (22)	2	190	60.12 ± 48.95
Cerebellum (6)	0.93	36.6	14.58 ± 12.42
Brainstem (7)	1	15.54	8.01 ± 5.25

Table 4: Relationship of Volume of Hematoma with Extension of Hematoma

Relationship between Volume of Hematoma with Extension of Hematoma				
Volume (cm3)	Cases (n)	Subarachnoid extension	Ventricular Extension	Midline Shift
< 5	15	1 (6.67%)	4 (26.67%)	1 (6.67%)
5.1 - 15	32	4 (12.50%)	7 (21.87%)	11 (34.37%)
- 30	12	5 (41.66%)	6(50%)	6 (50%)
>30	32	7(21.88%)	18 (56.25%)	27 (84.37%)
Total	91	17 (18.68%)	35 (38.46%)	45 (49.45%)
P Value	0.4733			

Table 5: Comparison of Clinical Outcome among Patients with Intraparenchymal Hemorrhages on the basis of GCS Score, IVH and Midline Shift

Comparison of Clinical Outcome among Patients with Intraparenchymal Hemorrhages based on GCS Score, IVH and Midline Shift					
Parameters	No of Patients (n=91)	Improved	Deteriorated	Mortality	P Value
GCS Score(At the time of discharge)					
< 8	32	1 (3.45%)	28 (96.55%)	3 (9.37%)	
> 8	59	51 (86.44%)	8 (13.56%)	0	< 0.0001
Total	91	52 (59.09%)	36 (40.91%)	-	
IVH					
Present	35	12 (34.28%)	23 (60.53%)	1 (2.63%)	
Absent	56	38 (71.70%)	18 (32.14%)	2 (3.77%)	0.0072
Total	91	50 (54.94%)	41 (45.05%)	3 (3.29%)	
Midline Shift					
Present	45	22 (48.88%)	24 (53.33%)	2 (4.44%)	
Absent	46	27 (58.69%)	16 (34.78%)	1 (2.17%)	0.32
Total	91	49 (53.84%)	40 (43.95%)	3 (3.29%)	

hemorrhages were managed surgically while 12.50% were managed conservatively.

Majority of cases i.e. 96.55% cases with GCS Score < 8 deteriorated while only 13.56% cases with GCS > 8 deteriorated against 86.44% who improved and this difference was statistically significant (p-value < 0.0001).

While comparing the patients with Intraventricular hemorrhages, 60.53% cases with Intraventricular hemorrhages deteriorated with mortality of 2.63% while majority patients with absent Intraventricular hemorrhages i.e. 71.7% showed improvement. However, mortality in this group was 3.77% and this difference was found to be statistically significant (p-value 0.0072).

When studying the patients with midline shift on Computed Tomography, 53.33% with midline shift deteriorated and 4.44% cases expired while majority 58.69% cases with no midline shift improved against 34.78% cases who deteriorated with 2.17% mortality. However, this difference was not statistically significant (p-value 0.32).

None patient in the study group had ICH Score 6. Among 4 and 2 patients with ICH Score 4 and 5 respectively, none improved. 50% cases with ICH Score 3 deteriorated while only 5.55% cases with Score 0 deteriorated against 94.44% of those who improved and this difference was observed to be statistically significant (p-value 0.00039).

DISCUSSSION

Spontaneous intracerebral hemorrhages, second most common subtype of stroke, is defined as non-traumatic bleeding into the brain parenchyma. This has reported around 5.3 million cases and over 3 million deaths worldwide in 2010.3 Case fatality is extremely high reaching approximately 60% at 1-year post-event. Among the patients who survive, only 20% cases are independent within 6 months.[3]

In our study, we found that among the patients with intraparenchymal hemorrhages, out of 91 patients, 32 patients had basal ganglia bleed (35.16%) followed by 22 cases of lobar bleed (24.17%), 11 cases of thalamic bleed (12.08%), 13 patients of thalamo-ganglia bleed (14.29%), 6 patients of cerebellar bleed (6.59%), 7 patients of brainstem bleed (7.69%).

Dabilgou et al. (2019), in his study to determine the prevalence, clinical profile, causes, and mortality risk factors of spontaneous subarachnoid hemorrhages, concluded that spontaneous subarachnoid hemorrhages accounted for 3.2% of all stroke. The mean age of patients was 60 years (range 20-93 years). There was a female predominance in 55.9%. The common vascular risk factors were hypertension (79.7%) and chronic alcohol consumption (16.9%). The main symptoms were headache (76.2%), motor weakness (74.5%), and consciousness disorders (62.7%). Hypertension was the most common cause of spontaneous subarachnoid hemorrhages.[9]

Tangella et al (2020), in a prospective cohort study on 80 patients with mean age 56.25 + 14.3 years (72.5% were male and 27.5% were female) with Intra Cranial Hemorrhages subjected to CT scan brain to locate the site and volume of hemorrhages concluded Hypertension to be one of the commonest risk factor for ICH (80%) and also stated that the volume of bleed more than 60 cc was associated with 100% mortality. He found that majority of the patients presented with ganglio – capsular hemorrhages (64%), however, cerebellar and lobar hemorrhages had mortality of 66.7% and 100% respectively.[10]

Hegde et al (2020), studied 905 patients including 638 males and 267 females with the mean age at presentation 58.10 ± 12.76 years with 523 patients (57.8%) diagnosed hypertensive. The most frequent locations of hematoma were basal ganglia (478), thalamus (202), lobar (106), cerebellar (61), brainstem (31), and primary intraventricular hemorrhages (27). The mean volume of the clot on admission was 23.45 ± 19.79 ml, and clot progression was seen in 5.08% cases while secondary intraventricular extension was seen in 425 (47%) patients. Surgical evacuation (craniotomy) was done in 147 (16.8%) patients, and external ventricular drainage was placed in 56 (6.2%) patients. It was observed that the overall 3–month mortality was 30.1% (266 patients) and hence concluded that spontaneous intracerebral hemorrhages predominantly affects a younger population in India in comparison to the Western society. Elderly age,

poor GCS on admission, clot volume above 30 ml and intraventricular extension remain the most consistent predictors of death and poor outcome.[11]

CONCLUSION

We conclude that a diagnostic flowchart that incorporates size, location, clinical risk factors, presenting complaints along with a careful assessment of age, and pathogenesis, when considered together, can lead to a more accurate diagnosis and, thus, a better management of Intracranial hemorrhages. Regarding neuroimaging, CT provides an excellent imaging modality for early detection and proper management of intracranial hemorrhages which definitely improves patients' functional outcome, life expectancy, as well as the wellbeing of the patient.

With the arrival of computed tomography of the brain, diagnosis of intra-cerebral hemorrhages has been taken from the age of calculated speculation with details of clinical features, angiograms and 'bloody taps' to the present day of arrival of a definitive diagnosis in a matter of minutes.

A significant reason for the decreasing trend of mortality in intra-cerebral hemorrhage patients in our country has also been due to the identification of the factors which might adversely affect the outcome, stratifying patients and instituting prompt acute stroke care. However, this is a very small study and therefore larger randomized trials are needed to validate our findings.

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