Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Radiology and Imaging

Computed Tomograhic Evaluation of Spontaneous Intracerebral Hemorrhage in a Tertiary Care Hospital of Bangladesh

Dr. Ummay Salma Sharkar[1*], Dr. Shah Muhammad Mustaquim Billah[2], Dr. Nafisa Jafreen[3]

[1]Ex Resident, Department of Radiology and Imaging, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh
[2]Professor, Department of Radiology and Imaging Bangladesh Shishu Hospital & Institute, Bangladesh
[3]Junior Consultant, Obs & Gynae, Nasirnagar Upazilla Health Complex, Brahmanbaria, Bangladesh

DOI: 10.36347/sjams.2023.v11i01.015 | **Received:** 03.12.2022 | **Accepted:** 10.01.2023 | **Published:** 15.01.2023

***Corresponding author:** Dr. Ummay Salma Sharkar
Ex Resident, Department of Radiology and Imaging, Sylhet MAG Osmani Medical College, Sylhet, Bangladesh

Abstract **Original Research Article**

Introduction: Intracerebral hemorrhage (ICH) accounts 10 -15% of strokes which is second leading cause of death worldwide. To explore the risk factors, pattern and location of ICH through neuroimaging technique in a developing country like Bangladesh. ***Methods:*** It was a prospective study conducted over two year's period in Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet. All clinically and radiologically diagnosed hemorrhagic stroke patients except pure subarachnoid, intraventricular & subdural hemorrhages, brain tumors and post traumatic cases were included in the study and their medical and computer tomography records were analyzed. ***Results:*** Total 552 patients were included with male to female ratio 1.46:1. Among the various risk factors, the single most common was hypertension 70% cases, followed by smoking in 38% cases, and diabetes in 33% cases Cerebellar hemorrhage was more in elderly aged group 62.9±10.1 compare to lobar, deep cerebral and brain stem groups (54.3±14.2; 55.7±15.9; 51.2±12.7). Lobar and deep cerebral hemorrhage was found almost similarly distributed among< 40 years and > 70 years aged group, but among 40-49 years aged group deep cerebral hemorrhage was found more (72.73%) compare to lobar (27.27%). ***Conclusion:*** Incidence of spontaneous intracerebral hemorrhage is more in our middle aged populations with maximum having hypertension, smoking and diabetes mellitus as risk factor. Change in life style and food habits may help reduce the incidence of ICH.

Keywords: Intracerebral hemorrhage, spontaneous, Computed Tomography, CT scan, pattern, risk factors, location.

INTRODUCTION

Spontaneous intracerebral haemorrhage (ICH) is defined as a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma [1]. Spontaneous intracerebral hemorrhage (ICH) accounts for 10% to 15% of all strokes [2], with a worldwide incidence of 24.6–29.2/100,000 person-years [3, 4]. Stroke is the second leading cause of death worldwide and one of the leading causes of disability [4]; hemorrhage is associated with a higher morbidity and mortality than ischemic stroke. ICH may be lobar hemorrhage (LH) or deep cerebral hemorrhage (DCH) (including brain stem and cerebellar). The anatomical distribution of the haemorrhage and its extension to other compartments (subarachnoid, subdural and intraventricular) may bring clues to identify the underlying cause of the bleeding. Knowledge of ICH patterns may give some insight into the etiology of ICH and help to reduce its burden particularly among Bangladeshis where health infrastructure is not well developed. Spontaneous ICH results from several distinct underlying vasculopathies. Several interacting and overlapping risk factors may play a role in the vessel rupture. The risk of ICH increases with age, being 9.6-fold higher in people over 85 years old compared with those less than 45 years of age [5]. The incidence rates and location of spontaneous ICH have been shown to vary between population and races [6]. ICH incidence is higher in men, especially in Asian populations [6]. In spite of advances in medical and neurosurgical treatment, ICH remains a condition with poor outcome, with an overall mortality of 40% to 50% [7]. Identification of factors determining and modifying the clinical presentation and outcome of ICH is, therefore, very important for every population. Among the etiologies implicated in spontaneous intracerebral hemorrhage, chronic arterial hypertension is considered the most important [7]; other established causes include cerebral amyloid angiopathy, arterio-venous malformation, infections, alcohol consumption[8], anti-coagulant treatment, and to a lesser extent, anti- platelet

use, thrombolytic therapy, and use of amphetamines or cocaine and possibly statins [6].

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 scan is widely available, affordable, non-invasive and relatively accurate investigation used in cases of stroke and is the modality of choice as an initial investigation in patient with stroke. The purpose of CT is to differentiate ischemic stroke from intracranial hemorrhage and to rule out other pathological processes such as tumour and vascular malformations which may present as stroke.

Brain CT detects symptomatic ICH within minutes of symptom onset but may lack sensitivity if delayed for >1 week after ICH onset [9]. Hyperacute CT angiography followed by a post-contrast scan may identify a 'spot sign' – one or more hyperintense spots in the haemorrhage –representing a contrast leak [10]. Its presence might suggest a risk of haematoma expansion, poor outcome and mortality, even if the translation of its value into clinical practice remains controversial [11]. CT venography is useful to search for a venous thrombosis [12].

Few studies have reviewed the pattern, location and risk factors of ICH in Asian population like Bangladeshi. As far our knowledge there is no such study in Bangladesh on the pattern, location or risk factors of ICH using CT scan.

This study present a evaluation of ICH using a modern neuro imaging technique with the aims to determine the pattern and location of ICH with risk factors among patients presenting in a tertiary hospital in Bangladesh.

METHODOLOGY

This study was conducted at the radiology and Imaging department of Jalalabad Ragib-Rabeya Medical College hospital, Sylhet, Bangladesh. Total 552 cases were included during the period November, 2017 to October 2019, who was referred for CT scan of brain from the emergency or neurology unit with clinical features of stroke and having ICH on CT scan. A detailed cardiovascular and neurological examination was done by the attending physician. Important clinical history and risk factors (i.e. hypertension, diabetes mellitus, coronary artery disease, smoking, alcohol intake, previous history of TIA/ stroke) were taken from each patient's record file.

Technique

CT scan was performed in a multi detector (Siemens SOMATOM Definition AS 128-slice) CT scanner with the patient lying with his or her back to the table. Axial images were obtained and reconstructed in the coronal plane and sagittal plane using Head Neuro protocol: Series-Head 5.0 H31s, kV-120, mAs-450, CTDIvol-51.345, RotTime-1.00, Pitch-0.80, Coll.-0.60, Slice-5.00, Recon increment-5.0, Images-25, Kernel-H31s, Window-Base-Orbita.

The Following Points were used as a Guide to Assess Brain CT to Demonstrate/Exclude ICH

1. **Brain Parenchyma:** Is there asymmetry anywhere or obliteration of the gyri sulci pattern?, abnormal gray-white matter differentiation?, hypo/hyperdense abnormalities?
2. **Hemorrhage:** A sharply delineated hyperdensity on the CT without contrast (HU around +40, consistent with blood), type/cause/location, subarachnoid cisterns; obliteration of the W shape, pentagon, moon shape, Sylvian fissure, mass effect or signs of herniation, is there still space around the brain stem?
3. **Ventricular System:** Hydrocephalus?, intraventricular blood?
4. **Bone:** Extracranial soft tissue swelling?, fracture? Pneumocephalus? Normal air content of the sinuses and the mastoid? Air-fluid (blood) levels in the sinus?
5. **Old Examinations:** New findings?

All the medical and Computed Tomography (CT) records of patients with a clinical diagnosis of hemorrhagic stroke were included. All cases of pure subarachnoid and intraventricular hemorrhages, subdural hematomas, brain tumors, and post-traumatic cases were excluded. In cases of repeat scan, each scan was recorded separately if the scan showed a different result. Relevant data were collected, and statistical analysis was performed using SPSS version 23 (IBM Corporation, New York, USA).

RESULTS

A total of 552 (16.23%) out of 3400 scans done for strokes were analyzed. 328 were males (59.02%) and 224 (40.58%). were females. Male female ratio was 1.46:1. The mean age of the patients was 52.3 ± 15.11 (range: 35–89) years. There was no statistical difference between the mean age of men and women (53.2 ± 12.3 vs. 51.9 ± 13.8. P = 0.65).

Risk factors were present in most 403(73 %) of the patients presenting with ICH. Among the various risk factors, the single most common was hypertension seen in 387 cases, followed by smoking in 210 cases, and diabetes in 182 cases (table 1). 247 patients had multiple combinations of the above risk factors. Among the multiple risk factors, combination of hypertension, diabetes and smoking was the commonest seen in 143

cases, followed by hypertension and diabetes in 128 cases (table 1).

About 11.59% had additional intraventricular hemorrhage and 4.35% subarachnoid hemorrhage besides ICH [Figure 1].

The frequency of LH and DCH were 44.44% and 55.6%, respectively. Around four percent (24/552) of all hemorrhages occurred in the cerebellum (Table 1). Age distribution of the location of ICH showed that LH peaked at 16– 39 years while DCH peaked at 40-49 years [Figure 2]. Cerebellar hemorrhage, however, peaked at 70 years and above. The mean age of occurrence of hemorrhages of different locations is shown in Table 2. There was no statistically significant difference between mean ages of occurrence of LH and hemorrhages of other locations.

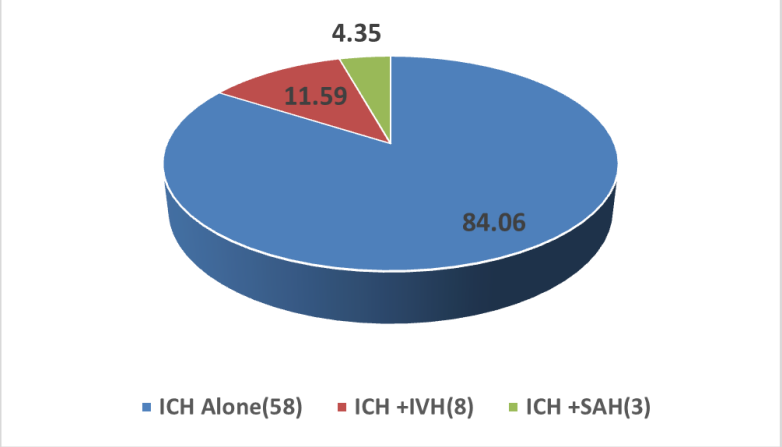


Figure 1: Pattern of intracerebral hemorrhage

Table 1: Distribution of Risk factors (n=403)

Risk factor	Male (%) n=328	Female (%) n=224	Total (%) n=552
Hypertension	297 (90)	90 (40.1)	387 (70.1)
Diabetes	93 (28.3)	89 (39.7)	182(33)
Smoking	191(58.2)	19(8.4)	210(38)
Alcohol intake	25(7.6)	0	35(6.3)
Coronary artery disease	20(6)	8(3.5)	28(5)
Previous history of TIA / Stroke	31(9.4)	8(3.5)	39(7)
Hypertension+ Diabetes+ Smoking + Alcohol intake	140(42.6)	0	140(25.3)
Hypertension+ Smoking	90(27.4)	3(1.3)	93(16.8)
Hypertension+ Diabetes	89(27.1)	39(17.4)	128(23.1)
Diabetes+ Smoking	109(33.2)	0	109(19.7)
Smoking + Alcohol intake	35(10.6)	0	35(6.3)

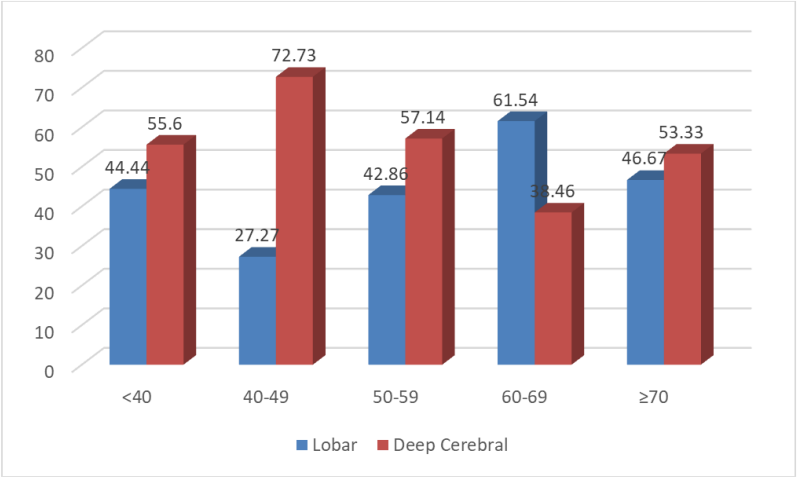


Figure 2: Age distribution of deep cerebral and lobar hemorrhages

Table 2: Distribution of ICH by gender and age groups n(%)

Variables	Lobar	Deep Cerebral*	Total	Brain Stem	Cerebellar
Gender					
Male	152(46.34)	176(53.66)	328(59.02)	16(4.88)	24(7.31)
Female	96(42.86)	128(57.14)	224(40.58)	8(3.57)	0(00)
Age groups(years)					
<40	32(12.90)	24(7.89)	72(13.04)		
40-49	24(9.68)	40(13.16)	88(15.94)		
50-59	72(29.03)	104(34.21)	168(30.44)	24(100)	8(33.3)
60-69	64(25.81)	88(28.95)	104(18.84)		16(66.67)
≥70	56(22.58)	48(15.79)	120(21.74)		
Total	**248(44.93)**	**304(55.07)**	**552(100)**	**24(100)**	**24(100)**

*Deep includes internal capsule, thalamic, putamen, brain stem and cerebellar hemorrhages. ICH=Intracerebral hemorrhage

Table 3: Distribution of mean ages of occurrence of ICH:

Type of ICH	Mean Age(SD)
Lobar	54.3(14.2)
Deep Cerebral*	55.7(15.9)
Brain Stem	51.2(12.7)
Cerebellar	62.9(10.1)

SD: Standard deviation, ICH: Intracerebral Hemorrhage,
*Deep includes internal capsule, thalamic, putamen, brain stem and cerebellar hemorrhages

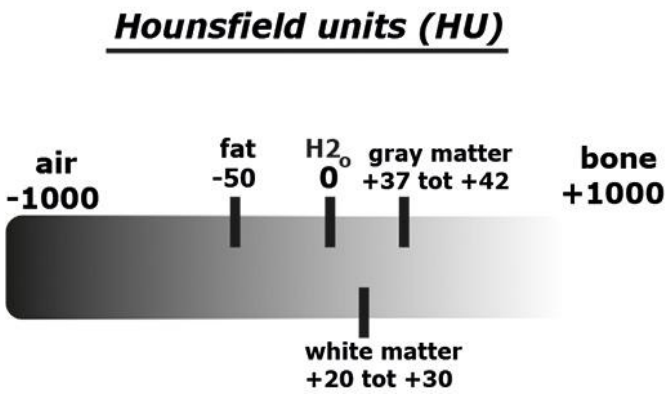
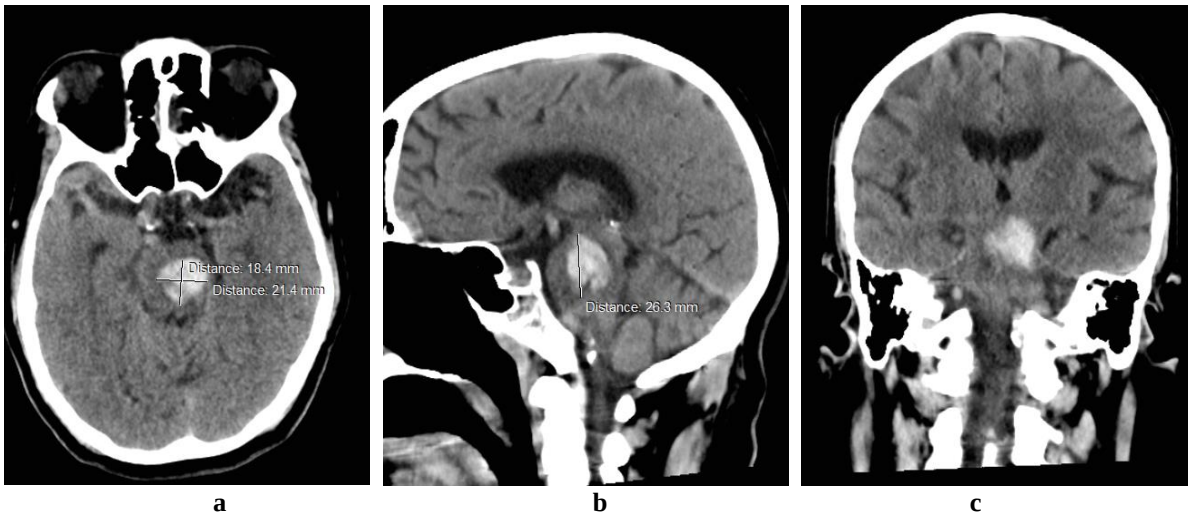
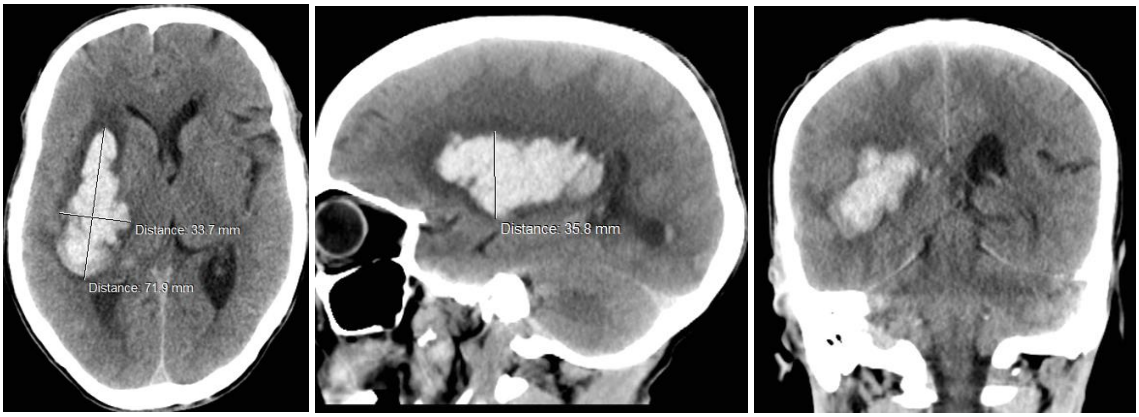


Figure 3: Hounsfield unit (HU) scale



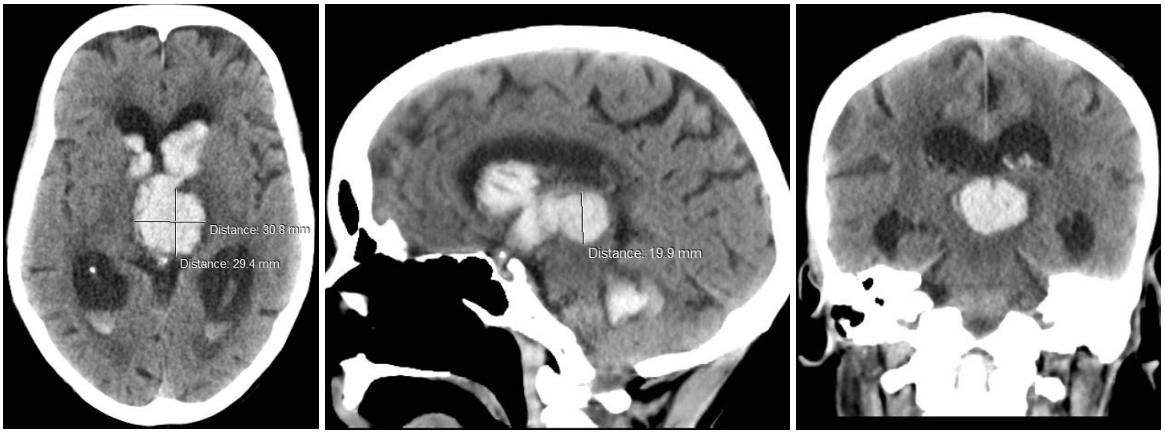
a **b** **c**

Figure 4 a, b & c: CT scan of brain axial, sagittal & coronal images of a 60 years aged male showing brainstem hemorrhage



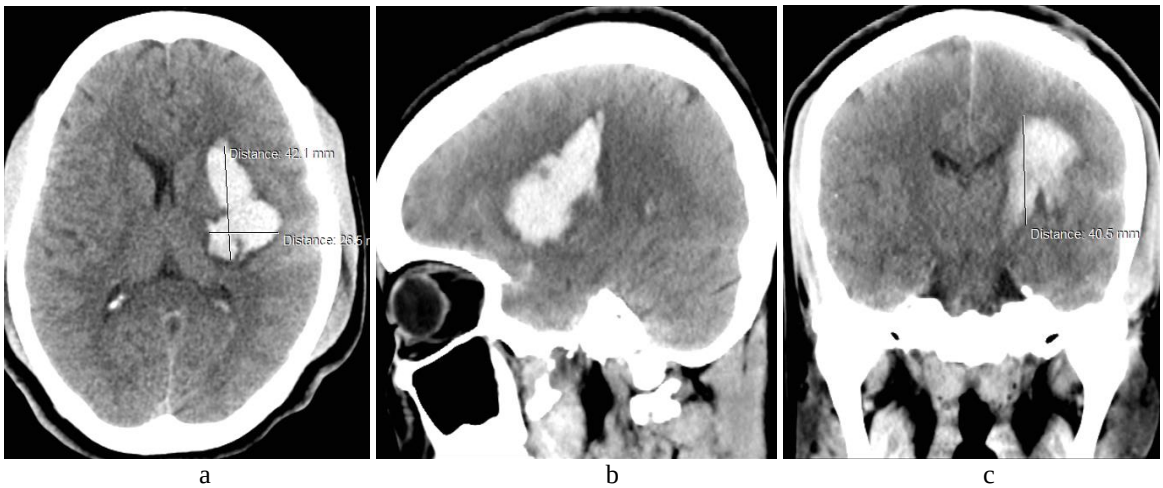
a b c

Figure 5 a, b & c: CT scan of brain axial, sagittal & coronal images of a 60 years aged male showing lobar hemorrhage with minimal intraventricular extension



a b c

Figure 6 a, b & c: CT scan of brain axial, sagittal & coronal images of a 55 years aged male showing lobar hemorrhage with intraventricular extension



a b c

Figure 7 a, b & c: CT scan of brain axial, sagittal & coronal images of a 60 years aged male showing lobar hemorrhage with minimal intraventricular extension and sub arachnoid hemorrhage

DISCUSSION

The clinical and epidemiological scenario of ICH has been changing in the last decades [13, 14]. Despite an overall stable incidence of ICH, the incidence among people older than 75 years has increased and the incidence among people younger than 60 years has decreased, with a larger proportion of lobar haemorrhages, suggesting that vasculopathies more strongly associated with the elderly, particularly cerebral amyloid angiopathy (CAA), represent an increasing proportion within the aetiological

distribution of ICH [14]. The poor prognosis of ICH may be partly due to our poor understanding of this heterogeneous disease.

The importance of the diagnostic work-up relies on the identification of the ICH but also of other neuroradiological biomarkers that can give clues to detect the underlying cause of the haemorrhage. The appearance of intracranial hemorrhage on CT is comparatively straightforward. The attenuation properties of intracranial blood are determined by the aggregation of globin molecules in the hematoma [15]. There is a linear relationship between CT attenuation, protein content (mainlyhemoglobin), and hematocrit [16]; however, artifacts located close to the skull base can easily mimic hemorrhage on spiral-CT scans [17]. Immediately after the hemorrhage, freshly extravasated blood exhibits a markedly heterogeneous appearance with mixed density values in the range of 40-60 Hounsfield units (HU; Fig. 1a) [18]. During the early hours of hemorrhage, the CT density values within the hematoma rapidly increase upto 60-80 HU (Fig. 1b). [19]. During the first week, intracranial hematomas appear on non-contrast CT scans as well-demarcated hyperdense lesions [20]. As the hematoma matures, clot retraction ensues. This increases the attenuation coefficient to 80-100 HU in the center of the hematoma. During the weeks after the acute event, the density of the hematoma decreases by an average of 0.7-1.5 HU per day, due to chemical breakdown of globin molecules [21]. This process begins in the periphery and proceeds toward the center of the hematoma. Usually, the decrease in density does not correspond to the decrease in mass effect; thus, even when the density change is no longer present, persistent mass effect on CT can give a clue to the previous episode of hemorrhage in the brain. After a few weeks or even months, macrophages digest the blood breakdown products, ultimately resulting in resolution of the clot. Other end- stage appearances of an old hematoma include: an area of hypodensity (dueto tissue loss); focal atrophy; calcification; or ventricular enlargement [21, 22]. After contrast administration in the acute stage, there is no enhancement of the hematoma; however, with time, there develops neovascularization in the surrounding brain tissue. This will enhance on contrast ad- ministration due to breakdown of blood-brain barrier (BBB) in the vascularized capsule [23]. The resulting ring-like enhancement can lead the radiologist or clinician to diagnose this condition erroneously as a brain tumor or abscess.

At admission, after the first brain image demonstrating the ICH, it is not always clear whether, how and when to undertake further radiological investigation. In a survey of current practice in three European countries, younger patient age strongly influenced whether further investigation of ICH was performed, followed by the absence of prestroke hypertension and lobar ICH location [24]. There is no general consensus concerning the choice and the timing of neuroradiological work-up for ICH [25]. The American Heart Association/American Stroke Association [26] recommended rapid brain computed tomography (CT) or magnetic resonance imaging (MRI) to distinguish ICH from ischaemic stroke and further vessel examination when structural underlying lesions are suspected. Besides vessel imaging, exploring brain parenchyma with MRI might bring additional clues to the underlying vessel disease [27].

The present study highlights the risk factors and distribution of ICH in Bangladeshi subjects. No data had previously described the location and risk factors of ICH among Bangladeshi.

The pooled 1-year survival estimate in nine population-based studies was 46 % (95 % confidence interval [CI] 43 to 49%), while when location is considered the 1-year survival was 45–59 % after lobar ICH, 45 to 59 % after deep ICH and 40 to 54 % after infratentorial ICH [28].

A total of 552 patients were analyzed. 328 (59.02%) were males and 224 (40.58%) were females. Male female ratio was 1.46:1. In a study conducted among 100 stroke patients admitted to CMCH between July 2001 and June 2003; 74% were males and 26% were females, with 39% suffering from haemorrhagic stroke (HS) [29]. In another study, 100 stroke patients were hospitalized at CMCH between November 2003 and May 2004; (20%) had HS, the ratios of male: female patients were 3·00 : 1·00 HS [30].

The mean age of the patients was 52.3 ± 15.11 (range: 35–89) years. The age-specific frequency of DCH decreased after 59 years. The peak age of DCH (50–59 years) supports findings by Ruíz-Sandoval *et al.*, [31].

Hypertension is a known predictor for deep cerebral / brainstem and cerebellar hemorrhage and was found in 70.1% of cases. This finding is not surprising considering the predominant age group studied and the high prevalence of hypertension in Bangladeshi. Hypertension is more prevalent in males than females [32] and increases with age. This may be explained not only by the increasing incidence and prevalence of hypertension but by factors such as smoking and sedentary lifestyle especially among hypertensive individuals.

In a study [29] Hypertension (63%) was the main risk factor for stroke, followed by heart disease (24%), diabetes mellitus (DM) (21%), and hyperlipidaemia (7%) [5]. In another study, hypertension was the most common risk factor, followed by cigarette smoking, DM, oral conception use, and previous history of TIA [30]. According to the study, the most common brain area affected by stroke

was the cortical region, followed by the basal ganglia, internal capsule, insula, thalamus, cerebellum, and multifocal areas [30].

In a study [33] at Mymensingh Medical College Hospital, there were 50 stroke cases that occurred between January and June 2008 and affected relatively young patients (mean age 35·8 ±7·39 years), majority of the cases (60%) were ischemic and the rest were hemorrhagic stroke (40%). Again, the majority of the patients were males; the ratio of male: female patients was 1·27: 1·00 [33]. However, the most common stroke-related risk factor found in this study [33] was dyslipidaemia and not hypertension, perhaps due to the smaller sample size. Other stroke-related risk factors were hypertension in the patient or in family members, smoking, DM, ischaemic heart disease in the patient or in family members, alcohol consumption, and family history of stroke [33]. The data also indicated that Ischemic stroke patients had a higher likelihood of recovery (68%) compared to hemorrhagic stroke patients (32%). Hemorrhagic stroke contributed significantly to overall inpatient mortality (12%), while no mortality rate was reported for ischemic stroke [33].

Another study investigated the association between metabolic syndrome and stroke occurrences among randomly selected stroke patients (n=50) hospitalized in DMCH between July and December 2009 [34]. Metabolic syndrome encompasses atherogenic dyslipidaemia, high blood pressure, hyperglycaemia, and centrally distributed obesity [34]. Although 46% of patients observed had metabolic syndrome, overall, a higher contribution was seen in the IS cases (65·2%) than in the HS cases (34·8%). Of the 54% of patients without metabolic syndrome, 55·6% were HS patients [35]. The data indicated that the majority (64%) of stroke patients had high TG levels and 68% had low HDL levels [35]. Of those with high blood pressure, 69·60% were HS patients and 59·3% were IS patients. Forty-four per cent of the patients were also hyperglycaemic with fasting blood glucose greater than 100 mg/dl [35]. Only 12% of patients were categorized as obese [35].

Stroke-related risk factors have also been studied among patients (n=400) hospitalized in DMCH between July and December 2007 [36]. The male: female ratio was 1·20: 1·00 and the patients were classified into different stroke subtypes according to the findings from computed tomography (CT) scanners or magnetic resonance imaging (MRI). The data indicated that 56·25%, 38·25%, and 5·50% of the patients had cerebral infraction, intracerebral haemorrhage and subarachnoid haemorrhage, respectively; the risk factors present in the stroke cases included hypertension (present in 58·62% of the stroke cases), cigarette smoking (53·79%), lipid disorder (48·01%), heart diseases (25·75%), DM (20·01%), and previous history of stroke (10·61%) [36]. In another study [37], the risk factors for stroke were investigated in 85 young patients (aged 14 to 45 years) hospitalized at the DMCH between January 2008 and July 2009, the majority (61·18%) suffered from an IS, while others had intracerebral haemorrhage (29·40%), subarachnoid haemorrhage (8·24%), or aneurysm (1·18%); common risk factors for both IS and HS were hypertension (60·00%), hypercholesterolaemia (38·80%), diabetes (35·20%), smoking (32·90%), premature atherosclerosis (8·20%), and oral contraceptive use (3·8%) [37].

The most frequent cause of deep ICH is deep perforating vasculopathy that supervenes mostly in small perforating arterioles (50–700 μm in diameter) originating from the middle cerebral artery and from the basilar artery, thus explaining the classic location in the basal ganglia and brainstem. Deep ICH may be restricted to brain parenchyma or may extend to the ventricles. Intraventricular haemorrhage (IVH) is a frequent complication occurring in nearly 50 % of ICH patients, and it is a predictor of poor outcome [38]. The risk of bleeding in patients with deep perforating vasculopathy might be enhanced in patients receiving oral anticoagulants [39] and among patients with heavy alcohol consumption history.

Lobar ICH can result from several distinct diseases. The most common is CAA. The pathological process seen in CAA occurs in small- to medium-sized leptomeningeal and cortical vessels (especially in the occipital and temporal regions), [40] while vessels in deep areas (thalamus, basal ganglia as well as brainstem) are usually spared. A recent population-based study showed an important increase (~80 %) in ICH incidence among people aged ≥75 years. This result was attributed to a two fold increase in lobar ICH, concomitantly with an observed rise in the premorbid use of antithrombotics at this age. These results suggest that some bleeding-prone vasculopathies in the elderly are more likely to bleed when antithrombotic drugs are used, as illustrated by the rise in the incidence of lobar ICH, in which CAA may be strongly implicated [14]. Intracranial vascular malformations, brain tumours, cerebral venous thrombosis (CVT), haemorrhagic transformation (HT), other vasculopathies and systemic diseases may also lead to lobar ICH.

In the present study DCH (55.07%) occurred predominantly, DCH and LH peaked at 50-59 years. The patient characteristics in the current study argued that with increasing age increases the risk of hemorrhagic stroke [3, 4]. Flaherty *et al.*, [3] reported an excess of DCH notably in middle-aged persons similar to findings in the present study.

The higher rate of LH among subjects <40 years may be due to smoking & use of alcohol, some other factors that may be contributory. LH increased after 49 years and decreased after 59 years. Rates of LH

and DCH at 50-59 years (in whom amyloid angiopathy becomes a significant cause of hemorrhage) were 29.03% and 34%, respectively which may reflect higher rates of hypertension in this age group. Most elderly individuals with LH may also have had hypertension for many years [31]. Current estimates show that almost 50% of ICHs in the lobar region are related to amyloid angiopathy [15].

Limitations

This is a single hospital based study; therefore, there is a possibility that very severe cases may be over represented and fatal cases totally excluded. Furthermore, data about the size of hemorrhages was not consistently stated. Case ascertainment was done through radiological reports. A multi-center study including clinical features and outcome may be preferable.

CONCLUSION

Incidence of deep cerebral hemorrhage (DCH) appears more in our population with maximum having hypertension, smoking and diabetes mellitus as risk factor. Smoking and alcohol consumption play role in higher incidence of ICH in the male. Change in life style and food habits may help reduce the incidence of ICH. Further multicentre studies are required to establish the risk factors and outcome of LH and DCH in our environment.

REFERENCES

1. Sacco, R. L., Kasner, S. E., Broderick, J. P., Caplan, L. R., Connors, J. J., Culebras, A., ... & Vinters, H. V. (2013). An updated definition of stroke for the 21st century: a statement for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*, *44*(7), 2064-2089.
2. Qureshi, A. I., Tuhrim, S., Broderick, J. P., Batjer, H. H., Hondo, H., & Hanley, D. F. (2001). Spontaneous intracerebral hemorrhage. *New England Journal of Medicine*, *344*(19), 1450-1460.
3. Flaherty, M. L., Woo, D., Haverbusch, M., Sekar, P., Khoury, J., Sauerbeck, L., ... & Broderick, J. P. (2005). Racial variations in location and risk of intracerebral hemorrhage. *Stroke*, *36*(5), 934-937.
4. Van Asch, C. J., Luitse, M. J., Rinkel, G. J., van der Tweel, I., Algra, A., & Klijn, C. J. (2010). Incidence, case fatality, and functional outcome of intracerebral haemorrhage over time, according to age, sex, and ethnic origin: a systematic review and meta-analysis. *The Lancet Neurology*, *9*(2), 167-176.
5. Algra, A., & Klijn, C. J. (2013). Understanding racial differences in intracerebral haemorrhage. *Nature Reviews Neurology*, *9*(7), 364-365.
6. Fogelholm, R., Nuutila, M., & Vuorela, A. L. (1992). Primary intracerebral haemorrhage in the Jyväskylä region, central Finland, 1985-89: incidence, case fatality rate, and functional outcome. *Journal of Neurology, Neurosurgery & Psychiatry*, *55*(7), 546-552.
7. Waga, S., Miyazaki, M., Okada, M., Tochio, H., Matsushima, S., & Tanaka, Y. (1986). Hypertensive putaminal hemorrhage: analysis of 182 patients. *Surgical neurology*, *26*(2), 159-166.
8. Calandre, L., Arnal, C., Ortega, J. F., Bermejo, F., Felgeroso, B., Del Ser, T., & Vallejo, A. (1986). Risk factors for spontaneous cerebral hematomas. Case-control study. *Stroke*, *17*(6), 1126-1128.
9. Warlow, C., Sudlow, C., & Dennis, M. (2003). Stroke. *Lancet*, 362(9391), 1211–24.
10. Boulouis, G., Dumas, A., Betensky, R. A., Brouwers, H. B., Fotiadis, P., Vashkevich, A., ... & Greenberg, S. M. (2014). Anatomic pattern of intracerebral hemorrhage expansion: relation to CT angiography spot sign and hematoma center. *Stroke*, *45*(4), 1154-1156.
11. Romero, J. M. (2012). Stroke: is spot sign the answer for intracerebral haemorrhage?, *Nature Rev Neurol*, 8, 300–1.
12. Bousser, M. G., & Ferro, J. M. (2007). Cerebral venous thrombosis: an update. *Lancet Neurol*, 6, 162–70.
13. Howard, G., Cushman, M., Howard, V. J., Kissela, B. M., Kleindorfer, D. O., Moy, C. S., ... & Woo, D. (2013). Risk factors for intracerebral hemorrhage: the REasons for geographic and racial differences in stroke (REGARDS) study. *Stroke*, *44*(5), 1282-1287.
14. Béjot, Y., Cordonnier, C., Durier, J., Aboa-Eboulé, C., Rouaud, O., & Giroud, M. (2013). Intracerebral haemorrhage profiles are changing: results from the Dijon population-based study. *Brain*, *136*(2), 658-664.
15. Dolinskas, C. A., Bilaniuk, L. T., Zimmerman, R. A., Kuhl, D. E., & Alavi, A. B. A. S. S. (1977). Computed tomography of intracerebral hematomas. II. Radionuclide and transmission CT studies of the perihematoma region. *Am J Roentgenol*, *129*, 689-692.
16. New, P. F., & Aronow, S. (1976). Attenuation measurements of whole blood and blood fractions in computed tomography. *Radiology*, *121*(3), 635-640.
17. Bahner, M. L., Reith, W., Zuna, I., Engenhart-Cabillic, R., & Van Kaick, G. (1998). Spiral CT vs incremental CT: is spiral CT superior in imaging of the brain?. *European radiology*, *8*(3), 416-420.
18. Bergström, M., Ericson, K., Levander, B., Svendsen, P., & Larsson, S. (1977). Variation with time of the attenuation values of intracranial hematomas. *Journal of computer assisted tomography*, *1*(1), 57-63.
19. Brooks, R. A., Di Chiro, G., & Patronas, N. (1989). MR imaging of cerebral hematomas at different field strengths: theory and applications. *J Comput Assist Tomogr*, *13*(2), 194-206.

20. Tans, J. T. (1977). Computed tomographyof intracerebral hematoma. *Clin Neurol Neurosurg*, 79, 285-295.
21. Dolinskas, C. A., Bilaniuk, L. T., Zimmerman, R. A., & Kuhl, D. E. (1977). Computed tomography of intracerebral hematomas. I. Transmission CT observations on hematoma resolution. *American Journal of Roentgenology*, *129*(4), 681-688.
22. Kreel, L., Kay, R., Woo, J., Wong, H. Y., & Nicholls, M. G. (1991). The radiological (CT) and clinical sequelae of primary intracerebral haemorrhage. *The British Journal of Radiology*, *64*(768), 1096-1100.
23. Laster, D. W., Moody, D. M., & Ball, M. R. (1978). Resolving intracerebral hematoma: alteration of the" ring sign" with steroids. *American Journal of Roentgenology*, *130*(5), 935-939.
24. Cordonnier, C., Klijn, C. J., van Beijnum, J., & Al-Shahi Salman, R. (2010). Radiological investigation of spontaneous intracerebral hemorrhage: systematic review and trinational survey. *Stroke*, *41*(4), 685-690.
25. Salman, R. A. S., Labovitz, D. L., & Stapf, C. (2009). Spontaneous intracerebral haemorrhage. *BMj*, *339*, b2589.
26. Morgenstern, L. B., Hemphill III, J. C., Anderson, C., Becker, K., Broderick, J. P., Connolly Jr, E. S., ... & Tamargo, R. J. (2010). Guidelines for the management of spontaneous intracerebral hemorrhage: a guideline for healthcare professionals from the American Heart Association/American Stroke Association. *Stroke*, *41*(9), 2108-2129.
27. Wijman, C. A., Venkatasubramanian, C., Bruins, S., Fischbein, N., & Schwartz, N. (2010). Utility of early MRI in the diagnosis and management of acute spontaneous intracerebral hemorrhage. *Cerebrovascular Diseases*, *30*(5), 456-463.
28. Poon, M. T. C., Fonville, A. F., & Salman, R. A. S. (2014). Long-term prognosis after intracerebral haemorrhage: systematic review and meta-analysis. *Journal of Neurology, Neurosurgery & Psychiatry*, *85*(6), 660-667.
29. Hossain, A. M., Ahmed, N. U., Rahman, M., Islam, M. R., Sadhya, G., & Fatema, K. (2011). Analysis of sociodemographic and clinical factors associated with hospitalized stroke patients of Bangladesh. *Faridpur Medical College Journal*, *6*(1), 19-23.
30. Siddique, M. A. N., Nur, Z., Mahbub, M. S., Alam, M. B., & Miah, M. T. (2009). Clinical presentation and epidemiology of stroke: a study of 100 cases. *Journal of Medicine*, *10*(2), 86-89.
31. Ruíz-Sandoval, J. L., Cantú, C., & Barinagarrementeria, F. (1999). Intracerebral hemorrhage in young people: analysis of risk factors, location, causes, and prognosis. *Stroke*, *30*(3), 537-541.
32. The World Bank Group. Poverty and equity. Available at http://povertydata.worldbank.org/poverty/region/SAS (accessed 3 February 2012).
33. Miah, M. T., Hoque, A. A., Tarafder, B. K., Romel, S. M. A., & Hassan, M. K. (2008). Stroke in young age-study of 50 cases. *Journal of Medicine*, *9*(1), 10-15.
34. Arenillas, J. F., Moro, M. A., & Dávalos, A. (2007). The metabolic syndrome and stroke: potential treatment approaches. *Stroke*, *38*(7), 2196-2203.
35. Iqbal, M. J., Siddiqui, M. M. R., Islam, Q. T., Hossain, A., Mustafa, E., Shipa, M. R. A., ... & Sina, H. (2010). Association between acute stroke and metabolic syndrome. *Journal of Medicine*, *11*(2), 124-127.
36. Badiuzzaman, M., Mohammed, F. R., Chowdhury, F. R., Bari, M. S., Alam, M. B., & Ahasan, H. N. (2009). Prevalence of modifiable risk factors among stroke patients in a tertiary care hospital in Dhaka. *Journal of Medicine*, 18-21.
37. Hossain, M. Z., Ahmed, S. U., Sarder, M. H., Dasgupta, R., Das, A., Sarker, R. N., ... & Mohammad, Q. D. (2009). Analysis of risk factors associated with stroke in young adults: a prospective study. *Journal of Dhaka Medical College*, *18*(2), 95-99.
38. Tuhrim, S., Horowitz, D. R., Sacher, M., & Godbold, J. H. (1999). Volume of ventricular blood is an important determinant of outcome in supratentorial intracerebral hemorrhage. *Critical care medicine*, *27*(3), 617-621.
39. Dequatre-Ponchelle, N., Hénon, H., Pasquini, M., Rutgers, M. P., Bordet, R., Leys, D., & Cordonnier, C. (2013). Vitamin K antagonists–associated cerebral hemorrhages: what are their characteristics?. *Stroke*, *44*(2), 350-355.
40. Rosand, J., Muzikansky, A., Kumar, A., Wisco, J. J., Smith, E. E., Betensky, R. A., & Greenberg, S. M. (2005). Spatial clustering of hemorrhages in probable cerebral amyloid angiopathy. *Annals of neurology*, *58*(3), 459-462.