CLINICAL PROFILE OF INTRACEREBRAL HAEMORRHAGE

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ABSTRACT

OBJECTIVES
Among all the strokes, the intracerebral haemorrhage comprises of about 8-13%. Hypertension accounts for majority about 50% of cases. Spontaneous Intracerebral Haemorrhage (SICH) is a type of stroke usually caused by a vessel rupture followed by spontaneous leakage of blood in the brain parenchyma.

METHODS
A prospective study was conducted among patients admitted with intracerebral haemorrhage in the Department of General Medicine, Government Stanley Hospital, Chennai, during March to September 2015.

RESULTS
Among 60 study subjects, 55% were of the age group 61-70 years. 70% of them were males. Only 20% had no co-morbid conditions. 41.7% had MAP 110-130 mmHg, 23% had 130-150 mmHg, 11.7% had MAP more than 150 mmHg. 46.7% presented with motor weakness in the form of hemiplegia, 56.7% was capsuloganglionic and 20% was of lobar location. 46.7% had ICH volume <30% 31.7% had 30-60 cub. mm, 21.7% had >60 cub. Mm, 23% had associated intraventricular extension, 8.3% had lethal outcome.

CONCLUSION
The lethal and poorer outcome is more with increased age, more in males compared to females. The more the mean arterial pressure MAP, lethal and poorer was the outcome. Lesser MAP was associated with a better the outcome. The Glasgow Outcome Score (GOS score) has a direct correlation with Glasgow Coma Scale (GCS) and inverse correlation with NIHSS score. A poorer outcome was observed with more haematoma volume, infratentorial location, and the presence of intraventricular extension.

KEYWORDS
Intracerebral Haemorrhage (ICH), Stroke, Clinical Profile of Intracerebral Haemorrhage (ICH), Glasgow Outcome Scale (GOS).


INTRODUCTION
Intracerebral haemorrhage makes about 10% of all strokes and carries a 50% case fatality rate. The modifiable risk factors include hypertension, anticoagulant use, thrombolytic therapy, alcoholism, drug abuse (particularly cocaine). The non-modifiable risk factors include increasing age, Negroid ethnicity, cerebral amyloid angiopathy, coagulopathies, vasculitis, Arteriovenous Malformations (AVMs), and intracranial neoplasms.

OBJECTIVE
To assess the clinical profile and radiological features of the patients presenting with intracerebral haemorrhage.

METHODS AND MATERIALS
A prospective study was done among the patients admitted with intracerebral haemorrhage in the Department of General Medicine at Government Stanley Medical College Hospital, Chennai, during March to September 2015. 60 patients were selected who had spontaneous intracerebral bleed confirmed by CT admitted within 2 weeks of the neurologic manifestations. Patients with traumatic intracerebral haemorrhage, coagulation disorders, intracerebral haemorrhage of more than 14 days duration. Patients on thrombolytics, anticoagulants, and intracerebral haemorrhage not proven by CT or MRI, haemorrhagic transformation were excluded from the study. They were enrolled in this study after obtaining informed consent from their close relatives in case of altered sensorium patients.

Socioeconomic demographic details and the details about preceding illness were recorded. Neurological assessment was done by taking the history. The onset and severity of stroke was assessed by Glasgow coma scale and neurological deficit on admission by NIH stroke scale. The final outcome at discharge was evaluated by Glasgow Outcome Score (GOS). CT scan plain was done and the following aspects of intracerebral bleed were noted. Its location supratentorial or infratentorial and specific location as well like capsuloganglionic region, cerebellum, brain stem, lobe location, and multiple lobe involvement, mass effect, and presence of intraventricular extension. The haematoma volume was calculated based on the formula A*B*C/2.
The data collected was analysed using SPSS Version 16. Appropriate descriptive and inferential statistics was used to analyse the data.

RESULTS
Of the 60 study participants, the mean age was 62.78 years (SD 9.06 years). Most of them were males. Only 20% of them had no associated comorbid conditions. Most of them were nonalcoholic. Among patients with lethal outcome, 41.2% (50-60), 23.5% (61-70), 11.8% >70 yrs., 76.5% were in the above 60 yrs. 24.5% were <50 yrs. This implies that mortality is increasing with increasing age. This observation is consistent with the previous studies in the literature like Sunil et al1 and Maya et al2.

Among patients with lethal outcome of GOS 1-about 33.3% were males, 16.7% were females. With poor outcome-GOS 2 2.4% were males, 5.6% were females. GOS 3-23.8% were males and 22.2% were females. With better outcome-GOS 4-21.4% were males, 22.2% were females. GOS 5-19.0% were males and 33.3% were females. The lethal and poorer outcome was more in males compared to females. These observations are consistent with previous studies like study by Sunil et al.1 46.7% presented with motor weakness in the form of hemiplegia. 5% presented with sensory impairment. 6.7% presented with ataxia. 28.3% had alteration in sensorium with loss of consciousness. 6.7% had seizures. 6.7% presented with isolated headache as the isolated manifestation (Table 1).

<table>
<thead>
<tr>
<th>Clinical Presentation</th>
<th>Frequency N=60</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weakness hemiplegia</td>
<td>28</td>
<td>46.7%</td>
</tr>
<tr>
<td>Sensory</td>
<td>3</td>
<td>5.0%</td>
</tr>
<tr>
<td>Ataxia</td>
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<td>LOC</td>
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</tr>
<tr>
<td>Seizures</td>
<td>4</td>
<td>6.7%</td>
</tr>
<tr>
<td>Isolated headache</td>
<td>4</td>
<td>6.7%</td>
</tr>
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</table>

Table 1: Clinical Presentation

Among patients with Mean Arterial Pressure more than 150 mmHg, 71.4% had died, 28.6% had survived with poor outcome GOS3. With Mean Arterial Pressure between 130-150 mmHg - about 42.9% had died, 28.6% had survived with a poorer outcome, and 28.6% had survived with a better outcome. With Mean Arterial Pressure between 110-130 mmHg 16% had died, 28% had survived with a poorer outcome, and 56% had survived with a better outcome. (GOS4 and 5). With Mean Arterial Pressure between less than 110 mmHg, only 14% had died, 21% had survived with a poorer outcome, and 64.3% had survived with a better outcome (GOS 4 and 5).

![Fig. 1: Age and Glasgow Outcome Score (GOS)](image1)

38.3% had admission GCS of 15 without any worsening during hospital stay. 18.3% had admission GCS 11-14. 23.3% had admission GCS 5-10. 20% had admission GCS <5. 28.3% had lethal outcome (GOS 1). 3.3% had GOS 2, 23.3% had GOS 3 had poor outcome, 21.7% had GOS 4, and 23.3% had GOS 5 (better outcome).

![Fig. 2: Mean Arterial Pressure (MAP) and Glasgow Outcome Score (GOS)](image2)

Among patients with lethal outcome of GOS 1, 64.7% had GCS <5, 29.4% had GCS 5-10. Among patients with lethal outcome of GOS 2, 0% GCS <5, 50% had GCS 5-10, 50% >GCS 10. Among patients with lethal outcome of GOS3, 0% GCS <5, 35.7% had GCS 5-10, 42.9% >GCS 10. Among patients with lethal outcome of GOS 4, 7.7% had GCS <5, 23.1% had GCS 5-10, 23.1% >GCS 10. Among patients with lethal outcome of GOS 5, 0% had GCS <5, 0% had GCS 5-10, 100% >GCS 10. Patients had lethal outcome if GCS<5, a poorer outcome for GCS 5-10 and a better outcome if GCS>10. (Table 2).
Among patients with lethal outcome of GOS 1 - 5.9% had NIHSS <5, 11.8% had NIHSS 5-15, 11.8% had NIHSS 15-25, 70.8% had NIHSS >25.

Among patients with lethal outcome of GOS 2-0% had NIHSS <5, 100% had NIHSS 5-15, 0% had NIHSS 15-25, 0% had NIHSS >25. Among patients with lethal outcome of GOS 3 - 35.7% had NIHSS <5, 57.1% had NIHSS 5-15, 7.1% had NIHSS 15-25, 0% had NIHSS >25. Among patients with lethal outcome of GOS 4 - 38.5% had NIHSS <5, 30.8% had NIHSS 5-15, 15.4% had NIHSS - 15-25, 15.4% had NIHSS >25. Among patients with lethal outcome of GOS 5 - 92.9% had NIHSS <5, 28.3% had NIHSS 5-15, 8.3% had NIHSS - 15-25, 0% had NIHSS >25 (Fig. 3).

Patients who presented with NIHSS >25, 85.7% had died and only 14.3% had survived. But, among patients who presented with NIHSS <5 - significant proportion of about 92.9% had a better outcome GOS 5.

This again emphasises that GOS has inverse correlation with NIHSS. The correlation is statistically significant (p<0.000**).

Among the patients who had lethal outcome, GOS 1 about 58.8% were diabetic patients, 17.7% had isolated or hypertension associated diseases, and in about 17.6% of patients the aetiology was unidentifiable (Table. 3).

<table>
<thead>
<tr>
<th>Location</th>
<th>Frequency</th>
<th>Percent</th>
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<tbody>
<tr>
<td>Capsuloganglionic</td>
<td>34</td>
<td>56.7</td>
</tr>
<tr>
<td>Lobar</td>
<td>12</td>
<td>20.0</td>
</tr>
<tr>
<td>Mixed</td>
<td>3</td>
<td>5.0</td>
</tr>
<tr>
<td>Brain stem</td>
<td>5</td>
<td>8.3</td>
</tr>
<tr>
<td>Cerebellum</td>
<td>4</td>
<td>6.7</td>
</tr>
<tr>
<td>Subarachnoid haemorrhage</td>
<td>2</td>
<td>3.3</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Table 3: ICH Location

With respect to location of bleed, 56.7% was capsuloganglionic, 20% was of lobar location, 8.3% brain stem, 6.7% cerebellum, 5% mixed, and 3.3% in subarachnoid space. This is about 85% of bleed in supratentorial and 15% of the bleed in infratentorial location. Calculating the volume of the haematoma using the formula, 46.7% had intracerebral haematoma volume <30, 31.7% had 30-60 cub. mm, 21.7% had >60 cub. mm. About 23.3% had associated intraventricular extension along with the intracerebral bleed.

**Fig. 3: National Institutes of Health Stroke Scale (NIHSS) and Glasgow Outcome Score (GOS)**

In supratentorial location group and patients with lethal outcome, GOS 1 - 15.4% with 30 mL, 23.1% with volume between 30-60 mL, and 81.5% with volume greater than 60 mL and patients with better outcome GOS 5 - 81.8% with 30 mL, 18.2% with volume between 30-60 mL, and 0% with volume greater than 60 mL (Fig. 4).

**Fig. 4: ICH Volume and Supratentorial Location**

With infratentorial location and patients with lethal outcome GOS 1, 7.5% with 30 mL, 0% with volume between 30-60 mL, and 25% with volume greater than 60 mL (Fig. 5).

**Fig. 5: ICH Volume and Infratentorial Brain Stem Location**

**Fig. 6: ICH Volume and Capsuloganglionic Location**
Among patients with intracerebral bleed with capsuloganglionic location with volume less than 30 mL - 28.6% was associated with lethal outcome, 42.9% had better outcome GOS 5 (Fig. 6).

Among patients with intracerebral bleed with lobar location - lethal outcome was seen in 72% with volumes greater than 60 mL and a poor outcome was seen in 27.3% with volume between 30-60 mL (Fig. 7).

But, a better outcome was observed in patients with lobar location in 50.1% with the volume of the bleed between 30-60 mL and 9.1% of the patients with volume of the bleed greater than 60 mL. Among patients with intracerebral bleed with brain stem location even with the volume of bleed less than 30 mL, 75% had a lethal outcome, 25% had a poorer outcome GOS 3, and none had a better outcome.

Among patients with lethal outcome of GOS 1 - about 52.9% had intraventricular extension and 47.1% did not have associated intraventricular extension. With lethal outcome of GOS 2 - 14.3% had intraventricular extension, 0% had no IVH, GOS 3 - 21.4% had IVH, 78.6% did not have associated intraventricular extension. With lethal outcome of GOS 3 - 0% had intraventricular extension, 100% did not have associated intraventricular extension. With lethal outcome of GOS 5 - 0% had IVH and 100% did not have associated intraventricular extension (Fig. 8).

The lethal outcome GOS 1 was seen in 44.4% of infratentorial and 25.5% with supratentorial locations. The better outcome GOS 5 was seen with 78.6% supratentorial and 21.4% with infratentorial regions.

The volume the intracerebral bleed less than 30 mL had a better outcome with GOS 4 and 5. Among the patients with the volume, the intracerebral bleed between 30-60 mL, 15.8% had lethal outcome, 28.3% had poorer outcome, and 57.9% had better outcome. With the volume, the intracerebral bleed more than 60 mL - 69.2% had lethal outcome, 14.8% had poorer outcome, and only 15.4% had better outcome (Fig. 9).

Among the patients with the outcome, MRS 6 - lethal outcome was seen in 69.2% with the volume of bleed more than 60 mL, 15.8% with 30-60 mL, 17.9% with less than 30 mL. With the outcome of severe disability, MRS 5 - 3.6% with less than 30 mL, 0% with 30-60 mL, 7.7% with more than 60 mL. With the outcome of moderate disability MRS 4 - 28.6% with less than 30 mL, 26.3% with 30-60 mL, 7.4% with more than 60 mL. With the outcome of slight disability, MRS 2 - 7.1% with less than 30 mL, 15.8% with 30-60 mL, 7.1% with more than 60 mL. With the outcome of NO disability, MRS 1 - 28.6 with less than 30 mL, 15.8% with 30-60 mL, 7.7% with more than 60 mL.

This implies that outcome modified Rankin Score directly correlates with volume of the intracerebral bleed. The correlation is statistically significant (p-0.011*).

**DISCUSSION**

The mortality is increasing with increasing age. This observation is consistent with the previous studies in the literature like Sunil et al.² and Maya et al.²

The lethal and poorer outcome was more in males compared to females. These observations are consistent with previous studies like study by Sunil et al.¹

The most common manifestation is focal neurological deficit followed by alteration in the level of consciousness followed by headache and vomiting, which are consistent with the previous study by Sahini et al.³ The higher the MAP, poorer the outcome, and the correlation is statistically significant (p - 0.001*). The admission GCS directly correlates with GOS at discharge. The correlation is statistically significant (p - 0.000**) and similarly GOS has inverse correlation with NIHSS, which was statistically significant (p - 0.000**).
The volume of ICH has a direct correlation with the outcome GOS at the time of discharge. The correlation is statistically significant \( (p=0.005^*) \). These observations are consistent with previous studies in the literature. More volume of intracerebral bleed was associated with lethal and poor outcome with the bleed in supratentorial location. But, with infratentorial even small volume of the bleed was associated with lethal outcome. The correlation is statistically significant \( (p=0.001^*) \).

This implies that a poor outcome was observed with whether the volume of the intracerebral bleed was more or even less in the brainstem location. However, patients with cerebellar bleed as well as subarachnoid haemorrhage in the study had a better outcome. But, the number of patients in the study group was very less to infer any conclusions in this observation. The correlation is statistically significant \( (p=0.011^*) \) for lobar bleed, but for other locations, the \( p \) value was not significant.

The presence of intraventricular extension is associated with a poorer or a lethal outcome whereas the absence of intraventricular extension had better outcome GOS 5 at the time of discharge. The correlation is statistically significant \( (p=0.000^{**}) \).

The infratentorial location has a comparatively poorer outcome when compared to supratentorial location. Hypertensive bleed occurs form rupture of small aneurysms.\(^4,5\) The intracerebral haemorrhage has three distinct stages or phases: (1) haemorrhage, which occurs initially, (2) expansion of the haematoma, and (3) oedema surrounding the haematoma.\(^6\) The prognosis and the end result relies on the second and third phase of the haemorrhage. The expansion of the haematoma, which occurs some hours later after the onset of symptoms. This consists of rise in Intracranial Pressure (ICP), which interferes with the integral functioning of the brain tissue as well as the blood-brain barrier: In addition, the resultant venous outflow obstruction initiates tissue thromboplastin release. This results in coagulopathy locally.\(^7\)

The expansion of the haematoma is associated with hyperglycaemia, hypertension, and use of anticoagulants in about 33% of the cases. The haemorrhage volume at the time of presentation and the rapidity of hematoma expansion subsequently are important factors in prognostication.

Subsequent to the expansion of the haematoma, cerebral oedema develops around the bleed as a result of inflammatory mediators and the breakdown of the blood-brain barrier, which is an important cause for worsening of the neurological status. The oedema starts developing over the next two days after the bleed. The overall mass effect can cause elevation of intracranial pressure, which can lead to the displacement of adjacent brain tissue and may result in herniation with consequences.\(^8\)

The correlation between GOS and comorbid diseases was not statistically significant. These observations differed from the observations in the study by Maya et al\(^9\) and Arboix et al\(^9\) had stated that the presence of diabetic status is an important independent factor prognosticating mortality in patients with intracerebral bleed.

**CONCLUSION**

The lethal and poorer outcome is more with increased age, more in males compared to females. The more the mean arterial pressure (MAP), lethal and poorer was the outcome. Lesser MAP was associated with a better the outcome. The Glasgow Outcome Score (GOS score) has a direct correlation with Glasgow coma scale (GCS) and inverse correlation with NIHSS score.

More the volume of intracerebral bleed was associated with lethal and poor outcome with the bleed in supratentorial location. The volume of ICH has a direct correlation with the outcome GOS. With infratentorial location even small volume of the bleed was associated with lethal outcome. The presence of intraventricular extension is associated with a poorer or a lethal outcome.

**REFERENCES**