

Clinical and Radiological Presenting Features and Post-Operative Complications of Intracerebral Haematoma by Burrhole Operation Using Urokinase

Mohammad Nazrul Hossain¹, Israt Zerin Eva², Junayed Ahmad³, Nazmul Hassan Saki⁴, Monowar Ahmad Tarafdar⁵

¹Associate Professor, Department of Neurosurgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet, Bangladesh.

²HMO, Dept of medicine, Dhaka Medical College Hospital, Dhaka, Bangladesh.

³IMO, Department of Neurosurgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet, Bangladesh.

⁴IMO, Department of Neurosurgery, Jalalabad Ragib-Rabeya Medical College Hospital, Sylhet, Bangladesh.

⁵Professor, Department of Community Medicine, ZH Sikder Women's Medical College, Dhaka, Bangladesh.

*Correspondence:

Mohammad Nazrul Hossain, Associate Professor, Department of Neurosurgery, Jalalabad Ragib-Rabeya Medical College, Sylhet, Bangladesh, E-mail: drmnh2003@gmail.com.

Received: 12 September 2017; Accepted: 06 October 2017

Citation: Nazrul Hossain M, Zerin Eva I, Ahmad J, et al. Clinical and Radiological Presenting Features and Post-Operative Complications of Intracerebral Haematoma by Burrhole Operation Using Urokinase. J Med - Clin Res & Rev. 2017; 1(2): 1-5.

ABSTRACT

This was a prospective and interventional type of study conducted in the Neurosurgery Department of Dhaka Medical College Hospital (DMCH), Dhaka from July 2010 to December 2010 with a sample size of 30 to observe the presenting feature, radiological findings and complications of burrhole aspiration of intracerebral haematoma by using urokinase. Purposive Sampling technique was used using a semi-structured data collection sheet designed for this study. Equipments used for the study were CT Scan, Soft catheter and Standard burrhole instrument. It was observed that 73% of the cases were within 9-12 GCS. Basal ganglion was involved in about 77% cases while fronto parietal lobe was involved in only 3% cases. CT scan revealed that half [15 (50.00%)] of the patients' haematoma was in the left hemisphere. Without considering the volume of extension into the ventricles, the average intracerebral haematoma volume was 41.43 ml and the volume ranged from 20.0 to 80.0 ml. 18 (60.0%) of the patients' had ventricular extension of the haematoma. Three-fifth of the haematomas were complicated with ventricular extension. More than one-fourth of the patients [8 (26.67%)] died before 3rd POD. In a single (3.33%) patient GCS decreased. Out of the remaining 21 cases, GCS increased 1 to 5 points. Pneumocephalous was the complication in about 17% cases, while accidental catheter withdrawal and aspiration pneumonia were the complications in about 7% each. It was revealed that 50% deaths were due to respiratory failure and all these deaths were before 3rd POD. Involvement of different areas of the cerebral hemisphere had strong association in outcome, when only death was considered; i.e. only 14.3% patients with cortical haematoma died on the contrary, 85.7% patients with ICH in the basal ganglia with ventricular extension (3 or more ventricles) died. Early treatment (within 24 hours of occurrence) by using minimally invasive technique and clot removal by urokinase mediated clot lysis can improve the consequences especially those with haematoma volume <40 ml, lobar haematoma and without ventricular extension.

Keywords

Spontaneous intracerebral haematoma, Burrhole operation, GCS level, Urokinase.

Introduction

Spontaneous intracerebral haematoma (SICH) is one of the most devastating forms of cerebrovascular disease accounting for about 10% of all strokes. It is associated with high morbidity and mortality [1]. The role of surgery in the management of these cases is controversial. It is possible that some cases will benefit from surgical evacuation. Current practice favours surgical intervention in following situations: lobar haemorrhage, clot volume between 20 to 80 ml, worsening neurological status, relatively young patients, and haemorrhage causing midline shift or raised intracranial pressure (ICP) [2]. Surgical indications in cerebellar haematomas however are more accepted. Haematomas above 3 cm diameter and those causing hydrocephalus, generally require surgical evacuation [1,3,4].

Primary intracerebral hematoma (ICH) is associated with a high mortality and severe disability. The treatment of choice is still controversial and may be surgical or non-surgical (conservative) [1,5]. Theoretically, clot removal is beneficial because it reduces hematoma volume and may therefore also lower intracranial pressure [6], reduce the chance of oedema formation, and improve perfusion in the affected hemisphere [3,7]. The effect on perifocal ischemia resulting from hypoperfusion is unclear; as such an ischemia itself is refuted by a recent publication [8].

Secondary enlargement of the hematoma [9] and neurotoxic oedema due to high levels of thrombin and blood degradation products [10] also may be reduced by clot removal. However, a classical open craniotomy may further traumatize brain tissue, and there is no unequivocal evidence that it reduces mortality [11]. Minimally invasive surgery (MIS) combines benefits of surgical clot removal with limited tissue damage and shorter surgery duration with the possibility of using local anaesthesia. Ultrasound-guided endoscopic clot removal, tested in a clinical randomized trial by Auer et al. (1985), suggested improved outcome after MIS [12].

Clinical Features depends on the site and size of the hematoma. Patients with a large hematoma usually have a decreased level of consciousness [13] as a result of increased intracranial pressure and the direct compression or distortion of the thalamic and brain-stem reticular activating system [14]. Decreased central benzodiazepine-receptor binding on cortical neurons in the presence of small, deep lesions may also contribute to altered consciousness [15].

Expansion of the hematoma is the most common cause of underlying neurologic deterioration within the first three hours after the onset of haemorrhage. Worsening cerebral oedema is also implicated in neurologic deterioration that occurs within 24 to 48 hours after the onset of hemorrhage [16]. Infrequently, late deterioration is associated with progression of oedema during the

second and third weeks after the onset [9].

Materials and Methods

This was a prospective and interventional type of study conducted Neurosurgery Department of Dhaka Medical College Hospital (DMCH), Dhaka from July 2010 to December 2010. All the Spontaneous Intracerebral Haematoma patients full filling the inclusion criteria were the study population of this study with a sample size of 30, Purposive Sampling technique was used using a semi-structured data collection sheet designed for this study. Equipments were used for the study were CT Scan, Soft catheter and Standard burrhole instrument.

Results

In this study, information was collected from 30 subjects who recruited for the study as per the inclusion and exclusion criteria in Neurosurgery Unit of DMCH during July 2010 to December 2010.

GCS	Frequency	Percent
6 – 8	3	10.0
9 – 12	14	73.3
13 - 15	5	16.7
Total	30	100.0

Table 1: Distribution of respondents (n=30) by GCS. Mean \pm SD 8.00 \pm 1.661 Mode 6 Range 6 to 11.

Site of haematoma	Frequency	Percent
Basal ganglia	23	76.7
Parietal lobe	2	6.7
Temporo-parietal lobes	2	6.7
Parieto-occipital lobes	2	6.7
Fronto-parietal lobes	1	3.3
Total	30	100.0

Table 2: Distribution of the cases by the site of haematoma.

CT scan revealed that half [15 (50.00%)] of the patients' haematoma was in the left hemisphere Without considering the volume of extension into the ventricles, the average intracerebral haematoma volume was 41.43 ml and the volume ranged from 20.0 to 80.0 ml. 18 (60.0%) of the patients' had ventricular extension of the haematoma. Three-fifth of the haematomas were complicated with ventricular extension. In most of the complicated cases [8 (26.67%)] the volume measured 41 to 60 ml. On an average the delay from ictus to intervention was 41:43 hours with a range from 7:00 to 72:00 hours. Catheter in the Ventricle was inserted in 2 (6.6%) patients. Catheter was placed in the centre of in 23 (76.7%) of patients. On an average the patients received 5.7 doses (instillation and aspiration) of urokinase with a range from 2 to 10 times. Most [14 (46.7%)] of them received 4 to 6 doses.

More than one-fourth of the patients [8 (26.67%)] died before 3rd POD (the first day for postoperative assessment of changes achieved through intervention). In a single (3.33%) patient GCS decreased. Out of the remaining 21 cases, GCS increased 1 to 5

points.

Complication		Frequency	Percent
No complication		15	50.0
Complications	Pneumocephalous	5	16.7
	Accidental catheter withdrawal	2	6.7
	Aspiration pneumonia	2	6.7
	Chest pain	2	6.7
	Others (Meningitis, Re-stroke, Re-bleeding, Psychosis)	4	13.3
Total		30	100.0

Table 3: Distribution of the cases by their postoperative complications.

		Time of death			
		Before 3rd POD	Before discharge	Before 30th POD	Total
Cause of death	Respiratory distress then death	7			7
	Sudden chest pain, then death		3		3
	Meningitis		1		1
	Aspiration pneumonia	1			1
	Re-bleeding		1		1
	2nd stroke			1	1
Total		8	5	1	14

Table 4: Distribution of the cases by their time and cause of death.

Involvement of different areas of the cerebral hemisphere had strong association in outcome, when only death was considered; i.e. only 14.3% patients with cortical haematoma died on the contrary, 85.7% patients with ICH in the basal ganglia with ventricular extension (3 or more ventricles) died.

Discussion

This study was aimed at reporting the clinical feature, radiological finding and complications of burrhole aspiration by urokinase mediated clot lysis in spontaneous intracerebral haematoma at DMCH.

Only 3 (10.0%) patients did not lose consciousness and the remaining 27 (90.0%) were unconscious at admission. Cent percent of the patients developed motor weakness. Exactly half of the cases [15 (50.0%)] had 'Rt. sided weakness' and other 15 (50.0%) patients had 'Lt. sided weakness'. In 27 (90.0%) patient 'vocal disturbance' could not be assessed. Of the remaining, 2 (6.7%) patients had aphasia and 1 (3.3%) experienced dysphasia. Different combinations of clinical presentations found among the patients. More than two-fifth 13 (43.333%) patients were suffering from limb weakness, unconsciousness and vomiting. More than a quarter [8 (26.67%)] had limb weakness with unconsciousness. 3 (10.0%) were suffering from limb weakness, unconsciousness and disturbance in phonation. The remaining 20.0% cases presented with 5 different combinations of various signs. All patients had

some degree of contralateral hemiparesis or hemiplegia, and 3 patients had additional aphasia [17]. All patients had some degree of neurological deficit such as contralateral hemiparesis, hemiplegia, and dysphasia [18]. Das S [19], found that all of the patients had history of alteration of level of consciousness, 36 (90.00%) had hemiplegia/hemiparesis and 8 (20.00%) had aphasia/dysphasia. Islam T [20], found that sudden alteration of level of consciousness, hemiplegia, hemiparesis, aphasia and dysphasia which were present in 100%, 73.3%, 26.7%, 13.4% and 10.0% respectively.

On admission the GCS level ranged from 6 to 11 and average was 8. PAI et al. found, fifty-one patients (53%) were in Glasgow Coma Scale (GCS) 6-8, 18 (19%) between 9-12 and 11 patients (12%) in GCS 13-15 and 16 patients (17%) in GCS <6 pre-operatively. Das S [19], found 21 (52.5%) patients the GCS was in 6-8, 17 (42.5%) between 9-12 and 2 (5%) in GCS 3-5. The mean \pm SD of GCS was (7.98 \pm 1.98) with a range of 5 to 12. Rahman A [22], found that mean \pm SD of GCS was (8.9 \pm 1.96) with a range of 6 to 12 and Islam T [20], found that 20 patients (66.67%) had GCS score 6 to 8 and 10 patients (33.33%) had GCS score 9 to 12.

Half [15 (50.00%)] of the patients developed 'Lt. sided hemiplegia', more than two-fourth [13 (43.33%)] developed 'Rt. sided hemiplegia' and the remaining 2 (6.67%) developed 'Rt sided hemiparesis'. CT scan revealed that half [15 (50.00%)] of the patients' haematoma was in the left hemisphere. More than three-fourth [23 (76.7%)] of the intracerebral haematomas were in basal ganglia. The remaining 7 (23.3%) were in various lobes at cortical level. PAI et al. [21], found seventy-two patients (75%) had basal ganglionic haematomas involving the putamen and globus pallidus, 24 (25%) had lobar haematomas and 2 (2%) had thalamic extension of the hematoma. Das S [19], found that 21 (56.75%) had left sided haemorrhage and 16 (43.25%) patients had haemorrhage in the right side. Among them majority 20 (50.00%) of ICH were in the basal ganglia, 17 (42.5%) in the lobar and 3 (7.5%) in the cerebellar region respectively. Rahman A [22] found 3 (10.3%) had lobar haematoma, 19 (65.5%) had putaminal haematoma and 7 (24.1%) had thalamic haematoma. Islam T [20] found 17 (56.7%) patients had left sided haemorrhage and 13 (43.3%) patients had haemorrhage in the right side. Among them 15 (50%) of ICH were in the basal ganglia region and 15 (50%) in the lobar.

Without considering the volume of extension into the ventricles, the average intracerebral haematoma volume was 41.43 ml and the volume ranged from 20.0 to 80.0 ml. PAI et al. [21], found the volume of the hematoma varied from 30 ml to above 70 ml. Das S [19], found that median haematoma volume was 40.00 ml and the volume ranged from 30-90 ml. Rahman A [22], found ,the median haematoma volume was 46.50 ml and the volume of haematoma varied from 32-66 ml. Islam T [20], found the median haematoma volume was 44.04 and the volume ranged from 20-70 ml.

More than 90 per cent [28 (93.33%)] of the patients' CT scan showed midline shift. Three-fifth [18 (60.0%)] of the haematomas

were complicated with ventricular extension. In most of the complicated cases [8 (26.67%)] the volume measured 41 to 60 ml. Das S [19], found the ventricles were involved radiologically in 11 (27.5%) of patients. Islam T [20], found the ventricles were involved radiologically in 15 (50%) patients.

Catheter in the Ventricle was inserted in 2 (6.6%) patients. Catheter was placed in the centre of the haematoma in 23 (76.7%) of patients and the remaining [5 (16.7%)] were benefited by both (centre of haematoma and ventricle) procedures. On an average the patients received about 6 doses (instillation & aspiration) of urokinase with a range from 2 to 10 times. Most [14 (46.7%)] of them received 4 to 6 doses. Out of total 40 patients with ICH in 25 (62.5%) patient's burr hole aspiration of haematoma was done, in 8 (20.00%) patients external ventricular drainage, in 5 (12.50%) patient's craniectomy and decompression, in 2 patient's (5.0%) craniotomy and evacuation of haematoma was done [19]. Out of total 29 patients with ICH in 8 (27.56%) patients external ventricular drainage, in 7 (24.13%) patient's craniectomy and decompression, in 14 (48.27%) patient's craniotomy and evacuation of haematoma was done [22]. Out of total 30 patients with ICH in 13 (43.3%) patients craniotomy and evacuation of haematoma was done, in 13 (43.3%) patients burr hole aspiration of haematoma was done, in 3 (10%) patients craniectomy and evacuation of haematoma was done, in 1 (3.3%) patient external ventricular drainage (EVD) was done [20].

More than one-fourth of the patients [8 (26.67%)] died before 3rd POD (the first day for postoperative assessment of changes achieved through intervention). In a single (3.33%) patient GCS decreased. Out of the remaining 21 cases, GCS increased 1 to 5 points. Again, among the live patients the GCS level had increased, i.e. on admission the level ranged from 6 to 11 and on 30th POD the lowest level was 10. GCS and also, the average of GCS had increased from 8.00 (On admission) to 13.44 (On 30th POD). There is positive correlation between the changes of GCS level among the alive cases. {Correlation [GCS (on admission and on 3rd POD)] = 0.724; R = 0.740 (p = 0.001); Correlation [GCS (on admission and at discharge)] = 0.846; R = 0.846 (p = 0.000) and Correlation [GCS (on admission and on 30th POD)] = 0.787; R = 0.641 (p = 0.002)}.

Near about three-fourth [22 (73.33%)] of the patients had unfavourable outcome; 14 (46.67%) 'Death' [among them 1 (7.14%) patient from lobar haematoma, 3 (21.42%) patient's from basal ganglia without ventricular extension, 4 (28.57%) patient's from basal ganglia with less than three ventricular extension and remaining 6 (42.82) patient's from basal ganglia with more than three ventricular extension] and 8 (26.67%) 'Sever disability' [one from lobar (12.5%), six from basal ganglia with less than three ventricle extension (75.0%) and one from basal ganglia with more than three ventricle extension (12.5%)]. The remaining 8 had favourable outcome; 5 (16.67%) 'Moderate disability' [two from lobar (40.0%) and 3 (60.0%) basal ganglia without ventricular extension] and 3 (10.0%) 'Good recovery' (all from lobar haematoma). Das S [19], found that in basal ganglia

mortality is 25%, severe disability 50% and moderate disability is 25%. In lobar haematoma mortality is 41.2%, severe disability is 35.5% and moderate disability is 23.5%. In Basal ganglia with ventricular extension, mortality is 100% and lobar haematoma with ventricular extension mortality is 71.4%. Islam T [20], found that in basal ganglia mortality is 73.30% and in lobar haematoma mortality is 6.7%.

50% (15) of the interventions were uncomplicated. Iatrogenic pneumocephalus was the most [5 (16.7%)] occurring complication, 2 (6.7%) individuals each had 'accidental catheter withdrawal', aspiration pneumonia and chest pain. Other complications like, meningitis, re-stroke, rebleeding, psychosis accounted for a single (3.3%) case. Near about half [14 (46.67%)] of the patients died. Out of the deaths, most [8 (26.67%)] occurred before 3rd OPD (1st date for assessment of outcome). 'Respiratory distress' was cause of 7 (50.0%) deaths. MI was responsible for 3 (21.48%) deaths. Re-bleeding, meningitis, aspiration pneumonia and re-stroke were the cause of remaining 4 (28.52%) deaths. Jin et al. reported, nine patients' (16.9%) died before hospital discharge (one from cardiac problems and eight from respiratory failure).

Involvement of different areas of the cerebral hemisphere had strong association in outcome, when only death was considered; i.e. only 14.3% patients with cortical haematoma died. On the contrary, 85.7% patients with ICH in the basal ganglia with ventricular extension (3 or more ventricles) died. Outcome achieved depended on initial GCS (on admission). The ratio of death was higher in patients with lower GCS level group. All of the patients with 'good recovery' had a GCS of 9 to 12 and patients with 'moderate disability' had initial GCS more than 6. Death rate was higher in patients with lower GCS on admission; the difference was statistically significant ($\chi^2 = 3.519$, $p < 0.05$). Outcome of haematoma in the basal ganglia was not good, as out of 23 (76.67%) haematoma 13 (43.33%) died. All 3 'good recovery' had cortical haematoma. Although the death rate was higher in patients with haematoma in the basal ganglia, the difference was not statistically significant; [$\chi^2 = 3.846$ ('p value' just above 0.05)]. Outcome of large haematoma volume was not good; death rate was high among patients with larger haematoma (>40 ml). All 3 'good recovery' was with small volume of haematoma (<40 ml). The difference was statistically significant between death and haematoma volume ($\chi^2 = 0.117$, $p < 0.05$). The ratio of death was higher among patients with ventricular extension of the haematoma. 10 (33.33%) died out of 18 (60.0%) with ventricular extension. On the contrary, only 4 (13.33%) died among 12 (40.0%) the cases without ventricular extension. The death rate was 1.67 times higher in patients with ventricular extension. All the patients with 'good recovery' did not have any ventricular extension of the haematoma. The difference in death among patients with ventricular extension of haematoma was statistically significant; ($\chi^2 = 1.429$, $p < 0.05$). The cortical haematoma showed time dependant outcome, 'the earlier the intervention the better the recovery'. The single death in patient suffering from ICH in the cortex, had a 72 hours delay from incidence to start of intervention. There was statistically significant difference in death and interval between ictus and intervention (χ^2

= 0.153, $p < 0.05$); better results with early intervention. Outcome of study by Jin et al. [18], was - at discharge, 25 patients (47%) had achieved good recovery (17 patients GOS 3, 6 patient GOS 4, and 2 patients GOS 5), and 19 patient (35.8%) remained vegetative (GOS 2). At 6 months' follow up, 29 patients (55%) had achieved good recovery (17 patients GOS 3, 8 patient GOS 4, and 4 patients GOS 5) and 15 patient (28.3%) remained vegetative (GOS 2).

Conclusions and Recommendations

Early treatment (within 24 hours of occurrence) by using minimally invasive technique and clot removal by urokinase mediated clot lysis can improve the consequences especially those with haematoma volume < 40 ml, lobar haematoma and without ventricular extension. Bad prognostic factors were GCS level < 8 , haematoma in the basal ganglia, ventricular extension of the haematoma. Volume > 40 ml and delay in intervention. This was a small scale study done at a single centre over a brief duration. A large scale, multi-centre study over long duration with repeated post-discharge follow-ups until 6 months to 1 year will give an elaborate picture on outcome of aspiration of SICH using urokinase and factors influencing the outcome. To minimise the complications and death i) should follow the standard burrhole procedure to avoid excessive entry of air in to the brain, ii) post-operative chest physiotherapy to avoid pneumonia and limb physiotherapy for improving functional status of the limbs, iii) aspiration pneumonia can be prevented by following appropriate NG feeding procedure.

References

1. Ojemann RG, Heros RC. 1983. Spontaneous brain hemorrhage. *Stroke*. 1983; 14: 468-475.
2. Siddique MS, Mendelow AD. Surgical treatment of intracerebral hemorrhage. *Br Med Bull*. 2000; 56: 444-456.
3. Lejeune JP, Thines L. Neurosurgical management of spontaneous cerebral hemorrhage. *J Neuroradiol*. 2003; 30: 332-335.
4. Escosa BM, Sola RG. Surgical indications in non traumatic intracerebral hemorrhage. *Rev Neurol*. 2001; 32: 1060-1062.
5. Unwin DH, Batjer HH, Greenlee RG. Management controversy: medical versus surgical therapy for spontaneous intracerebral hemorrhage. *Neurosurg Clin N Am*. 1992; 3: 533-537.
6. Chambers IR, Banister K, Mendelow AD. Intracranial pressure within a developing intracerebral haemorrhage. *Br J Neurosurg*. 2001; 15: 140-141.
7. Nath FP, Kelly PT, Jenkins A, et al. Effects of experimental intracerebral hemorrhage on blood flow, capillary permeability, and histochemistry. *J Neurosurg*. 1987; 66: 555-562.
8. Zazulia AR, Diringner MN, Videen TO, et al. Hypoperfusion without ischemia surrounding acute intracerebral hemorrhage. *J Cereb Blood Flow Metab*. 2001; 21: 804-810.
9. Zazulia AR, Diringner MN, Derdeyn CP, et al. Progression of mass effect after intracerebral hemorrhage. *Stroke*. 1999; 30: 1167-1173.
10. Lee KR, Kawai N, Kim S, et al. Mechanisms of edema formation after intracerebral hemorrhage: effects of thrombin on cerebral blood flow, bloodbrain barrier permeability, and cell survival in a rat model. *J Neurosurg*. 1997; 86: 272-278.
11. Prasad K, Shrivastava A. Surgery for primary supratentorial intracerebral haemorrhage. *Cochrane Database Syst Rev*. 2000; 2: CD000200.
12. Auer LM, Ascher PW, Heppner F, et al. Does acute endoscopic evacuation improve the outcome of patients with spontaneous intracerebral hemorrhage. *Eur Neurol*. 1985; 24: 254-261.
13. Mohr JP, Caplan LR, Melski JW, et al. The Harvard Cooperative Stroke Registry: a prospective registry. *Neurology*. 1978; 28: 754-762.
14. Andrews BT, Chiles BW, Olsen WL, et al. The effect of intracerebral hematoma location on the risk of brain-stem compression and on clinical outcome. *J Neurosurg*. 1988; 69: 518-522.
15. Hatazawa J, Shimosegawa E, Satoh T, et al. Central benzodiazepine receptor distribution after subcortical hemorrhage evaluated by means of [123] iomazenil and SPECT. *Stroke*. 1995; 26: 2267-2271.
16. Mayer SA, Sacco RL, Shi T, et al. Neurologic deterioration in noncomatose patients with supratentorial intracerebral hemorrhage. *Neurology*. 1994; 44: 1379-1384.
17. Montes JM, Wong JH, Fayad PB, et al. Stereotactic computed tomographic-guided aspiration and thrombolysis of intracerebral hematoma: protocol and preliminary experience. *Stroke*. 2000; 31: 834-840.
18. Jin SC, Hwang SK, Cho DS, et al. Urokinase Thrombolysis for Nonaneurysmal Spontaneous Intraventricular Hemorrhage. *J Korean Neurosurg Soc*. 2005; 38: 281-286.
19. Das S. Role of surgery in primary spontaneous intracerebral haemorrhage- Analysis of early outcome of surgery in relation to site and size of haemorrhage; MS (Neurosurgery thesis). 2004; BSMMU.
20. Islam TKM. Analysis Of early outcome of surgery in relation to preoperative Glasgow coma scale (GCS) score and volume of haematoma; MS (Neurosurgery thesis). 2010; DMC.
21. Pai SB, Varma RG, Parthiban JKBC, et al. Keyhole craniectomy in the surgical management of spontaneous intracerebral hematoma. *Neurology Asia*. 2007; 12: 21-27.
22. Rahman A. To compare the outcomes between surgical and medical treatment of spontaneous supratentorial intracerebral haemorrhage and also to compare the outcomes according to Glasgow Coma Scale (GCS), Glasgow outcome scale (GOS), Modified Rankin Scale (MRS) and mortality between two groups; MS (Neurosurgery thesis). 2008; CMC.