Original Article

Outcome of Traumatic Subarachnoid Hemorrhage

Nazar Husain, Muhammad Akmal Husain, Tariq Ahmad

ABSTRACT

Objectives: Traumatic Subarachnoid Hemorrhage (Tr SAH) is a part of traumatic brain injury (TBI). Traumatic brain injury has worst impact on society converting useful citizens to severely disabled and vegetative patients. Traumatic subarachnoid hemorrhage is a negative prognostic factor. We have tried to study the contribution of traumatic subarachnoid hemorrhage to this lethal entity. **Methods:** Thirty patients with head injury with traumatic subarachnoid hemorrhage, diagnosed in emergency ward, were included and managed. They were assessed by GCS and Fissure grading at admission. Outcome was assessed by Glasgow outcome scale at the time of discharge and at thirty days after discharge. **Results:** Traumatic subarachnoid hemorrhage is a negative prognostic factor. Outcome depends upon clinical condition at time of admission (mild head injury & severe head injury $p<0.05$), distribution of the blood in the brain (F2 & F4, F2 & F3 $p<0.05$) and associated brain injury ($p<0.05$). **Key Words:** Traumatic subarachnoid hemorrhage, Vasospasm, Hydrocephalus, Traumatic brain injury.

INTRODUCTION

Traumatic brain injury is a common cause of morbidity and mortality world wide.[1] The incidence of TBI in industrialized and non-industrialized countries varies from 150 – 250 cases per 100,000 population per year.[2] Primary head injury is preventable and secondary head injury is treatable.[3] Traumatic subarachnoid hemorrhage is a common finding in the setting of TBI.[4] It is difficult to estimate the real incidence of traumatic SAH in TBI but the medical literature quotes its incidence as 25 to 30% in cases of moderate and severe head injury[5]. As a result of Tr.SAH, there is mechanical tearing, stretching or laceration of blood vessels leading to bleeding in subarachnoid space.[6] Whether tr.SAH is an independent causative factor for worse clinical outcome following TBI due to vasospasm, or mere a marker of more severe incurred head injury, is not clear.[7] Patients with tr.SAH may develop vasospasm, hydrocephalus, brain oedema and ischemia etc. The mortality rate was greater in patients who had cisternal clot >1mm. Recovery rate and daily activity were lower in patients with intraventircular hemorrhage. Patients with severe SAH have worse clinical outcome.[8]

METHODS

The patients with head injury were received in emergency ward. They were assessed by Glasgow coma scale. CT scan brain was done. Thirty patients with traumatic subarachnoid hemorrhage were included. Depending on the distribution of blood on CT scan, Fissure grading was done (Table I). Head injury was managed according to standard protocol by maintaining air way, administrating IV fluid and Mannitol. Nimodipine was given to a group of patients. Patients were observed for complications of traumatic subarachnoid hemorrhage. Outcome was assessed according to Glasgow outcome scale (I = dead, II = vegetative, III severe disability, IV mild disability, V good recovery) at the time of discharge and at thirty days after discharge.

RESULTS

It is a prospective study of 30 patients with traumatic subarachnoid hemorrhage, age ranged form 7 to 75 years, with average age was 30. 16 years and M:F 4:1.. The causes of traumatic SAH in patients include road traffic accident (RTA) 12 (73.33%), fall 4(13.33%), fight 3(9.9%) firearm injury 1(3.33%).

CT scan brain was done which showed the anatomical distribution of subarachnoid hemorrhage like.

Interhemispheric fissure bleed, 10(33.33%) patients
Sylvian fissure bleed 3(3.33%)
Cortical Bleed 7(23.33%)
Perimesencephalic bleed 2(6.66%)
Intraventricular bleed 2(6.66%)

ASSOCIATED PATHOLOGIES INCLUDED

Brain contusions 5(16.66%)

Extradural Hematoma (EDH) 1(3.33%), Acute Subdural Hematoma (SDH) 2(6.66%)

Linear fracture 1(3.3%) Depressed fracture 1(3.33%)

Foreign body 1(3.3%) ,

Brain edema 5(6.66%).

The conscious level of the patient was assessed according to Glasgow coma scale and was like

GCS 13-15, 11(36.66%) patients
GCS 9-12 9(3.33%) = 30%
GCS 8 and below 10 = 33.3%

Patients were admitted in Emergency Ward (E/W) and were managed in intensive care unit (ICU) Neurosurgery ward. Nimotop was given to eight patients. Inj. Mannitol and fluids were given. Outcome was assessed by Glasgow outcome scale at the time of discharge and at 30 days after discharge (Table 2).

Depending upon the thickness of the blood on CT Scan, patients were graded by Fissure grading like:

Table 1:

F Grading	Blood distribution on CT scan	No. of patients
F1	Blood not visible	0
F2	Thickness of blood < 1 mm	14(46.66%)
F3	Thickness of blood >1mm	14(46.66%)
F4	Intraventicular hemorrhage	2(6.66%)

INCLUSION CRITERIA

All Patients with traumatic SAH.

Small extradural hematoma (EDH) or single small contusion was acceptable

EXCLUSION CRITERIA

All patients with major accidental other brain and systemic injuries effecting the outcome

Postoperative patients

Major metabolic and systemic ailments

Patients with F1 fissure grading of subarachnoid hemorrhage

The results are as follows

Table 2:

Overall Outcome

Sr. No	Discharge		Follow-up at 30 days	
1	G-V	4(15.38%)	G-V	19=61%
2	G- IV	16(53.33%)	G-IV	6=23%
3	G-III	5(16.66%)	G-III	0
4	G-II	3(10%)	G-II	3=10%
5	G-I	2(6.66)	G-I	2=6.66%

It is clear from results (Table 2) that the overall immediate and follow up outcome was good in GOC III, IV & V.

Brain edema + Basal ganglia contusion 6 patients

At the time of discharge

G-IV=1(3.33%)
G-II=4(13.33%)
G-I=1(3.33%)

Follow-up

G-V=1(3.33%), G-II=4(13.33%), G-I=1(3.33%)

Grade III.IV,V (Glasgow outcome scale) favorable group

I, II (Glasgow outcome scale) unfavorable group

Table 3:

group * outcome Crosstabulation

Count

		outcome		
		overall outcome	head injury+brain oedema	Total
group	favourable group	25	1	26
	unfavourable group	5	5	10
Total		30	6	36

chi- square value = 11.077

df = 1
p-value (1-sided) = 0.005
(Both groups have significant statistical difference in results. P<.05)

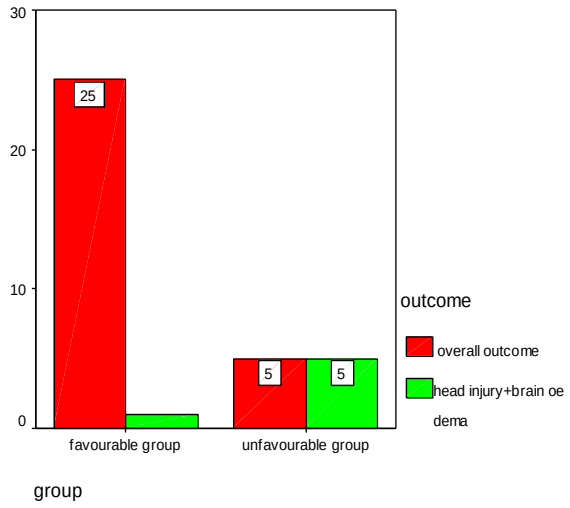

Table 4:
At the Time of Discharge

	G-V	G-IV	G-III	G-II	G-I
Good 11(13–15)	3 9.9%	8 26.4%	0	0	0
Moderate 9(9 – 12)	1 3.3%	-6 19%	1 3.3%	0	1 3.3%
Severe 10 (<8)	0	1 3.3%	4 13.2%	4 13.2%	1 3.3%

Table5:
Follow-up at 30 days of Discharge

	G-V	G-IV	G-III	G-II	G-I
(13 – 15)	14(46.66%)				0
(9 – 12)	4(10%)	2(6.66%)	0	0	1
(<8)	1(3.33%)	3(9.99%)	0	4(13.33%)	1(3.33%)

It was found that patients with GCS (13-15) at admission had 46.66% favorable outcome as compared to patient with severe head injury which had (13.32%) favorable and unfavorable 16.66% (p value 001).

Table6:

group * condition of the patient at follow up Crosstabulation

Count

		condition of the patient at follow up		
		good	moderate	Total
group	favourable group	14	6	20
	unfavourable group		1	1
Total		14	7	21

chi- square value = 2.1
df = 1
p-value (1-sided) = 0.0735
Mild and moderate head injury do not have statistical significant difference in results. P>0.05

Table7:

group * condition of the patient at follow up Crosstabulation

Count

		condition of the patient at follow up		
		good	severe	Total
group	favourable group	14	4	18
	unfavourable group		5	5
Total		14	9	23

chi- square value = 9.938
df = 1
p-value (1-sided) = 0.001
(Both groups have significant statistical difference in results. P<0.005)

Table 8:

group * condition of the patient at follow up Crosstabulation

Count

		condition of the patient at follow up		
		moderate	severe	Total
group	favourable group	6	4	10
	unfavourable group	1	5	6
Total		7	9	16

chi- square value = 2.861
df = 1
p-value (1-sided) = 0.0455
(Statistically difference acceptable)

Table 9:
At time of Discharge
Follow-up at 30 days of Discharge

	G-V	G-IV	G-II	G-II	G-I	G-V	G-IV	G-III	G-II	G-I
F2	1(3.3%)	13(43.2%)	1(3.3&)			14(46.6%)	1(3.3%)			
F3	3(9.9%)	2(6.6%)	3(9.9%)	3(9.9%)	2(6.6%)	3(9.9%)	2(6.6%)	3(9.9%)	3(9.9%)	2(6.6%)
F4			1(3.3%)	1(3.3%)			1(3.3%)		1(3.3%)	

Table 10:

group * fissure grading at follow up Crosstabulation

Count

		fissure grading at follow up		
		F2	F3	Total
group	favourable group	15	8	23
	unfavourable group		5	5
Total		15	13	28

chi- square value = 7.023
df = 1
p-value (1-sided) = 0.004

Table 11:

group * fissure grading at follow up Crosstabulation

Count

		fissure grading at follow up		
		F2	F4	Total
group	favourable group	15	1	16
	unfavourable group		1	1
Total		15	2	17

chi- square value = 7.969
df = 1
p-value (1-sided) = 0.0025

Table 12:

group * fissure grading at follow up Crosstabulation

Count

		fissure grading at follow up		
		F3	F4	Total
group	favourable group	8	1	9
	unfavourable group	5	1	6
Total		13	2	15

chi- square value = 0.096
df = 1
p-value (1-sided) = 0.378
F2 & F3 and F2& F4 have statistically significant difference in results $p<0.05$ but F3 & F4 have no significant statistical difference in result. P = 0.378
There was not significant change in outcome in patients with Nimodipine intake and those who didn't get the Nimodipine.

DISCUSSION

Breast In our study we have tried to examine the SAH in isolation. That is why we have not included patients with traumatic SAH with other major brain and systemic injuries contributing the major role in outcome. We do admit that there is always associated some traumatic brain injury as it was also evident in Marmarou model of traumatic SAH where modified weight was dropped which also caused some diffuse brain injury thus not allowing to study tr SAH in isolation[9]. Our results show better overall outcome of 84% in favorable group (GOCS III,IV,V) at time of discharge (Table 2). It was also found that patients with good GCS (13-15) at admission had 46.60% favourable outcome as compared with patients with severe head injury which had 13.32% favourable outcome and 16.66% unfavourable outcome.

Paiva et al showed better outcome in favorable group (GOC IV,V) as compared with unfavorable group (GOC I,II,III). However I grouped like favorable group as (GOC III,IV,V) and unfavorable group like GOC I and II. There was 6.6% mortality which was present only in patients with low GCS less than 12. Distribution of the blood in basal cisterns also played a role like F2 has 50% favourable outcome as

compared with 27% in F3. The outcome was even low in F4. Armin et al had shown that the mortality was higher in patients with clot >1mm on CT scan. The recovery rate and daily life activity on GOC were lower in intraventicular heamorrhage.[7]

The patients with severe tr.SAH had worst prognosis[7]. The amount of extravasated clot on CT scan (fissune grade) and level of GCS on admission, are the most important determinant of predicting mortality of SAH patients[10]. Moreover, when patient had associated brain injury like brain edema or contusion, the outcome was poor like 17.66% favorable (GOCS III,IV,V) outcome and 13.66% unfavorable outcome as compared with overall outcome. We also have observed patients for complications of traumatic SAH. Vasospasm is a known entity in SAH. Our patients did not show vasospasm clinically. Oertel et al had shown through transcranial doppler ultrasono graphy that heamodynamically significant vasospasm does occur frequently in TBI. How ever despite the findings, he was unable to clarify relationship between radilogically detected vasospasm and neurologically deterioration.[11] Moreover the vasospasm could be demonstrated in TBI in absence of tr.SAH.[11]We did not detect vasospasm in our study. It may be due to small size of study as compared with study of 299 patients by Oertel et al. He reports frequent occurrence of vasospasm in tr.SAH.[11]Vasospasm in tr.SAH and TBI is thought due to stretching, tearing and laceration of blood vessels.[6] Positran emission tomography and advanced MRI studies are needed in future to determine the extent to which vasospasm causes celeberal ischemia or infarction, and whether its development is directly affected by Tr.SAH, is not clear.[3]Our two patients had IVH but none of these developed HCP as assessed by clinical examination and C.T. Scan. However, in large series of 301 patients by Oertal et al. hydrocephalus was found in 11.9% cases and it was associated with increasing age, intraventricular hemorrhage, increased thickness and distribution of the blood.[12] Calcium channel blockers are in use to counteract the vasospasm. A recent Cochrane review and meta analysis had found no beneficial effect of CCB in TBI overall but showed a statistically significant although, relatively small beneficial effect in this trSAH subgroup in terms of reducing unfavorable outcome (death and severe disability).[13] Many investigators believe that even in the setting of aneurysmal SAH, CCBS exert the modest benefit through mechanism other then vasospasm.[13]

CONCLUSION

Traumatic Subarachnoid Hemorrhage is a negative prognostic factor. Outcome depends on clinical condition at admission (mild head injury & severe head injury $p<0.05$), distribution of the blood in the brain (F2 & F4, F2 & F3 $p<0.05$) and associated brain injury ($p<0.05$).

REFERENCES

1. Ewout W Steyeberg, Nino Mushkudioni, Pablo Perel, Isabella Butcher, Juan Lu, Gillian SMcHugh, G ordon D Murray, Anthomy Marmuarou, Ian Roberts J.DikF Habbema, Andrew IR Mass: Predicting outcome after TBI: Development and international validation of Prognostic scores based on admission characteristics. PlosMed 2008;5:165.
2. Lecon-Camon J, Dominguez-Morales Mdel R, Barroso Y Martin JM, Murillo-Cabezas F. Epidimiology of TBI tr SAH. Springer Science Pituitary 2006; 8:197-202.
3. Armin S.S, A.R.T. Colohon, J.H. Zhang:Vasospasm in TBI.Acta Neurochir Suppl 2008;104:421-425.
4. Z Wu, S.Li, J.Lei, D.An, E.M. Haacke: Evaluation of Tr SAH using susceptibility weighted imaging. The American Journal of Neurorodiolgy 31-1302-1310 August 2010.
5. European study Gruoup on Nimodipine in severe Head injury. A multicenter trial of the efficacy of nimodipine on outcome after severe head injury. F Neurosurg 1994;80:797-804.
6. Barker S: Subdural and primary SAH: a case study Neonatal Netoo 2007;26:143-51.
7. Armin SS, Colohan AR, Zhang JH. Traumatic subarachnoid haemorrhage: our current understanding and its evolution over the past half century. *Neurol Res.* 2006;28:445–452.
8. Paiva WS. De Andrade AF. De Amorim RL. Muniz PK, Bernardo LS, Figueiredo EG, Teixeira MJ : The prognosis of the TSAH : A prospective repot of 121 patients Int. Surg 2010;95:172-6.
9. Marmarou A, Foda MA, van den BW, Campbell J, Kita H, Demetriadou K. A new model of

diffuse brain injury in rats. Part I: Pathophysiology and biomechanics. J Neurosurg 1994;80:291–300.
10. Sang-Kyu Park, Hyoung-Joon Chun, Dong-Won Kim, Tai-Ho lm, Hyun-Jong Hong, and hyeong-Joong: Acute physiology and chronic health evaluation II and simplified acute physiology score II in predicting hospital mortality of Neurosurgical intensive care unit patients. J Korean Med Sci.2009;24:420-26.
11. Oertel M, Boscardin WJ, Obrist WD, Glenn TC, McArthur DL, Gravori T, Lee JH, Martin NA. Posttraumatic vasospasm: the epidemiology, severity, and time course of an underestimated phenomenon: a prospective study performed in 299 patients. *J Neurosurg.* 2005;103:812–824.
12. Tian HL, Xu T, Hu J, Cui YH, Chen H, Zhou Lf. Risk factors related to hydrocephalus after traumatic subarachnoid hemorrhage. Surg Neurol. 2008;69:241-6.
13. Armin S. S, Colohan A. R. T, Zhang J. H. Vasospasm in traumatic brain injury Acta Neurochir Suppl. 2008;1: 104: 421–425.

AUTHORS

- **Dr. Nazar Husain**
  Assistant Professor of Neurosurgery
  Punjab Medical College, Faisalabad
- **Dr. Muhammad Akmal Husain**
  Associate Professor of Neurosurgery
  Punjab Medical College, Faisalabad
- **Prof. Dr. Tariq Ahmad**
  Professor of Neurosurgery
  Punjab Medical College, Faisalabad