

PROGRESSIVE EXTRADURAL HAEMATOMA AND ITS CLINICAL FEATURES

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ABSTRACT

Introduction: Progressive extradural haematoma is a unique entity by virtue of its evolution over time. The current radiologic and neurointerventional development are the key factors which have contributed to the identification of this new entity among the head injured patients. Factors which might be related to its incidence are important for timely diagnosis and intervention.

Objective: The aim of this study is to identify the factors which predict the incidence of progressive extradural haematoma in adult head injury patients.

Methods: This is a retrospective chart review study, conducted at the department of neurosurgery, Hayatabad Medical Institute Peshawar between January and December 2015. Patients who were admitted with head injury and who later progressed to increase in the volume of epidural haematoma on follow-up CT during the same admission were included. Data was collected about patient demographics, arrival GCS, laboratory and clinical findings such as systolic BP at presentation, pupillary abnormalities, coagulation profile, platelet counts, comorbidities and CT findings such as initial and final volume of epidural hematoma. Patients who were not candidates for surgical evacuation on follow-up CT were excluded.

Results: Overall mean age was 33.79 ± 11.41 years, mean arrival GCS was 10.86 ± 2.45 (median: 11), mean systolic BP was 114.28 ± 18.41 mm of Hg, mean time since injury was 7.59 ± 3.93 hours, mean platelets count was $217 \times 10^3 \pm 119 \times 10^3$. The mean INR was 1.25 ± 6.3 . Overall mean volume at the first CT was 11.78 ± 6.3 mm³ (range: 0 – 23 cc). Similarly, mean volume at the follow-up CT was 43.14 ± 10.16 mm³ (range: 26 – 60 cc) with a mean time between the two scans of 15.28 ± 9.27 hours (range: 4 – 48 hours). The mean GCS at discharge was 12.3 ± 3.5 (median: 13.5).

Conclusion: Outcome in progressive extradural haematoma is affected by age, arrival GCS, systolic BP and the volume at the time of surgery. The time since injury, coagulation profile, platelets count and the volume at first CT is not associated with the outcome.

Keywords: Head Injury, Traumatic Brain Injury, Progressive Extradural Haematoma.

INTRODUCTION

Traumatic brain injury is a common cause of death and disability, and has serious consequences for patients living in the developing countries due to lower number of health facilities with neurosurgical trauma units, difficult transportation and high costs of treatment for the majority of poverty stricken population. Emergency treatment of epidural haematoma is reportedly one of the most cost effective surgical interventions with excellent outcome if intervened on time.^{1,4} Progressive epidural haematoma is one form of the clinico-radiologic presentation of this entity which appears to affect 10-20% of patients with epidural haematoma.^{2,3,4}

Progressive epidural haematoma is the appearance of a new haematoma or increase in the size of

pre-existent epidural haematoma as diagnosed on initial computed tomographic scan of the brain.^{3,4,5,6}

The widespread availability of CT scan and its rapidity has helped neurosurgeons and trauma specialists to develop guidelines which recommend performing sequential scans of head trauma patients according to the currently available clinical criteria.

Repeated CT scanning of head is indicated in all patients with severe brain trauma (GCS ≤ 8) and in those who show signs of neurologic deterioration or signs of increasing intracranial pressure (vomiting, seizures, decreasing GCS). Studies have shown that repeat CT scanning of the brain will lead to intervention in over one third of patients if performed at right time and according to right indications.^{7,8}

Mechanisms leading to and factors associated with the evolution of progressive epidural haematoma have been extensively investigated and their significance tested in various clinical studies.^{3,4} However, focused studies investigating the clinical, laboratory and radiological features which might be predictive of the occurrence of PEDH are scarce. This study looks into the factors which might predict the incidence of

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PEDH during the early admission period along with their impact on early postoperative outcome.

METHODS

This is a retrospective chart review of patients who were labelled with progressive epidural haematoma during the 12-months period (January to December 2015). Approval was obtained from the institute's ethical committee.

Patients with closed head injury with no initial indication for immediate evacuation of epidural haematoma were included. Patients with a traumatic lesion who had no indications for surgical intervention on initial CT scan underwent repeat CT scanning, twice within 24 hours of presentation and once thereafter when patient was fully examined neurologically, up to 7th day of admission as was guided by the neurologic status.

Similarly, those patients who had an epidural haematoma of less than 25 cc were also listed to undergo repeat CT scan at a specified time interval or if there were an observable neurologic deterioration or signs of increasing intracranial pressure. Those patients who were diagnosed as having an increasing epidural haematoma size on repeat CT scan or those who showed evolution of new haematoma and subsequent surgical intervention were included in the study.

Patients with penetrating brain injuries, recurrent epidural haematoma, clear indications for decompressive craniotomy/craniectomy in case of a traumatic mass lesion and those who died before undergoing a repeat CT scan were excluded.

Volume of haematoma was calculated by using the formula $A \times B \times C \times 0.5$, where A is the length of the haematoma, B is the thickness and C is the height or number of CT scan cuts in which the haematoma appears.⁹ Surgical indications for a decompressive craniectomy include a haematoma of ≥ 25 cc in supratentorial regions and ≥ 10 cc in the infratentorial compartment or a midline shift of more than 5 mm or progressive neurologic deterioration.¹⁰

The presence of vomiting, headache and seizures, GCS at arrival, pupillary status, volume of haematoma on initial CT scan (if any), volume on subsequent CT, interval between the two scans and approximate interval since the time of injury were recorded. Coagulation profiles (PT/INR, APTT), platelets count, the presence or absence of skull fracture on operation were also noted. For outcome analysis, total hospital stay, complications, GCS at discharge and any neurologic deficits were recorded. Patients were then followed in the outpatient department and Glasgow outcome score (GOS) recorded at three months.¹¹

Good neurologic outcome was defined as a GOS of 4 and 5, while a GOS of 1, 2 or 3 was labelled as bad outcome.¹¹

Progressive epidural haematoma was defined as the evolution of a pre-existent haematoma to a size which crosses the cut-off limit of 25 cc, those with radiologic evidence of increasing haematoma size and neurologic deterioration and those who show a midline shift of more than 5 mm.

Data was analysed using the SPSS software version 20.0. Continuous variables are presented as mean \pm standard deviation. Independent samples t-test was run for determining mean differences across the two outcome groups. Binary logistic regression model was used to identify the factors which may predict the development of PEDH.

RESULTS

56 patients were operated for PEDH, out of 390 extradural haematoma patients (6.96% incidence). Overall mean age was 33.79 ± 11.41 years, mean arrival GCS was 10.86 ± 2.45 (median: 11), mean systolic BP was 114.28 ± 18.41 mm of Hg, mean time since injury was 7.59 ± 3.93 hours, mean platelets count was $217 \times 10^3 \pm 119 \times 10^3$. The mean INR was 1.25 ± 6.3 .

Overall mean volume at the first CT was 11.78 ± 6.3 mm³ (range: 0 – 23 cc). Similarly, mean volume at the follow-up CT was 43.14 ± 10.16 mm³ (range: 26 – 60 cc) with a mean time between the two scans of 15.28 ± 9.27 hours (range: 4 – 48 hours). The mean GCS at discharge was 12.3 ± 3.5 (median: 13.5). The median GOS at the end of follow-up period was 4.0 (mean: 4.05 ± 1.1).

The most common mode of injury was road traffic accidents in 60.7% (n=34) patients, which was followed in frequency by falls in 21.4% (n=12), assault in 10.7% (n=6) and fall of heavy object on the head in 7.1% (n=4) patients.

Headache was the presenting complaint in 32.1% (n=18) patients, vomiting in 82.1% (n=46). Seizures at the scene of trauma were reported in 39.3% (n=22) patients. Pupillary abnormalities were encountered in 13 (30.2%) patients while midline shift was noted in 27 (48.2%) patients.

Favourable outcome was recorded in 43 (76.8%) of patients, while 13 (23.2%) patients remained in the unfavourable outcome group at 3-month follow-up. Overall, there was 8.9% (n=5) mortality.

Chi-squared test showed a strong association of pupillary abnormalities, comorbidities, and midline shift with the outcome in terms of Glasgow outcome score. Table 1

The independent samples t-test was performed to determine the significance of mean difference of various continuous variables with regard to the two outcome groups according to GOS. We observed that there was a significant mean difference for age, GCS at arrival, systolic BP and the volume at follow-up CT in the two outcome groups. Table 2

Table 1: Clinical features

Clinical variable	Favourable Outcome n = 43		Unfavourable Outcome n = 13		P-value
	n	%	n	%	
Gender					
Male	28	65.1	9	69.2	0.78
Female	15	34.9	4	30.8	
Headache	15	34.9	3	23.1	
Vomiting	34	79.1	12	92.3	
Seizures	15	34.9	7	53.8	
Pupillary abnormalities	13	30.2	9	69.2	0.01
Skull fracture	30	69.8	8	61.5	0.57
Comorbids	-	-	6	46.2	<0.001
Midline Shift	17	39.5	10	76.9	0.01

Table 2: Group wise descriptive statistics

	Outcome group	n	Mean	Std. Deviation	P-value
Age of the Patient	favourable	43	32.00	10.191	0.03
	unfavourable	13	39.69	13.573	
GCS at Arrival	favourable	43	11.72	2.074	<0.001
	unfavourable	13	8.00	1.000	
systolic BP	favourable	43	122.9767	10.34981	<0.001
	unfavourable	13	85.5385	3.52646	
Time Since Injury	favourable	43	7.70	4.400	0.71
	unfavourable	13	7.23	1.641	
Platelets Count	favourable	43	227162.79	67151.191	0.25
	unfavourable	13	183461.54	220448.261	
INR	favourable	43	1.2374	.13988	0.42
	unfavourable	13	1.2769	.20064	
Volume at First CT Scan	favourable	43	11.7907	6.31912	0.9
	unfavourable	13	11.7692	6.50838	
Time between Scans	favourable	43	14.7907	9.37717	0.47
	unfavourable	13	16.9231	9.11465	
Volume at Second CT Scan	favourable	43	39.5116	8.49193	<0.001
	unfavourable	13	55.1538	4.14017	

DISCUSSION

Follow-up CT is an important monitoring tool for head trauma patients, because it is a well-known fact that head injury and intracranial lesion development is not a static phenomenon but a dynamic process. Indications for repeat CT are usually neurologic deterioration, new onset deficit, progressive and unrelenting symptoms, and clinical features like the presence or appearance of pupillary.^{12,13,14,15,16}

In this study, we observed PEDH in 6.96% patients

which is similar to that reported by Chen et al and other authors.^{3,5,6} We have developed a policy to perform a repeat CT at an interval of 6 to 12 hours after the initial CT scan, usually dictated by the neurologic status or the first CT scan findings. All of our patients had a repeat CT scan within the first 12 hours. Most of the cases showed an increase in the volume of a haematoma over the first 6 to 8 hours, while beyond 12 to 24 hours, there is very rare increase in the size of the haematoma. Similar findings have been noted in a study by Chen et al where only one patient had a PEDH after day three

of admission.³

The majority of the patients belonged to the age range of the third and fourth decade. This finding is similar to the findings of other similar studies by Servadei and Lobato et al.^{1,4} The statistical analysis has shown a significant correlation between the age of a patient and outcome of the operative intervention for PEDH.

The GCS at arrival and during the clinical course is a well-known prognostic and diagnostic instrument which can guide the decisions about a patient overall condition while he is under observation for progressive traumatic brain injury. Our findings included a strong association between the arrival GCS and the functional outcome at 3-months follow-up ($p < 0.001$)

The time since injury to the arrival in emergency room and the volume of the haematoma noted on the first CT scan were not significantly different for the outcome groups. This observation is contrary to the observation of Chen et al, who observed a positive correlation between the time to arrival (or first CT) of less than 2 hours and the likelihood of progressive haemorrhage on subsequent CT scan.³ The reason might be lower overall severity of trauma in our patients or it might be the association of other comorbidities. As mentioned above, in this study we did not find any difference of platelet count and INR for the two outcome groups. This is also contrary to the findings of studies by Talying et al and Lobato et al.^{3,17,18,19} Coagulation abnormalities are obviously related with higher chances of progressive haemorrhage, however, among the younger age groups, such as in our study, may not be having comorbidities for which the use of anticoagulants or antiplatelet might be in use. Another reason might be the presentation GCS. Severity of head trauma is directly correlated with the incidence of post-TBI coagulopathy.¹⁹ These findings were concurrent with the study by Chen et al and Lobato et al where coagulation profile derangements, presence or absence of skull fracture and time between scans (not initial CT scan) were not a determinant of progressive haemorrhage neither had an effect upon the final outcome in terms of GCS at discharge or GOS at 3 months.^{3,4}

In our study, however, a lower GCS at arrival influenced the 3-month outcome in terms of GOS ($p < 0.001$) than those who presented at higher GCS. Studies by Lobato et al and Chan et al have demonstrated that patients in the severe TBI group require repeat CT sooner and at higher rate than patients in the moderate and mild TBI groups.^{3,4,5}

Ding et al has demonstrated the effectiveness of repeat CT scans and clinical vigilance in detecting progressive or evolving pathology in head trauma patients, especially in the moderate and severe TBI groups.²⁰ Talbott et al has recommended close monitoring and repeat CT scanning of those who presents with skull fractures, because this subset of patients frequently develops progressive epidural haemorrhage.¹⁶

CONCLUSION

Progressive epidural haematoma is one component of the spectrum of progressive traumatic brain injury. Outcome in progressive extradural haematoma is affected by age, arrival GCS, systolic BP and the volume at the time of surgery. The time since injury, coagulation profile, platelets count and the volume at first CT is not associated with the outcome. Moreover, CT scan findings, such as midline shift may determine the final outcome.

REFERENCES

1. Servadei F, Vergoni G, Staffa G, Zappi D, Nasi MT, Donati R, Arista A. Extradural haematomas: how many deaths can be avoided? Protocol for early detection of haematoma in minor head injuries. *Acta Neurochir (Wien)*. 1995;133(1-2):50-5.
2. Oertel M, Kelly DF, McArthur D, Boscardin WJ, Glenn TC, Lee JH, Gravori T, Obukhov D, McBride DQ, Martin NA. Progressive hemorrhage after head trauma: predictors and consequences of the evolving injury. *J Neurosurg*. 2002 Jan;96(1):109-16.
3. Chen H, Guo Y, Chen SW, Wang G, Cao HL, Chen J, Gu Y, Tian HL. Progressive epidural hematoma in patients with head trauma: incidence, outcome, and risk factors. *Emerg Med Int*. 2012;2012:134905.
4. Lobato RD, Rivas JJ, Cordobes F, et al. Acute epidural hematoma: an analysis of factors influencing the outcome of patients undergoing surgery in coma. *J Neurosurg*. 1988 Jan;68(1):48-57.
5. Milo R, Razon N, Schiffer J. Delayed epidural hematoma. A review. *Acta Neurochir (Wien)*. 1987;84(1-2):13-23.
6. Riesgo P, Piquer J, Botella C, Orozco M, Navarro J, Cabanes J. Delayed extradural hematoma after mild head injury: report of three cases. *Surg Neurol*. 1997 Sep;48(3):226-31.
7. Lobato RD, Gomez PA, Alday R, Rivas JJ, Dominguez J, Cabrera A, Turanzas FS, Benitez A, Rivero B. Sequential computerized tomography changes and related final outcome in severe head injury patients. *Acta Neurochir (Wien)*. 1997;139(5):385-91.
8. Chan KH, Mann KS, Yue CP, Fan YW, Cheung M. The significance of skull fracture in acute traumatic intracranial hematomas in adolescents: a prospective study. *J Neurosurg*. 1990 Feb;72(2):189-94.
9. R. U. Kothari, T. Brott, J. P. Broderick et al., "The ABCs of measuring intracerebral hemorrhage volumes," *Stroke*, vol. 27, no. 8, pp. 1304-1305, 1996.
10. Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury. *Journal of Neurotrauma*. 2007;24 (supplement 1):S1-S95.
11. B. Jennett and M. Bond, "Assessment of outcome after severe brain damage. A practical scale," *The Lancet*, vol. I, no. 7905, pp. 480-484, 1975.
12. Lobato RD, Alen JF, Perez-Nuñez A, et al. Value of

- serial CT scanning and intracranial pressure monitoring for detecting new intracranial mass effect in severe head injury patients showing lesions type I-II in the initial CT scan. *Neurocirugia (Astur)*. 2005 Jun;16(3):217-34.
13. Stein SC, Spettell C, Young G, Ross SE. Delayed and progressive brain injury in closed-head trauma: radiological demonstration. *Neurosurgery*. 1993 Jan;32(1):25-30.
 14. Stein SC, Spettell CM. Delayed and progressive brain injury in children and adolescents with head trauma. *Pediatr Neurosurg*. 1995;23(6):299-304.
 15. Ding J, Yuan F, Guo Y, et al. A prospective clinical study of routine repeat computed tomography (CT) after traumatic brain injury (TBI). *Brain Inj*. 2012;26(10):1211-6.
 16. Talbott JF, Gean A, Yuh EL, Stiver SI. Calvarial Fracture Patterns on CT Imaging Predict Risk of a Delayed Epidural Hematoma following Decompressive Craniectomy for Traumatic Brain Injury. *AJNR Am J Neuroradiol*. 2014 Jun 19.
 17. Greuters S, van den Berg A, Franschman G, Viersen VA, Beishuizen A, Peerdeman SM, Boer C. Acute and delayed mild coagulopathy are related to outcome in patients with isolated traumatic brain injury. *Crit Care*. 2011;15(1):R2.
 18. Franschman G, Greuters S, Jansen WH, Posthuma LM, Peerdeman SM, Wattjes MP, Loer SA, Boer C. Haemostatic and cranial computed tomography characteristics in patients with acute and delayed coagulopathy after isolated traumatic brain injury. *Brain Inj*. 2012;26(12):1464-71.
 19. Talving P, Benfield R, Hadjizacharia P, Inaba K, Chan LS, Demetriades D. Coagulopathy in severe traumatic brain injury: a prospective study. *J Trauma*. 2009 Jan;66(1):55-61.
 20. Ding J, Yuan F, Guo Y, Chen SW, Gao WW, Wang G, Cao HL, Ju SM, Chen H, Zhang PQ, Tian HL. A prospective clinical study of routine repeat computed tomography (CT) after traumatic brain injury (TBI). *Brain Inj*. 2012;26(10):1211-6.

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