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ORIGINAL ARTICLE

Traumatic extradural hematoma - role of non-surgical management and reasons for conversion

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Abstract

Background To study the role of conservative management and various reasons for conversion to surgical intervention in traumatic extradural hematoma (EDH).

Setting Government Medical College Jammu.

Materials and methods One hundred-twenty trauma patients diagnosed as extradural hematoma on CT scan were managed during 1 year period.

Outcomes Role of conservative management and various reasons for conversion to surgical intervention were studied.

Results Out of these 120 patients admitted 67 were managed conservatively, 53 cases were operated upon. 31 were operated upon immediately and 22 were operated upon as delayed. There was 1 death and 4 had poor outcome in this group of patients.

Conclusion It is stressed that small size <10 ml, GCS >12 and locations other than temporal area are the criteria for conservative management. Twenty-two patients out of 89 were needed to be treated surgically during the course of conservative management due to neurodeterioration, increase in size of hematoma on CT, bradycardia, hemiparesis, pupillary abnormalities, delay in referral and only 18% had poor outcome. A strict vigilance is to be kept for clinical deterioration and various reasons mentioned above are to be kept in mind and patients should be subjected to repeated CT scan. Early diagnosis and immediate surgical intervention had good outcome, which shows the fruitful results of early diagnosis and intervention.

Keywords Extradural hematoma · Head injury · Surgical management · Conservative management.

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Introduction

Extradural hematoma (EDH), considered to be the most serious preventable complication of head injury, requiring immediate diagnosis and surgical intervention, encountered in 2% of patients with head injuries and 5–15% of patients with fatal head injuries, is a traumatic accumulation of blood in the potential space between the inner table of the skull and the stripped off dural membrane [1], prognosis is considered excellent if treated aggressively. EDH usually is stable, attaining maximum size with in minutes of injury, a hypothesis some authors disagree [2]. However it may progress during first 24 hours after injury. Rebleeding or continuous oozing presumably causes this progression. Occasionally EDH runs a chronic course and is detected only days after injury. A patient with small EDH may be treated conservatively though close observation is advised, as delayed, yet sudden neurological deterioration

may occur. Though surgical evacuation constitutes the definitive treatment of this condition but many patients can be saved from craniotomy with watchful repeated neurological assessments. Aim of our study was to study the role of conservative management and various reasons for conversion to operative intervention during the treatment of traumatic EDH.

Patients and methods

A prospective study of traumatic EDH treated conservatively and surgically was done over a period of 1 year in Government Medical College, Jammu from 01.08.2003 to 31.07.2004. Our study included only patients diagnosed as EDH after CT scan. Cases with associated cerebral lesions were also included, but only if the relevant lesion was the extradural effusion. The first clinical assessment was made in our emergency department on admission.

Initial resuscitation efforts included assessment and stabilization of airway patency, breathing and circulation. A thorough trauma evaluation was done. Detailed history especially about mode of injury, lucid interval and the physical examination included a thorough evaluation for evidence of traumatic sequelae and associated neurological deficits, skull fractures, hematoma, lacerations, bradycardia, hypertension, CSF ottorrhoea or rhinorrhoea, hemotympanum, GCS score, anisocoria, weakness, aphasia, visual field defects, numbness, ataxia. Repeated neurological examinations were done to assess the development of raised intracranial pressure. Apart from routine laboratory investigations relevant X-ray imaging studies were done. This study was done by dividing patients with EDH into groups according to different clinical parameters and treatment modalities and neuroradiological findings at the time of admission and hospital stay. Group-A: who required immediate surgical evacuation of the hematoma. Group-B: was further divided into two: B1, patients who were treated conservatively. B2, patients who were well with initial conservative treatment despite visible EDH on the first CT scan and who required operation in the course of conservative management and delayed appearance of EDH on CT scan.

Neurological evaluation was immediately undertaken following cardiopulmonary resuscitation, skull and chest X-ray, if time permitted. CT scan head was done in all patients. The dehydrating agent Mannitol (Neurotrol) was given in certain cases that had associated brain injury, edema, threatening to coma. Dexamethasone (Dexona) was administered for first few days and then gradually tapered off. Conservative management was immediately terminated and a craniotomy performed if the patient showed signs of local brain compression or herniation.

Outcome was measured according to Glasgow outcome score by Jannett and Bonds criteria which include death, vegetative state, severe disability, moderate disability and good recovery. A moderate disability who recovered independence (GOS-3, 2 and 1) were considered to have a good outcome, whilst patients who were severely disabled, vegetative or died were included together in the poor outcome group. The distribution of good and poor outcome with respect to each of the prognostic factors was examined statistically using a Chi-square test [3].

Results

Out of these 120 patients admitted, Table 1 shows various reasons for delayed operative intervention in 22 patients with neurodeterioration being the commonest reason involving nine patients. Four located at temporoparietal, 2 each in temporal and parietal, 1 in occipital region, followed by delayed development on CT, 1 each in frontal, parietal and temporoparietal region. Pupillary changes and hemiparesis were seen in 3, located in temporoparietal region. Increasing bradycardia was reason in 2 located at temporal and occipital region and persistent vomiting in 2 located at frontal and posterior fosse. Delay in referral was seen in 2, 1 each in frontal and temporoparietal region and increase in size of hematoma was seen in 1 located at frontal region as shown in Figure 1. There was 1 death and 4 had poor outcome in this group of patients.

67 patients were managed conservatively as shown in Table 2. All patients had GCS >12 except 6. Three patients died, all had associated intracranial lesions. There were 38 clots <10 ml, 32 of them were located in the temporoparietal region and there were 29 clots >10 ml, 4 were located at temporoparietal region and 11 were located at frontotemporal or frontal region as shown in Figure 2. Fifty-three cases were operated upon, 52 by craniotomy and 1 by craniectomy. Thirty-one were operated upon immediately and 22 were operated upon as delayed. Out of those operated immediately, 7 frontal, 3 temporal, 8 parietal, 2 occipital and 4 temporoparietal clots were >25cc and the GCS was <12 in 20, abnormal pupillary signs in 5, hemiparesis in 1 and 3 were located in the temporal region. Two patients died, one having temporal and the other temporoparietal clot. Seven patients had clot <25 ml, 2 each in frontal and temporal, 1 each in parietal, occipital and temporoparietal region, all had good outcome, as shown in Table 3.

Discussion

The history of operative intervention of the ailment related to skull dates back to Master Surgeon Sushrutha which are mentioned in “Sushrutha Samhita” (800 B. C.). Hippocrates recommended that the cranium be perforated when injuries might be followed by serious consequences such as the extravasation of blood. By trephining patients for EDH and evacuating the blood resulted in the recovery of the patients

Table 1 Details of cases converted from conservative treatment to surgical evacuation

Site	Case no.	Size ml.	Case no.	Reason for conversion	Outcome		Deaths
					Good	Poor	
Frontal	4	>25	2	Vomiting (1), Increase in size (1),	1	1	0
		<25	2	Delay in development (1)	2	0	0
				Delayed referral (1)			
Parietal	3	>25	2	Neurodeterioration (2)	2	0	0
		<25	1	Delayed development (1)	1	0	0
Temporal	3	>25	1	Increasing bradycardia (1)	1	0	0
		<25	2	Neurodeterioration (2)	2	0	0
Temporo -parietal	9	>25	9	Neurodeterioration (4)	6	3	1
				Hemiparesis (1), Pupllary changes (2), Delay in development (1), Delayed referral (1)			
		<25	0		0	0	0
Occipital	2	>25	2	Increasing bradycardia (1)	2	0	0
				Neurodeterioration (1)			
		<25	0		0	0	0
Posterior fossa							
	1	>25	0		0	0	0
		<25	1	Vomiting (1)	1	0	0
Total	22		22		18	4	1

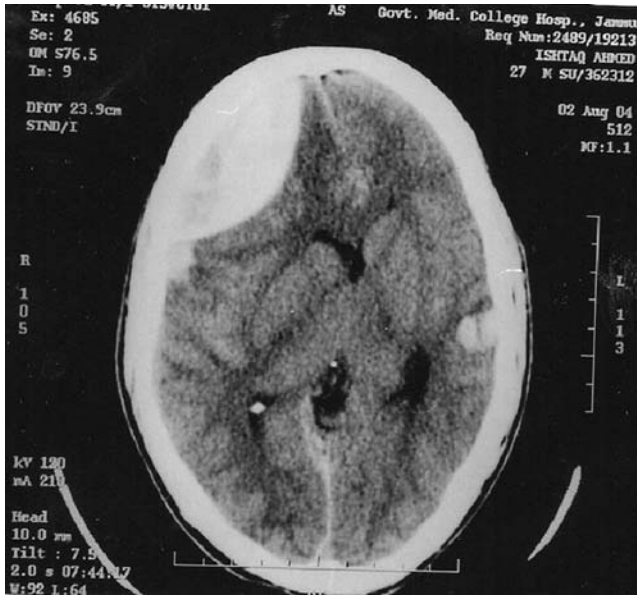

Fig. 1 Showing right frontal EDH as increase in size on repeat CT scan which required surgical intervention

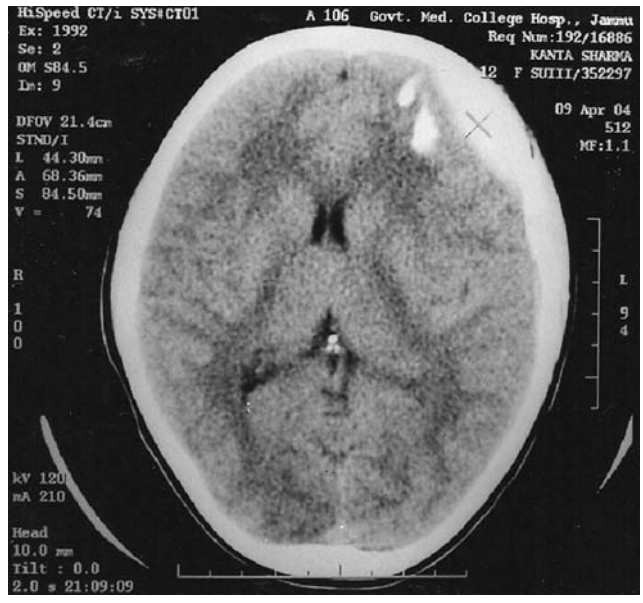

Fig. 2 Showing left frontal EDH with associated frontal contusions managed conservatively

during 18th century [4]. Celsus advised waiting until untoward symptoms appeared before operating. Operated mortality was high during early years of 20th century [5]. There was unrecognized high lethality in comatose patients due to traumatic acute EDH with no difference in outcome with regard to sex, mode of injury, or the presence of or the absence of contusion or shift in the CT scan. The motor score immediately before the operation was the most powerful preoperative predictor of outcome. So it was recommended to immediate evacuation of EDH i.e. when they are first

Table 2 Detail of cases treated by conservative measures

Site	Case no.	Size	Case no.	Outcome		Deaths
				Good	Poor	
Frontal and fronto-temporal	23	<10 ml	12	11	1	0
		>10 ml	11	11	0	0
Parietal	23	<10 ml	12	12	0	0
		>10 ml	11	11	0	0
Temporoparietal and temporal	11	<10 ml	7	5	2	0
		>10 ml	4	2	2	2
Occipital	8	<10 ml	5	5	0	0
		>10 ml	3	2	1	1
Posterior fossa	2	<10 ml	2	2	0	0
		>10 ml	0	0	0	0
Total	67		67	61	6	3

Table 3 Detail of case treated by immediate surgery

Site	Case No.	Size	Case No.	Outcome		Deaths
				Good	Poor	
Parietal	9	<25 ml	1	1	0	0
		>25 ml	8	8	0	0
Temporal	5	<25 ml	2	2	0	0
		>25 ml	3	2	1	1
Frontal and fronto temporal	9	<25 ml	2	2	0	0
		>25 ml	7	7	0	0
Occipital	3	<25 ml	1	1	0	0
		>25 ml	2	2	0	0
Temporal parietal	5	<25 ml	1	1	0	0
		>25 ml	4	3	1	1
Total	31		31	29	2	2

noted on the CT scan rather than waiting for clinical motor deterioration [6].

Until 1980, only a few delayed EDH were recorded, when angiography was used to make the diagnosis. The interval between injury and development of clinical signs is an important factor in determining the better prognosis than the acute. Most sub acute hematoma arises from venous, often combined, low tension sources of bleeding. The importance of recognizing gradual neurological deterioration and performing craniotomy in these patients is rewarding [7].

CT diagnosis of hematoma before the development of signs of depressed cerebral functions has reduced morbidity and mortality but disability is the greatest risk for a patient whose sub acute hematoma is evacuated late [8]. In the case of chronic EDH, there is membranes development and liquification of the clot which may permit drainage of such collections through twist drills and burr holes. The time from development and the neuroimaging changes on CT and MRI can suggest the age and nature of the clot and thus permit timing of surgery so that drainage may be accomplished with a minor procedure [9]. In the present study 22 patients (25%) out of 89 deteriorated in course of conservative treatment (group B2) and required neurosurgical interventions. Various reasons responsible for delayed interventions are tabulated in Table 1. Of these 22 patients only 4(18%) had poor outcome and 1 (4.5%) died. There were 67 cases which were treated conservatively only with poor outcome in 6 (9%) and mortality rate of 4.47% (3 cases) only.

Very good results are obtained due to CT scan because surgery can be preformed quickly in patients in better neurological condition. Repeated CT scans are indicated in patients with worsening symptoms who may have an unusual delayed intracranial hematoma. In this study 31 patients where operated upon immediately after CT scan with poor outcome in only two patients. One patient died and the other (with multiple brain contusions)

was permanently disabled. There is no question that to withhold a CT scan of a patient who does not yet have a focal neurological deficit. Jamieson during 1969 stressed that; one must prevent appearance of classical clinical picture [10].

Conservative management

Sophisticated care of head injury patients in the emergency department does not demand sophisticated knowledge of neurosurgery. Instead it depends upon meticulous attention to the fundamental principles of resuscitation, prevention of secondary insult to brain which can further injure the traumatized brain.

For many years it has been known that some patients can tolerate the presence of an intracranial hematoma and will recover even if it is not surgically removed. Cases of EDH, sometimes of considerable size which were not surgically treated were regularly reported in the literature. Traumatic epidural hematomas which are minimally symptomatic do not require surgical intervention. Despite the well performed clinical studies and the experience of many neurosurgeons using prompt evacuation of EDH, there have been number of reports suggesting non-operative management of selective EDH [11–18]. The use of CT scan in head trauma revealed a new class of EDH patients who may be treated conservatively. Such patients should be monitored with frequent neurological examinations and regular serial CT scans to demonstrate resolution of the hematoma and associated shift. With such clinical and radiographic monitoring, a subgroup of patients with acute EDH is detected in whom a return of normal mental status will follow loss of consciousness and in whom spontaneous resolution of their hematoma will occur [19]. In the past these hematoma were occasionally discovered even some weeks after the injury during the course of neurosurgery for other reason namely dural plastic surgery of ACF. Some expressed doubts concerning the need to remove EDH in patients affected by subjective disorders only and without neurological deficits [20]. One of the several mechanisms to explain the resorption of the hematoma was the transfer of the clot into the epicranial space through the skull fracture [21].

In some studies the size of the hematoma, rather than its location, the degree of midline shift were the most influential in deciding in favor of surgical treatment in asymptomatic significant EDH [22, 23]. But temporal location of EDH with heterogeneous density in patients whose CT scan was performed <6 hours after trauma had a higher risk of hematoma growth and thus should be treated surgically [24]. In our study involving 67 patients managed conservatively, out of 38 clots <10 ml, only 3 have poor outcome, 2 were located at temporoparietal region and one at fronto temporal region and none of the patient died. Out of 29 patients with clot size >10 ml, three patients had poor outcome, 1 with occipital hematoma and 2 with temporoparietal clots and there were 3 deaths, 2 had temporoparietal clots and 1 with occipital clot. All patients with frontal and occipital hematoma having small size had good outcome. Eleven patients out of 23 patients with temporal and temporo-parietal location of the EDH as compared to only 11 out of the rest 66 patients on conservative management needed to be converted to surgical management which was statistically significant ($p < 0.002$). Sixty-one patients had GCS > 12 only 6 patients had GCS <12. It is stressed that small size <10 ml, GCS >12 and locations other than temporal area may be considered as the criteria for conservative management.

Conclusions

In 67 patients continued conservatively, out of 38 clots <10 ml, only 3 have poor outcome and none of the patient died. Out of 29 patients with clot size >10 ml, three patients had poor outcome and there were three deaths. All patients with frontal and occipital hematoma having small size had good outcome. Sixty-one patients had GCS >12 only six patients at GCS <12. Eleven patients out of 23 patients with temporal and temporoparietal location of the EDH as compared to only 11 out of the rest 66 patients on conservative management needed to be converted to surgical management which was statistically significant ($p < 0.002$). It is stressed that small size <10 ml, GCS >12 and locations other than temporal area are the criteria for conservative management. Twenty-two patients out of 89 were needed to be treated surgically during the course of conservative management due to neurodeterioration, increase in size of hematoma on CT, bradycardia, hemiparesis, pupillary abnormalities, delay in referral and only 18% had poor outcome. A strict vigilance is to be kept for various reasons mentioned above and patients should be subjected to repeated CT scan. 29 patients out of 31 who were early diagnosed and immediately operated upon had good outcome, which shows the fruitful results of early diagnosis and intervention. Outcome was measured according to Glasgow outcome score by Jannett and Bonds criteria, six patients died 3 each in operative and conservative group. One hundred eight patients out of 120 had good outcome. It is stressed that a group of patients can be treated conservatively without undergoing surgical intervention provided a strict vigilance is required to assess clinically supplemented by repeat CT scan.

References

1. Bell C (1816) Surgical observations. Longman, Hirst Rees Edv Arrie and Brown: 466
2. Frankhauser H, Kiener M (1982) Delayed development of extradural hematoma. Acta Neurochir (Wien) 60:29–35
3. Jennett B, Bond M (1975) Assessment of outcome after

severe brain damage, a practical scale. Lancet 1: 480–484
4. Hill J (1772) Case in surgery particularly of cancers and disorders of the head from external violence and observation. J Balfour, Edinburgh: 263
5. Munro D, Maltby GL (1941) Extradural hemorrhage. Ann Surg 113(2):192–203
6. Seeling JM, Marshal LF, Tautan SM, et al. (1984) Traumatic acute EDH, unrecognized lethality in comatosed patients. Neurosurg 15:617–620
7. Pozzati E, Frank F, Frank G, et al. (1980) Subacute and chronic EDH, A study of 30 cases. J Trauma 20(9):795–799.
8. Zuccarello M, Fiore DL, Pardatscher K, et al. (1983) Chronic EDH. Acta Neurochirurgica 67:57–66
9. Kaufman HH, Herschberger J, Kopitnik T, et al. (1992) Chronic EDH, Indications for surgery. Br J Neurosurg 6:359–364
10. Jamieson KG, Yelland J DN (1968) Extradural and subdural hematoma. Report of 167 cases. J Neurosurg 299:13–23
11. Aoki N (1981) Rapid resolution of acute epidural hematoma. Report of 2 cases. J Neurosurg 54:258–261
12. Tochio H, Waga S, Tashiro H, et al. (1984) Spontaneous resolution of chronic epidural hematomas. Report of 3 cases. Neurosurg 15:96–100
13. Illingworth R, Shawdon H (1983) Conservative management of intracranial extradural hematoma presenting late. J Neurol Neurosurg Psychiatry 46:558–560
14. Pang D, Horton JA, Henon JM, et al. (1989) Non-surgical management of extradural hematomas in children. J Neurosurg 59:958–971
15. Tuncer R, Kazan S, Ucar T, et al. (1993) Conservative management of epidural hematomas. Prospective study of 15 cases. Acta Neurochir (Wien) 121:48–52
16. Bullock R, Smith RM, Van Dellen JR (1985) Non-operative management of extradural hematoma. Neurosurg 16: 602–606
17. Ceylan S, Kuzeyli K, Ilbay K, et al. (1992) Non-operative management of acute extradural hematoma in children. J Neurosurg Sci 36:85–88
18. Chen TY, Wong CW, Chang CN, et al. (1993) The expectant treatment of asymptomatic supratentorial EDH. Neurosurg 32(2):176–179
19. Weaver D, Pofereskin L, Jane JA (1981) Spontaneous resolution of EDH. Report of two cases. J Neurosurg 54: 248–251
20. Trowbridge WV, Poster RW, French JD. (1954) Chronic EDH. Arch Surg 69:324–330
21. Tuncer R, Acikbas C, Ucar T, Kazan S, Karasoy M, Saveren M (1997) Conservative management of extradural haematoma. Effects of skull fractures on resorption rate. Acta Neurochirurgica 139(3):203–207
22. Cook RJ, Dorsch NW, Fearnside MR, et al. (1988) Outcome prediction in EDH. Actaneurochir (Wein) 95(3–4):90–94
23. Servadei F, Faccai G, Rocella P, et al. (1989) Asymptomatic EDH, results of a multicenter study of 158 cases in minor head injury. Acta Neurochir (Wein) 96:39–45
24. Bezircioglu H, Ersahin Y, Demircivi F, et al. (1996) Non-operative treatment of acute extradural hematomas: analysis of 80 cases. J Trauma 41:696–698