

Disappearance of Intracranial Extradural Hematomas: Role of Diastatic Cranial Fracture and Intracranial Pressure - An Institutional Experience

Abstract

Context: The intracranial extradural hematoma (EDH) occupies space and creates a mass effect on the brain but the tenacious-adhesions of dura to the inner table of skull counters this effect. The intracranial pressure also pushes the hematoma back while it is held by dural tensile-force. **Aims:** The exploitation of a diastatic fracture, overlying an EDH, by the intracranial pressures to decompress a hematoma out of extradural space into subgaleal/subperiosteal space without surgical intervention. **Settings and Design:** In a period of 15 years, a group of 11 patients among 729 EDHs were managed conservatively. **Materials and Methods:** The retrospective study of 11 EDH patients was conducted in the Department of Neurosurgery from January 2000 to December 2014 in 15 years. **Statistical Analysis Used:** The statistical law of variance was used as applicable. **Results:** Analysis of spontaneous disappearance of intracranial EDH among 11 patients revealed that only 1.5% (11/729) EDHs resolved conservatively. The most cases (63.6%) were children and the youngest being 9 months old. All the patients had a diastatic fracture overlying-EDH and were fully conscious. The cause of head injury in most was the fall from height. The hospital stay ranged from 2 to 4 days. All the patients had a good recovery at the time of discharging. **Conclusion:** The trial of the conservative or spontaneous disappearance of an EDH through a diastatic fracture into the subgaleal space is similar to burr-hole drainage without surgical intervention but depends upon the neurological status, the intracranial pressure of the patient, and the availability of all the modern neurosurgical gadgets.

Keywords: Intracranial extradural hematoma, intracranial pressures, skull fracture, spontaneous decompression

Introduction

The extradural hematoma (EDH) is a neurosurgical emergency and is usually fatal in children and young adults if not treated surgically in time. However, infants and elderly patients, due to the flexibility of cranial vault and less brain volume respectively, may not show symptoms and signs of deterioration and may be managed either surgically or medically. Furthermore, the fact is that 1.5% of all patients treated for head trauma have EDHs for which surgery is needed, but spontaneous resolution may also occur.^[1] The risk of managing intracranial EDHs conservatively may be reduced in the modern-era with the availability of modern diagnostic tools, electronic monitoring, keep-ready advanced surgical equipment, and a state-of-art neurosurgical operation-room and intensive critical care unit. However, the hazards of follow-up serial computed tomography (CT)

scans and unnecessarily prolonged hospital stay may be ethically questionable. However, a selective group of those patients who attain criteria of several factors such as a wide fracture-line over the EDH; a fully alert and conscious patient; nontemporal EDH; patient with no associated severe brain injury; a non-threatening volume and thickness of EDH or a midline shift may be managed medically. The analysis of 11 EDH patients in the present study reveals the clinical features and the main factors responsible for the disappearance of the intracranial EDH.

Materials and Methods

The Department of Neurosurgery, the single and high patient-inflow neurosurgical center in the state, analyzed 729 EDHs among head-injury patient-population retrospectively, in which 11 spontaneous disappearances of intracranial EDHs occurred, in 15 years from January 2000 to December 2014.

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The spontaneous resolution or disappearance of EDHs was monitored only when the cranial vault harbored a fracture line at the site of EDH, and the patient was fully conscious (even after an initial brief spell of unconsciousness at the time of injury). All patients were subjected to all the basic investigations such as complete blood chemistry, X-ray chest, cervical spine, pelvis, ultrasound abdomen, and the initial X-ray of the skull anteroposterior and lateral views, plain and three-dimensional CT scan. The patients were admitted to the intensive critical care unit with the serial CT scans ready as and when required and keeping the surgical option open and ready as may be needed in case of deteriorating patient. The exclusion criteria's were temporal EDH, bilateral EDH; underlying brain contusions, acute subdural hematomas and; a Glasgow coma scale (GCS) score of 12 and lower; volume of EDH 30 ml and more; thickness of EDH 15 mm and more; midline shift of more than 6 mm and wound in the scalp at the site of fracture. The data were compiled and resulted dawn. The statistical law of variance was used wherever applicable.

Results

A mere 1.5% (11/729) EDHs decompressed spontaneously in 15 years. All the patients were received within 30 min

to 3 h of the alleged history of head injury. About 72.7% patients had a history of a brief spell of unconsciousness before resuming full alertness in the hospital. Among a total of 11 cases of intracranial EDH which spontaneously drained into the extracranial space (subperiosteal and subgaleal spaces), there were 63.6% (7/11) children (youngest being 9 months male infant and eldest was 12 years male child), and 4 adults of which eldest was 52 years male and youngest was a 23 years male [Table 1]. The X-ray skull showed a diastatic fracture in more than 81% cases [Figure 1]. The cause of EDH was fall from height in about two-third of the patients. More than 70% of patients had a parietal bone fracture and underlying EDH. About 90% of patients had an admission GCS score of 13–15. The patients were monitored and subjected to serial CT scans, with a maximum of five scans each in three cases and four times each in five cases while a minimum of two scans each were performed in two patients [Figures 1 and 2]. The initial CT scan of 54.5% patients showed the volume of EDH as 20–26 ml, while 26 ml was the highest volume, the lowest was 14 ml. The thickness of hematoma at the widest point was 10 mm to 13 mm in more than 72% patients while the minimum thickness of 7 mm was seen in one patient [Table 1]. A significant

Table 1: Analysis of Glasgow coma scale scoring and computed tomography findings in 11 extradural hematoma-disappearance cases

Patient number	Cause of injury	Age/sex	Admission GCS score	CT scan findings					Outcome score
				EDH/fracture site	Volume (ml)	Thickness (mm)	MS (mm)	Time of disappearance	
1	Fall	9 months Male child	Alert/ active	Parietal	17	10	3	12	Alert/active
2	Fall	2 years Female child	13	Parietal	25	13	6	5	Good recovery
3	Fall	4 years Female child	15	Frontal	15	9	0	16	Good recovery
4	Fall	7 years Male child	14	Parietal	22	10	5	18	Good recovery
5	RTA	10 years Male child	15	Parietal	18	10	4	10	Good recovery
6	Fall	11 years Male child	13	Parietal	20	11	5	15	Good Recovery
7	Fall	12 years Male child	13	Frontal	17	9	3	18	Good recovery
8	RTA	23 years Male	14	Parietal	26	12	5	12	Good recovery
9	Fall	45 years Male	13	Frontal	25	10	5	17	Good recovery
10	RTA	50 years Female	15	Parietal	14	7	0	22	Good recovery
11	Assault	52 years Male	14	Parietal	20	11	4	20	Good recovery

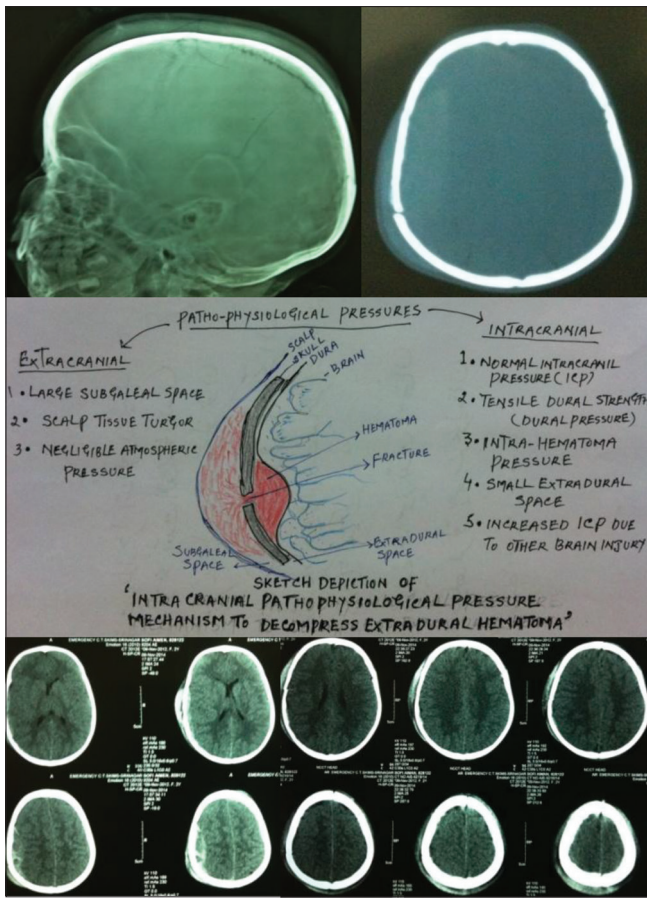


Figure 1: X-ray skull lateral view and noncontrast computed tomography head with bone window (before and after disappearance of extradural hematoma) show a vertical diastatic fracture in the right parietal bone, a cephalhematoma, and an extradural hematoma. A manual sketch diagram depicts the mechanism of disappearance of extradural hematoma

midline shift of 6 mm to opposite side was found in only one (9%) patient of 2 years old with a 25 ml volume EDH, decompressing in only 5 h. Although two patients (18%) had no midline shift at all but an insignificant midline shift of 3 mm was noticed in other 18% patients. All the patients had clinically an uneventful course till their good recovery which was confirmed radiologically by a CT scan head. The time of disappearance of the EDH was calculated from the time of alleged history of head injury to the time of a CT scan that showed the complete disappearance of the EDH. A period of 22 h was the maximum time an EDH of 14 ml took to decompress itself fully into the extracranial subgaleal or subperiosteal space through a fracture at the parietal eminence. The time of disappearance of EDHs in most of the patients (81.8%) was 10–20 h, though only 4 cases (36.3%) took 18 h and more [Table 1]. The cephalhematoma of 81.8% patients increased in size when EDH decompressed itself into it. The maximum hospital stay for 63.6% patients was 4 days; 3 days for 18.1% patients; and 2 days for another 18.1% patients.

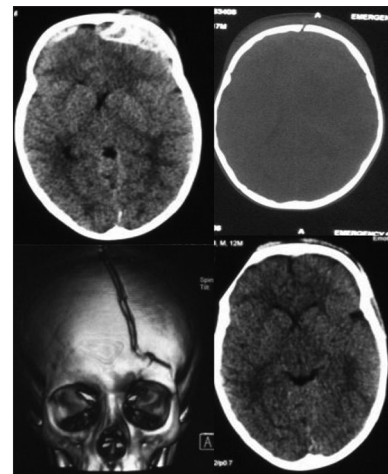


Figure 2: Plain and three-dimensional computed tomography scan head and bone window display a diastatic fracture in the left to midline frontal bone with evident cephalhematoma, extradural hematoma and its disappearance

Intracranial pressure mechanism

The existent physiological intracranial pressure and the pathological intracranial pressure, which builds up after the formation of a traumatic EDH, sum up to push the EDH against the inner table of the skull. However, in the presence of a diastatic fracture-skull at the site of hematoma, the fracture line acts as a conduit to allow transportation or shifting of hematoma into extracranial compartment [Figure 1]. The probable intracranial physiological and pathological pressures are (1) the normal intracranial pressure (physiological), (2) the normal dural tensile pressure (physiological); the dura is adherent in extremes of age to the inner table of calvarium which reduces the extradural space normally to zero volume, and the pressure of hematoma which strips dura off the bone to create a space is resisted by the dural tensile-strength (stretch-pressure), (3) The pressure within the hematoma itself (pathological), causing tamponade on the bleeding dural-vessel, i.e., intra-hematoma pressure, exerts equal pressure on all sides, i.e., the bone as well as dura, and (4) The associated brain-injury if any (pathological), in the form of brain edema, acute subdural hematoma, contusions, etc., increases the intracranial pressure further to resist intracranial accommodation of any objective-pressure like hematoma. In comparison to intracranial space, the pressures within the extracranial space are negligible. The potentially huge subperiosteal/subgaleal space, with a negligible pressure in lying down position, can accommodate a substantial amount of blood. The insignificant atmospheric pressure and the scalp turgor are the least resistant forces. This gradient in intra- and extra-cranial pressures in the presence of a “patent-conduit” like a diastatic fracture-line creates a one-way transportation of hematoma resulting in the decompression of intracranial EDH into extracranial large subgaleal space [Figure 1].

Discussion

Since a potential communication existed in the form of a mild diastatic linear fracture in all the cases, the EDH (under high pressure) in the completely occupied (with brain, CSF, and blood) cranial cavity oozed out extra-cranially in the large subgaleal space which subsequently enlarged in size. The diastatic fracture-line is a significant potential-communication or temporary-conduit, though not alone a factor responsible, for the decompression of EDH. The difference between the pressures of extra- and intra-cranial spaces has also played a significant role in forcing the hematomas out into subgaleal space [Figure 1]. A significant midline shift of 6 mm was found in only one (9%) patient of 2 years old with a 25 ml volume EDH, decompressing in only 5 h in comparison to a period of 22 h taken, as the maximum time in this series, by a 14 ml EDH to decompress extra-cranially in an elderly female. The observations point to the intracranial pressure variations which occur due to physiological, degenerative, and pathological causes in different age groups. Thereby, an intracranial hematoma should increase in size if more space is available and intracranial pressure is reduced in the case of an elderly patient with brain atrophy. Similarly, extracranial soft tissue spaces also exhibit interstitial pressures which are liable to changes from atmospheric pressures, thickness, and turgor of the soft tissue coverings, posttraumatic open wounds, pressure dressings, and bandages. These multi-factorial mechanisms of intra- and extra-cranial pathological and physiological pressures may explain the exploitation of diastatic fractures by the existing EDH to leak out of intracranial extradural space. Thus, rather than resolution of hematoma in a given period, the process of disappearance of hematoma from extradural space appears merely to be the transportation of a hematoma with an increase in size of subgaleal space. Otherwise, the resolution of blood should occur simultaneously in both the extradural as well as in subgaleal spaces without an increase in the size of any space in question. But on the contrary, in the present series, the volume of extracranial space (cephalhematoma) increased in the size in all cases. It is reported that EDH is a serious complication of head injury which requires rapid diagnosis and early surgical evacuation as a standard management.^[2,3] However, conservative follow-up has also been reported in the literature.^[2,4,5] Many reported cases of spontaneous rapid resolution encourage the opinion of conservative follow-up.^[6] Various theories have been proposed to explain the underlying mechanism of these cases.^[3,6-8] The conservative management may be encouraged and facilitated nowadays by the increase in the number of intensive care units, which enhances the close monitoring and follow-up of these patients, the availability of the neurosurgeons in health care centers and easy access to increasingly available CT machines, essential in the diagnosis and follow-up of such patients. The rapid

spontaneous resolution of epidural hematoma has been reported in children as well as in adults. The search in the available literature reveals that 8 cases of EDH in pediatric population have been reported to have spontaneously resolved.^[7] Similarly, five cases of “spontaneous EDH resolution” were seen and reported in adult population.^[8] The oldest patient reported in the literature was 65 years old.^[9] The present study, a series of 11 cases, had 9 months infant as the youngest patient while 52 years female as the oldest patient. Among various theories that have been proposed to explain the underlying mechanisms of rapid spontaneous resolution is the existence of potential communication with a fracture between intra- and extra-cranial spaces.^[7,10] The increased intracranial pressure creates a pressure gradient between EDH and extracranial soft tissue spaces so that hematoma is forced out of extradural space through fracture line. The reported literature points out the second probable mechanism in spontaneous resolution as the pressure-induced re-distribution of the EDH secondary to brain swelling along the inner table of the cranial vault.^[11] However, dissipation of the hematoma seems harder due to tenacious adhesions between the dura mater and skull.^[9,12,13] Another theory emphasizes that the extracranial blood may be pushed into the extradural space through a fracture line, due to an increased extracranial subgaleal interstitial pressure and the pressure gradient after injury. Hence, as the subgaleal interstitial pressure decreases, the blood from intracranial extradural space leaks back. However, the process takes about 18 h to complete.^[13] Because of this fallacy, the theory is insufficient to explain the underlying mechanism for all the cases especially for those where hematoma resolved in <18 h. Looking at the resolution times in pediatric cases, the longest time for the resolution was 72 h,^[6] and the shortest period was only 1 h.^[11] The present series revealed that the disappearance time of EDHs in 63.6% (7/11) cases was <18 h. Bullock *et al.* reported that epidural hematoma <30 cm in volume, <15 mm in thickness and leading to <5 mm midline shift in patients with a GCS score of more than 8 without any focal neurodeficit could be managed conservatively. These patients need serial computed tomographic scans and close monitoring and observation in a neurosurgical center.^[14] This study noticed that volume of EDH in any of the 11 cases was not more than 26 ml, neither any patient had an EDH of more than 13 mm thick. However, a midline shift of 5 mm and less was found in all cases but one patient who had a midline shift of 6 mm and was fully conscious. All the patients had an admission GCS score of 13–15 and were discharged to home with good recovery with a hospital stay of 2 (48 h) to 4 (96 h) days. Eventually, in such situations, there is a need for the individualization in the management of each case differently depending on the condition of the patient, imaging, and neurosurgical team. However, the presence of a fracture at the site of EDH may be the cause of EDH as well as the existing treatment for it. This study showed that none of the 11 cases had a temporal EDH. The reason

could be that either the EDHs in the temporal area did not decompress spontaneously despite an existing fracture, possibly due to the strong extracranial temporalis muscular resistance and/or temporal EDHs were not given a chance of spontaneous-decompression or conservative trial owing to the high and frequent risk of coning and death.

Conclusion

The role of a diastatic skull fracture and the intracranial (physiological and pathological) pressures, to decompress an EDH into extracranial space, is evident. The chances of such disappearances of EDHs, though exist, but are as low as 1.5%. The risk of exploiting a diastatic fracture for conservative trial of an EDH may also be used in the near future by the neurosurgeons for minimally-invasiveness.

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Conflicts of interest

There are no conflicts of interest.

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