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Traumatic Extradural Hemorrhage Management: An article Review

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ABSTRACT

Extradural hemorrhage (EDH) is an accumulation of blood in the potential space between the inner table of the skull and the stripped off dural membrane, its prognosis is considered excellent if treated immediately. EDH occurs in about 2% of all cases of head injury but 5 to 15% of cases of fatal head injury. Around 30% of cases are acute, 30% subacute and 10% chronic. The clinical criteria to suspect EDH during the period of latency include symptoms as headache, restlessness, nausea, vomiting, dizziness, confusion, lack of responsiveness and sometimes seizures. In addition, signs as contralateral hemiparesis and ipsilateral occulomotor nerve paresis are alarming as it may be followed by decerebrate rigidity, arterial hypertension, cardio respiratory irregularity, apnea, and death. The primary diagnostic investigation for extradural hematoma is a CT without contrast in the majority of cases. Surgery is management of choice in patients with EDH as it represents 80% of management of EDH patients. The aim of the current study to review the possible modalities management for traumatic EDH.

Keywords: Traumatic Extradural Hemorrhage ; Signs ; Diagnosis ; Management

Introduction

Extradural hematoma (EDH) has been encountered in 2% of patients with head injuries and 5-15% of patients with fatal head injuries. It is an accumulation of blood in the potential space between the inner table of the skull and the stripped off dural membrane, Its prognosis is considered excellent if treated immediately **(1).**

Extradural hemorrhage is most often due to a fractured temporal or parietal bone damaging the middle meningeal artery or vein with blood collecting between the dura and the skull. It is typically caused by trauma to the temple just beside the eye, although it may also follow a tear in dural venous sinuses **(2).**

Small sized extradural hematoma can be considered special entity. It is usually stable. However, it may progress during first 24 hours after injury. Re-bleeding or continuous oozing presumably causes this progression. Such EDH runs a chronic course and is detected only days after injury. A patient with small EDH may be treated conservatively through close observation, yet sudden neurological deterioration may occur **(3).** Though surgical evacuation constitutes the definitive treatment of this condition but many patients can be saved from craniotomy with watchful repeated neurological assessments **(4).**

Children are less likely to have an associated skull fracture than adults. Around 30% of cases are acute, 30% subacute and 10% chronic **(5).**

An extradural haematoma usually occurs inside the head, most commonly after a fractured skull caused by a head injury. The fractured skull bone can cause separation of the dura mater from the inside of the skull and can cause damage to a blood vessel. The damaged blood vessel causes blood to leak and collect in the potential epidural space between the skull and the dura mater. This build-up of blood can cause the pressure inside

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the head to rise. This can put pressure on the brain and cause injury to the brain if it is not treated quickly **(6).**

Clinical Picture

The typical presentation is an initial loss of consciousness following trauma, a complete transient recovery ("often termed as a lucid interval"), culminating in a rapid progression of neurological deterioration. This occurs in 14% to 21% of patients with an EDH. However, these patients may be unconscious from the beginning or may regain consciousness after a brief coma or may have no loss of consciousness **(6)**. Therefore, the presentations range from a temporary loss of consciousness to a coma. Beware that the lucid interval is not pathognomonic for an EDH and may occur in patients who sustain other expanding mass lesions. The classic lucid interval occurs in pure EDHs that are very large and demonstrate a CT scan finding of active bleeding. The presentation of symptoms depends on how quickly the EDH is developing within the cranial vault. A patient with a small EDH may be asymptomatic, but this is rare. Also, an EDH may also develop in a delayed fashion **(7)**.

A posterior fossa EDH is a rare event. This kind of EDH may account for approximately 5% of all posttraumatic intracranial mass lesions. Patients with posterior fossa EDH may remain conscious until late in the evolution of the hematoma, when they may suddenly lose consciousness, become apneic, and die. These lesions often extend into the supratentorial compartment by stripping the dura over the transverse sinus, resulting in a significant amount of intracranial bleeding **(8)**.

This enlarging hematoma leads to eventual elevation of intracranial pressure which may be detected in a clinical setting by observing ipsilateral pupil dilation (secondary to uncal herniation and oculomotor nerve compression), the presence of elevated blood pressure, slowed heart rate, and irregular breathing. This triad is known as the "Cushing reflex." These findings may indicate the need for immediate intracranial intervention to prevent central nervous system (CNS) depression and death **(9)**.

Deterioration of consciousness is the most important sign. It is obvious in patients who have not been concussed, or in those who recover before compression develops, i.e. in the lucid interval. Lethargy and drowsiness lead to compelling sleep. At this stage, there is a real danger that the pathological lowering of level of consciousness may be mistaken for normal sleep. The patient is allowed to sleep for kindly reasons, but the error becomes apparent when he cannot be aroused **(10)**.

Drowsiness associated with confusion, restlessness renders a patient conscious for many hours, the onset of compression fails to attract attention and in consequence, operation may be delayed until too late. In these, regularly repeated examinations with standard stimuli are of great importance in establishing a further depression of response. The restlessness which occurs during recovery from concussion is associated with increasing responsiveness and purposeful reactions to stimuli, while, the restlessness which may accompany cerebral compression is characterized by decreasing reactions to stimuli **(11)**. The state of consciousness depends to a considerable degree on the location of the hematoma. A high portion of patients with frontal hematoma remain conscious, in contrast to patients with posterior fossa hematoma, who are usually unconscious at the time of surgery. The rate of development of clinical signs varies from less than an hour to days and, on rare occasions, even a few weeks. The slowest development occurs in those who are

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unconscious throughout and those with frontal hematomas. Posterior fossa EDH causes compression of the brain stem **(12)**.

Bonow et al. (13) have defined the following five stages of unconsciousness associated with extradural hematoma: (1) No initial loss of consciousness and continued consciousness until time of treatment; (2) No initial loss of consciousness, but subsequent unconsciousness at the time of treatment; (3) Initial unconsciousness, with recovery and relative alertness; (4) Subsequent loss of consciousness; and (4) Unrelieved unconsciousness from the time of impact.

The Glasgow Coma Scale (GCS) was modified to assess the severity of neurologic injury, provide a standard for measuring different intervention techniques, and assess the prognosis. Patients admitted with Glasgow Coma Scale of 4 or less had 45% mortality, as compared to 3% for those with scores of 9-12 on admission **(14)**.

Investigations

Although the neurological examination provides the most rapid and clinically relevant assessment of a head injured patient, several adjunctive diagnostic measures are available and should be utilized to facilitate emergency determination of the precise location and nature of the intracranial lesions, radiological investigations facilitate rapid management of intracranial hematomas. Of all the potentially lethal complications of head injury, extradural hematorna is the most readily diagnosed. In many cases of extradural hematoma, damage to other parts of the brain may complicate the picture. In these cases, diagnosis without the help of neuroradiologic procedures is difficult **(15)**.

Imaging studies such as a computed tomogram (CT) scan comprise the mainstay of diagnosis. Laboratory studies such as INR, partial thromboplastin time (PTT), thromboplastin time (PT), and liver function test (LFT) may be obtained to assess for increased bleeding risk or underlying coagulopathies **(16)**.

CT scan is the most common imaging modality to assess for intracranial bleeding. Its popularity is related to its widespread availability in emergency departments. The majority of EDHs are identifiable on a CT scan. The classic presentation is a biconvex or lens-shaped mass on brain CT scan, due to the limited ability of blood to expand within the fixed attachment of the dura to the cranial sutures. EDHs does not cross suture lines **(17)**.

Generally, radiologists use a standard formula for estimating the amount of blood present in an EDH. It is as follows: The maximum hemorrhage diameter on the CT slice with the largest area of hemorrhage, the maximum diameter 90 degrees to A on the same CT slice and the number of CT slices with hemorrhage multiplied by the slice thickness in centimeters **(18)**.

There are, however, other CT findings that may need to be taken into account when evaluating EDH. For example, continued bleeding may be indicated by areas of low density, or a "swirl-sign." The latter may be used for prognosis, and often indicates the need for surgical intervention. If the EDH abuts brain tissue that is hemorrhagic or contused, it may appear shallow, and thus, may be overlooked if the CT scan is not carefully examined **(17)**.

Several factors may lead to a non-diagnostic CT scan. These are as follows: a low-density blood collection may result from severe anemia (thus leading to misinterpretation), arterial extravasation may be reduced secondary to severe hypotension, a positive finding on CT requires that enough blood accumulates for visualization. If the CT is obtained too soon after trauma, there may not be sufficient accumulation for appropriate interpretation

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and if the EDH is secondary to venous bleeding, blood accumulation may be slow. This could potentially result in difficulty with CT interpretation **(18).**

The investigation of choice for extradural hematoma is a CT without contrast in the majority of cases this will show a hyper dense leniform (biconvex) extra-axial collection adjacent to calvarium **(Fig. 1)**. However, in about 5% of cases, the extradural hematoma may be crescent (concavo-convex) **(19).**

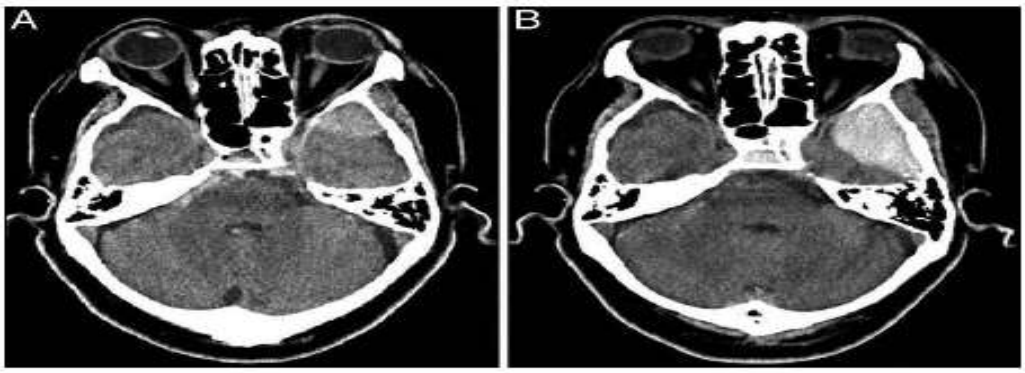

Figure (1): A case in which deterioration of a temporal tip EDH showed (A)Admission head CT scan obtained in a 51-year-old man revealing a temporal tip EDH measuring 20 mm in thickness, and opacified ethmoidal sinuses.(B) 6 hrs later, the hematoma was found to have increased in size, and it was therefore surgically evacuated **(19)**.

Type II (subacute) hematomas have homogenous high attenuation which has been attributed to a clotted hematoma observed in patients scanned 12 hours to days after injury **(20)**. Type III (chronic) hematomas are characterized by formation of a dense peripheral rim representing neomembrane of granulation tissue **(20)**.

Brain MRI is more sensitive than a CT scan, particularly when assessing for EDH in the vertex. It should be obtained when there is high clinical suspicion for EDH, accompanying a negative initial head CT scan **(Fig. 2)**. In the situation of a suspected spinal EDH, a spinal MRI is the preferred imaging modality, as it affords higher resolution versus a spinal CT **(18)**. The MRI appearance of acute extradural hematoma is isointense on T1-weighted images and extremely hypointense on T2 weighted images. As the hematoma ages, it becomes hyperintense on all imaging sequences. The membrane formed around chronic extradural hematoma enhances with gadolinium contrast **(21)**.

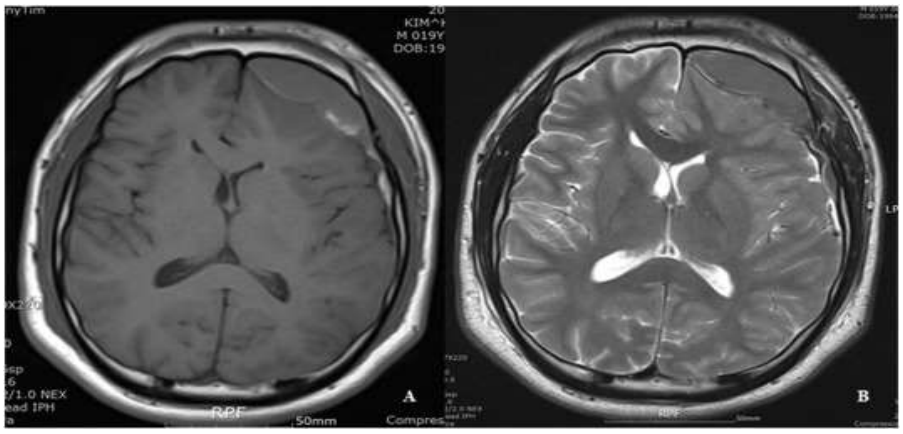

Figure (2): Brain MRI acquired 7 days after trauma revealed an increased amount of EDH, with iso-signal intensity of T1- (A) and T2-weighted (B) axial images

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If the decision is made to manage acute EDH non-surgically, close observation with repeated neurological examinations and continuous surveillance with brain imaging is required, as the risk for hematoma expansion and clinical deterioration is present. The recommendation is to obtain a follow-up head CT scan within 6 to 8 hours following brain injury **(18)**.

Operative management:

In patients with acute and symptomatic EDHs, the treatment is craniotomy and hematoma evacuation. Based on the available literature, "trephination" (or burr hole evacuation) is often a crucial form of intervention if more advanced surgical expertise is unavailable; it may even decrease mortality. However, the performance of a craniotomy, if feasible, can provide a more thorough evacuation of the hematoma **(18)**.

The primary aim of surgery for extradural hematoma is to decompress the brain, relieving both the local pressure effects and the general effects of raised intracranial pressure. This obviously requires not only hematoma evacuation, but also strict attention to the hemostasis to prevent recurrence. In general, an extradural hematoma must be considered a neurosurgical emergency, and evacuation should ideally be achieved as soon as possible before there is deterioration in conscious state or neurological deficit with hematoma volume more than 30m1 volume, thickness more than 15mm &midline shift more than 5mm **(22)**.

Urgent evacuation of extradural hematoma is indicated in any patient in whom the extradural hematoma is associated with deteriorating conscious state, any abnormal neurological signs, and/or if there is any significant radiological mass effect particularly volume, midline shift or effacement of the ipsilateral ventricle **(23)**.

Small extradural hematomas with none of these associated features mentioned above may be treated expectantly in an alert and cooperative patient. However, it must be stressed that this is a potentially risky clinical strategy, and it would only be appropriate if very close neurological observation can be maintained and the facilities for emergency craniotomy are available. The potential for an extradural hematoma to rapidly enlarge must be considered, and it must also be recognized that to delay surgery until after there has been a significant deterioration in the patient conscious state will lead to a significantly reduced chance of good outcome **(24).**

Special considerations:

Mass effect identified on the CT scan (such as the degree of midline shift. effacement of the lateral ventricle, uncal or subfalcine herniation) must determine the urgency of treatment rather than the size of the hematoma. Associated intracranial lesions such as cerebral contusions, intracerebral hematomas, subdural hematomas and even contralateral extradural hematomas are not uncommon. It is therefore essential that the whole of the intracranial cavity is scanned and that these associated pathologies are recognized, as not only are they prognostically important and may require treatment in their own right, but they may also enlarge after the evacuation of the extradural hematoma **(25)**.

The CT scans should be printed with the lateral skull scout view and also with axial bone window views to allow the accurate assessment of skull fractures. Although these will usually not require specific treatment, multiple skull fractures may influence the choice of intraoperatively head stabilization **(26)**.

Aggressive hemostasis must be obtained at all the wound sites. A pentobarbital coma should be instituted if continued elevations in intracranial pressure were recorded despite

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hypocapnia, diuresis, and ventriculostomy drainage. With continued elevations of ICP, patients with a severely contused brain are taken to surgery for a lobectomy. A full neurological follow up must be completed before discharge and from 6 months to 1 year afterward **(27)**.

Delayed evacuation:

The Most frequent and important complication in treatment of extradural hematoma relates to a delay in the diagnosis or evacuation of the hematoma. This has devastating prognostic implications and its avoidance is the single most important factor in achieving good results in the treatment of patients with extradural hematoma **(25)**.

Re-accumulation:

Re-accumulation of the extradural hematoma can occasionally occur, although it should be extremely unusual if strict attention to hemostasis, combined with dural hitching sutures and/or wound drainage, have been performed during the operation **(28)**.

Failure to diagnose other intracranial pathology:

Between 25% and 40% of patients with extradural hematomas, and up to 75% of those who are comatosed, have other significant intracranial pathology, such as subdural hematoma, cerebral contusion, intracerebral hematoma or a second extradural hematoma. It is essential that these are recognized on the initial CT, as they not only may require treatment in their own right and adversely affect the prognosis, but must be recognized as having significant potential to enlarge after the evacuation of the extradural hematoma. These patients should therefore have extremely close postoperative neurological monitoring, and serious consideration should be given to both monitoring and routine postoperative CT scanning **(27)**.

Complications of surgery:

1) Infection:

The risk of infection should be very low after the evacuation of an extradural hematoma, although if it is associated with a compound fracture or significant scalp laceration the risk is elevated significantly **(22).**

2) Delayed postoperative extradural hematoma:

The avoidance of postoperative bleeding and prevention of extradural hematoma is essential for safe neurosurgery. A postoperative hematoma is often attributed to the pitfalls of surgical technique. Meticulous technique and appropriate use of topical hemostats, such as oxidized cellulose, Gelfoam, fpgiue, and, possibly, aprotinin, play a pivotal role in the control of surgical hemorrhage. In recurrent bleeding of EDH due to MMA bleeding embolization of middle meningeal artery may be done **(29)**.

3) Nerve injury:

Injury to the frontal branch of the facial nerve should not occur during evacuation of a hematoma if the preauricular skin incision is kept <1 cm anterior to the tragus and the temporalis fascia incised in line with this incision, with only a subperiosteal dissection of the zygomatic arch being used. Traction on the greater superficial petrosal nerve may occur,

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as the dura on the floor of the middle fossa is raised, the hematoma or iatrogenically during surgery. Although this can theoretically transmit traction to the facial nerve in the temporal bone, facial nerve palsy due to this is extremely rare. However, injury to the greater superficial petrosal nerve itself can result in a dry eye postoperatively **(22)**.

Prognosis:

In general, patients with pure EDHs have an excellent prognosis of a functional outcome after the surgical evacuation, when it is rapidly detected and evacuated. A delay in diagnosis and treatment increases morbidity and mortality **(30)**.

EDHs caused by arterial bleeding develop rapidly and can be detected quickly. But those due to a dural sinus tear develop more slowly. Thus, clinical manifestations may be delayed, with a resultant delay in recognition and evacuation. Generally, an EDH volume greater than 50 cm prior to evacuation results in a worse neurological outcome and consequent mortality **(18)**.

Factors that may influence the outcome are as follows: patient age, time elapsed between injury and treatment, immediate coma or lucid interval, presence of pupillary abnormalities and GCS/motor score, on arrival **(31)**.

Several markers that correlate with a poor prognosis of EDH include the following: a low GCS before surgery, or on arrival, abnormal pupil examination, in particular, un-reactive pupils (unilateral or bilateral), advanced age, the time between neurological symptoms and surgery and elevated ICP in the post-operative period

Certain head CT findings can correlate with a poor prognosis: hematoma volume of greater than 30 to 150 ml, a midline shift greater than 10 to 12 mm, “Swirl sign” indicating an active bleed and Associated intracranial lesions such as contusions, intracerebral hemorrhage, subarachnoid hemorrhage, and diffuse brain swelling **(18).**

Conservative of post-traumatic extradural hemorrhage versus surgery:

Advances in imaging tools encouraged neurosurgeons to present the conservative management as an option for treatment and the number of patients who were initially managed conservatively has risen. Small EDH thickness, with a conscious patient, supports the decision of conservative treatment **(32)**.

The volume of EDH above 30 ml should be treated surgically and many institutions all over the world follow those guidelines **(33)**.

Non-operative management comes out as an option after the CT development and its availability with affordable price. A second advantage of the conservative management is avoidance of the high-risk complications of the operative intervention like massive blood loss due to open venous sinuses or injured middle meningeal artery. The third advantage is that the patients who were treated conservatively had less hospital stay and less ventilator use **(34)**.

The role of conservative management and various reasons for conversion to surgical intervention in traumatic extradural hematoma (EDH). They 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

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repeated CT scan. Early diagnosis and immediate surgical intervention had good outcome, which shows the fruitful results of early diagnosis and intervention **(35)**.

On other hand, an unusual case of a remarkable epidural hematoma managed conservatively with a favorable clinical outcome. Posttraumatic epidural hematoma presenting a maximum thickness of 2.5 cm, a midline shift of 1.2 cm, and approximate volume of 30 ml can be successfully managed in a conservative fashion **(36)**.

Many factors may play a role in the patient's ability to tolerate a given clot volume, including rapidity of volume accumulation, presence of associated intradural lesions, elasticity of the brain, combined volume of the sulci and other anatomic variations, and the location of the hematoma. EDH, both supratentorial and in the posterior fossa, can be managed nonsurgically. A large volume EDH (>30 cm3) can be managed nonsurgically provided the GCS at presentation and follow-up remains the same, with symptomatic improvement **(37)**.

Conclusion:

The conservative management gives successful results and the good outcome conservative management of EDH has been increased in the last years ,it is increased from 1% to 60%, making conservative management an important issue to study so the patients can avoid complications of anesthesia especially when there is a medical problem; and complications of surgery, and the conservative management gives a successful results in the management of EDH according to the protocol with close neurological observation in a hospital equipped with a CT brain; following the GCS of the patient; the volume of EDH and the other factors of the protocol of the conservative management of EDH. It should be noted that the neurosurgeon must be ready at any time to do surgical evacuation of the EDH if any deterioration of conscious level or increase of hematoma volume.

Recommendation:

- The recommendation is to obtain a follow-up head CT scan within 6 to 8 hours following brain injury.
- If the decision is made to manage acute EDH non-surgically, close observation with repeated neurological examinations and continuous surveillance with brain imaging is required, as the risk for hematoma expansion and clinical deterioration is present.
- Once confirmed on imaging, rapid determination of those requiring surgical intervention should be based on the evolution of their neurological status from time of symptom onset to the latest neurological examination by a trained Neurosurgeon.
- Any patient without sign of improvement or any patient with deterioration in ASIA score should be emergently treated by surgery.
- Identify the optimal therapeutic solution including both surgical and conservative treatment are required to identify and substantiate more parameters.
- Continuous quality improvement program and auditing.
- Education and training of health care workers and adherence to a standardized protocol for Management and deal with Epidural Hemorrhage.

No Conflict of interest.

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