



A CASE OF THALASSEMIC CHILD PRESENTING WITH EXTRADURAL HEMATOMA

Neurosurgery

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ABSTRACT

Extradural hematoma (EDH), a collection of blood between the skull bone and the dura, is a commonly seen after head trauma. Neurosurgeons have to take urgent decisions in the emergency room and usually there is no time to address other issues apart from history of recent trauma. However, sometimes there are associated conditions which make the situation worse. **Case description** – This report describes a case of 9 year old male child with thalassemia and severe anaemia who presented to neurosurgery emergency following a road traffic accident with a right frontal EDH. **Conclusion** – EDH evacuation is an emergency procedure and when you have a patient who is thalassemic and severely anaemic, surgery becomes all the more necessary.

KEYWORDS

Extradural, hematoma, thalassemia

INTRODUCTION

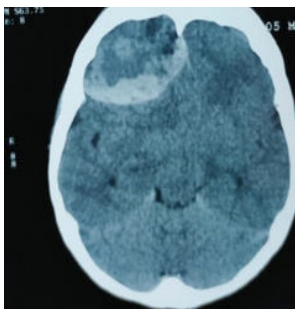
Extradural hematoma (EDH) is collection of blood between the skull bone and dural layer, commonly seen after trauma to head although sometimes it can occur spontaneously¹. It is most commonly seen after road traffic accidents followed by fall from height, however in children the latter is the more common cause^{2,3}. It is a neurosurgical emergency and if operated timely, more often than not it proves to be a life-saving procedure. Sometimes we have patients with significant medical history such as blood disorders like thalassemia, sickle cell anaemia, leukaemia or cardiac patients on antiplatelet medications. In these cases the neurosurgeon has added responsibility to take complete history and take necessary steps, simultaneously not delaying the surgical management for too long as “time is brain” for neurosurgeons.

CASE DESCRIPTION

A 9 year old male child presented to our emergency department with history of road traffic accident before 12 hours. On presentation patient had the following vitals Pulse rate- 62/min, Blood Pressure- 126/90mmhg His Glasgow coma scale (GCS) on presentation was E3V3M5. On local examination, he had a forehead swelling with ecchymosis of skin. CT brain done from outside showed a right frontal EDH approximately 30 ml in volume.

On further discussion, the parents gave the information that the child had low haemoglobin and they were taking him for blood transfusion when this accident happened. The child had beta thalassemia major and his fresh haemoglobin was 4.7g/dl, he required two transfusions per month usually. Both the parents were thalassemia carriers. No history of any surgical procedure or to be specific splenectomy was given. He was never on any chelation therapy for iron overload.

Clinically child was pale, there was no jaundice. On per abdomen examination, liver and spleen were not enlarged.



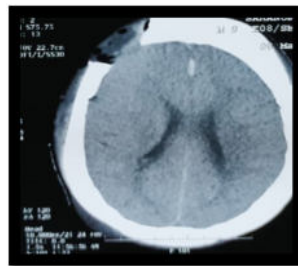
**Preoperative CT scan
MANAGEMENT**

This was not one of those EDH where we could just take the patient to operating room (OR) straightaway. Patient was severely anaemic and taking him straight to OR meant increasing the risk many times. The patient was transfused two units of packed red blood cells (PRBC) over

12 hours and during this time hourly GCS check was done to monitor any drop in response, more importance being given to best motor response and OR was kept ready for emergency burr hole. After two transfusions patient's hemoglobin rose to 7.8g/dl, coagulation profile normal, platelets on lower side at 83,000/microlitre. We took the patient to OR with PRBC and platelets kept ready for intraoperative transfusion.

Intraoperatively we planned for right frontal hairline incision starting in front of tragus and above zygoma, there was profuse bleeding starting from the skin incision. Skull bone had granular bluish appearance possibly due to chronic infarcts seen in thalassemic bone. As we tried to drill burr holes for craniotomy, the bone depth was too much to drill past the inner table. There was profuse bleeding from pericranium and diploic spaces (extensive marrow) and patients hemodynamics were changing rapidly with this amount of blood loss. We completed one burr hole using hudson brace and did a 4 cm craniectomy and evacuated the EDH. Bone edges were waxed and dural hitch sutures taken. Surgical placed over the craniectomy site and closure done. The entire procedure was completed in 30 minutes. One PRBC was transfused starting at the time of closure. Patient was extubated uneventfully and was stable hemodynamically. His GCS improved to E4V3M6 in the immediate post operative period.

Postoperatively patient did well apart from right periorbital swelling which gradually resolved. He was started on soft diet the next day and underwent CT brain on his first post operative day which showed no residual EDH. His post operative hemoglobin was 8.5g/dl.



DISCUSSION

EDH is a neurosurgical emergency and is common after traumatic head injury. Most of the cases are reported after road traffic accidents followed by fall from height and physical assault². The most common source of bleeding is middle meningeal artery followed by diploic veins, these get injured following fracture of skull bone which is found to be associated in 70-90 percent cases⁴.

EDH spans all ages and is also common in infants and children. It can occur in frontal, temporal, parietal or occipital regions. It can be easily diagnosed with plain CT brain and if diagnosed timely, the surgical management almost definitely saves the life of patient. Conservative

management is also an option in patients with less clot volume, less mass effect and GCS $\geq$ 14.

Thalassemia presents a challenge in the management of EDH, as these patients are usually anaemic which is sometimes severe. Also they have tendency of increased thrombosis which leads to chronic infarcts in skull bone which decreases the bone density. In hematological disorders, chronic extra-medullary hematopoiesis occurs. Hyper-proliferative bone marrow disrupts the inner and outer skull margins and precipitates extravasations of blood into the subgaleal and extradural spaces¹. This is one of the postulations for spontaneous component of EDH in such disorders. Some of them, mostly those with thalassemia major have enlarged spleen which may be consuming platelets and thus altering the coagulation processes in body. Massive splenomegaly causes concern about possible splenic rupture. Leucopenia or thrombocytopenia due to hypersplenism causes clinical problems (e.g. recurrent bacterial infection or bleeding)⁵.

Thus, any surgical procedure in such patients presents a challenge to the surgeon, all the more if it is a neurosurgical procedure. The patient must be transfused prior to surgery if he/she is anaemic and hemoglobin brought upto or above 7g/dl and to check the coagulation profile also. If patient has jaundice, that needs to be well informed to the anaesthetist so that they can manage accordingly. The surgeon needs to be alert about the possible complications and to manage the medical part also but still keeping in mind that patient needs surgery which cannot be delayed.

ACKNOWLEDGEMENTS

None

CONFLICT OF INTEREST

None

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