Scientific Research Publishing

Open Journal of Modern Neurosurgery, 2023, 13, 69-73
https://www.scirp.org/journal/ojmn
ISSN Online: 2163-0585
ISSN Print: 2163-0569

Intracranial Calcified Extradural Hematoma about a Case

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How to cite this paper: Kanikomo, D., Diallo, M., Tokpa, A., Sogoba, Y., Koumaré, I.B., Diarra, M.S., Sogoba, B., Diallo, O., Dama, M., Coulibaly, O., Coulibaly, M. and Kanikomo, A. (2023) Intracranial Calcified Extradural Hematoma about a Case. *Open Journal of Modern Neurosurgery*, **13**, 69-73.
https://doi.org/10.4236/ojmn.2023.132009

Received: November 25, 2022
Accepted: April 17, 2023
Published: April 20, 2023

Abstract

Introduction: Extradural hematoma is a common pathology in our department. The natural history of small-volume hematoma is usually towards resorption. In rare cases, the hematoma calcifies and compresses the brain. We report an extradural hematoma in a 15-year-old boy who had a road traffic accident on February 2011. The brain CT-scan performed the following day revealed a small right frontal extradural hematoma. During hospitalization, he had a fever of 40 degrees with convulsions and the blood culture isolated the coagulase-negative staphylococci and Group D streptococci. When his conscience was restored, hemiparesis and dysarthria were observed. The control brain CT-scan performed 2 weeks after the trauma showed early resorption of the hematoma. Two months after the trauma, the brain CT-scan showed calcification of the hematoma. He underwent surgery in April 2011 to remove the calcification. The sequels were hemiparesis and posttraumatic epilepsy. **Conclusion:** The evolution of a small non-operated extradural hematoma can be done towards calcification thus requiring surgery.

Keywords

Extradural Hematoma, Calcification

1. Observation

A male student of 15-year-old was involved in a road traffic accident (motorcycle skid) on February 2011 in Sikasso, a region 369 km from Bamako. He was admitted to the regional hospital in a state of brain injury with loss of conscious-

ness and trauma to the left thigh. On admission to the intensive care unit of the Gabriel Touré University Hospital, his Glasgow coma scale was evaluated at 7/15. The brain CT scan performed revealed a small right frontal extradural hematoma with no mass effect (Figure 1). View of the persistence of the loss of consciousness which lasted one week, he underwent a tracheotomy. The course was marked by the onset of a fever at 40 degrees during hospitalization associated with seizures. Blood culture isolated coagulase-negative staphylococcus and streptococcus from Group D. On regaining consciousness, hemiparesis and dysarthria were observed. A follow-up CT-scan three weeks after showed the beginning of hematoma resorption. In April 2011, because of seizures a control CT was performed and showed a calcification of the hematoma on the right frontal side (Figure 2). The patient was operated on for removing the calcification. A frontal bone flap allowed access to the calcification which was extracted. He also had a calcification on his left thigh. The course was favourable marked by a regression of the hemiparesis but persistence of the seizures.

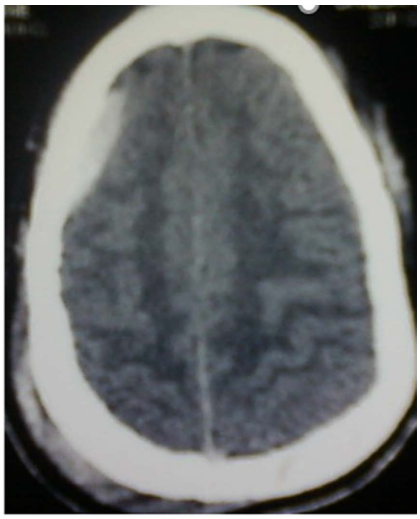

Figure 1. Brain CT-scan without injection, showing small right frontal hematoma.

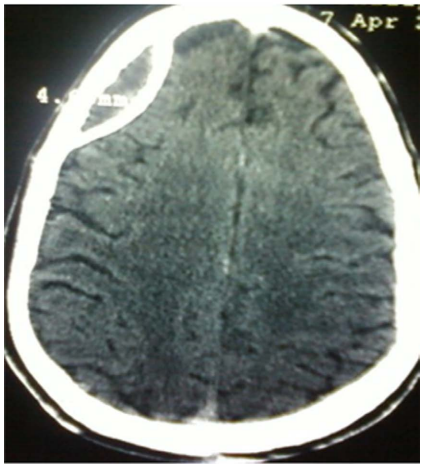

Figure 2. Brain CT-scan, showing calcified right frontal extradural hematoma.

2. Discussion

The non-operated extradural hematoma usually progresses to absorption in 3 to 15 weeks [1]. The evolution can be done, in rare cases, through encapsulation [2] or calcification [3]. Several mechanisms of resorption of extradural hematoma are described in the literature: fibrovascular neomembrane (covering the dural face of the chronic EDH) [4], arteriovenous shunts that develop in the extradural space during haemorrhage [5], to the infiltration of blood through the fracture line in relation to the EDH [6] [7].

Calcification of the extradural hematoma remains rare, accounting for 4% - 30% of non-operated EDH [8]. It occurs within 3 months to 3 years after the trauma in the literature. This delay was 62 days in our observation compared to 73 days for Chang *et al.* [9]. However, in the literature calcification occurs earlier between ten [10] and twelve days [11] [12]. A maximum delay of 40 years has been reported by Negan [13]. Calcification affects all ages, but it is thought to be more common in children and adolescents like ours. All of the four patients of Quenum *et al.* [14] and the two patients of Kawata *et al.* [12] were less than fourteen years old. On the other hand, Trodi's case was 36 years old [11].

The mechanism of calcification remains poorly understood. It is due to the local inflammation that gives a proliferation of fibroblasts that cause a neocapsule on the dural face of the hematoma around the fourth day according to Chen [15]. Poor circulation, insufficient cell degradation and necrosis would facilitate calcium deposit [10]. Early ossification in this child may be due to disproportionate tissue repair following acute injury [16] [17].

Calcification of the extradural hematoma is done on the external side of the dural unlike that of the chronic subdural hematoma where it is done on the inner side according to Nagane *et al.* [13].

Surgical treatment is advocated by some authors [9] [10] [11] [17]. Like Dawar *et al.* [17] we opted for surgical treatment despite the absence of the mass effect to have a cerebral and dural expansion. Similarly Shim *et al.* [18] suggested that surgical removal of calcified EDH should be considered, even if the patient's condition is good, as this carries a risk of excessive ossification. On the other hand Do Yeon *et al.* [19] proceeded to the conservative treatment (calcification confused with the internal table of the skull). The prognosis remains good in the literature.

3. Conclusion

Calcification of non-operated extradural hematoma remains a rare evolutionary mode. Thus, conservative treatment of extradural hematoma requires clinical and scannographic monitoring. The treatment of calcification is essentially surgical with a good prognosis.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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