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ORIGINAL ARTICLE

Clinical profile of subdural hematomas: dangerousness of subdural subacute hematoma

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Abstract Subacute subdural hematomas are a poorly individualized nosological entity, often equated clinically to chronic subdural hematomas. Yet, their neurological deterioration which is usually rapid seems to distinguish them from chronic subdural hematomas. We wanted to show this dangerousness by establishing the clinically evolving profile of the three types of subdural hematomas. This was a prospective and retrospective study of 63 subdural hematoma (18 acute, 13 subacute, and 32 chronic) patients admitted between 2012 and 2014 in the neurosurgery unit of Lomé University Hospital. Hematomas were classified according to the elapsed time after head injury and blood density on CT. The main parameter studied was the evolution of the Glasgow Coma Score (GCS) in the 3 months following the trauma, enabling to establish an evolving profile of each type of hematoma. The average age of patients was 58.1 years for chronic subdural hematomas and 47.6 years for subacute subdural hematomas. Disease duration before admission was 13.1 days for chronic against 36.6 h for subacute hematoma. The clinical profile shows acute worsening within hours during the second week for patients with subacute hematoma, while it is progressive for patients with chronic hematoma. We noted two deaths, all victims of a subacute hematoma (one operated, one patient waiting for surgery). Iso-density hematoma on CT, especially in a young person, must be considered as a predictive factor of rapid neurological aggravation suggesting an urgent care or increased monitoring by paramedics.

Keywords Subdural hematomas · Clinical profile

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Introduction

Subdural hematomas are the collections of blood localized between the dura mater and arachnoid. These are common complications of head injuries (TC) and classified according to the delay of head injuries and the age of the blood in imagery into acute, subacute, and chronic [9, 12]. Unlike the other two, the clinical characteristics of subacute subdural hematoma are poorly reported and not well known. Subacute subdural hematomas are a poorly individualized nosologic entity, often clinically put in the same category as chronic subdural hematomas by several authors [15]. Yet, the young age of their patients and the rapid neurological aggravation seem to distinguish them from chronic subdural hematomas. This study aims to show the dangerousness of the subacute hematomas by comparing the clinical profile of the three types of subdural hematomas, while recalling the different physiopathological hypotheses that characterize them.

Materials and methods

This was a prospective and retrospective study on 63 subdural hematoma (18 acute, 13 subacute, and 32 chronic) patients treated between January 2012 and 2014 in the neurosurgery unit of Lomé University Hospital. Inclusion criteria were as follows:

– All patients treated for an acute subdural hematoma (ASDH) (head injury less than 7 days associated with a

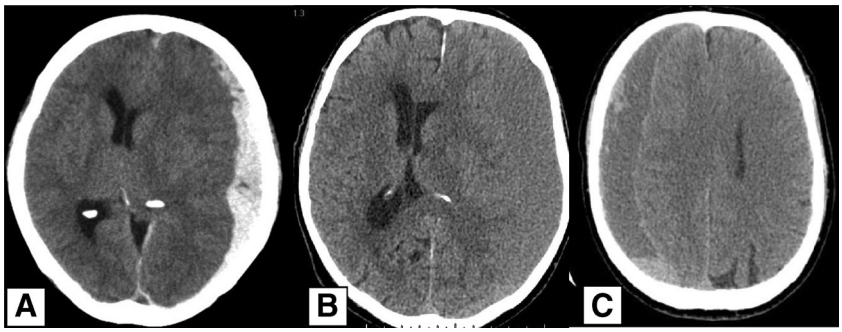

Fig. 1 Left acute subdural hematoma (**a**), left subacute subdural hematoma (**b**), and right chronic subdural hematoma (**c**)

hyperdensity on CT) (Fig. 1a) with a Glasgow Coma Score (GCS) >8 on admission who were operated or not

– Patients admitted for subacute subdural hematoma (SASDH) (cranial trauma between 8 and 21 days associated with an isodensity on the CT) (Fig. 1b) and chronic subdural hematoma (CSDH) (trauma of more than 21 days and with hypodensity or heterogeneous density on CT) (Fig. 1c), the Glasgow score of which was notified clearly after the cranial trauma

The parameters evaluated for each patient were age, the evolution of the GCS in the 3 months following the trauma, the appearance of the clinical signs and their duration, and the evolution. These parameters allowed to establish a progressive profile on the Glasgow score of each type of hematoma.

Results

The average age of patients was 58.1 years for CSDH and 47.6 years for SASDH. Symptoms occurred within an average of 27.4 days after the cranial trauma for CSDH against 12.6 days for SASDH. The duration for the development of symptoms before admission was 13.1 days on average for CSDH against 36.6 h for SASDH. Contortions or aftereffects of contortions were present respectively in 100 % of ASDH cases, 23.1 % of SASDH cases, and 5.7 % of CSDH cases. We noted two deaths, all victims of a subacute hematoma (one operated, one patient waiting for surgery). The assessment or the notification of the Glasgow score the days following the head injury allowed to establish the progressive clinical profile of patients, victims of the three types of subdural hematomas. This profile (Fig. 2) shows a rapid worsening within a few hours during the second week with improvement after surgery for SASDH, whereas it is progressive and less abrupt for CSDH.

Discussion

The classification of subdural hematomas into acute, subacute, and chronic phases according to the time limit and the characteristics of the blood in imagery is admitted [9, 12]. However, the distinction between subacute and chronic hematoma is not always obvious, both in the definition of time limit and in the imagery [8]. Even if the major principles of blood evolution in the imagery are known, the dating of subdural hematomas is influenced by hematocrit, hemoglobin, coagulation, and invasion of cerebrospinal fluid (CSF) [6, 18]. So in

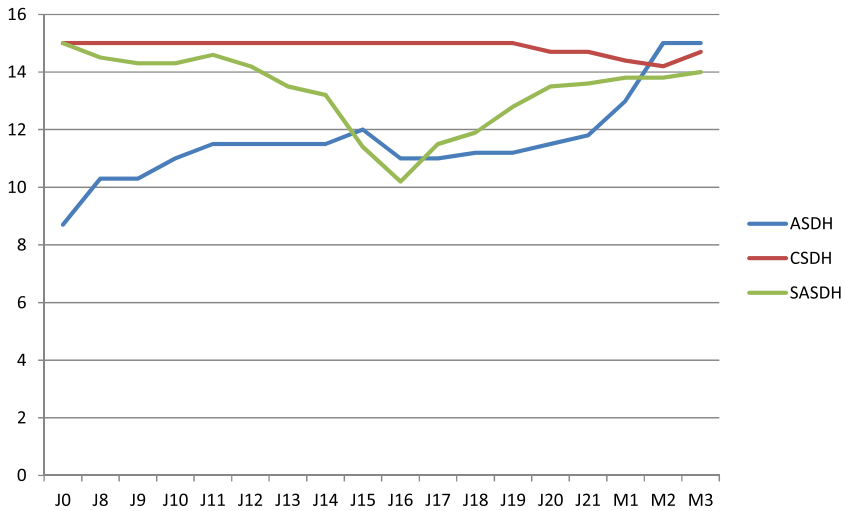

Fig. 2 Clinical progressive profile concerning the Glasgow score of the three types of subdural hematomas: acute subdural hematoma (*ASDH*), subacute subdural hematoma (*SASDH*), and chronic subdural hematoma (*CSDH*)

the presumed phase of a chronic subdural hematoma, it is possible to find a scanning isodensity simulating a subacute hematoma, which is responsible for confusion. The operative indications of acute subdural hematomas are based on their size, associated lesions, the clinical condition, and the technical capacity of neurointensive care [3]. For patients who underwent surgery, the improvement is slow but progressive in time. When the hematoma is minimal and pauci-symptomatic, it is possible to carry out a conservative treatment with a favorable prognosis [5, 7, 10, 14], but in some cases, there is a secondary aggravation mostly in the subacute phase [10, 11, 20]. What are the factors of that aggravation? Two mechanisms have been evoked for the explanation: first is the invasion of subdural hematoma by the LCS during the subacute phase caused by the difference in osmotic pressure on either side of the arachnoid, and second is the cerebral hyperperfusion at the subacute phase that follows the hypoperfusion of the acute phase [1, 2, 4, 11, 16, 17, 19]. So does the SASDH constitute a progressive form of the ASDH? The literature is controversial [11, 15]. Strictly speaking, the answer is yes, but it may also be the result of a progressive bleeding constitution after the initial venous rupture as in CSDH, and bleeding masked on the initial scan by the LCS invasion. Indeed, of the 13 patients operated for subacute subdural hematoma, 5 had an ASDH on the initial scan, 3 a normal scan and 5 had not made scan because they belong to the group 1 of Masters classification [13]. Although the physiopathology of SASDH is hypothetical and seems to integrate the characteristics of the other two, we should insist on their progressive dangerousness because we usually think that the dangerousness of subdural hematoma decreases when the traumatic phase is going away. Thus, it is crucial at the clinical course level to distinguish them from CSDH, even if their treatment is identical. But when analyzing their progressive profile in our series, we realize that they are different according to the mean age of the patient (47.6 years for SASDH against 58.1 years) and the rapidity of neurological worsening that is usually acute (36.6 h). This progressive searing intensity was responsible for a high mortality of SASDH (15.4 %). In addition, intraoperatively, we noticed that SASDH do not have a shell unlike CSDH. Thus, although the shell is a pejorative recurrence factor, it appears to be a protection factor against rapid worsening due to the fact that it constitutes an insurmountable anatomic barrier against the easy invasion of CSF, which is not the case for SASDH which does not have it. All these elements should be in favor of nosological individualization of SASDH.

Conclusion

The isodensity hematoma, especially in a young person, should be considered as a predictor of rapid neurological worsening suggesting the rapid care or increased monitoring by paramedics, especially as our working conditions do not always allow a quick charge socket (lack of financial means).

Conflict of interest The authors declare that they have no competing interests.

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Comments

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Dr. Kpelao and colleagues describe a rare sub-entity of subdural hematomas. In their retrospective study, the authors identified a distinct subgroup of patients suffering from an isodense hemorrhage with a short recent history of trauma and a tendency for a rapid neurological deterioration. Obviously, the paper from Togo is afflicted with the usual drawbacks of retrospective studies. The conclusion of the authors that patients suffering from subdural hematomas with a rapid clinical deterioration should be subjected to a fast surgical evacuation points out a rare but an important condition in clinical practice. Furthermore, the manuscript represents a noteworthy attempt to initiate an academic culture in African neurosurgery that so far is only rarely visible within the international neurosurgical literature. For this reason, I wish to congratulate our colleagues from Togo for their efforts in publishing scientific clinical articles and wish them success for the future.