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Case Report

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Post-traumatic subdural hygroma: About two cases

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Abstract

We report two clinical cases of subdural hygroma (SDH) in two male patients respectively aged 37 years and 44 years whose etiological factor was a cranio-encephalic traumatism. The cerebral scansperformed had shown a bi-fronto-parietal SDH in one patient and a right fronto-parieto-occipital SDH in the other. They had been placed under corticotherapy. The disease course was marked by a transformation of the SDH into subdural hematoma in one of the two patients.

Keywords: Subdural hygroma, Cranio-encephalic traumatism, Lubumbashi.

INTRODUCTION

The hygroma or subdural hygroma is defined through the existence of a subdural fluid collection most often bilateral, generally fronto-temporally localised and occurring preferentially in the aged patient [1]. It is facilitated by the existence of a cerebral atrophy, a dehydration or a traumatism in the past history [1,2]. We report two clinical cases of SDH in two male patients aged respectively of 37 years and 44 years whose etiological factor was a cranio-encephalic traumatism.

CLINICAL OBSERVATION

CASE 1

S.B., a male patient aged 37 years, was brought in emergency in excitement state to the Centre Médical du Centre –Ville (CMDC) after being hit by a motorcycle about 12 hours before. There is a notion of loss of consciousness for about 12 hours after the accident. He was a driver and mechanic by profession. No particular past history had been noted. During complementary anamnesis, it was reported that he was vomiting in spurts. The general state was marked by restlessness. The neurologic examination revealed a temporo-spatial disorientation, an impairment of recollection and fixation memories, and a disorder in symbolic functions (auditory agnosia). He also had neck stiffness at the end of flexion and a deep osteo-tendon hyperreflexia. The pollico-mental reflex was present bilaterally. The sucking reflex was also present. From the psychiatric point of view, a psychomotor excitement, a logorrhoea, and a silly euphoria had been observed.

We had inferred a frontal syndrome probably secondary to an intracranial post-traumatism cranio-encephalic expansive process. The patient was placed under a treatment of corticoids (Dexamethasone). The cerebral scan performed on the 14th day had revealed a bi- fronto-parietal SDH with diffuse left temporo-parietal axonal lesions. No bone lesion was identified (Figure 1).

In his disease course, from the neurological point of view, the patient had displayed an ideomotor sluggishness, yet he was well oriented in time and space.

From the psychiatric point of view, the patient had a depressive mood. Thecerebral scan performed on the 777th day had revealed tiny left frontal and parietal subdural hematomas (Figure 2).

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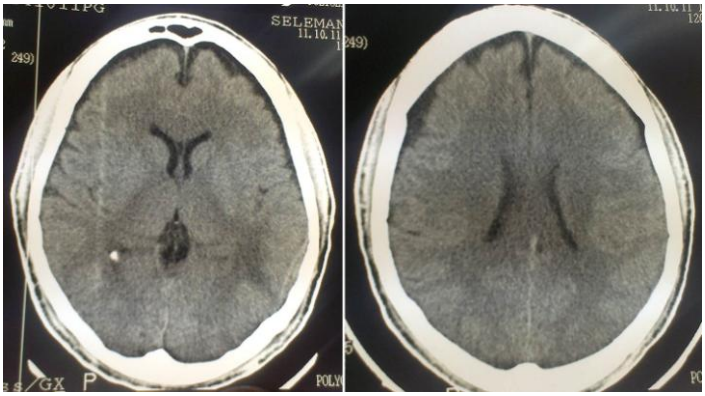

Figure 1: CT-Scan picture showing a bi-fronto-parietal SDH (14th day post-traumatism)

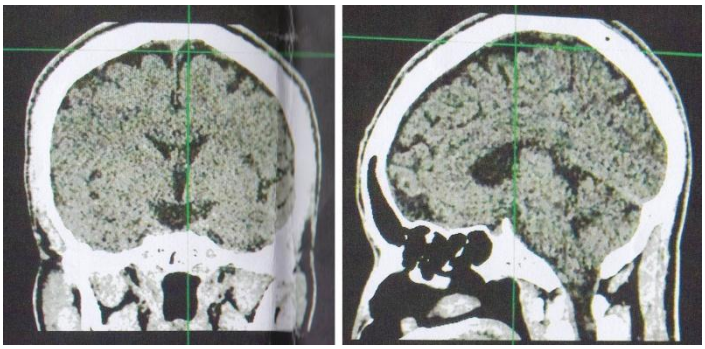

Figure 2: CT-Scan picture showing tiny left frontal and parietal subdural hematomas (777th day post-traumatism)

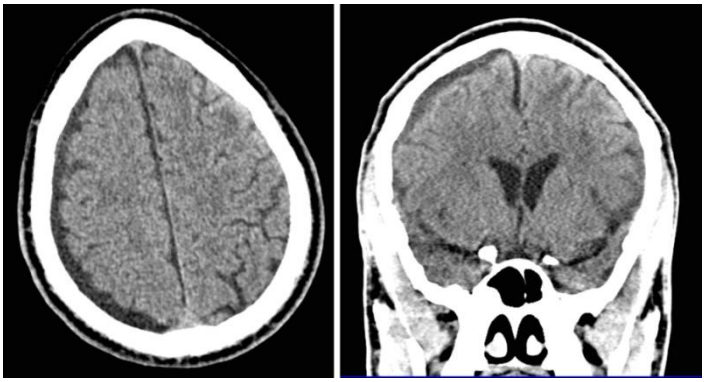

Figure 3: CT-Scan picture showing a right fronto-parieto-occipital SDH (15th day post-traumatism)

CASE 2

R.K., a male patient aged 44 years, was brought in emergency to Centre Médical du Centre –Ville (CMDC) for behaviour disorder consisting in incoherent words, excessive forgetfulness, talking nonsense, and night fugue. It started two weeks before the consultation following a cranio-encephalic traumatism secondary to a road traffic accident (the patient had fallen off his motorcycle that he was riding). Therefore, he had a loss of consciousness for about 24 hours. He was a mechanic by profession. No particular history had been noted. During the further anamnesis, he had frontal headaches of average intensity relieved by habitual antalgesics, an epistaxis and a facial puffiness. The neurosomatic examination revealed a subconjunctival hemorrhage of the left eye, an impairment of recollection and fixation memories, a temporo-spatial disorientation, an acalculia, a prosopagnosia, an astereognosis and inadapted gestures to questions asked. A deep osteo-tendon hyperreflexia and a bilateral pollico-mental reflex were also observed. Urinary and fecal incontinence was noted. We had inferred a frontal syndrome secondary probably to an intracranial post-traumatism cranio-encephalic expansive process.

The cerebral scan performed on the 15th day had revealed a right fronto-parieto-occipital SDH giving rise to a discrete left deviation of 2 millimetres of median structures (Figure 3). No bone lesion had been clearly identified. The patient had been placed under a treatment of corticoids (Dexamethasone). From the clinical point of view, the disease course wasmarked by a neat improvement of intellectual and symbolic functions on the 100th day of hospitalisation.

DISCUSSION

Severe cranial traumatism is by far the most frequent cause of SDH [3]. Out of 3002 patients admitted for cranial traumatism, Jaccard had found 70 cases of SDH, that is to say a frequency of 2.3% [4].

The mechanism of the occurrence of SDH is not clearlyelucidated, involving probably a splitting of the interface dura mater-arachnoid, and then accumulation of fluid by effusion from serum or from cerebrospinal fluid with a secondary formation of a neo-membrane. The latter may be vascularised and the bleeding at its level may entail blood accumulation within the cavity [5,6]. An insignificant traumatism may cause a splitting of the interface dura mater-arachnoid, which is the basic condition for the development of a subdural hygroma. According to Lee, cerebral atrophy, excessive dehydration, and the reduction of the intracranial pressure are necessary conditions for the development of the SDH and the latter may develop itself through a passive effusion [1,3,5].

The SDH is exceptionally symptomatic and its occurrence is most often casual [6]. In our two patients, we had thought about an intracranial post-traumatism cranio-encephalic expansive process of which the nature was determined by cerebral scan performed two weeks after the traumatism. In the series of Zanini, the average time of the diagnosis of hygroma was of 9 days [7].

The SDH does not often require a surgical treatment. It can regress or on the contrary be transformed in chronic subdural hematoma [2,6]. Jaccard had foundin his series that the surgical treatment, compared to conservative treatment, had been rarely followed by a significant improvement of patients [4]. This transformation in subdural hematoma occurs between 0 to 50% of cases, according to the type of study and the time of evolution [8] and depends on the persistence of necessary conditions beyond several weeks [1]. In Wang's study, the average time of transformation varied from 14 to 100 days after traumatism [9].

CONCLUSION

Cranio-encephalic traumatism is the most frequent etiological factor of subdural hygroma. The latter found casually and the cerebral scan allows making the diagnosis. Its evolution may be favourable without resorting to neurosurgery.

Authors' Contribution

All the authors have taken part in the caring of patients and in the writing of the manuscript. All the authors approve the final version of the manuscript.

Conflicts of interest

The authors do not state any conflict of interest.

Abbreviation

SDH: subdural hygroma

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