Atypical presentations of chronic subdural hematomas

The article by Alugolu[1] describes atypical neurological presentation of the chronic subdural hematoma. This case confirms the statement that the medicine is not like mathematics. Sometimes radiological findings do not correlate with neurological symptoms. Every case/patient should be considered individually.

A widely accepted classification divides subdural hematomas into acute, subacute and chronic according to the time elapsed between the head injury and the onset of clinical symptoms. The borderline between subacute and chronic hematomas is variously defined by different authors. Most authors define chronic hematoma as clinically manifesting after more than 2 or 3 weeks following an injury. The data for chronic hematomas estimate the frequency of preceding head injury at less than 50% of cases.[2,3] In some cases, because of rather trivial head trauma, some patients (especially older) forget about any trauma which happened some time ago and thus do not associate it with the development of hematoma. Consequently, these patients of members of their families do not report such head trauma to the personnel of the hospital.[3] Other risk factors which can cause development of chronic subdural hematoma are alcohol abuse, seizures, cerebrospinal fluid shunts and coagulopathies (including therapeutic anticoagulation).[4] Bilateral hematomas are reported to account for 20-25% of chronic hematomas.[5] Many chronic subdural hematomas probably start out as acute subdural hematomas. Blood within the subdural space evokes an inflammatory response. Within days, fibroblasts invade the clot and form neomembranes on the inner (cortical) and outer (dural) surface, followed by in-growth of neocapillaries, enzymatic fibrynolisis and liquefaction of blood clot. Fibrin degradation products are reincorporated into new clots and inhibit hemostasis. The course of chronic subdural hematoma is determined by the balance between plasma effusion and/or rebleeding from the neomembranes and the reabsorption of fluid.[6]

Trepanation is chosen as the primary surgical technique in the vast majority of patients with chronic subdural hematomas. Remaining patients undergo craniotomy. Clinical improvement is observed when the subdural pressure is close to zero, which usually occurs after about 20% of the collection is removed.[7] Patients with high subdural fluid pressure tend to have more rapid expansion and clinical improvement than patients with low pressure. Importantly, in elderly patients, rapid decompression following evacuation of hematoma may be associated with hyperemia of the cerebral cortex directly inferior to the hematoma, which may give rise to an intracerebral hematoma or epileptic seizures.[8]

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