AUTOPSY OF AN INTRADURAL HAEMATOMA CASE

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Abstract: This paper describes the microscopy of dura matter in a case of intradural haematoma, explaining diagnostic criteria on the basis of one real case in which the haematoma became associated with alcoholism and trauma, producing death.

Keywords: Intradural haematoma, internal haemorrhagical pachymeningitis-thanatogenerator syndrome, Romania.

The intradural haematoma is a overflowed blood which develops slowly and progressively within the intradural space, without a traumatic cause, and compressing the brain.

It has a low frequency: 0.1-0.2% in cranio cerebral pathology and is formed through a layer of fibrous tissue lined by arachnoida. The dura matter has one external, large, dense, and fibrous layer and another which is internal, thin, of low density, and fibrous. Between these Hannah described an abundant capillary net, classified by Pfeiffer, as follows:

First layer: an external arterial net, formed by very soft vessels, which join in large meshes and are fed from the dura mater vessels and from the arterial anastomoses of the diploe.

Second layer: a venous net, anastomosing to the external arterial net. It contains many vessels that finish at the end of the receptaculum. This layer, together with capillary arteries which meet in right angle, form what Langer called "duck beaks".

Third layer: sinuous arteries and scattered veins.

Fourth layer: this is the deepest layer and is composed of capillaries resembling a layer. They have very thin walls, and their microscopic aspect suggests an important capacity for transudation and reabsorption.

This rich vascular layer represents the source of the intradural hematic accumulation.

Intradural haematomes are more frequent in aged persons, often appearing bilaterally, and sometimes simultaneously.

Associated factors include those which favor it (vascular fragility), determinant modifications of dura matter, and releasers (effort, a feverish condition, a minor cranio-cerebral trauma). Most authors mention modifications of the pachymeninx in the genesis of the haematoma.

Favouring factors produce venous microbleeding: the blood accumulates between the two layers of dura matter, in the Hannah net. This blood can appear bilaterally (through modifications of dura), at mid level on the surface of brain.

The accumulations develop for a long time, rapidly in presence of the releaser factor; intracerebral balance is lost with the appearance of the intracranian expansive process symptomatology.

The haematoma has a fibrous, conjunctive external membrane and an internal membrane formed by newer conjunctive tissue. The dura matter changes histologically, presenting internal haemorrhagical pachymeningitis lesions. Simultaneous with this evolution (possibly for months), the external membrane becomes denser (1-2mm), and the blood remains perfectly encapsulated.

The volume is 50-150 ml, rarely more. Intradural haematoma has been reported to disappear through gradual reabsorption, but we consider that difficult.

Case description:

B.V., male, 52 years old, left a train while intoxicated and was beaten repeatedly by an officer with a stick. The victim lied in front on the building until the second day when he died (aprox. 8 a.m.). He suffered alcoholism and complained of headaches and dizziness, but received no treatment. Bruises covered 15-20% of the body and there were some superficial wounds.

Autopsy showed a discreet hemorrhagical epicranian infiltrate, frontal and temporal, on the right side (approximately 5 x 3 cm). Head bones were not fractured. Dura matter white, mother-of-pearl dark, shiny and adhered to the endobase. The right parieto-temporal part had two thickened layers (the external part 1 mm larger), limiting a cavity with 180ml of liquid brown yellow blood and a brown-violet, shiny clot with a yellowish hue.

On the left parieto-occipital side there was another cavity between layers of dura matter, with approximately 40 ml of blood. The right hemisphere corresponding to the intradural haematoma was depressed 2cm (11x8cm). The principal vessels of the brain were sinuous, with a rigid wall and with small plates by atroconversion on the intimate surface.

The chest and abdomen autopsy showed bronchial and lung sclerosis, aorta-coronarian atheromatosis, micronodular hepatitis cirrhosis and renal distroptic modifications.

Histopathological results:

-Dura matter (right hemisphere) with recent hematic suffusions. In some areas thickened, vascularised and with circular corpuscle inflammatory spots, with close accumulation of macrophages. Dura matter (opposite part) resembling hemorrhagical pachymeningitis.

-Visceral staza, general arterosclerosis, micronodular cirrhosis, pancreatic

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fibrosis and lipodistrofia, diffuse glomerulonephritis.

-Alcoholemia: 1g% and alcoholuria 1.40g%, at moment of death.

**DISCUSSION:**

The haematoma had a greater volume in the right side where it was compressive. There was evidence of a compensatory mechanism operating before the trauma for at least three weeks, showing that the haematoma was not caused by the attack.

Rather than any isolated factors (bruises, haematoma, alcohol), we believe that death was caused by a combination of factors and was not a result of the officer’s action. The autopsy does not support charges of murder.

Although in this case the histopathological exam confirmed the macroscopic diagnosis, microscopy is always required for reliable conclusions.

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