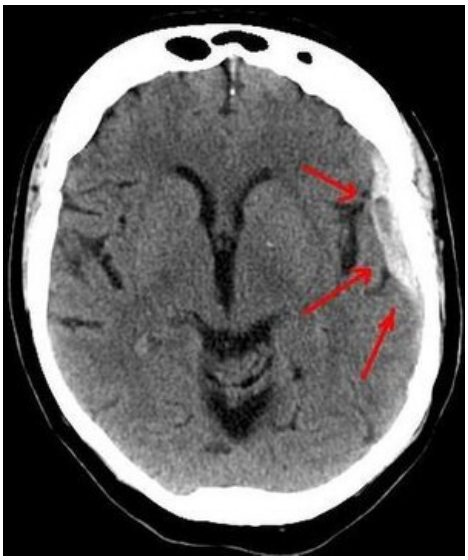
Chronic subdural hematoma

The essence of **chronic subdural hematoma** (CSDH) is bleeding from the bridging veins in patients with variously expressed cortical **atrophy** (elderly patients, alcoholics), as well as in infants. CSDH was first described by Rudolf Virchow in 1857 as "*pachymeningitis haemorrhagica interna*" [1], after which Wilfred Totter described the basics of CSDH pathophysiology and thus defined that hemorrhage from the bridging veins is its root cause and named it "*subdural hemorrhagic*" [2].

It is important to adequately distinguish CSDH from acute subdural hematoma. Compared with CSDH, acute SDH typically occurs in younger individuals primarily as a result of more severe cranio traumas and is associated with symptomatic compression of the brain parenchyma, which typically manifests within 72 hours. Conversely, in the case of CSDH, bleeding is **clinically asymptomatic for a longer period of time**, on the order of several weeks or months, most often occurring after **minor injuries**. The incidence of CSDH in the population is around 1.72 / 100,000 inhabitants per year, in the population aged 70–79 this value increases to 7.35 / 100,000 per year, thus further confirming the fact of a higher risk of developing CSDH in the elderly.

Risk factors



Subdural hematoma

The basic risk factors include **cerebral atrophy**, associated with older age. In direct proportion to aging, the space between the calva and the parenchyma itself increases from the original 6% to 11%. This allows the brain to move sideways, so in the event of trauma, bridging veins can be more easily damaged, resulting in CSDH. [3]. However, trauma alone is not the only cause of CSDH, patients with **anticoagulant medication**, **alcoholism** or **epilepsy** are also affected (up to 10% of patients with CSDH are alcoholics or people with a history of seizures). [4].

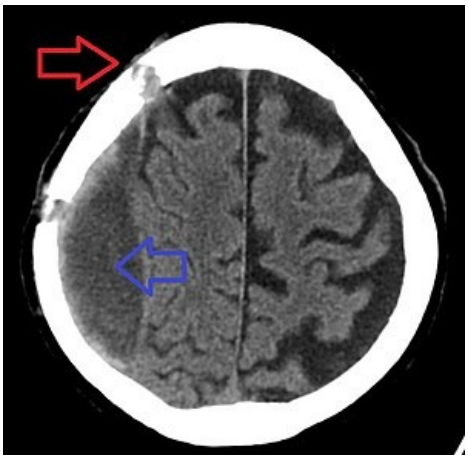
Pathophysiology and symptomatology

Damage to the bridging veins (eg in the case of trauma) results in bleeding into the subdural space, with a thin layer of fibrin and fibroblast forming around the hematoma surrounding the hematoma. Fibrin and fibroblasts further proliferate and migrate, forming a kind of membrane (approximately 4 days after the incident), which then progressively enlarges. Some CSDH is **resorbed spontaneously**, but most grows in volume and results in **clinically symptomatic CSDH** [5], neurologically **manifested** by headache, confusion or, at a more advanced stage, contralateral hemiparesis.

Diagnosis

CT is used to image the hematoma. It clearly displays the resulting hematoma. It is typically a hypodense extracerebral bed, see picture.

Therapy



Chronic subdural hematoma in an elderly patient with evident cortical atrophy. The blue arrow points to the hematoma site, the red one points to a trepanning hole.

In the case of large CSDHs without expansive tendencies and clinically good patient status, it is not necessary to evacuate the hematoma, **conservative treatment** consists in close monitoring of the patient [6]. In the case of a progressive symptomatic lesion, we indicate a **trepanation return**, ie the evacuation of covalent blood and the introduction of temporary subdural drainage from the return site under local anesthesia. In some cases, **craniotomy** is used, especially in patients with recurrent hematomas, resp. in persons showing blood re-accumulation after trenapulation return, or the presence of a solid hematoma. Rehydration of the patient and care for the patency of the drainage under strictly sterile conditions is important, while a control CT is performed after 3 days and the drain is removed if the finding is satisfactory. [7][8]

Links

Related articles

- Craniocerebral trauma

- Acute subdural hematoma
- Subdural hematoma
- Epidural hematoma
- Subarachnoid hemorrhage

References

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