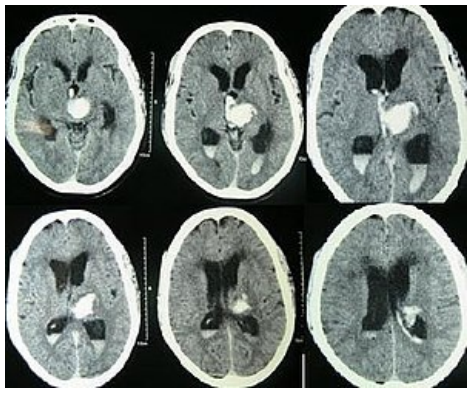
Bleeding into the capsula interna

Bleeding into the capsula interna is a hemorrhagic stroke. The most common cause is hypertension or rupture of a perforating artery. Less common causes include arteriovenous malformation and hemorrhagic diathesis. In the elderly, cerebral amyloid angiopathy is to blame. In younger patients, drug addiction (amphetamine, cocaine,...)

Symptomatology of hemorrhagic stroke



CT image of hemorrhagic stroke

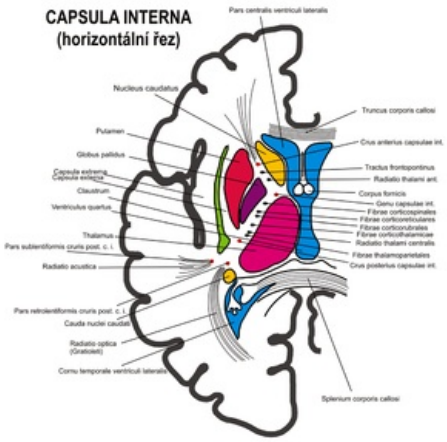
Depends on the size, character, etc. It is divided into major and minor bleeding.

Major bleeding

These are splinter hemorrhages, expansive in nature. There is the destruction of brain tissue. They have a severe neurological deficit. They present with headache, vomiting, and impaired consciousness (edema of the brain, intracranial hypertension). There is a risk of ventricular seepage (haematocephalus). Most patients die.

Minor bleeding

In contrast to the previous case, minor hemorrhage only compresses the brain tissue, it does not degenerate. It acts expansively (hematoma). Focal symptoms are predominant, depending on the localization of the hemorrhage.



Capsula interna (horizontal cut)

Most common localizations of cerebral hemorrhages

basal ganglia (*putamen, capsula interna*) 35-50%

thalamus 10-20%

brainstem 10-15 %

cerebellum 10-20 %

nucleus caudatus 5 %

Division by localization of hemorrhage (minor hemorrhage)

Infratentorial hemorrhage

Bleeding in the posterior cranial fossa is life-threatening. When the volume is increased by a hematoma or increasing brain swelling, it can rapidly displace neural structures upward transtentorial or, more commonly, downward into the foramen magnum with the development of an occipital conus, which very quickly leads to brain death.

Supratentorial hemorrhage

The primary symptom is a disappearing lesion dependent on the location, size, and propagation of the hematoma. In some localizations, clear focal expression may be absent and psychological attenuation or deterioration predominates. Arterial haemorrhages usually have an apoplectic onset; even very severe focal symptomatology can develop in seconds. Patients often suffer from nausea or vomiting. Headache is frequent, not only diffuse in intracranial hypertension syndrome but also localized.

Coma as an early symptom usually means extensive bleeding into the basal ganglia or directly into the brainstem with involvement of the ascending reticular formation. Initial unconsciousness lasting a few seconds or minutes may be followed by a severe neurological deficit. The local manifestation of hemorrhage is e.g. hemiparesis, hemihypesthesia or aphasia.

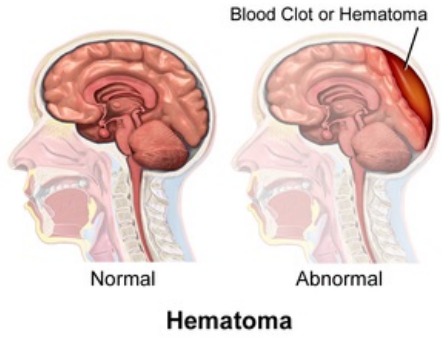
Typical hematomas

Supratentorial hematomas are **typical** (in the basal ganglia) and **atypical** (all others). Typical hematomas are deep-seated, affecting the basal ganglia (*capsula interna*) and thalamus. Affected patients have hypertension and are over 55 years of age. The main volume of the hematoma is typically in the putamen and the source is usually the *a. lenticulostriata* or other perforating arteries. The clinical manifestation is usually a complete neurological

deficit because the ***capsula interna*** area, the pyramidal pathway, is also affected. Quantitative impairment of consciousness is the rule, and a deep coma with the need for controlled ventilation may be present from the beginning.

Treatment and prognosis

Depends on the initial CT image. Unfortunately, hemorrhage cannot be reliably distinguished from ischemia. Expansive atypical hematomas, which can lead to the development of cone symptoms, are indicated for acute angiography because neurosurgical intervention with positive findings can treat both the hematoma and its source at the same time. Small hematomas tend to have better results with conservative treatment, and therefore angiography waits until the blood is absorbed or the expansive hemorrhage is corrected.



Hematoma

The mainstay of conservative management is therapy for *intracranial hypertension*. In large hematomas over 50-70 ml in volume, when hypertension is always present, treatment must be very aggressive. The duration of treatment depends on the size and localization of the hematoma, the degree of expansive behaviour, i.e. how much it leads to ventricular compression, etc. In a severe clinical course with impaired consciousness and respiratory disturbances, intubation and controlled ventilation are necessary, usually initially with controlled hypocapnia.

Complications of hemorrhagic stroke

Salt wasting syndrome

Otherwise known as cerebral salt wasting syndrome, is caused by increased production of certain natriuretic factors - atrial natriuretic factor, brain natriuretic factor and some other substances. Monitoring not only the actual ion values but also their balance and the overall fluid balance is therefore very important.

In treatment, it must be remembered that very rapid compensation of severe hyponatremia by administration of a markedly hypertonic *NaCl* solution can lead to central pontine myelinolysis, which is a severe irreversible and often fatal damage to the white matter of the brainstem and, preferentially, the pons.

Inappropriate therapy

Inadequate fluid and ion supply leading to hypovolemia and hypotension are devastating. However, excessive fluid intake, especially free water in glucose solutions or markedly hypoosmolar solutions, is equally erroneous.

Links

Related articles

- Capsula interna
- Motor system

Used literature

- Kalina, M. (2002). Akutní mozková hemoragie - diagnostika a léčba. Interní Med., 4(6), 22-28.
- AMBLER, Z – RŮŽIČKA, E. *Klinická neurologie – část speciální.* 1. edition. Prague : Triton, 2010. ISBN 978-80-7387-157-4.

References