Cerebral abscess

Pathology

Intracerebral abscess may occur as a result of direct spread from air sinus infection, following surgery or from haematogenous spread especially associated with respiratory infection, endocarditis or dental infection. In around 25% of cases no cause is found.

Patients at increased risk of cerebral abscess formation include those with cyanotic heart disease and those who are immunocompromised: patients with diabetes, solid organ transplant, haematological malignancy or long-term steroids.

A 2- to 3-day period of early cerebritis with inflammatory cell inflammation is followed by late cerebritis over days 4–9, with the formation of a necrotic core and increasing numbers of macrophages and fibroblasts. After 10 days, a capsule begins to form and is firm and mature by day 14. In a non-compromised host the causative organisms include anaerobic and micro-aerophilic streptococci, staphylococci, enterobacteria and anaerobes. Immunocompromised hosts are also susceptible to other infections including *Nocardia*, *Listeria*, *Aspergillus*, *Candida*, *Cryptococcus* and *Toxoplasma*.

Diagnosis

Presentation is with focal signs, seizures and raised ICP, as with other mass lesions, but the time course is often short. Patients may be febrile or have a raised peripheral white cell count or inflammatory markers. However, many patients with cerebral abscess show no markers of systemic infection. An intracerebral abscess appears as a ring-enhancing mass lesion on CT or MRI and the lesions may be multiple in the case of haematogenous spread. Differential diagnosis includes primary brain tumour and cerebral metastasis. MRI may help differentiate between a tumour and an abscess.

Treatment

Cerebral abscess is usually treated by surgical drainage followed by administration of intravenous antibiotics for at least 6 weeks. Antibiotic therapy is rationalised according to microbiological culture and sensitivity. Multiple small abscesses may be treated medically with antibiotics targeted against organisms isolated from systemic sources.

Steroids are reserved for cases with significant oedema or mass effect and their routine use is discouraged because of their negative impact on antibiotic therapy. Owing to the high risk of seizures, patients should also be treated with anticonvulsants.

Subdural empyema

Subdural empyema is less common than cerebral abscess but carries a high mortality (5–10%). Infection usually spreads locally from sinusitis and the pus collects over the cerebral convexity and in the parafalcine space. Location in the subdural space encourages cortical venous thrombophlebitis with consequent venous infarction. Causative organisms are usually streptococci such as *Streptococcus viridans* or *Streptococcus milleri.*

Presentation is with headache, fever and meningism. Seizures are common and focal neurological deficits may progress quickly to altered mental state and coma.

Diagnosis by non-contrast CT can be difficult and a high index of suspicion must be used – a small parafalcine collection of fluid may be all that is visible. MRI is useful when the diagnosis is doubtful.

Treatment is by craniotomy and thorough drainage of the pus, followed by intravenous antibiotic treatment, anticonvulsants and radiological surveillance as for cerebral abscess.

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