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24.11.3 Intracranial abscesses 6097 Goh KJ, et al. (2000). Clinical features of Nipah virus encephalitis among pig farmers in Malaysia. N Engl J Med, 342, 1229–35. Granerod (2010). Causes of encephalitis and differences in their clin- ical presentations in England: a multicentre, population-based pro- spective study. Lancet Infect Dis, 10, 835–44. Gyure KA (2009). West Nile virus infections. J Neuropathol Exp Neurol, 68, 1053–60. John CC (2015). Global research priorities for infections that affect the nervous system. Nature, 527, S178–86. Landry (2009). Herpes simplex type 2 meningitis: presentation and lack of standardised therapy. Am J Med 122, 688–91. Mansfield KL, et al. (2009). Tick-borne encephalitis virus—a review of an emerging zoonosis. J Gen Virol, 90, 1781–94. McGill F, Griffiths MJ, Bonnett L, Geretti AM, Michael BD, Beeching NJ, McKee D, Scarlett P, Hart IJ, Mutton K, Jung A, Adan G, Gummery A, Sulaiman WAW, Ennis K, Martin A, Haycox A, Miller A, Solomon T, on behalf of the UK Meningitis Study Investigators. Viral meningitis in UK adults - a multicentre prospective observational cohort study of incidence, aetiology and sequelae. Lancet Infect Dis (2018) In Press. Messacar K (2015). A cluster of acute flaccid paralysis and cranial nerve dysfunction temporally associated with an outbreak of enterovirus D68 in children in Colorado, USA. Lancet 385, 1662–71. Muñoz LS, Barreras P, Pardo CA (2016). Zika Virus-Associated Neurological Disease in the Adult: Guillain-Barré Syndrome, Encephalitis, and Myelitis. Semin Reprod Med, 34(5), 273–79. Epub 2016 Sep 9. Ooi MH, et al. (2007). Human enterovirus 71

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these patients. Intracranial abscesses can be categorized according to their ana- tomical location, occurring in the extradural space, subdural space, or intraparenchyma. Occasionally, abscesses exist in more than one tissue plane. Intracerebral and subdural abscesses may rupture into the subarachnoid space and be accompanied by meningitis. Intracerebral pus may rupture into the ventricular system and pro- duce ventriculitis.

section 24 Neurological disorders 6098 Aetiology The aetiology can be divided according to the route of transmission: • Direct—microorganisms spreading from a local source of infec- tion such as otitis media, mastoiditis, paranasal sinusitis, or dental infection. • Haematogenous—microorganisms spreading from a distant source such as a right-to-left shunt secondary to cyanotic con- genital heart disease in children, or subacute endocarditis, bron- chiectasis, and lung abscesses in adults. Abscesses can also form after intracranial surgery or cranial trauma. Immunodeficient patients are at increased risk of developing ab- scesses, either secondary to conditions such as AIDS or leukaemia. It is also thought that ischaemic brain tissue may encourage bac- terial invasion and, therefore, abscesses can develop in closed head injuries and following cerebral infarcts, although this is rare. The most common intracranial abscess is found within the intracerebral compartment, with about 60% related to middle-ear infection and 20% to frontal sinusitis. In about 10% of cases no pri- mary source of infection can be identified. Owing to their strong connection with sinus and middle-ear disease, most intracerebral abscesses are found within the frontal or temporal lobes, or within the cerebellum. Infection disseminated through the bloodstream from more distant sites may result in multiple abscesses in any part of the brain. Microbiology The most common organisms associated with intracranial ab- scesses are aerobic, anaerobic, and microaerophilic streptococci, Staphylococcus aureus, and Bacteroides. However, up to 25% of cul- ture samples are sterile. Cerebral abscesses associated with otitis media, mastoiditis, and nasal sinusitis usually show a mixed growth of anaerobic and aerobic organisms including anaerobic and microaerophilic streptococci and Bacteroides, Strep. Viridans, and Staph. aureus are also frequently seen. Listeria tend to produce areas of focal cerebritis rather than true abscesses. Abscesses associated with frontoethmoidal sinusitis are usually due to Strep. milleri and Strep. angiosus. With trauma the organism is often Staph. aureus or one of the Enterobacteriaceae. Pathology There are four distinct histological stages in the pathogenesis of intracranial abscesses. Spread of microorganisms, either direct or haematogenous, causes parenchymal damage after occlusion of the small vessels. The initial stage is early cerebritis (days 0–3) char- acterized by a local inflammatory response with perivascular and parenchymal neutrophil infiltration. During late cerebritis (days 3–9) a central necrotic area forms with macrophage infiltration. A reticulin network forms around the necrotic area in the early capsular stage (up to day 14) with parenchymal oedema. This is followed by the formation of a collagen capsule with distinct zones of neovascularization during the late capsular stage (after day 14). Perilesional cerebral oedema continues, and gliosis takes place. The areas of oedematous brain may exert considerable mass effect. In the case of direct spread, infection within an accessory air sinus or the petrous bone may cause an area of localized osteitis just above the dura. This can then spread intracranially. Initially it may be en- tirely confined to the extradural space, but will eventually penetrate the dura and spread subdurally. If the adjacent arachnoid is stuck to the inflamed patch of dura then it may spread into the subarach- noid space to give meningitis. If the subarachnoid space has been obliterated, it may penetrate the brain to produce initially a focal cerebritis. Large intracerebral abscesses can rupture into the ven- tricular system, producing a ventriculitis. Clinical features Presentation depends upon the site, size, and number of lesions, and

the involvement of neighbouring structures such as the cerebral ventricles and the venous sinuses. The signs are, therefore, legion but the diagnosis should be considered in any case where there is an ob- vious primary source of infection associated with evidence of raised intracranial pressure, focal neurological signs, headache, seizures, or meningeal irritation. Extradural abscess may be difficult to detect clinically, but can sometimes manifest as severe, unremitting, localized headache in association with sinusitis or mastoiditis. Patients with subdural empyema frequently appear toxic, with a swinging pyrexia, severe headache, a depressed level of consciousness, contralateral hemipar- esis, papilloedema, meningeal irritation, and seizures. There is often an accompanying frontal sinusitis with tenderness of the forehead, erythema, and swelling of the eyelids. Diagnosis If a brain abscess is suspected, predisposing sources of infec- tion, including possible distant sites, should be carefully sought. Intracranial abscesses derived from haematogenous spread are often more fulminating in their course than those associated with direct spread. CT scans of the skull base, including views of the mastoids and other skull sinuses, should be performed. The investigations of choice for all forms of suspected intracranial abscess are either CT scanning, with and without contrast, or MRI (with and without enhancement). CT will normally demonstrate both extradural and subdural empyema and show intracerebral abscesses as ring-enhancing lesions with low-attenuation centres (Fig. 24.11.3.1). The CT and MRI appearances correlate closely with the pathogenesis. During the early stages of cerebritis areas of hypodensity may be very subtle with minimal contrast enhance- ment. At this stage MRI may reveal oedema and some ring enhance- ment not evident on CT. Subdural empyema may initially be thinly spread over the cerebral cortex, producing relatively little midline shift, appearing virtually isodense with brain on CT. Under such circumstances, contrast-enhanced MRI (particularly with coronal views) is of great value. Diffusion-weighted imaging with MRI can help differentiate a pyogenic abscess from other ring-enhancing le- sions. Proton MR spectroscopy can be used to show peaks of lactate and cytosolic amino acids present in abscesses.

24.11.3 Intracranial abscesses 6099 The principal differential diagnoses in an intracranial abscess are meningitis, subdural haematoma, and intracranial tumour. It is not always possible to differentiate between intracerebral abscess and tu- mour on a CT scan, particularly when there is an appearance of ring enhancement, and it is largely for this reason that the biopsy of sus- pected cerebral tumour is advocated in nearly all such cases. MRI, however, tends to show a low-signal capsule on T2-weighted images and may be helpful in making this differentiation. One obvious concern is to differentiate between bacterial menin- gitis and intracerebral abscess. Both may present with pyrexia, neck stiffness, and some focal signs, but if there is any evidence of raised intracranial pressure, or any other supportive evidence of cerebral abscess, a lumbar puncture should be strictly avoided until a neuro- surgical opinion has been sought. Lumbar puncture in the presence of cerebral abscess can lead to tonsillar or tentorial herniation. The cerebrospinal fluid can be entirely normal in intracranial abscesses. Management Treatment includes identifying and treating the underlying cause and image-guided surgical aspiration or excision of the abscess by craniotomy combined with long-term antibiotics. Selected pa- tients can be managed without surgical intervention. This tends to be limited to those who have multiple or inaccessible abscesses and those in whom surgery is contraindicated due to poor general med- ical condition. However, without biopsy the diagnosis cannot be confirmed and the pathogens cannot be identified. Therefore, tissue should be obtained if at all possible. Early diagnosis and treatment are essential for good outcomes. The damaging effect of abscesses results from parenchymal dis- placement and oedema rather than destruction of tissue, so neurological deficits may be reversible. Treatment is aimed at identifying the causative organism, obtaining antibiotic sensitiv- ities, and

reducing mass effect. Early management includes the fol- lowing steps: culturing blood and any extracranial infective lesion, obtaining tissue and starting appropriate intravenous antibiotics, administration of anticonvulsant agents, and, in cases of grossly depressed level of consciousness and massive cerebral oedema seen on a CT scan, giving intravenous dexamethasone. Steroids decrease cerebral oedema but are also thought to prevent antibiotic penetration. Therefore, they should only be used in patients with mass effect on CT. Pus from the suspected primary site of infection should be col- lected immediately and both aerobic and anaerobic cultures obtained. The intracranial pus must be similarly cultured. High dose intravenous empirical antibiotics should be commenced im- mediately without waiting for the culture report, and subsequently changed in the light of the sensitivity findings. The antimicrobial regimen should include metronidazole and, usually, a β-lactam such as ceftriaxone, depending on the likely source of infection and the infective agent. Meropenem is an alternative. Although antibiotics have good penetration into cerebrospinal fluid, the acidic environ- ment of the abscess means that the efficacy of the antibiotic may be reduced. Intravenous antibiotics should be continued for sev- eral weeks before reverting to oral medication. Infection specialists should be involved in decisions regarding length and nature of anti- biotic treatment. Most supratentorial abscesses can be treated by aspiration through a burr hole. Aspiration is often repeated several times, but in about 30% of cases a single aspiration will suffice. Once the abscess is sterile, the capsule will shrink and finally form an irregular gliotic scar within the brain. Shrinkage of the abscess must be checked by serial CT or MRI scans. Subdural empyema should be evacuated through a craniotomy rather than burr holes, as very frequently the pus can spread widely, particularly alongside the falx cerebri. Extradural empyema is evacuated through a burr hole, or through a craniotomy for larger collections to allow complete excision of the abscess. Advances in image-guided surgery have resulted in far more accurate and safe targeting of all intracranial lesions including ab- scesses and should be used to aid any surgical intervention. Cerebellar abscesses, when diagnosed early, may be aspirated through a burr hole, but immediate total excision is often recom- mended because the small volume of the posterior cranial fossa leaves little latitude in terms of tonsillar herniation and death. Prognosis The mortality rate from intracranial abscesses has been reduced from 40–60% to around 10% since the introduction of CT scanners. The main problems remain those of late diagnosis and resistant bac- teria. Epilepsy and permanent disability still occur in up to 50% of cases. Temporal lobe abscess, subdural empyema, and abscesses that rupture into the ventricles have a worse prognosis. Fungal abscesses can occur in immunosuppressed individuals and carry a much higher mortality rate. FURTHER READING Helweg-Larsen J, et al. (2012). Pyogenic brain abscess, a 5-year survey. BMC Infect Dis, 12, 332. Kao PT, et al. (2003). Brain abscesses: clinical analysis of 53 cases.
J Microbiol Immunol Infect, 36, 129–36. Kastrup O, Wanke I, Maschke M (2008). Neuroimaging of infections of the central nervous system. Semin Neurol, 28, 511–22. Fig. 24.11.3.1 Contrast CT scan showing large frontal cerebral abscess (A) with surrounding oedema (B) and ventricular compression (C).

Revision #1
Created 2026-01-22 16:43:20 UTC by Omar Ayman
Updated 2026-01-22 16:43:20 UTC by Omar Ayman