INFECTIOUS DISORDERS

CAUSES AND MANAGEMENT OF BRAIN ABSCESS

The microbiology and recent developments in the diagnosis and management of brain abscess in children are reviewed from Georgetown University School of Medicine, Washington, DC. The intracranial infection originates from various sites: 1) direct extension from chronic otitis media or sinusitis; 2) contiguous spread from venous thrombophlebitis; 3) infected open fractures or surgical sites; and by 4) hematogenous spread from a distant focus (eg. cyanotic congenital heart disease, lung infection, bronciectasis, dental abscess). Clinically, the child presents with fever, headache, drowsiness, seizures, vomiting, papilledema, ataxia, and hemiparesis. Diagnosis may be delayed for up to 2 weeks in 2/3rds of patients, and longer in abscesses localized to the frontal and parietal lobes. The predominant organisms are *Staphylococcus aureus, Streptococcus pneumoniae, Streptococcus pyogenes,* and alpha-hemolytic streptococci. Lumbar puncture is only considered when a mass and increased intracranial pressure have first been excluded by CT or MRI. Low CSF sugar (<40 mg/dL) occurs in one-third and elevated protein (>40 mg/dL) in two thirds of patients. CSF leukocytes may reach 100,000/mm3 or more when an abscess ruptures. CSF cultures are positive in <10% patients, unless the abscess ruptures. Cultures should include aerobic, anaerobic, fungi, and acid-fast staining. MRI is the preferred imaging method for diagnosis, and a diffusion MRI will differentiate between cerebral tumor, stroke, and abscess. MRI in patients with cerebritis may mimic findings in stroke. Since the advent of MRI, mortality has fallen by 90% and is <5-15%. EEG may show a focus of high voltage slow waves, but is less sensitive and non-specific. Ultrasonography and contrast enhanced CT may detect subdural empyema in infants and differentiate it from subdural effusion. *Treatment.* Antimicrobial therapy and measures to control the increased intracranial pressure are essential in the early stages, before encapsulation has occurred. Once the abscess has formed, surgical excision or drainage with a long course of antibiotics (4-8 weeks) is the treatment of choice. Antibiotics alone may be successful in patients who are stable without increased intracranial pressure, with symptoms for less than 2 weeks, and an abscess <2 cm in diameter. Repeated CT-guided needle aspirations are preferred to complete excision in patients with multiple abscesses or concomitant meningitis. Corticosteroids are controversial: steroids can retard encapsulation, increase necrosis, reduce antibiotic penetration into the abscess, alter CT images, and produce a rebound effect when discontinued. When used to reduce cerebral edema and increased intracranial pressure, steroid therapy of short duration may be life saving. *Outcome* correlates with abscess size and mass effect: those <1.7 cm (range 0.8-2.5 cm) usually respond to antimicrobial therapy alone, while those with an average size of 4.2 cm (range 2.0-6.0 cm) require surgical intervention. Duration of symptoms before diagnosis is also important: those with symptoms <1 week have a more favorable outcome than those with symptoms >1 week. Early antibiotic therapy can prevent progression from cerebritis to formation of abscess and need for surgery, but proper selection of antibiotics, with regard to organism sensitivity and penetration into abscess and CNS, requires expert consultation. (Brook I. Microbiology and management of brain abscesses in

children. **J Pediatr Neurol** July-Sept 2004;2:125-130). (Respond: Itzhak Brook, MD, MSc, 4431 Albemarle St NW, Washington, DC 20016).

COMMENT. The experience of brain abscess at Children's Hospital, Boston, between 1981 and 2000, is reported by Goodkin et al. 2004 (see **Ped Neur Briefs** June 2004;18:43-44). Congental heart disease was the most common predisposing factor. In post-1981 compared to pre-1981 cases (n=55 cf 94), the annual rate of abscess was similar, those associated with otitic or sinus infection had decreased in frequency, abscess in infants was more common, acute immunosuppressive disease was a more frequent predisposing factor, treatment with antibiotics alone had become successful in >20% cases, but mortality rate had shown no significant change (24% vs 27%). Despite improvements in diagnosis using neuroimaging, brain abscess continues to result in high rates of neurologic impairment and death. A greater awareness of presenting symptoms and predisposing factors and earlier diagnosis may be expected to improve outcome in the future. New-onset headache and seizure in a child with congenital heart disease or recent ear or sinus infection, and especially a positive culture for *Streptococcus milleri (S intermedius)*, or fungal disease in an acutely immunosuppressed patient, should alert the physician to the diagnosis of cerebral abscess.

SEIZURES COMPLICATING BACTERIAL MENINGITIS

The clinical data of 116 patients, 1 month to <5 years of age, admitted for bacterial meningitis, and grouped according to those with and without seizures during hospitalization, were compared in a study at Buddhist Dalin Tzu Chi General Hospital, Chang Gung Memorial Hospital and other centers in Taiwan. Fifty-five (47%) had seizures in the acute phase, especially prevalent in the older infants, and 12 of these had one or more afebrile seizures after completing treatment for acute meningitis. Seizures occurring 2 weeks after treatment were associated with hydrocephalus, subdural empyema, or cerebral infarction. Seizure prevalence was high in meningitis caused by *E coli, Salmonella species, S agalactiae, Hemophilus species, and N meningitides.* At 1-year follow-up, 26 had good outcomes and in 29 the outcome was poor. In those who had seizures in the acute phase, the outcome was worse than those without. Neuroimaging abnormalities correlated with the occurrence of seizures during hospitalization for meningitis. Factors that increased the risk of seizures included disturbed consciousness on admission, abnormal neuroimaging, and low glucose and elevated CSF protein. (Chang C-J, Chang H-W, Chang W-N, et al. Seizures complicating infantile and childhood bacterial meningitis. **Pediatr Neurol** Sept 2004;31:165-171). (Respond: Dr Cheng-Hsien Lu MD, MSc, Department of Neurology, Chang Gung Memorial Hospital, 123, Ta Pei Road, Niao Sung Hsiang, Kaohsiung Hsien, Taiwan).

COMMENT. Seizures are common during hospitalization in the child with bacterial meningitis, and the occurrence of seizures during the acute phase is strongly correlated with late seizures, especially during the first 5 years of follow-up. Treatment of neurologic complications and aggressive antimicrobial therapy are essential for optimal outcome.

Afebrile seizures provoked by minor infections carry a 5.7% risk of subsequent unprovoked afebrile seizures by 5 year follow-up in a study at National Neuroscience Institute, Singapore (Lee W-L, Ong H-T. **Pediatr Neurol** 2004;31:157-164).