INFECTIOUS DISORDERS

SPINAL AND INTRACRANIAL EPIDURAL ABSCESS

Presentation, epidemiology, diagnosis and treatment of spinal epidural abscess (SEA) and intracranial epidural abscess (ICEA) are reviewed by researchers at The John's Hopkins University School of Medicine, Baltimore, MD, and Universidad de Santander, Columbia. Risk factors for SEA have increased in frequency and include injected-drug use, diabetes mellitus, invasive spinal procedures, spinal trauma, immunosuppression, skin infections, and bacteremia. The most common risk factor for ICEA is frontal sinusitis; 60-90% of cases are associated with otitis or sinusitis. Other factors include post-traumatic infections, nasal or mastoid surgical procedures, and congenital defects of the anterior cranial fossa. Gram-positive cocci, including Staphylococcus and Streptococcus are the most common causes of SEAs, with Staph aureus involved in 50-66% of cases. Mycobacterium tuberculosis is common in some geographic areas. Pseudomonas species are isolated from injected drug users with SEA. ICEAs are polymicrobial in origin, most commonly anaerobic gram-positive cocci, Staph and Strep spp (Strep anginosis) and gram-negative bacilli. CT and MRI are the preferred diagnostic tests. Medical and surgical treatments are reviewed in detail. Morbidity and mortality from SEA are high, especially in developing countries. Early diagnosis, specific microbiologic identification and prompt antimicrobial therapy can improve prognosis. In addition to broad-spectrum antibiotics, surgery is usually required in treatment of ICEAs. (Pradilla G, Hsu W, Rigamonti D, Pradilla Ardilla G. Epidural abscesses of the CNS. **Lancet Neurol** March 2009;8:292-300). (Respond: Daniele Rigamonti MD, The Johns Hopkins Hospital, Phipps Building, Room 104, 600 North Wolfe Street, Baltimore, MD 21287. E-mail: dr@jhmi.edu).

COMMENT. Prevalence of SEA although rare has increased, as a result of injected-drug users, while that of ICEA has decreased, following the introduction of more effective antimicrobial treatments. Prognosis is often poor due to delayed diagnosis. An awareness of the common risk factors leads to early recognition and prompt antimicrobial therapy. Most common causative factors are injected-drug use, immunosuppression and spinal surgical procedures in patients with SEA, and frontal sinusitis in ICEA.

SEIZURE DISORDERS

VISUAL FIELDS IN MOTHERS AND CHILDREN EXPOSED IN UTERO TO VIGABATRIN

Three mothers with 4 children exposed to vigabatrin in utero (but not breast fed) underwent perimetry and imaging of the retinal nerve fiber layer (RNFL) at the University Hospital of Wales and School of Optometry, Cardiff, UK. Two mothers showed vigabatrin-attributed visual loss and an abnormally attenuated RNFL. The third had an upper left quadrantanopia, consistent with previous temporal lobe surgery, and a normal RNFL. All four children, ages 6, 10, 15 and 18 years, had normal visual fields and RNFL thickness. Estimates of the in utero exposure to vigabatrin varied from 600 to 1410 mg/kg/day with a

mean of 1100. Children exposed pre-natally may be spared the visual toxicity of vigabatrin. (Lawthom C, Smith PEM, Wild JM. In utero exposure to vigabatrin: no indication of visual field loss. **Epilepsia** Feb 2009;50:318-321). (Respond: Dr John Wild, Cardiff School of Optometry and Vision Sciences, Cardiff University, Maindy Road, Cardiff CF24 4LU, Wales, UK. E-mail: wildjm@cardiff.ac.uk).

COMMENT. Vigabatrin-induced visual field loss manifests as a bilateral concentric constriction. It occurs in 30% of patients treated, and the incidence increases with duration and extent of exposure to the drug. Visual field perimetry examination is unsatisfactory under 9 years of age, but attenuation of the retinal nerve fiber layer thickness, estimated by optical coherence tomography, is a sensitive and specific test for vigabatrin toxicity.(Wild et al, 2006) Unlike the temporal quadrant atrophy seen in optic neuritis, vigabatrin toxicity is characterized by nasal quadrant constriction while the temporal quadrant is spared. Children exposed to vigabatrin by placental transfer only, in a dose 10 times that given to infants with infantile spasms, appear to be spared the visual field defect. Infants exposed after 6 months of age are 2.5 times more likely to show vigabatrin toxicity compared with those exposed before 6 months of age (Westall et al, 2007; cited by Lawthom et al).

Vigabatrin-induced visual field loss and age of exposure. Visual fields of 16 children treated with vigabatrin for infantile spasms were examined by Goldmann kinetic perimetry at age 6-12 years, in a study at Helsinki University Central Hospital, Finland (Gaily E et al. **Epilepsia** Feb 2009;50:206-216). Vigabatrin was started at a mean of 7.6 (range, 3.2-20.3) months. Mean duration of therapy was 21 months. Visual fields were normal in 15 children; a mild visual field loss occurred in one child who was treated with vigabatrin for 19 months. Children treated in infancy are less susceptible to vigabatrin-induced visual field loss than patients treated at a later age.

SEIZURE-INDUCED BRAIN DAMAGE IN THE NEONATE

The pathophysiology of neonatal seizures, and evidence for seizure-induced brain damage are reviewed by researchers from Montreal Children's Hospital, and Universite de Montreal, Quebec, Canada. Electrographically documented seizures with or without clinical manifestations are the most accurate concept of neonatal seizures. Incidence is 1.5-3.5 per 1000 live births, varying with risk factors such as low birth weight, prematurity, perinatal complications, and NICU availability. Etiology is the major determinant of outcome, but the seizure itself may be a factor. In animal models, neonatal seizures impair cognition, alter behavior, increase anxiety, and are associated with epileptogenesis. Clinically, the reported prevalence of epilepsy and abnormal neurodevelopment after neonatal seizures varies, ranging from 6.5% to 56% for epilepsy and from 19% to 67% for neurological abnormalities. Electrographic neonatal seizures, with or without clinical manifestations, correlate with increased morbidity and mortality. Risk factors for epilepsy include diffuse abnormalities on cranial imaging (more than focal), prolonged use of anticonvulsants, poor response of neonatal seizures to phenobarbital, abnormal EEG background, and acquired CNS infections. The efficacy of both phenobarbital and phenytoin in neonatal seizures is 50%, and no randomized controlled trial is reported to show improvement in neurodevelopmental outcome or prevention of epilepsy. Controlled trials of newer anticonvulsants and neuroprotective