

seizures and FSE, with durations of 43, 45, and 60 minutes. CSF findings were normal and bacterial cultures were negative. A child aged 3 months to 5 years who presents with a first or recurrent FS should be considered for LP if one or more of the following indications are present: neurologic signs of meningitis, systemic signs of toxicity, complex seizure with prolonged postictal obtundation of consciousness, or pretreatment with antibiotics. Complex FS alone is not an absolute indication for LP.

In a retrospective study at Children's Hospital Boston to assess the rate of acute bacterial meningitis among 526 children who present with their first complex febrile seizure, 2.7% had CSF pleocytosis and 3 patients (0.9%) had acute bacterial meningitis. One appeared well clinically; of 2 with *Streptococcus pneumoniae* cultured from CSF, 1 was nonresponsive clinically, and the other had a bulging fontanel and apnea. (Kimia A, et al. **Pediatrics** 2010 Jul;126(1):62-9).

THALAMOCORTICAL STRUCTURAL AND FUNCTIONAL CONNECTIVITY IN JUVENILE MYOCLONIC EPILEPSY

Researchers at King's College, Institute of Psychiatry, London and other centers in the UK, US, and Germany discovered changes in an anterior thalamo-cortical bundle during tests of structural connectivity, as measured by diffusion tensor imaging, in a cohort of 28 subjects with juvenile myoclonic epilepsy. An alteration in task-modulated connectivity was detected in a region of frontal cortex connected to the thalamus via the same anatomical bundle, and overlapping with the supplementary motor area. In patients with active seizures, the degree of abnormal connectivity is related to disease severity in those with active seizures. These results point to abnormalities in a specific thalamo-cortical circuit, with reduced structural and task-induced functional connectivity that underlies this idiopathic epilepsy. (O'Muircheartaigh J, Vollmar C, Barker GJ, et al. Abnormal thalamocortical structural and functional connectivity in juvenile myoclonic epilepsy. **Brain** 2012 Dec;135(Pt 12):3635-44). (Response: Dr Mark P Richardson, E-mail: mark.richardson@kcl.ac.uk).

COMMENT. The characteristic generalized spike and wave discharges in the EEG of juvenile myoclonic epilepsy implicate thalamo-cortical interactions, and the discharges are most prominent in frontal regions. The functional and diffusion MRI and diffusion tensor imaging used above provide anatomic evidence for the role of the thalamus and a specific thalamo-cortical circuit dysfunction in JME. JME is a lifelong disorder and a structural cerebral defect may explain the necessity to continue treatment indefinitely. (Wandschneider B, et al. Frontal lobe function and structure in juvenile myoclonic epilepsy: a comprehensive review of neuropsychological and imaging data. **Epilepsia** 2012 Dec;53(12):2091-8).

MUSCULAR DYSTROPHY-DYSTROGLYCANOPATHY AND EPILEPSY

Investigators from the University of Catania, and other centers in Europe have identified a novel genetic glycosylation disorder, DPM2-CDG (part of the DPM synthase complex) in 3 infants with severe hypotonia, progressive muscle weakness and wasting, elevated CK, absent psychomotor development, intractable epilepsy with onset at 1 week

to 5 months, and early mortality. (Barone R, Aiello C, Race V, et al. DPM2-CDG: a muscular dystrophy-dystroglycanopathy syndrome with severe epilepsy. **Ann Neurol** 2012 Oct;72(4):550-8). (Respond: Dr Dirk J Lefeber. E-mail: D.Lefeber@neuro.umcn.nl or Dr Gert Matthijs. E-mail: Gert.Matthijs@uzleuven.be).

COMMENT. Serum N-glycosylation screening and/or enzyme analysis of DPM synthase are recommended in the workup of infants born with unsolved dystroglycanopathies.

INFECTIOUS DISORDERS

SUBDURAL EMPYEMA IN BACTERIAL MENINGITIS

Researchers at the University of Amsterdam, the Netherlands, evaluated the occurrence, treatment, and outcome of subdural empyema as a complication of community-acquired bacterial meningitis in 28 (2.7%) adults. Predisposing conditions in 26 (93%) patients included spread of otitis or sinusitis to the subdural space in 21 (75%). Presenting symptoms in 23 patients (82%) were neurologic and consisted of paresis, focal seizures, and dysesthesia contralateral to the empyema. The organism cultured from the CSF was *Streptococcus pneumoniae* in 26 patients (93%) and *Streptococcus pyogenes* in 1 (3%). One patient had negative CSF cultures. Complications leading to an unfavorable outcome in 68% cases were seizures (50%), focal neurological abnormalities (54%), and hearing impairment (39%). Five patients with empyema causing midline shift were treated by neurosurgical evacuation of the empyema. (Jim KK, Brouwer MC, van der Ende A, van de Beek D. Subdural empyema in bacterial meningitis. **Neurology** 2012 Nov 20;79(21):2133-9). (Response and reprints: Dr van de Beek, E-mail: d.vandebeek@amc.uva.nl).

COMMENT. Symptoms or signs indicative of subdural empyema in adults with meningitis are otitis or sinusitis, focal neurologic deficits, or seizures. In patients suspected of having developed subdural empyema, the diagnosis was confirmed by MRI with diffusion-weighted imaging. Lumbar puncture may be associated with a risk of brain shift and sudden clinical deterioration and requires careful monitoring.

In a pediatric study of intracranial empyema at the University of Paris Descartes, 33 of 38 patients presented with subdural empyema and 5 with extradural empyema. Ten were infants <1 year of age, all related to bacterial meningitis, and 28 were children mainly associated with otitis or sinusitis infections. In children with subdural empyema, factors associated with poor prognosis were neurological deficit and cerebral herniation on admission CT scan. (Legrand M, et al. **Eur J Pediatr** 2009 Oct;168(10):1235-41).

BRAIN ABSCESS FROM A PERITONSILLAR ABSCESS

Researchers at Louisiana State University, Shreveport, LA, report the case of a 9-year-old immunocompetent girl diagnosed with a left frontal brain abscess accompanied by fever, headache, and weight loss for a 3-month period. A left-sided peritonsillar abscess was the presumptive source of the brain abscess. A review of the literature