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VASCULAR DISORDERS

ENDOVASCULAR TREATMENT OF CEREBRAL VENOUS SINUS THROMBOSIS

Investigators at Frenchay Hospital, Bristol, UK, retrospectively reviewed case notes and imaging in 9 consecutive children (age range 18 months to 16 years) with cerebral venous sinus thrombosis (CVST) who were treated using endovascular methods after medical therapy with heparin had failed. Six had superior sagittal sinus and transverse sinus involvement, 6 had straight sinus and 5 had vein of Galen and internal cerebral vein involvement. Three had preprocedural parenchymal hemorrhage and 6 showed edema/venous infarction. Predisposing conditions were anemia, diarrhea, and vomiting, nephrotic syndrome, and hypoplastic left heart; none was identified in 5 children. A thrombolytic agent (rtPA) was used in 8 patients. Diagnosis of CVST was made by CT, CT venography, MRI, or MR venography. Seven children were comatose, one had raised intracranial pressure with progressive cranial nerve palsies, 5 had suffered hemiparesis, 3 had suffered seizures, and one had a fluctuating hemiparesis at time of endovascular treatment. Endovascular methods used included local tissue thrombolytic plasminogen activator (in 8 patients), microguidewire and catheter disruption (6 patients), balloon angioplasty (in 2), and thromboaspiration using the Penumbra mechanical thrombectomy device (in 4). Partial recanalization was achieved in all and excellent recanalization in 2 patients. Good functional outcomes were obtained in 8 (89%). One child died with uncontrolled intracranial venous hypertension. Endovascular therapy may have a role in treatment of CVST in children when conventional medical therapy has failed and outcome is poor and deteriorating. (Mortimer AM, Bradley MD, O'Leary S, Renowden SA. Endovascular treatment of children with cerebral venous sinus thrombosis: A case series. *Pediatr Neurol* 2013 Nov;49(5):305-12). (Response: Dr Mortimer, Dept. of Neuroradiology, Frenchay Hospital, Bristol, UK. E-mail: alex_mortimer@hotmail.com).

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COMMENT. Children with CVST and severe neurological deterioration despite anticoagulation may have a favorable response to endovascular treatment. Clinical deterioration after medical treatment is an indication for endovascular therapy. Larger scale studies are required to establish the role of endovascular treatment of deteriorating cases of pediatric CVST. Indicators of a poor prognosis are coma, involvement of the deep venous system, and parenchymal hemorrhage. Anticoagulation may be associated with an increase in size of a hematoma or de novo hemorrhage (Moharir MD, Shroff M, Stephens D, et al. Anticoagulants in pediatric cerebral sinovenous thrombosis: a safety and outcome study. **Ann Neurol** 2010 May;67(5):590-9).

Stroke therapy for children is discussed in an editorial (Roach ES. **Pediatr Neurol** 2013 Nov;49(5):301-2). Until age-specific studies are available, pediatric neurologists must accept methods and results of trials in adults. Thrombolysis with tPA in adults must be administered within 4.5 hours of symptom onset for a favorable risk-benefit ratio to be maintained. Administration of tPA after this time increases the risk of hemorrhage. Use of tPA in children is not approved by the FDA, but a trial is now underway (Amlie-Lefond C, et al. Thrombolysis in acute childhood stroke: design and challenges of the thrombolysis in pediatric stroke clinical trial. **Neuroepidemiology** 2009;32(4):279-86).

ENCEPHALITIS AND ENCEPHALOPATHIES

DIAGNOSTIC ALGORITHM FOR ENCEPHALITIS

Diagnostic algorithm for initial evaluation of encephalitis in children is proposed with a consensus statement from the International Encephalitis Consortium, a committee begun in 2010 to serve as a practical aid to clinicians evaluating patients with suspected encephalitis. *Encephalitis is defined as inflammation of the brain parenchyma associated with neurologic dysfunction.* Major diagnostic criterion is an altered mental status lasting >24 hours. Minor criteria include fever, seizures, focal neurologic findings, CSF WBC count >5/cubic mm, MRI parenchymal lesion, or EEG abnormality indicative of encephalitis (>3 required for probable or confirmed encephalitis).

Routine studies proposed include CSF, serum, imaging (MRI preferred), EEG, throat sample for *Mycoplasma pneumoniae* PCR, and throat and stool specimens for enterovirus PCR. Specific signs and symptoms of encephalitis include abnormal behavior, psychotic features, seizures or movement disorder indicating need for NMDAR antibody test in serum and CSF; vesicular rash indicating VZV PCR from CSF; respiratory symptoms indicating *Mycoplasma pneumoniae* PCR in CSF; and limbic symptoms indicating autoimmune limbic encephalitis testing HHV6/7 PCR (CSF). (Venkatesan A, Tunkel AR, Bloch KC, et al. Case definitions, diagnostic algorithms, and priorities in encephalitis: Consensus statement of the International Encephalitis Consortium. **Clin Infect Dis** 2013 Oct;57(8):1114-28). (Response: Ann Venkatesan MD, PhD, Johns Hopkins University School of Medicine, 600 N Wolfe St, Baltimore, MD 21287. E-mail: avenkat2@jhmi.edu).

COMMENT. The definition proposed is chosen to capture both encephalitis and encephalopathy. Encephalopathy is a clinical state of altered mental status, manifesting as