INHERITED PROTHROMBOTIC RISK FACTORS IN MIGRAINE, STROKE, OR TRANSIENT ISCHEMIC ATTACK

The prevalence and association of inherited prothrombotic risk factors in children with established diagnoses of stroke, transient ischemic attack, or migraine were studied at Zagreb University School of Medicine, Croatia. Genotypic analyses were performed for factor V G1691A, factor II G2010A, MTHFR C677T, and 4 common platelet glycoprotein polymorphisms. Only factor V was significantly associated with increased risk for arterial ischemic stroke (AIS) in childhood and perinatal arterial ischemic stroke (PAS). Heterozygosity for factor V G1691A was associated with a 7-fold increased risk for AIS, PAS, and transient ischemic attack (TIA). Carriers of human platelet alloantigens had an increased risk of TIA. Human platelet alloantigen-2b allele was associated with a 2.23-fold increased risk for migraine, whereas factors V and II were not implicated. A trend toward an increased risk for migraine in children homozygous for MTHFR C677T, especially migraine with aura, was not statistically significant. (Herak DC, Antolic MR, Krleza JL, et al. Inherited prothrombotic risk factors in children with stroke, transient ischemic attack, or migraine. Pediatrics April 2009;123:e653-e660). (Respond: Renata Zadro PhD, Clinical Hospital Center, Kispaticeva 12, Zagreb 10000, Croatia. E-mail: rzadro@mef.hr).

COMMENT. Factor V G1691A is important in susceptibility to arterial ischemic stroke in childhood and the perinatal period, and transient ischemic attacks. Platelet glycoprotein polymorphisms may increase the risk of TIA and migraine.

INFECTIOUS DISORDERS

COMPARISON OF SENSITIVITY OF SERUM AND CSF SAMPLES IN IMMUNODIAGNOSIS OF NEUROCYSTICERCOSIS

Paired serum and CSF samples were obtained from 91 patients with neurocysticercosis (NCC) for detection of Taenia solium (TS) antibodies and antigens, in a study at centers in Lima, Peru, Belgium, and the USA. TS antibodies were detected using an enzyme-linked immunotransfer blot (EITB) assay, and antigens, using a monoclonal antibody-based enzyme-linked immunosorbent assay (ELISA). NCC was intraparenchymal in 48 and extraparenchymal in 43 patients. For the intraparenchymal NCC group, the EITB antibody assay yielded more true positive results on serum samples, whereas the ELISA antigen assay yielded slightly more positive results for CSF samples (differences not significant). Patients with calcified NCC were antibody positive and antigen negative. For extraparencymal disease, all samples were antibody positive, and all but 2 were antigen positive, mostly with high antigen levels. (Rodriguez S, Dorny P, Tsang VCW, et al, for the Cysticercosis Working Group in Peru. Detection of *Taenia solium* antigens and anti-T. solium antibodies in paired serum and cerebrospinal fluid samples from patients with intraparenchymal or extraparenchymal neurocysticercosis. J Infect Dis May 2009;199:1345-1352) (Respond or reprints: Dr Hector H Garcia, Dept of Microbiology, Universidad Peruana Cayetano Heredia, Av H Delgado 430, SMP, Lima 31, Peru. E-mail: hgarcia@jhsph.edu).

COMMENT. The authors conclude that the EITB antibody-detecting assay is equally sensitive on serum and CSF samples. The ELISA assay for antigen detection is more sensitive when performed on CSF samples than serum, but less sensitive than the EITB assay. ELISA, using either serum or CSF samples, is better than EITB in the differentiation of active and inactive NCC. High antigen levels detected by ELISA suggest the presence of subarachnoid NCC, associated with a worse prognosis.

Since neuroimaging is often nonspecific for NCC, immunodiagnosis is usually necessary for confirmation. CSF samples offer no advantage over serum for detection of antibodies by EITB assay. In intraparenchymal cases, although the use of CSF samples for antigen detection by ELISA assay may yield a 13% increase in case identification, the increase over serum samples is not significant. With extraparenchymal disease, most patients are strongly seropositive by EITB assay on either serum or CSF.

The findings in the above study suggest that serum antibody detection by an EITB assay, using purified antigen, is the assay of choice for diagnosis of NCC.

ANTI-N-METHYL-D-ASPARTATE RECEPTOR ENCEPHALITIS WITH CATATONIA TREATED BY PLASMAPHERESIS

The case of a 12-year-old girl with anti-methyl-D-aspartate receptor (NMDAR) encephalitis is reported by researchers from Augsburg and Bonn, Germany, and Oxford, UK. She was admitted with an episode of paresthesia and hypotonia of the left leg, and rare episodes of head turning with clonic movements of the left and all extremities. The symptoms were preceded by a 2-day episode of diarrhea 3 weeks before. The EEG, brain MRI, and CSF were normal, and she was discharged with a suspected diagnosis of psychogenic seizures. She was readmitted 2 days later with agitation, hyperventilation and intermittent ocular deviation, followed by intermittent catatonic postures with fever, hyperidrosis, chewing movements and tongue protrusion. She required tube feeding and intermittent oxygen. EEG now showed continuous slowing. The MRI was unremarkable. IgM antibodies against Campylobacter jejuni were elevated. Prednisolone was ineffective. At 6 weeks after admission, CSF showed IgG antibody reactivity with hippocampal neurophil, and subsequently, serum antibodies to NMDAR were demonstrated. Plasmapheresis was followed within 2 weeks by speaking words, walking and almost full recovery in 4 weeks. CSF antibody reactivity was no longer detected, and ultrasound and CT were negative for teratoma in the pelvis and mediastinum. (Schimmel M, Bien CG, Vincent A, Schenk W, Penzien J. Successful treatment of anti-N-methyl-D-aspartate receptor encephalitis presenting with catatonia. Arch Dis Child April 2009;94:314-316). (Respond: Dr Johannes Penzien, Department of Paediatrics, Klinikum Augsburg, Stenglinstrasse 2, 86156 Augsburg, Germany. E-mail: johann.penzien@klinikum.augsburg.de).

COMMENT. This 12-year-old girl appears to be the youngest patient reported with anti-NMDAR encephalitis. Her presentation with neuropsychiatric symptoms progressing to seizures, catatonia, autonomic dysfunction, hypoventilation, orofacial dyskinesia and dysphagia is typical. Teratoma, absent in this child, may occur in 65% of patients, and sometimes develops 7 years following encephalitis (Dalmau J et al. **Lancet Neurol** 2008;7:327-340)(Iizuka T et al. **Neurology** 2008;70:504-511). An apparent response to plasmapheresis and the association with *Campylobacter* infection are of interest.