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SEIZURE DISORDERS

CYTOKINES, VIRAL INFECTION AND FEBRILE SEIZURES

Immune responses to a common viral factor, double-stranged ribonucleic acid (dsRNA), were examined in children with and without a history of febrile seizures (FS), in a study at Saga University, Japan. The blood levels of IL-1B and interferon alpha (IFN-a) cytokines were measured using immunosorbent assays at least 4 weeks after the last febrile episode. IL-1B production was significantly increased in a group of 27 FS patients compared to 18 control children. The levels of IL-1B were 703.7 +/- 1318.4 pg/mL in the FS group cf 94.5 +/- 112.1 pg/mL for controls (P=0.0007). IL-1B production was not significantly different in 9 patients with a single prior FS compared to 18 with multiple FS. IL-1B levels of 3 patients with prolonged FS were 567.1, 206.0, and 20.6 pg/mL, respectively. IFN-a levels were low, often undetectable, in both FS and control groups; controls had significantly higher levels than FS patients (5.5 +/- 4.6 pg/mL cf 1.28 +/- 3.2 pg/mL, P=0.0009). Genotyping of IL-1B and IL-1 receptor antagonist polymorphisms showed no significant differences in allelic distribution among FS patients and controls, IL-1B production was not influenced by genotype. (Matsuo M, Sasaki K, Ichimaru T et al. Increased IL-1B production from dsRNA-stimulated leukocytes in febrile seizures. Pediatr Neurol August 2006;35:102-106), (Respond: Dr Marsuo, Department of Pediatrics, Faculty of Medicine, Saga University, 5-1-1 Nabeshima, Saga, 849-8501, Japan).

COMMENT. Viral infections play a role in the etiology of febrile seizures by more than one possible mechanism: (1) fever per se; (2) a degree of fever that exceeds the individual threshold convulsive temperature; (3) viral neurotropism or viral reactivation; and (4) an elevated cytokine or abnormal immune response to infection. Fever is the essential factor, and the threshold to FS is dependent on the height of the body temperature, not the rate of temperature rise.

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Fever induced by infection is regulated by components of the immune response, particularly the proinflammatory cytokines, interleukin-1B (IL-1B), IL-6, and tumor necrosis factor (TNF)-alpha, and the anti-inflammatory cytokine IL-10. Several recent studies have examined the levels of production of various components of the immune response in febrile children with and without seizures, with varying results. Despite some negative findings, most studies favor a cytokine role in febrile seizures. Serum levels of IL-6 and IL-10 are useful indicators of the severity of influenza-associated febrile seizures (Kawada J-1 et al. J Infect Dis 2003;188:690-698; Millichap JG, Millichap JJ. J Infect Dis 2004;189:564-5; and idem Pediatr Neurol Sept 2006;35:165-172).

ELECTROCLINICAL FEATURES OF ABSENCE EPILEPSY

Clinical and EEG features of absence seizures in 47 children with newly diagnosed, untreated childhood absence epilepsy (CAE) were analyzed using video-EEG recordings, in a study at University of Otago, Wellington, New Zealand; British Columbia Children's Hospital, Vancouver, BC, Canada: and Royal Children's Hospital, Melbourne, Australia, CAE was defined as daily typical absence seizures presenting between the ages of 2 and 10 years, and without other seizure types. In 339 absence seizures analyzed, the average seizure duration was 9.4 seconds (range 1 to 44 sec). Clinical features included arrest of activity, loss of awareness, staring (upward in 33% patients, lateral in 28), and 3-Hz eve blinking. Eve opening occurred in 59% of seizures, when eves were initially closed. Automatisms, predominantly oral, were seen in 41% of seizures and 81% of patients. Hyperventilation induced seizures in 83% of patients. Ictal EEG showed regular 3-Hz generalized spike and wave (GSW), with one or two spikes per wave, and postictal slowing. Interictal EEG showed GSW fragments (brief, <2 seconds, epileptiform discharges without clinical seizures), posterior bilateral delta activity (notched in 40% cases), and focal discharges. The recently proposed ILAE criteria for CAE were fulfilled by only 5 of the 47 (11%) unselected patients in this study that used 1989 ILAE criteria. The authors conclude that the newly proposed criteria will be of limited value in the diagnosis of CAE and prediction of prognosis. (Sadleir LG, Farrell K, Smith S, Connolly MB, Scheffer LE, Electroclinical features of absence seizures in childhood absence epilepsy. Neurology August (1 of 2) 2006;67:413-418). (Reprints: Dr Lynette Grant Sadleir, Department of Paediatrics, Wellington School of Medicine and Health Sciences, University of Otago, PO Box 7343, Wellington, South New Zealand).

COMMENT. The clinical and EEG features of childhood absence epilepsy are heterogeneous, and the less restrictive 1989 ILAE diagnostic criteria used in the present study are of greater practical value than the criteria proposed in 2000. The more recent criteria restrict the age to 4 to 10 years, seizure duration >4 sec, no myoclonic features, no photic-induced seizures, <3 spikes per slow wave, and no disorganized discharges. CAE is defined as frequent daily absence seizures in otherwise normal school age children with EEG showing bilateral synchronous symmetric 3-Hz spike-wave and normal background activity (ILAE Classification. Epilepsia 1989;30:389-399). The majority of children with CAE that fulfill these criteria have a good prognosis; 60% do not develop generalized tonic clonic seizures, and 95% outgrow their absence seizures (Loiseau P, Panayiotopoulos C. 2002).