

of chest, abdomen and pelvis revealed a 1.9 cm right ovarian dermoid and mycoplasma pneumonia opacities in the lungs. Paraneoplastic limbic encephalitis was suspected, and recovery followed removal of the teratoma and 5 plasmapheresis treatments. (Reid DK, Clardy SL. Anti-NMDA-receptor encephalitis: unusual presentation of an uncommon condition. **J Neurol Neurosurg Psychiatry** 2013 Jan;84(1):69-70). (Response: Dr Stacey L Clardy, Penn State MS Hershey Medical Center, Department of Neurology, EC037, PO Box 859, Hershey, PA 17033. E-mail: staceyllynncardy@yahoo.com).

COMMENT. The authors considered this case to be a less severe phenotype in the clinical spectrum of anti-NMDA-receptor encephalitis. They refer to cases presenting with new-onset epilepsy and psychosis. (Niehusmann P, Dalmau J, Rudlowski C, et al. **Arch Neurol** 2009 Apr;66(4):458-64).

INFANTILE SEIZURES

***PRRT2*, INFANTILE CONVULSIONS, PAROXYSMAL DYSKINESIA, AND MIGRAINE**

Researchers at the Institut de Neurobiologie de la Mediterranee (INMED), Marseille, and other centers in France have extended the spectrum of PRRT2 mutations and phenotypes to hemiplegic migraine and other types of migraine. Previously, they and others had identified PRRT2 (proline-rich-transmembrane protein) as the gene causing infantile convulsions with paroxysmal kinesigenic dyskinesia syndrome (IC/PKD). Thirty-four additional families with either typical IC/PKD or IC/PKD with migraine were analyzed, and 2 known and 2 novel PRRT2 mutations were detected in 18 families. The proportion of migraineurs among PRRT2 mutation carriers (10/37) was significantly increased as compared with the overall migraine prevalence (~12%)($p=0.02$). (Cloarec R, Bruneau N, Rudolf G, et al. PRRT2 links infantile convulsions and paroxysmal dyskinesia with migraine. **Neurology** 2012 Nov 20;79(21):2097-103). (Response and reprints: Dr Pierre Szepetowski. E-mail: szepetowski@inmed.univ-mrs.fr).

COMMENT. No less than 6 articles and one editorial in the Nov 20, 2012 issue of *Neurology* are devoted to PRRT2 gene mutations and their link to infantile convulsions, paroxysmal dyskinesia, ataxia, and hemiplegic migraine. PRRT2 gene mutations are also occasionally reported in patients with febrile seizures, febrile seizures plus, and absence epilepsy (Scheffer IE, et al. PRRT2 phenotypic spectrum includes sporadic and fever-related infantile seizures. **Neurology** 2012 Nov 20;79(21):2104-8).

The increasing number of reports on phenotypes associated with PRRT2 mutations emphasizes the role of non-ion channel genes in the pathogenesis of various paroxysmal neurologic disorders. (Guerrini R, Mink JW. Editorial. **Neurology** 2012 Nov 20;79(21):2086-8). Confirmation of PRRT2 mutations in patients with infantile seizures, paroxysmal dyskinesia, or both provides reassurance that the seizures are likely to be benign and self-limited but the risk of dyskinesia during childhood is increased. An increased risk of PRRT2-related hemiplegic migraine, migraine, or episodic ataxia requires further study.