

VASCULAR DISORDERS

PREDICTORS OF PERINATAL HEMORRHAGIC STROKE

The prevalence and predictors of perinatal hemorrhagic stroke were determined in a case-control study of infants born from 1993 to 2003 in the Northern California Kaiser Permanente Medical Care Program, Oakland, CA, and reported from the University of California, San Francisco, CA. Among 323,532 live births, 20 cases of perinatal hemorrhagic stroke were identified of which 19 were intracerebral hemorrhage and 1 subarachnoid hemorrhage. Three (15%) were premature (31 weeks gestation), and 5 (25%) postmature; birth weights were <2500g in 3 (15%) and >4000g in 3. The prevalence of perinatal hemorrhagic stroke was 6.2 in 100,000 live births, or 1 in 16,000 live births. In comparison, 93 cases of perinatal arterial ischemic stroke were identified, a prevalence of 29 in 100,000 or 1 in 3500 live births. Cases of hemorrhagic stroke presented with encephalopathy (100%) and seizures (65%). The etiology was idiopathic in 15 (75%), thrombocytopenia in 4, and cavernous malformation in 1. Neuroimaging abnormalities were unifocal in 14 of 19 (74%) and unilateral in 15 (83%). Lesions were left-sided more than right-sided, and parietal and frontal more than temporal in location. Univariate predictors of hemorrhagic stroke were male gender, fetal distress, emergent cesarean delivery, prematurity, and postmaturity but not birth weight or difficult vaginal delivery; 49% of infants delivered by emergent cesarean had fetal distress, compared with 4.3% infants born by vaginal delivery ($P<.0001$). Fetal distress and postmaturity were independent predictors. (Armstrong-Well J, Johnston SC, Wu YW, Sidney S, Fullerton HJ. Prevalence and predictors of perinatal hemorrhagic stroke: results from the Kaiser Pediatric Stroke Study. **Pediatrics** March 2009;123:823-828). (Respond: Heather J Fullerton MD MAS, University of California, Department of Neurology, Box 0114, 505 Parnassus Ave, San Francisco, CA 94143. E-mail: fullertonh@neuropeds.ucsf.edu).

COMMENT. Published reports classify (Kirton A et al. **Pediatr Neurol** 2009;40:205-214) and define the prevalence and risk factors (Lee J et al. **JAMA** 2005;293:723-729) for perinatal arterial ischemic stroke (PAS), but perinatal hemorrhagic stroke (PHS) is studied infrequently (Laugesaar R et al. **Stroke** 2007;38:2234-2240). In the Kaiser Permanente study, fetal distress and postmaturity were the independent predictors of PHS. In contrast, previously reported risk factors for PAS include chorioamnionitis, prolonged rupture of membranes, preeclampsia, placental thrombi, intrauterine growth retardation, prothrombotic and hematological factors, congenital heart disease with cerebral thromboembolism, infection and inflammation. Congenital hemiplegia and epilepsy are the most common neurologic deficits resulting from PAS (Kirton A et al. **Pediatr Neurol** 2009;40:205-214). Encephalopathy and seizures are the most frequent presenting symptoms with PHS. Preterm children at 12 years of age, especially those with a history of perinatal cerebral hemorrhage and severe brain injury, have delays in cognitive function and require educational intervention. (Luu TM et al. **Pediatrics** 2009;123:1037-1044). The use of indomethacin to lower the risk of intraventricular hemorrhage in preterm infants did not affect intellectual function in surviving children.

In 85 infants and children (range 7 days to 17 years) with nontraumatic intracranial hemorrhage and stroke in a US study, risk factors were intracranial vascular anomalies in 24

(28%)(arteriovenous malformation in 11), congenital heart disease in 14 (16%), and brain tumor in 13 (15%). Infection was associated in 5 (6%) cases, and coagulation deficiencies in 4 (5%). (Wo WD et al. **Arch Neurol** 2008;65:1629-1633; **Ped Neur Briefs** 2009;23:16).

Of 251 patients with childhood stroke (aged 1 month through 16 years) admitted to Beijing Children's Hospital, China, 1996-2006, arterial ischemic stroke accounted for the majority of cases (62.5%) and hemorrhagic stroke for 37.5%. Vitamin K deficiency was a major etiology of hemorrhagic stroke in China, diagnosed in 72 (76.6%) of 94 cases, most occurring in breastfed infants who had received no vitamin K after birth. Cerebral vascular abnormality was present in 7 (7%) (AVM in 6 of these). Infection played a role in 10%, including viral encephalitis, varicella zoster, mycoplasma and Epstein-Barr virus infections.(Wang J-J et al. **Pediatr Neurol** 2009;40:277-281). Etiological factors associated with hemorrhagic stroke in China differ from those reported in Western countries, where AVM is the most frequent cause.

HEADACHE DISORDERS

MENSTRUAL MIGRAINE IN ADOLESCENTS

The relationship between migraines and the menstrual cycle in prepubertal and pubertal girls was analyzed retrospectively in 896 girls, aged 9 to 18 years (mean age 14.3 years), attending a pediatric Headache Center at the Cincinnati Children's Hospital, OH. At the initial evaluation, headaches had occurred with menstrual periods in 50.3% of menarchal girls and 36.9% of all girls in the cohort. Mean age of headache onset was 10.3 +/- 3.5 years, and mean age of initial presentation was 13.8 +/- 2.6 years. Of the total group, 830 (92.6%) reported headaches that met the ICHD-II criteria (migraine without aura in 80.4% and migraine with aura in 12.3%). Those not meeting ICHD-II criteria had 'probable migraine.' Average headache frequency was 17.2 +/- 10.7 per month and average severity was 6.5 on a scale of 0-10. Duration of headache was 12.3 +/- 19 hrs. Migraine with a menstrual pattern started between day -2 and +3 of the onset of the menstrual period (68% before and only 14% after the period started). Headache frequency, severity and duration were no different in girls with or without a menstrual pattern (p=0.10). Associated symptoms, including photophobia, nausea and vomiting, were increased in girls with menstrual migraine compared to those without (p<.01). A total of 160 had a monthly pattern to the headaches and potential menstrual related migraine before beginning menstruation. A progressive increase in prevalence of a monthly pattern of migraine was observed in adolescents who later developed menstrual related migraine. The prevalence plateaued at age 13 years in susceptible patients, coinciding with onset of the first period. Intermittent prophylaxis for adolescents with a predictable pattern of menstrual related migraine should be considered. (Crawford MJ, Lehman L, Slater S et al. Menstrual migraine in adolescents. **Headache** March 2009;49:341-347). (Respond: Andrew D Hershey MD, Children's Hospital Medical Center, Department of Neurology, 3333 Burnet Ave, MLC 2015, Cincinnati, OH 45229).

COMMENT. Menstrual migraine is defined as migraine without aura with 90% of attacks occurring between 2 days before and 3 days after onset of menstruation, in at least 2 out of 3 menstrual cycles. Falling levels of estrogen during the late luteal/early follicular phase of the menstrual cycle are a proposed mechanism for menstrual migraine.