presented with leg weakness and pain, followed in 2 to 6 days by loss of pain and temperature sensation, and finally complete myelopathy, with proprioceptive loss and urinary incontinence. One patient had brain involvement also, manifesting with delirium, spasticity, and arm weakness. Despite aggressive antifungal therapy, the patients died within 1 month of onset of symptoms. Postmortem findings showed multifocal meningeal exudates, cerebral and spinal hemorrhagic necrosis, and fungal abscess in the cord of one child, and aortic endarteritis, spinal necrosis and intramedullary hematoma in a second. A third, without postmortem, had shown CT and MRI evidence of a paravertebral mass with vertebral osteomyelitis, and a positive biopsy culture for *Aspergillus*. Blood and CSF cultures were negative. (Koh S, Ross LA, Gilles FH, Nelson MD Jr, Mitchell WG. Myelopathy resulting from invasive aspergillosis. Pediatr Neurol Aug 1998;19:135-138). (Respond: Dr Susan Koh, Miami Children's Hospital, Neuroscience Center, 3100 SW 62nd Avenue, Miami, FL 33155).

COMMENT. Aspergillosis is a potential cause of myelopathy in immunosuppressed children treated with broad spectrum antibiotics.

## CEREBRAL CANDIDIASIS SEQUELAE IN PREMATURE INFANTS

Cerebral candidiasis in two premature infants, severely handicapped on follow-up at 3 and 6 years, is reported from the Women's College Hospital, Toronto, Canada. Infant 1 had respiratory distress syndrome and was treated with ampicillin and gentamicin for 5 days. A *Candida* diaper rash with skin breakdown on day 12 was complicated by sepsis, treated with vancomycin and cefotaxime, and followed by systemic candidiasis, requiring amphotericin and 5-flucytosine. Head ultrasound showed echogenic fungal microabscesses in brain parenchyma that resolved after 37 days treatment. Follow-up at 6 years revealed mild cerebral palsy, severe aphasia, and ADHD. Infant 2 had respiratory distress syndrome, low Apgars, skin rash, sepsis treated with ampicillin, vancomycin and gentamicin, and at 6 days, candidiasis treated with amphotericin and flucytosine. Head ultrasound showed scattered echogenic microabscesses, and later, calcifications in the basal ganglia and periventricular areas. At 3 year follow-up, the child had spastic quadriplegia, speech impairment, and cognitive delay. (Scott PA, Ohlsson A. Sequelae associated with cerebral candidiasis in two premature infants. Acta PaediatrOct 1998;87:1090-2). (Respond: Dr PA Scott, NICU, Women's College Hospital, 76 Grenville Street, Toronto, Ontario, Canada M5S 1B2).

COMMENT. Previous reports of candidiasis involving the brain have involved autopsy descriptions of scattered microabscesses. Large doses of amphotericin (1 mg/kg/d) and 5-flucytosine were thought to explain the survival of the two infants described above, although the resolution of infection did not prevent significant neurologic handicaps as sequelae. Treatment was continued for 37 days in case 1, with the better outcome, and 22 days in case 2, more severely handicapped. Perhaps more prolonged treatment may have improved the outcome, but serious side effects can be a problem. AAP 1997 Redbook recommends 6 weeks or longer treatment for systemic candidiasis in high risk patients.

## TOXOPLASMIC ENCEPHALITIS AND HYPER-IgM SYNDROME

A 9-year-old boy with hyper-IgM syndrome complicated by impaired consciousness and disseminated toxoplasmosis is reported from Nagoya University School of Medicine, Japan. X-linked hyper-IgM syndrome was diagnosed at 5 months of age and treated with IV immunoglobulin. On admission, he had involuntary movements and convulsions. CT and MRI showed multiple lucencies

in subcortical white matter of frontal lobes and basal ganglia, without mass effect. Disseminated toxoplasmosis was diagnosed at autopsy, and microscopy showed necrotizing toxoplasmic encephalitis. (Tsuge I, Matsuoka H, Nakagawa A et al. Necrotizing toxoplasmic encephalitis in a child with the X-linked hyper-IgM syndrome. Eur J Pediatr Sept 1998;157:735-737). (Respond: Dr I Tsuge, Department of Paediatrics, Nagoya University School of Medicine, Tsurumai-cho 65, Showa-ku, Nagoya 466, Japan).

COMMENT. X-linked hyper-IgM syndrome is an immunodeficiency disorder characterized by recurrent infections with opportunistic organisms, such as *Pneumocystis carinii and Cryptosporidium*, and associated with low serum levels of IgG and IgA, and normal to increased IgM. Disseminated toxoplasmosis is now added to the potential complications of this disorder.

## CEREBRAL TUMORS

### INTRACRANIAL ARACHNOID CYSTS AND ASSOCIATED LESIONS

The clinical manifestations and associated brain anomalies of intracranial arachnoid cysts are reported in 30 children treated at the National Taiwan University Hospital, Taipei. Onset of symptoms ranged from 1 day to 14 years (mean, 4 years, 7 months), and age at diagnosis was 10 days to 16 years (mean, 6 years, 2 months). The majority of cysts were located in the middle fossa, usually unilateral (17 cases); 7 were in the posterior fossa. Presenting clinical symptoms included seizures in 11 cases (37%), headache in 6 (20%), and macrocephaly (2 cases). EEG showed focal temporal or central epileptiform discharges in 13 of 17 patients. In patients with intractable seizures, surgery resulted in only partial reduction in cyst size and seizure frequency. Progressive hydrocephalus and increased intracranial pressure occurred in 2 patients and were relieved by ventriculo-peritoneal shunt. Associated anomalies included brain tumor, nevocellular nevi, achondroplasia, microphthalmia, intracystic hemorrhage, corpus callosum dysgenesis, and heterotopia. (Wang P-J, Lin H-C, Liu H-M, Tseng C-L, Shen Y-Z. Intracranial arachnoid cysts in children: related signs and associated anomalies. Pediatr Neurol Aug 1998;19:100-104). (Respond: Dr Wang, Department of Pediatrics, National Taiwan University Hospital, 7 Chung-Shan South Road, Taipei, Taiwan).

COMMENT. Intracranial arachnoid cysts (AC) may be asymptomatic and are recognized incidentally by CT or MRI for head trauma or at autopsy. Some are identified by neuroimaging performed because of headache, seizures, ataxia, or macrocephaly. The pathophysiology is a congenital malformation of the leptomeninges. The temporal lobe or middle cranial fossa is the most common location. The conservative approach to treatment, usually favored by neurosurgeons, is based on lack of sustained benefit and a 30% risk of cyst recurrence after surgery.

In addition to headaches and seizures, the most common presenting symptoms of AC, attention deficit hyperactivity disorder has been associated with temporal lobe AC, the TAC/ADD syndrome. (Millichap JG. Temporal lobe arachnoid cyst - attention deficit disorder syndrome: role of the electroencephalogram in diagnosis. Neurology May 1997;48:1435-1439). A causal association was considered plausible based on the coincidental learning and language disabilities that might be explained by temporal lobe and sylvian region pathology in the the initial 3 cases reported. Since this publication, I have diagnosed one further case and colleagues have provided me with case reports of 3 examples of the TAC/ADD