NEUROLOGICAL ASPECTS OF LEAD POISONING

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The neurological manifestations of lead poisoning are varied, and the major syndromes are: acute lead encephalitis; chronic lead encephalitis; cerebellar ataxia; convulsive disorder; behavior disorder; learning and perceptual disorders; mental retardation; and peripheral neuropathy. These are the eight major syndromes we have to consider.

Acute encephalopathy. Convulsions, delirium, coma and vomiting are the presenting symptoms and signs. The convulsions may be generalized or focal in pattern. Acute encephalopathy may develop after an initial period of irritability, anemia and anorexia, and it may be precipitated by acute infection or metabolic disturbance. In some cases, however, there are no premonitory symptoms or signs. Neurologic examination reveals papilledema, fixed dilated pupils, hyperreflexia or depressed deep tendon reflexes, Babinski signs, and often, nuchal rigidity. The skull x-ray shows spreading of the sutures, and the cerebrospinal fluid is under increased pressure with pleocytosis and elevated protein. This syndrome is most frequent in young infants during the summer months. If the acute encephalopathy is corrected, complete recovery is unusual and sequelae include hemiparesis, ataxia, mental retardation, convulsions, and optic atophy.

Chronic encephalopathy. After repeated nonfatal episodes of acute encephalopathy a chronic syndrome emerges resembling a progressive degenerative cerebral disorder, manifested by convulsions, ataxia, and mental deterioration.

Cerebellar ataxia. This may occur as the initial and main manifestation of central nervous system involvement but more frequently as a complication of acute or chronic encephalopathy.

Convulsive disorder. Convulsions may be generalized or focal in pattern. They may occur spontaneously or in association with fever and inter-current infections. They may be misdiagnosed as simple febrile seizures if they are short in duration and unaccompanied by changes in the cerebrospinal fluid.

Behavior disorders. These consist of irritability, frequent crying, inattention, impulsiveness, temper tantrums, and hyperactivity. Anemia, anorexia and loss of weight are frequently concomitant, but not invariable.

Learning disorders. Perceptual deficits include visual-motor incoordination and auditory imperception and the child with the learning disorder complicated by subtle neurologic abnormalities such as incoordination may be classified as having minimal brain dysfunction. Despite average intelligence, the child fails to achieve in school to the level of his potential. Improvements in behavior and learning have been reported after chelation therapy.

Mental retardation. Delays in development and regressions in intellect and behavior occur as neurologic sequelae of acute and chronic lead encephalopathy and have been reported in the absence of overt signs and symptoms of lead intoxication.

In one study of Perlstein and Attala in 1966,[1] among 58 children treated for asymptomatic lead poisoning, five, or 9%,

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were observed at follow-up examination to be mentally retarded. Admittedly, this study was retrospective, and one cannot be certain that mental retardation did not antedate lead poisoning. Pica, a common prelude to plumbism, may be a manifestation of emotional disorder or mental retardation, and the milder symptoms of lead poisoning are nonspecific and their significance often difficult to interpret.

Polyneuropathy. Wrist drop due to radical nerve involvement, a common manifestation of lead poisoning in adults, is uncommon in children. When neuropathy occurs during childhood, it usually affects the peroneal nerves and results in foot drop. Motor nerves and muscles subjected to the greatest use and fatigue are those chiefly affected. Diaphragmatic paralysis is a rare complication.

Differential Diagnosis

Acute lead encephalopathy must be distinguished from other forms of toxic encephalopathy, including arsenic and thallium poisoning and Reye's syndrome. Meningitis, particularly tuberculous meningitis, brain tumor and brain abscess should be excluded. Lead poisoning may occur at blood lead levels below 60 and even 50 mg per 100 ml, but some children with blood lead levels well beyond 100 mg per 100 ml appear well and asymptomatic. The finding of an elevated blood lead level alone should not be accepted as pathognomonic evidence of lead in the etiology of convulsions and coma.

Treatment of Encephalopathy

Convulsions should be controlled with phenobarbital and the cerebral edema treated with steroids and mannitol. Urine flow is established by appropriate fluid therapy, and the body temperature

should be reduced to normal levels. Residual lead is removed from the bowel by enema and treatment with 2,3-dimercaptopropanol (BAL) and EDTA by intramuscular injection is initiated and continued for five to seven days.

The use of surgical decompression is controversial and is usually reserved for patients in whom increased intracranial pressure persists beyond the initial five-day treatment period and in chronic encephalopathy.

Prognosis of Encephalopathy

A mortality of 20 per cent is to be expected and survivors sustain a variable amount of permanent nervous system injury. In one study of 425 children with lead poisoning, 39 per cent had some evidence of neurologic sequelae and among 59 children with lead encephalopathy, 82 per cent were left with handicaps.

Further efforts must be devoted to the prevention of lead poisoning, but the recognition of early symptoms and signs of intoxication and prompt therapy should reduce appreciably the number of fatalities and permanent neurologic sequelae.