LEUCOENCEPHALOMYELOSIS IN NONHUMAN PRIMATES ASSOCIATED WITH LEAD POISONING

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LEUCOENCEPHALOMYELOSIS IN NONHUMAN PRIMATES ASSOCIATED WITH LEAD POISONING

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Abstract: Lead poisoning was diagnosed in five of seven primates affected with leucoencephalomyelosis that were necropsied at the National Zoological Park and the Antwerp Zoo. Diagnoses of lead poisoning were made by various means including th detection of acid-fast intranuclear inclusions in renal proximal tubular cells and the presence of excess lead in the liver specimens.

The implications of the concurrence of lead intoxication and leucoencephalomyelosis is discussed with regard to etiology and pathogenesis.

Leucoencephalomyelosis is a demyelinating disease of captive Old World primates. This disease has been described many times in zoo monkeys under a variety of names including cage paralysis; confluent leucoencephalosis and leucomyelosis; confluent leucoencephalosis and perivascular myelosis; subacute combined degeneration; and others.[2,3,5,8-12]

The clinical and pathologic manifestations of leucoencephalomyelosis are divided into the spinal type, characterized by flaccid paralysis of the extremities and associated with foci of demyelination in the spinal cord; the cerebral type, typified by convulsions and associated with focal demyelination in the brain; and the optic type, manifest by blindness and demyelination of optic nerves or tracts. These clinico-pathologic types may occur singly or in various combination.[2,3,5,10]

The cause of leucoencephalomyelosis has not been determined, but evidence indicates that a deficiency of vitamin B_{12} may be involved.[5,8,9] This disease has also been reported to affect primates simultaneously with acute amaurotic epilepsy,[2] a syndrome caused by lead intoxication.[3,4]

The simultaneous occurrence of leucoencephalomyelosis and lead poisoning in a primate at the National Zoological Park (NZP)[11] stimulated a review of all NZP cases of leucoencephalomyelosis. It also prompted the acquisition and study of tissues from affected primates from the Antwerp Zoo. The purpose of this report is to present further evidence that lead poisoning may be associated with leucoencephalomyelosis.

MATERIALS AND METHODS

A search of records at NZP (1959-1971) disclosed nine cases of leucoencephalomyelosis. The cases were studied with regard to history and clinical signs. Four of the nine were necropsied and the diagnosis in these primates was verified by comparison of microscopic neural lesions to descriptions of leucoencephalomyelosis by others.[2,3,5,8,10,12] In addition, tissue specimens were obtained from three primates affected with leucoencephalomyelosis that were described in a major report of this disease from the Antwerp Zoo.[3]

The NZP and Antwerp Zoo primates were further studied for evidences of lead poisoning including exposure to lead, compatible clinical signs, acid-fast intra-

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nuclear inclusions in renal proximal tubular cells, and lead in formalin-fixed specimens of liver.*

RESULTS

All of the primates with leucoencephalomyelosis studied had histories and signs compatible with lead poisoning.[4,6,7,13,14] Paint containing 3.5% to 30.0% lead was found on all cages that housed affected NZP monkeys; lead-containing paint has been found on some primate cages at the Antwerp Zoo.[14]

Acid-fast intranuclear inclusions characteristic of lead poisoning occurred in renal proximal tubular cells of the four affected NZP primates that were examined post mortem. Liver from these four monkeys contained 3.8, 6.7, 10.0, and 45.0 parts per million (ppm) lead. Amounts of lead in the livers of control Zoo monkeys previously tested ranged from 0.3 to 2.5 ppm.[14] One of the three affected Antwerp Zoo primates (case 238/35)[8] had typical renal acid-fast inclusions and 200 ppm lead in the liver. The other two Antwerp Zoo monkeys (299/38 and 424/36)[8] had no inclusion bodies and only 0.3 and 2.8 ppm lead in their livers.

DISCUSSION

Lead poisoning was diagnosed in five of seven necropsied primates affected with leucoencephalomyelosis from NZP and the Antwerp Zoo. The lack of renal acid-fast inclusions and non-toxic amounts of lead in the livers of two monkeys with leucoencephalomyelosis indicates that this syndrome may occur in the absence of lead intoxication. These findings suggest to us that lead poisoning may be etiologically related to leucoencephalomyelosis, but that it is not a necessary causative factor.

There are many possible explanations for the frequent concurrence of leucoencephalomyelosis and lead poisoning. The possibility that the two diseases are unrelated and were merely coincidently found to affect five of seven primates at the same time cannot be discounted. It seems more likely to us, however, that one disease may predispose toward, or tend to aggravate the other. Some possible mechanisms for this contention are: 1. A diet deficient in vitamin B_{12}, which is thought to cause leucoencephalomyelosis, may well be deficient in other nutrients resulting in pica and the ingestion of non-food substances such as leaded paint. 2. Chronic lead poisoning may result in interference with the absorption or utilization of vitamin B_{12}. 3. There may be an additive or synergistic effect, e.g. both lead poisoning and deficiency of vitamin B_{12} can independently result in loss of myelin. In combination, demyelination may occur more rapidly and extensively than in either condition alone. It is also possible that chronic lead intoxication in certain susceptible primates may cause extensive demyelination of the central nervous system and that the clinical and pahologic manifestations in these individuals are indistinguishable from leucoencephalomyelosis.

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