Brain Lesions of Lead Poisoning in a Calf
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(Received 6 February 1990/Accepted 7 September 1990)


KEY WORDS: brain lesion, calf, lead poisoning.

Lead poisoning has been recognized in various species of domestic animals [10, 13, 15, 17, 24] and in man [12], and commonly occurred in calves [3, 4, 8, 23] because of their high susceptibility and their habits of licking and sucking at anything else left within their reach [2, 4]. The symptoms and lesions of the disease have been known to focus on its effects on the red blood cells, nervous system, kidney, and bone tissues [5, 7, 9, 11, 25]. Most cases are acute form in cattle and die within several hours from the onset [8, 16]. The present paper describes pathological findings in a calf suffered from lead poisoning, with special emphasis on the brain lesions.

On a farm located in Iwate Prefecture, in which fourteen Japanese Black cattle, 8 cows and 6 calves were kept, the disease occurred only in a female calf. The calf had been kept with her dam since the day of birth in a newly built pen apart from the barn where the other animals were raised. The calf suffered from the disease suddenly without any premonitory signs at 50 days old of age, on November 14, 1988. Clinical signs were anorexia and pronounced nervous symptoms such as blindness, pushing the head against the wall in a state of excitement, circling, opisthotonus, and staggering gait. She was not improved in spite of repeated administrations of electrolytes and antibiotics. The animal was killed by euthanasia on the 11th day after the onset of the disease because of its unfavourable prognosis and submitted to autopsy.

As the epidemic aspect, there was evidence that old water pipe coated with anticorrosive paint and laid on in the pen, had been licked by the animal, although the farmer had no chance to watch the animal licking the paint. The paint was easily flaked away when it was scraped with a knife.

Autopsy disclosed leptomeningeal congestion and edema, and somewhat flattened cerebral gyri. Frontal sections of the cerebrum revealed several focal and small areas of yellowish discoloration at the tips of gyri in the gray matter of the occipital and parietal lobes.

At autopsy, specimens of the organs and tissues from the whole body were fixed in 10% neutral formalin solution. Paraffin sections were stained with hematoxylin and eosin (HE), Luxol fast blue (LFB), and Ziel-Neelsen (ZN) methods.

Histologically, multiple focal or laminar lesions of neuronal necrosis were observed in the cerebral cortex, caudatum, and medial nuclei of thalamus, predominantly at the tips of gyri in the occipital and parietal lobes. The lesions spread occasionally to the deeper region of the gyri along the sulci. The affected neurons were shrunken and angular, sometimes triangular in outline with pale eosinophilic cytoplasm. The nuclei showed pyknosis, rhexis, or obscurity (Fig. 1). Edematous dilatation of perivascular and perineuronal spaces with spongiosis of neuropil was observed from the molecular layer to outer zone of the white matter. Pyramidal cell layer lost its normal arrangement, in which both necrotic and apparently normal neurons were scattered. Astrocytic proliferation was also observed. Blood capillaries were congested with enlarged and increased endothelial cells.

Meningeal blood vessels were prominently congested with mild lymphocytic infiltration. Edema of Purkinje cell layer in the cerebellum and mild neuronal degeneration in the nucleus ruber of the mesencephalon were seen. No lesions were observed in the retina and optic pathway.

In the kidney, were found epithelial degeneration confined to the proximal convoluted tubules as well as congestion. The epithelial cells were swollen and increased in the eosinophilic affinity. Eosinophilic intranuclear inclusions were observed accompanying with enlarge-

Fig. 1. There are eosinophilic neuronal degeneration (arrows) with karyo-pyknosis, rhexis, or lysis, edematous vacuolation of neuropil, and enlargement and proliferation of capillary endothelial cells in the cerebral occipital lobe.

HE stain. ×200.
ment of the nucleus with poor chromatin and occasional chromatin margination (Fig. 2). The inclusions were solitary or in groups of various sized particles in a nucleus, and acid-fast with ZN stain. Necrosis of tubular epithelia was confined to a few cells and no glomerular lesions were found.

Biochemical examination for lead performed by the dithiazone method disclosed 1.3 and 9.4 parts per million (ppm) on a wet weight basis of lead in the liver and kidney, respectively. Their values in a 2-month-old Japanese Black calf used as a control that had been kept on the other farm, were 0.9 and 0.6 ppm, respectively. Flakes of the anticorrosive paint removed from the water pipe were qualitatively positive for lead by the same method.

The diagnosis of lead poisoning in the present case was based on the pronounced nervous symptoms, multiple focal or laminar necrosis in the cerebral cortex and nephrosis with acid-fast intranuclear inclusions. More than 25 ppm of lead concentration in the renal cortex are considered to be lethal [22]. The renal lead level was 9.4 ppm in the present case. The cortical level would be higher than that value, since the renal tissue used for analysis included both cortex and medulla and lead level is known to be higher in the cortex than the medulla [1]. Accordingly, the present case seemed to have ingested more than normal amounts of lead. The anticorrosive paint coating water pipe was suspected to be the source of the lead because it had been licked and contained lead.

Clinical differential diagnosis of the disease is very important among neurologic diseases in cattle such as cerebrocortical necrosis (CCN), infectious thromboembolic meningoencephalitis [4, 16], and listeriosis [14]. It will be helpful for their differentiation to observe the response to therapies with calcium EDTA [6, 17], thiamine and antibiotics. It is also an indispensable aid for diagnosis to find some lead contaminants around the diseased animals.

Basic cerebral lesions in both lead poisoning and CCN have been understood to be vascular disturbance followed by ischemic change of neurons [5, 18]. Therefore, regarding the cerebral lesions it may be difficult to differentiate both diseases [5, 16], especially in their acute phase [5]. Christian and Tryphonas [5] suggest that the lesions in the lead intoxication are frequently located at the tips of gyri while prominent along the sulci in CCN. In the present case, the cerebral lesions were apt to be situated at the tips of gyri. But the lesionual distribution appeared not to be a clear point for the differential diagnosis as far as in the present case. A distribution of the affected neurons scattered in the gray matter in the present case might be useful for the differentiation from CCN, in which most of neurons were affected [19] in the disease courses other than the early stage when many neurons showed no evidence of degeneration [18]. The presence of acid-fast intranuclear inclusions in the kidney, which have been often seen in lead poisoning cases [21] and not in CCN [19, 20], will be a useful additional evidence for the diagnosis. Demonstration of significant amounts of lead in the renal or hepatic tissue will contribute to the more correct diagnosis.

REFERENCES