Pathology of Natural and Experimental Lead Poisoning in Cattle

Case Report

Gholami,*[1], M.R., Ottarod, V.,[2] Hablolvarid, M.H.[1] and Ezzi, A.[1]

1. Pathology Dept., Razi Vaccine & Serum Research Institute, P.O.Box 11365-1558, Tehran, Iran

2. Veterinary Organization of Iran, P.O.Box 14115-6349, Tehran, Iran

Received 14 Jan 2004, accepted 24 Apr 2004

Lead poisoning has been recognized in most domesticated animals species (Fenstermacher 1946, Jubb 1985, Link 1966, Allcroft 1951, Radostits 1994, Bywater 1937, Donawick 1966, Zook 1972) and the experimentally induced form has been studied in several species (Hammond 1964, Lampert 1967, Pentchew 1966, Rosenblum 1968, Staple 1955). Lead poisoning in cattle and other species is occurred via oral route or less commonly via the respiratory system or skin (inorganic lead). It affects the CNS, PNS, kidney, liver, bone marrow, bone, gastrointestinal tract, blood, vessels, reproductive and en ocrine systems, and seems to acute, subacute or chronic forms (Donald 2002). Th common sources of lead are lead bearing paints and metallic lead. After absorp n lead is excreted in bile, milk and urine, and the blood levels of lead provide iable indication of lead status of the animal. Deposition in tissues occurs par ularly in liver and renal cortex and medulla in acute poisoning and in the bones in chronic poisoning (Radostits 1994).

There is considerable variation between species in their susceptibility to lead and the young animals are more susceptible. Acute lethal single dose for calves is 400-600mg/kg body weight. Chronic poisoning for cattle is daily dose 6-7mg/kg body weight (equivalent to100-200mg/kg in the diet (Radoostits 1994). The toxic effects of lead are manifested in three main ways: lead encephalopathy, gastroenteritis, and

*Author for correspondence. E-mail:gholami@rvsri.com

degeneration of peripheral nerves. The mechanism by which the nervous signs of encephalopathy and the lesions of peripheral nerve degeneration are produced appears to be related to the degenerative changes seen in the nervous tissue (Radostits 1994). Histological tissue changes can be considerable diagnostic value in suspected cases of lead poisoning. Eosinophilic acid fast and intranuclear inclusion bodies with in renal epithelial are pathogonomic for diagnosis of lead poisoning (Zook1969, Donald 2001, Jubb 1985, Jones 1983).

In this study the pathology of natural and experimental lead poisoning in cattle was discussed.

Key words: lead, poisoning, intranuclear inclusion, demylination

Clinical history

In two cattle breeding farms 15 cases mostly calves with clinical signs of dullness, anorexia, blindness, incoordination and staggering, muscle tremor, frothy salivation, grinding of teeth, stare gazzing, neck contraction, deviation of head in one side, recumbency, high rate of heart beat and breathing and in some cases with traumatic lesions of head and muzzle as a result of pressing on the objects were noted. Based on field studies on rations of the farms a kind of mineral supplement was suspected to be the cause of the disease. For confirmation two calves each of 6 months age were selected. Calf A was fed with regular ration plus 250gr/day of suspected mineral and calf B fed without mineral. Clinical trial after 5 days symptoms of anorexia and blindness and in 20 days nervous disorder appeared in calf A, while the calf B did not show any symptom.

Chemical analysis. Two samples of suspected minerals supplement from the farms were analyzed by spectrophotometer; the range of lead was estimated 200-6756mg/kg.

Pathology. Experimental and natural cases were euthenised. Hyperhemia of abomasum and rumen with sloughing of epithelium, petechial hemorrhages of heart,

kidney, and spleen were noted. The liver and kidney showed gray color and the liver enlarged slightly. The brain showed hyperhemia of meninges and foci of hemorrhages in different parts of cut surface. In some cases on occipital lob especially gyri the color of brain changed to pail; while there were no any pathologic lesions on control calve. Fomalin fixed brain, liver and kidney were submitted to pathology department of Razi institute. After processing 5μ paraffin block sections were prepared and stained by H&E method. The most obvious and consistent lesions in the brain were in blood vessels. Many arteriols, venuls and capillaries were considerably distended with blood and endothelial cells were swollen, some dilated capillaries were necrosis and disintegrated. Altered vessels were often surrounded by extravasated erythrocytes, which varied in size, but were generally small. Proliferation of endothelial cells (Figure 1) and budding of new capillaries were seen in two cases. The proliferate changes were most pronounced in the cerebral cortex gray matter, especially adjacent to the gyral tips of the subcortical white matter.

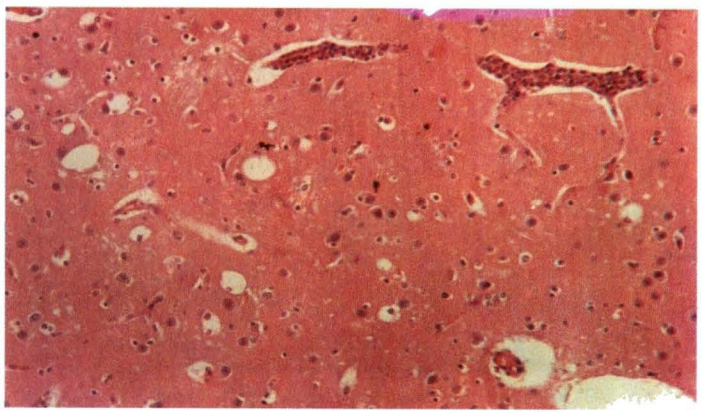

Figure 1. *Brain: endothelial cells proliferation of blood vessels and increases perineuronal, perivascular space (edema). H&E×200*

Degeneration and necrosis of neurons in the cortical gray matter were noted. Affected areas contained many shrunken eosinophilic neurons and marked perineuronal spaces and spongiosis depend on severity and duration of disease, were

relatively prominent in thalamous, cerebellum, medula oblangata and cervical spinal cord (Figure 2). Neuronal sattelitosis in some areas and moderate demylination of white matter were noted (Figure 3). The lesions of lead encephalopathy in children, cattle, monkey, and dogs are similar in most respects (Zook 1972).

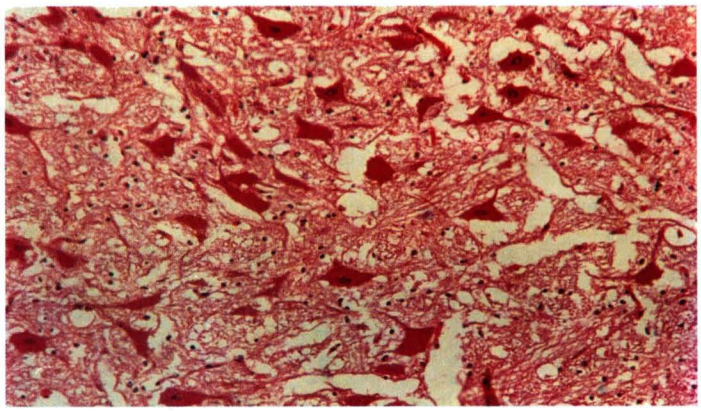

Figure 2. ***Brain: neuronal degeneration and necrosis, perineuronal edema and spongiosis.*** *H&E×200*

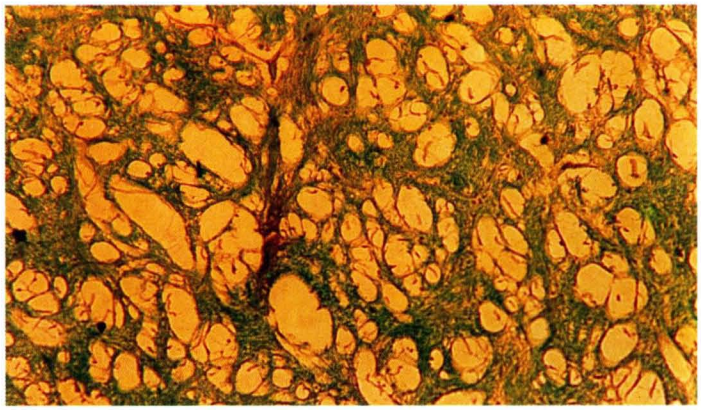

Figure 3. ***Brain: severe spongiosis of white matter. Luxal Fast Blue×200***

Degeneration and necrosis of renal tubular epithelium were seen in calf A and natural affected cases. The changes were virtually confined to proximal tubules; most epithelial cells were enlarged, finely vacuolated and had irregular apical borders encroaching upon the lumen of their tubules. Some tubules especially in proximal

area were completely necrotized. Using Charbol-Fuschin staining of kidney most eosinophilic and acid fast intranuclear inclusions were seen in straight segments of proximal tubules, which were contained in the proximal covoluted portion. A few inclusions in medula of kidney were found (Figure 4). In order to be ascertained the inclusions are not artifact, two normal kidneys were processed and stained with Charbol-Fuschin, but any inclusion were not seen.

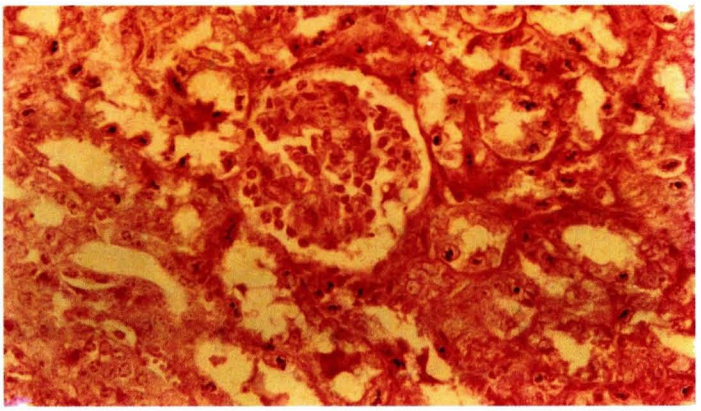

Figure 4. *Kidney: degeneration and necrosis of tubules and intra nuclear inclusions bodies. Charbol-Fuschin×200*

In the liver mild periportal fibrosis, congestion, fatty degeneration, and bile duct hyperplasia were noted. In some cases changes in hepatocytes were similar to those in the kidney, but less constant and noticeable. Many hepatocytes had slightly enlarged nuclei and severe degenerative changes, a few acid fast intranuclear inclusions were noted, but it was not so conspicuous as kidney. In these cases for the reasons of clinical signs, chemical analysis and high level of lead in the diet and experimental induced of the disease; histopathological changes and specific acid-fast inclusions confirmed the lead poisoning.

References

Allcroft, R. (1951). Lead poisoning in cattle and sheep. *Veterinary Record* 63:583-590.

Bywater, H.E. (1937). Lead poisoning in pigs. *Veterinary Record* 49:549-551.

Christian, R.G., Tryphonas, L. (1971). Lead poisoning in cattle, Brain lesions and hematologic changes. American Journal of Veterinary Research 32:203-216.

Donald, M., Carlton, W. and Zachary, F. (2001). *Special Veterinary Pathology* (3th edn.), London, Mosby, Inc.

Donawick, W.G. (1966). Chronic lead poisoning in a cow. *Journal of American Veterinary Medical Association* 148:655-661.

Fenstermacher, R., Pomeroy, B.S., Roepke, M.H. and Boyd, W.L. (1946). Lead poisoning of cattle. *Journal of Veterinary Medical Association* 108:1-4.

Hammond, P.B., Aronson, A.L. (1964). The mobilization and excretion of lead in cattle: A comparative study of various chelating agents. *Annual of New York Academic Sciences* 110:498-511.

Jubb, K.V., Kennedy, F., Peter, C. and Palmer, N. (1985). *Pathology of Domestic Animal* (3th edn.), London, Academic Press, Inc.

Jones,T.C., Hunt, R.D. (1983). *Veterinary Pathology* (5th edn.), Philadelphia, Lea & Febiger.

Link, R.P., Pensinger, R.R. (1966). Lead toxicosis in swine. American Journal of Veterinary Research 27:759-763.

Pentschew, A., Garro, F. (1966). Lead encephalo-myelopathy of the suckling rat. *Acta Neuropathologia* 6:266-278.

Radostits, O.M., Blood, D.C. and Gay, C.C. (1994). *Veterinary Medicine* (8th edn.), London, Bailliere Tindall.

Rosenblum, W.I., Johensen, M.G. (1968). Neuropathological changes in suckling mice produced by maternal feeding of lead. *Archives of Pathology* 85:640-648.

Staple, E.J. (1955). Experimental lead poisoning in dogs. *New Zealand Veterinary Journal* 3:39-46.

Zook, B.C. (1972). Lead poisoning in dogs. *Veterinary Pathology* 9:310-327.