

# Clinical and Laboratory Investigations on Chronic Bovine Fluorosis

A.A. Metwalli, S.A. El-Gharieb, and K.M. Ashry

Dept. of Vet Med. & Forensic Med. & Toxicology

Fac. of Vet. Med.

Alexandria University, Egypt

## Summary

During summer 1995, a clinical picture of chronic bovine fluorosis was observed in Fowa villages (Kafr El-Sheikh Governorate) where many brick kilns were established. A total number of 26 cows (5-9 years) of native breed was investigated in the present study. Sixteen animals were collected from different places around the kilns, another 10 animals (control group) were chosen from places away from the brick kilns zone. The affected cows showed reduced appetite, loss in general body condition, lameness and dental lesions in the form of staining, mottling and blackish discoloration specially of the incisors teeth. Biochemical analysis of serum of affected cows revealed significantly decreased level of serum calcium and total protein, whereas serum alkaline phosphatase activity, fluoride content, urea, and creatinine were significantly increased as compared with control animals. Haematological examination of affected cows showed significantly decreased values of Hb and PCV. On the basis of laboratory and circumstantial evidence, the problem was suspected to be fluorosis, possibly due to environmental pollution with fluorine-bearing smoke arising from the large number of brick kilns.

## Introduction

Environmental pollutants are the most deleterious agents to the biological life. Industrial fluorosis in livestock today is among the most important pollutant well known by veterinarian in all industrial countries (Ender, 1969). Cattle fluorosis has been observed in several parts of the world as a consequence of high fluoride concentration in phosphate rock deposits, water sources and manufacture bricks (Clarke and Clarke, 1975 and Bartik and Piskac, 1981). One of the most aggressive forms of fluoride intoxication is related to the intake of dust containing fluoride. In addition to the usual system effects, there is an abrasive local action on the respiratory system and on the digestive tract (Bartik and Piskac, 1981). Blood and Radostits (1989) recorded variation in the effects produced by chronic fluorine in-

toxication. The author stated that dental changes were the earliest and most diagnostic sign. The obvious signs were mottling with the appearance of staining and accelerated attrition, particularly of the incisor teeth. The author added that in heavy contamination there will be signs of generalized effects, reduced appetite, loss in general body condition, stiffness, lameness and painful gait. Similar signs were mentioned by Swarup and Singh (1989), Araya, *et al.* (1990), Botha, *et al.* (1993) and Singh and Swarup (1994).

The present study aimed to investigate the biochemical and haematological changes of chronic fluorosis in cattle due to contamination from a brick works in Fowa villages (Kafr El-Sheikh Governorate).

## Materials and Methods

### Animals:

A total number of 26 cows (5-9 years) of native breed was put under clinical and laboratory investigations. Diseased animals (16) were collected from different areas around the kilns which had been fired during the past few years for manufacturing bricks in Fowa (Kafr El-Sheikh Governorate). Clinically, cows showed signs of lameness, reduced appetite, loss of general body condition, paleness of mucous membranes and brownish or blackish discoloration of the incisor teeth. Animals of the control group (10) were chosen from an area far away from the brick kiln.

### Biochemical and haematological examinations:

Blood and serum samples were collected from diseased and control animals. Blood samples were used to determine haemoglobin concentration (Hb) and packed cell volume (PCV).

Serum samples were used for determination of fluoride level by fluoride electrode using ion analyzer (Fray and Taves, 1970), serum calcium (Gindler and King, 1972), serum inorganic phosphorus (Fiske and Subbarow, 1925), serum urea nitrogen (Fawcett and Scott, 1960), serum creatinine (Husdan and Rapapost, 1968), serum total protein (Wiechselbaum, 1946), serum albumin (Bartholemew and Delancy, 1966), serum al-

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kaline phosphatase (ALP) activity (King and King, 1954) and creatine kinase activity (CK), (Oliver, 1955.)

#### Statistical analysis:

Data were statistically analyzed according to the method of Snedecor and Cochran (1974).

### Results

Diseased cows in different areas in the vicinity of the brick kilns exhibited signs of lameness, general illness, reduced milk yield, poor appetite, weight loss and diarrhoea. Dental lesions were very striking and incisor teeth showed mottling, blackish discoloration and attrition.

Serum biochemical analysis for fluoride, calcium, inorganic phosphorus, alkaline phosphatase activity, urea, creatinine, total proteins, albumin, globulin, creatine kinase activity and some haematological parameters were presented in Table 1.

**Table 1.** Serum biochemical and haematological profile of healthy and fluorotic cows.

Parameters	Mean $\pm$ S.E.	
	Healthy cows	Affected cows
Calcium mg/dl	10.95 $\pm$ 0.09	8.28 $\pm$ 0.15**
Fluoride mg%	0.17 $\pm$ 0.03	0.73 $\pm$ 0.02**
Inorganic phosphorus mg/dl	6.24 $\pm$ 0.14	5.91 $\pm$ 0.22
Alkaline phosphatase UI	58.14 $\pm$ 1.46	134.37 $\pm$ 5.15**
Urea mg%	28.74 $\pm$ 0.55	58.33 $\pm$ 0.82**
Creatinine mg%	2.38 $\pm$ 0.15	7.16 $\pm$ 0.12**
Total protein gm/dl	7.31 $\pm$ 0.18	6.55 $\pm$ 0.13**
Albumin gm/dl	3.53 $\pm$ 0.13	3.22 $\pm$ 0.05*
Golbulin gm/dl	3.78 $\pm$ 0.11	3.32 $\pm$ 0.10**
Creatine kinase UI	69.32 $\pm$ 0.66	73.18 $\pm$ 1.36
Hb gm %	12.17 $\pm$ 0.20	9.14 $\pm$ 0.10**
PCV %	36.53 $\pm$ 0.35	27.03 $\pm$ 0.24**

\* Significant at  $P \leq 0.05$

\*\*Highly significant at  $P \leq 0.01$   
S.E. Standard error

Depending on the clinical findings, the presence of brick kilns in the affected area and the increased level of serum fluoride, we could decide that these animals were suffering from chronic fluorosis.

### Discussion

**Industrial fluorosis occupies a prominent position among environmental contamination problems. Sources of environmental pollution with fluorine usually arise from the large number of brick kilns as recorded by WHO (1984). Swarup and Singh (1989) and Singh and Swarup (1994).**

This investigation followed earlier reports of lameness, general illness and reduced milk yield in cows in Fowa villages. Clinical examination of the affected cows revealed that animals reared near the brick kilns appeared weak, emaciated with reduced appetite, pale mucous membrane and weight loss. Teeth lesions were the main clinical disorder recorded, where mottling, pitting, blackish discoloration and excessive rate of attrition were noticed. These affections were attributed to fluoride intoxication. Such clinical signs were in agreement with previous reports of bovine fluorosis (Suttie *et al.* 1985, Swarup and Singh, 1989, Araya *et al.*, 1990 and Botha *et al.*, 1993). Similar symptoms of chronic fluorosis were observed by Ibrahim (1983) and Singh and Swarup (1994) in buffaloes and Karram *et al.* (1990) in camels.

Fluoride accumulate inside the body until it reaches sufficient concentration to produce clinical manifestations. In accordance, Clarke and Clarke (1975) stated that when bone fluorine values become sufficiently high to be reflected as skeletal exostosis, lameness will appear clinically.

Also, the increased rate of wearing could be explained by the hypomineralization which had been ensured by the special histologic examination of Shearer *et al.* (1978).

Lameness usually occurs as a result of changes in bone structure and excessive mobilization of calcium and phosphorus to compensate their increased urinary excretion in conjunction with fluoride (Blood and Radostits, 1989). Other clinical findings recorded in the affected cows could be attributed directly or indirectly to fluoride toxicity (Swarup and Singh, 1989 and Singh and Swarup, 1994).

In comparison to healthy cows, the affected animals had significantly ( $P < 0.01$ ) decreased serum calcium and total proteins levels, increased levels of fluoride, urea, creatinine and alkaline phosphatase activity (Table 1).

Hypocalcemia in affected cows may be attributed to the adverse effect of fluoride on the parathyroid gland, which may be responsible for disturbed calcium and phosphorus metabolism and subsequent changes in their serum levels (Singh and Swarup, 1994). It is clear from our studies, an interaction between fluoride and calcium in the biological system was drawn; this relationship was documented by Karram (1982) and Karram *et al.* (1990).

Concerning the alkaline phosphatase activity, it was documented that fluoride stimulates osteoblastic activity resulting in increased activity of alkaline phosphatase in serum of affected animals in comparison to control group (Table 1), and this is considered to be an important finding in fluorosis (Swarup and Singh, 1989 and Botha *et al.*, 1993).

The increased urea and creatinine concentration in fluorotic cows may be due to renal dysfunction which can occur in fluorosis (Singh and Swarup, 1994). An increase in serum urea and creatinine was also noticed in fluorotic sheep by Kessabi *et al.* (1983).

As shown in Table 1, total proteins, albumin and

globulin were significantly decreased in affected group when compared with control one. These results are in parallel with those recorded by Mohiuddin and Reddy (1989) and Botha *et al.* (1993).

Haematological data revealed that the Hb content and PCV value of affected cows were significantly decreased when compared with healthy cows. This may be related to the adverse effect of fluorine on iron metabolism in bone marrow and inhibition of haeme synthesis in bone marrow reticulocytes noted in fluorosis (Karram, 1982). Our results in this respect agree with those of Mohiuddin and Reddy (1989) in sheep, Swarup and Singh (1989) in cattle and Karram *et al.* (1990) in buffaloes affected with chronic fluorosis.

**The clinical examination together with increased alkaline phosphatase activity and abnormally high fluoride in sera of affected cows, provided a strong evidence that the affected animals were suffering from chronic fluorosis.**

Since the affected area had large aggregates of brick kilns and the incidence of disease coincided with their operation, it was suspected that the source of fluoride was the dust and smokes generated by the kilns.

**Finally, it is recommended that brick kilns should be established away from animal and human populations to prevent or minimize the environmental pollution by fluoride compounds to provide enough protection for animal health and production.**

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