

## **Fluorosis**

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In recent years, there has been significant veterinary interest in fluorine toxicity. Generally fluorosis occurs in chronic form caused by the continued ingestion of small but above permissible amounts of fluorine in the ration or drinking water over a long duration. Acute toxicity arises by inhalation of fluorine smoke, vapors, dust from volcanic eruptions; dust from industries of aluminum, copper, enamel, glass, iron, steel, superphosphate etc. It also may be due to accidental ingestion of large amounts of fluorine.

According to WHO guidelines (1994) and Rajiv Gandhi National Drinking Water Mission (RGNDWM), GOI (1994), 1.5 mg/l. is the permissible limit for fluoride in potable drinking water. When concentration becomes higher than 1.5 ppm, it may cause dental and skeletal fluorosis in human beings.

Available reports revealed the prevalence of fluorosis in bovines in Andhra Pradesh and Uttar Pradesh (Dwivedi *et al.*, 1997), Punjab (Sharma *et al.*, 1997), Rajasthan (Choubisa, 1999), Karnataka (Muralidhara *et al.*, 2000), Orissa (Maiti *et al.*, 2004) and Bihar/ Rajasthan (Rajan *et al.*, 2009) in India.

#### **Etiology:**

The toxicity depends on the amount ingested/administered, its solubility and subsequent bio-degradability. Source of fluorine as rock phosphate or cryolite (>100 ppm in ration), Calcium fluoride or sodium fluorosilicate (>400 mg to 2 gm/kg of body weight), Sodium fluoride (> 50 ppm in dry ration) may induce fluorosis. When fluorine levels in water exceeds 2 ppm, it is toxic to animals, at 5 ppm, produces mild teeth lesions, at 10 ppm causes excessive wear and tear of teeth and if it is present at the rate of 30 ppm in water, it may produce more systemic effects. Bore water containing 12 – 19 ppm fluorine may initiate chronic intoxication (Ulemale *et al.*, 2010).

#### **Metabolism of fluorine:**

When fluoride enters the bloodstream of an animal, considerable amounts of it may be excreted through urine, the rest combines with calcium and

phosphorous and is stored in the bones and teeth. The stored fluorine is liberated slowly but continuously excreted in urine over a considerable period. Normal cattle have blood levels of up to 0.2 mg fluorine per deciliter of blood and 2-6 ppm in urine.

#### **Pathogenesis & Necropsy Findings:**

In continuous fluoride ingestion, principal damage appears to the bones and teeth. The deposition of fluorine occurs highly in long bones on periosteal surfaces and due to excessive mobilization of calcium and phosphorus, exostosis may occur at any age in life. Osteomalacia, osteofluorosis, osteoporosis, hip lameness and painful gait, stiffness of limbs, un-thriftiness are the characteristic clinical symptoms of chronic fluorosis. Enlargement of mandible, sternum, metacarpal etc, may occur and animals are prone to fracture. Periosteal hyperostosis or exostosis at joints or at places of attachment of ligaments and tendons cause shifting pain and lameness. Degenerative changes are seen in kidney, liver, adrenal glands, heart muscle, central nervous system, bone marrow. Also causes aplastic anemia. (Ulemale et. al, 2010)

#### **Clinical findings:**

According to the available literature, in field conditions, the incidence of fluorosis worldwide is 7.71 – 65 % with Indian conditions having an incidence in the range of 7.71 – 46.8 %.

Araya *et. al* (1993) reported osteofluorosis and dental fluorosis in cattle after a volcanic eruption in Chile. Osteo/dental fluorosis was also evidenced by other researchers from places like Turkey, Morocco, Canada, Egypt, South Africa, Poland etc (Kececi *et. al*, 2002; Kessabi *et. al*, 1983; Krook *et. al*, 1979; Maylin *et. al*, 1987; Raghieb *et. al*, 1994; Schultheiss & Godley, 1995 and Sobocinski *et. al*, 1984). Baum *et. al* (1981) correlate positively the fluoride concentration in bone of affected animals with fluorine in air. In South Africa, severe dental lesions, lameness and exostoses were reported after consumption of water from bore well and pastures with high fluorine content (Botha *et. al*,

1993). A case of sub-acute fluorine poisoning following ingestion of superphosphate causing hypocalcaemia and hypomagnesaemia was noticed in New Zealand (Clark *et. al*, 1976) whereas an outbreak of acute diarrhoea characterized by watery and bloody faeces with mortality in draught cattle was attributed to acute fluorine poisoning caused by the chemical fertilizers in China (Wang,1986). In an experimental exposure of fluorine in goat, high fluorine content was seen in the blood and also in bones and teeth, while muscles and milk showed very little (Crombet, 1980). Krook and Maylin (1979) recorded stunted growth and severe dental fluorosis where cows died, or had to be slaughtered after the third pregnancy in Canada. Mehedintu *et. al* (2000) found anaemia, hypocalcaemia and hypophosphataemia with elevated serum alkaline phosphatase in fluorosis in cattle. In Poland, cough, diarrhoea, salivation, skin lesions, anorexia, infertility and reduced milk yield were observed in dairy herds situated surrounding 5 km radius of an aluminium processing plant (Sobocinski *et. al*, 1984). Similarly, Raghiv *et. al* (1994) found general emaciation with stunted growth and rough falling hair, respiratory and digestive diseases and dental fluorosis in dairy Friesian cows nearby a aluminium factory in Egypt. Severely affected cases showed mottled teeth with bulging gums and periodontitis.

In India, Swarup and Singh (1989) made an epidemiological survey of the bovine population in a brick kiln congested locality with signs of lameness. Lameness having 'knee posture', reduced appetite, loss in general body condition, loss of hair with stiff rough skin coat, poor performance, anemia, mottled teeth, enlarged mandible, painful protuberances of metatarsal, metacarpal and facial bones were recorded. On the basis of clinical and circumstantial evidence, the condition was suspected to be fluorosis, possibly due to environmental pollution with fluorine-bearing smoke arising from the large number of brick kilns. Similar conditions were also noted in Ghaziabad by Singh and Swarup (1994) along with decreased milk yield in buffaloes and in cattle by Jagadish *et.al* (1998). Similar scenarios were also noted by other researchers (Patra *et.al*, 2000 and Swarup *et.al*, 2001) in Udaipur.

Kapoor et. al (1993) made an experiment on Twenty male calves (Thaparker X Holstein) by feeding them with sodium fluoride for 4.5 months, but did not get any clinical symptoms. Histopathologically, changes were noted in the liver, kidney and metatarsal bones of calves given higher doses. The liver showed centrilobular necrosis and mononuclear infiltration in the portal triad area. The kidneys showed atrophied glomeruli, peri-glomerular fibrosis, and tubular necrosis. Microsections of the metatarsal bones showed osteoporotic changes and periosteal hyperostosis indicating the first changes of fluorosis.

Dwivedi et. al (1997) found that the fluorosis in buffaloes is related with drinking water containing toxic levels of fluoride. In a human fluorosis endemic area of Punjab, overall prevalence of 7.71 % fluorosis in cattle and buffalo was recorded where fluorosis was associated with toxic fluoride level (0.42 ppm) in underground water. Dental mottling, lameness and higher respiration and heart rates were common symptoms. Anaemia, reproductive disorders like prolonged postpartum anoestrus and increased incidence of vaginal prolapse were also recorded (Sharma et. al, 1997). Maiti et. al (2003) in Orissa concluded that there is a positive correlation between prevalence of dental fluorosis in cattle and fluoride content of ground water. Cases of repeat breeding due to fluorosis in cattle in Bihar and Rajasthan were also noted by Ranjan et. al (2009).

Choubisa (1998) noted that buffaloes were found to have a higher prevalence and greater severity of dental and skeletal fluorosis when compared to cattle. The prevalence of dental fluorosis was higher in calves of both cattle and buffalo than in adults. At a fluoride concentration of 4.0 ppm in water, 100% of calves, 65.6% of buffaloes and 61.0% of cattle were affected with dental fluorosis to some degree. Intermittent lameness was observed in animals above 7 years of age at 2.8 ppm fluoride or more in the water (Choubisa, 1999). It was also found that prevalence of dental fluorosis is relatively higher in calves due to greater sensitivity and less tolerance to fluoride. In Dungarpur district of Rajasthan, evidence of osteo-dental fluorosis in mature animals with prevalence rate of dental and skeletal fluorosis were reported in mature

animals, being 55.9% and 48.3% respectively in buffaloes, followed by 48.0% and 39.8% in cattle, 10.7% and 8.4% in goats, 7.3% and 5.6% in sheep, and 5.3% and 5.3% in camels (Choubisa, 2011) was reported.

In acute intoxication cases, there is formation of hydrofluoric acid which causes irritation of stomach and results in ruminal stasis, constipation or gastroenteritis. Characteristic nervous signs including muscle tremor, weakness, pupillary dilatation, hyperesthesia and constant chewing occur, followed by tetany, collapse and death within a few hours.

In dental form, lesions do not occur in deciduous as well as permanent teeth that have grown prior to poisoning. In cattle lateral incisors show most marked changes. Mottling of tooth enamel, erosion of teeth, with the appearance of pigmented spots, opaque chalk like areas, very light yellow, green, brown or black colored spots having linear pigmented streaks and pits or bands arranged horizontally across the teeth are the earliest sign. Multiple dental caries is a constant feature of this poisoning.

In chronic poisoning, anemia is observed due to suppression of haemopoietic activity of the bone marrow and interference with the mineralization process. Reduced milk yield is observed when fluorine intake level is ranged between 150 – 200 ppm. Adverse effects on reproduction, significant increase in post calving anestrous, decline in fertility has been observed in cows receiving a diet containing 8-12 ppm fluorine for a year. (Ulemale *et. al*, 2010)

Fig. 1. Mottling consisting of diffuse yellow brownish discolouration in streaks and spots with appearance of chipped incisor teeth. (Trangadia et. al, 2011)



Fig.2: Moroccan cow with fluorosis, from industrial contamination

(Source: [http://en.wikipedia.org/wiki/File:Fluor%C3%B4ze\\_egzostozes1-800h.jpg](http://en.wikipedia.org/wiki/File:Fluor%C3%B4ze_egzostozes1-800h.jpg))



**Diagnosis:**

The following criteria may be helpful to recognize a developing fluoride toxicosis

1. Determination of the amount of fluorine in the diet, urine, bones, and teeth by chemical analyses;

2. Effects on tooth, in animals exposed at time of permanent teeth development;
3. Lameness, as the result of fluoride accumulation in bone; and
4. Systemic evidence as reflected by anorexia, inanition, and cachexia etc.

The normal levels of fluorine in livestock:

- <0.2 ppm in plasma,
- 1-8 ppm in urine,
- 200-600 ppm in bones, and
- 200-500 ppm in teeth.
- <5 ppm in urine

In borderline toxicity, urine contains 20-30 ppm, systemic signs appear >35 ppm. In cattle, toxicosis is associated with levels of >5,500 ppm in compact bone and >7,000 ppm in cancellous bone). (

<http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/211000.htm>)

**Treatment and Control:**

Acute toxicity:

Calcium gluconate (IV) and oral magnesium hydroxide or milk to bind fluoride before absorption is advocated.

Chronic toxicity:

Removal of the animal from affected areas helps in control of fluorosis. Feeding of calcium carbonate, aluminum oxide, aluminum sulfate, magnesium metasilicate, or boron may decrease absorption or increased excretion of fluoride, which may help in control of chronic fluorosis. However, no treatment has been shown to cure the chronic effects of fluorine toxicity.

<http://www.merckvetmanual.com/mvm/index.jsp?cfile=htm/bc/211000.htm>)

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