

## PREVALENCE OF ENDEMIC FLUOROSIS WITH GASTROINTESTINAL MANIFESTATIONS IN PEOPLE LIVING IN SOME NORTH-INDIAN VILLAGES

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**SUMMARY:** Numerous reports on epidemiological surveys of skeletal and dental fluorosis exist, but information is quite limited on non-skeletal manifestations of fluoride toxicity. The present study was conducted to assess the prevalence and severity of non-skeletal manifestations, especially gastrointestinal disturbances, in an area of skeletal and dental fluorosis.

The subjects, numbering 1958 inhabitants belonging to 489 families residing in four endemic villages of Faridabad District of Haryana State, were interviewed on health complaints. The information was recorded in a pre-coded questionnaire. Every drinking water source was analysed for fluoride content. This led to the identification of "safe" (fluoride 1 ppm or less) and "contaminated" (fluoride above 1 ppm) sources of water.

Results revealed that among the subjects were people affected with: 1) Dental fluorosis (58%), 2) Skeletal fluorosis (27%), 3) Non-skeletal manifestations (41%) and 4) Gastrointestinal complaints (26%). Those affected were consuming water contaminated with fluoride ranging from 0.25-8.00 ppm. Among the total of 78 sources of water, 20 were "safe" while the remaining 58 were more contaminated with fluoride from natural sources.

It is concluded that in an endemic zone, where the inhabitants are consuming water of high fluoride content, the occurrence of gastrointestinal complaints - viz., loss of appetite, nausea, abdominal pain, flatulence, constipation and intermittent diarrhoea - is one of the early warning signs of fluoride toxicity and fluorosis. When water with negligible amounts of fluoride (safe water) is provided, the complaints disappear within a fortnight.

**Key words:** Dental fluorosis; Endemic fluorosis; Epidemiology; Faridabad, Haryana (India); Gastrointestinal disturbances; Non-ulcer dyspepsia.

### Introduction

Endemic fluorosis is a form of chronic fluoride intoxication resulting from ingestion of excessive quantities of fluoride through drinking water. This form of chronic intoxication was first described in one of the southern States of India in 1937 (1,2). Cases of endemic fluorosis have been reported sporadically from almost all parts of the world, particularly from China (3), Japan (4), South Africa (5), North Africa (6), Argentina (7), the Persian Gulf (8), Saudi Arabia (9), United States of America (10,11), Canada (12) and Europe (13,14). Shortt *et al* (1,2) were the pioneers in recognizing the disease, from cases of dental and skeletal fluorosis among residents of Nellore District in Andhra Pradesh. Subsequently the condition was associated with ingestion of fluoride in drinking water. Pandit *et al* (15) made a comprehensive study in this area dealing with the etiological aspect. Daver (16)

reported endemic fluorosis from Hyderabad. Khan and Wig (17) reported chronic fluoride toxicity with bone affliction in Punjab. Siddiqui (18) noted symptoms of fluoride toxicity in immigrants within 1-4 years of entering an endemic village. Singh *et al* have reported extensively on dental, skeletal and neurological aspects of the disease (19,20). Anand *et al* (21) described cases of endemic fluorosis in the Delhi region. Jolly *et al* (22,23) studied dental fluorosis in schoolchildren and tried to correlate the incidence with the water fluoride level.

The large quantity of drinking water consumed in hot arid climates is supposed to contribute to a higher daily intake of fluoride resulting in incidence of clinical fluorosis (24,25). The work of Brouwer *et al* indicated that in the hot climate of Senegal both dental and skeletal fluorosis are more prevalent and severe than would be expected from the fluoride concentration in drinking water (26). Fisher *et al* (27) have reported a case of spinal cord compression with paraplegia as a result of endemic skeletal fluorosis. An epidemiological study by Evans (28) reported the dependence of dental fluorosis on fluoride exposure during the critical period of tooth development.

The major pathway by which fluoride enters the circulation is by absorption from the gastric and duodenal mucosa (29). Unlike most substances, fluoride can be absorbed in appreciable amounts from the stomach (30), which is why gastric and intestinal disorders are noticed in most of the cases of osteofluorosis (31). Gastrointestinal problems, most commonly abdominal pain, vomiting, nausea and anorexia, have recently been reported in fluorosis patients and patients on sodium fluoride therapy (32). The effect of a single dose of fluoride was tested in 12 healthy male and female volunteers who underwent endoscopies two hours after consuming fluoride (33). The stomach was videotaped and examined. Mucosal injury and structural damage were observed. However, no epidemiological survey on non-skeletal manifestations of fluorosis, with a focus on gastro-intestinal disturbances, has appeared so far. The present study was undertaken to assess the prevalence and severity of skeletal and dental fluorosis, and accompanying non-skeletal manifestations with gastrointestinal disturbances, among the people of four villages in Faridabad District of Haryana State.

### Material and Methods

The four villages Samaypur, Karnera, Sikrona and Bhanakpur of the Faridabad District were chosen randomly for survey work, because large numbers of patients from that area with backache, joint pain and pain in the neck and hip region, later diagnosed as cases of skeletal fluorosis, had visited the Outpatients Department of the All India Institute of Medical Sciences Hospital. A door to door survey with face-to-face interviews was carried out. The information collected was entered on a pre-coded questionnaire. Health complaints related to dental fluorosis, skeletal fluorosis, and non-skeletal manifestations, including gastrointestinal complaints, were recorded.

The teeth were examined for characteristic mottling and pigmentation, *viz.*, yellow-white patches, brown streaks or black patches on the enamel surface, and pitted, perforated or chipped-off enamel. Information was recorded on complaints of severe pain and the rigidity of back-bone, joints and neck and hip region through a set of simple tests illustrated in the Figure. Besides non-skeletal manifestations, *viz.*, aches and stiffness of muscles, muscle weakness, tingling sensation in hands

and feet, polydipsia and polyuria were also recorded. Complaints of abdominal pain, constipation, intermittent diarrhoea, bloated feeling, loss of appetite, feeling of nausea, and mouth sores were recorded under gastrointestinal disturbances.

Water samples were collected from all sources from each village. Fluoride estimation was done on an ION 85 ION ANALYZER (Radiometer, Copenhagen). The main sources of drinking water in these villages are open wells, hand pumps and municipal supply.

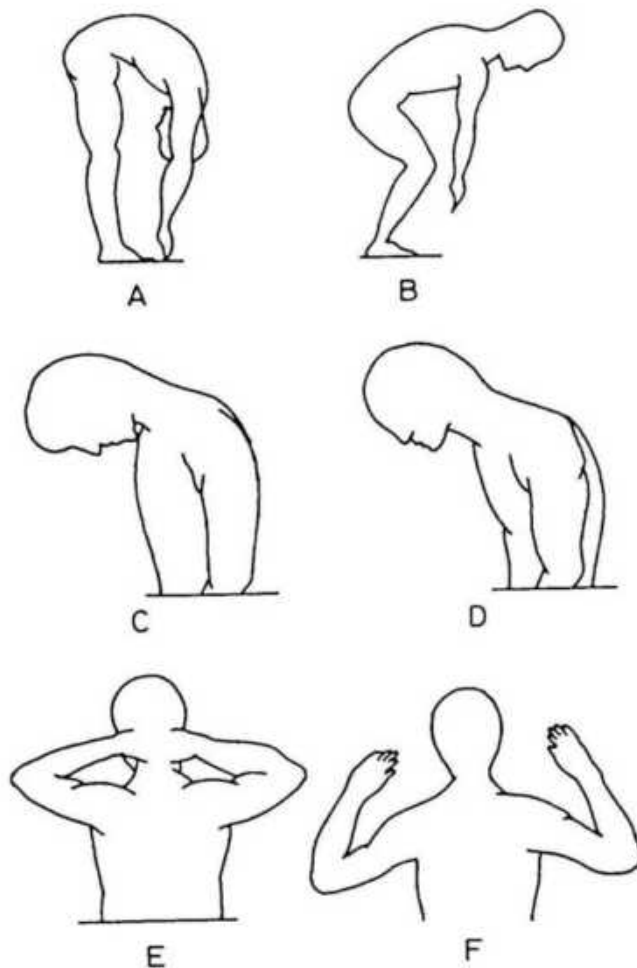
#### FIGURE LEGENDS

##### Normal healthy individual:

- A Can bend body and touch the floor/toes.
- C Can touch chest with chin.
- E Can stretch hands, fold arms and touch back of head.

##### Fluoride toxicity manifestation:

- B Unable to bend without folding knees.
- D Unable to bend neck - touching chest with chin not possible.
- F Unable to stretch hands, fold arms and touch back of head.



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## Results and Discussion

The results are summarized in Tables 1 and 2

TABLE 1  
Water quality and health status in four villages of Faridabad District

Village:	SAMAYPUR	KARNERA	SIKRONA	BHANAKPUR
Total families surveyed	89	79	99	222
Total population surveyed	288	315	518	837
Total no. and percent of cases:				
of dental fluorosis	190 65.9%	275 87%	317 61%	353 42%
with skeletal fluorosis	163 56.6%	135 42.8%	94 18%	141 16.8%
with non-skeletal manifestations	184 63.9%	214 67.9%	135 26%	264 31.5%
with gastrointestinal manifestations	151 52.4%	155 49%	61 11.7%	143 17%
Total no. of water sources	36	32	6	4
No. of safe water courses	11	4	2	3
Fluoride content of water:				
Min.	0.25 ppm	0.3 ppm	0.3 ppm	0.7 ppm
Max.	8.0 ppm	7.0 ppm	5.4 ppm	1.6 ppm
Mean	3.2 ppm	3.7 ppm	2.5 ppm	1.0 ppm

TABLE 2  
Summary of observations

No. of villages:	4		
No. of families:	489		
Total population surveyed	1953		
	Number	Percentage	
Total no. of cases with dental fluorosis	1135	58	
Total no. of cases with skeletal fluorosis	533	27	
Total no. of cases with non-skeletal manifestations	797	41	
Total no. of cases with gastrointestinal manifestations	510	26	
Total no. of water sources:	78		
Total no. of safe water sources	20		
Fluoride content in water:	Minimum	0.25 ppm	
	Maximum	8.0 ppm	

### Dental fluorosis

Mottled enamel or dental fluorosis is a well recognized entity (34,35) and one of the overtly visible signs of excessive intake of fluoride during the period of teeth eruption. In this survey the overall prevalence of dental fluorosis in the four villages was 58%. Fluoride concentration in drinking water ranged between 0.25 and 8.0 ppm. The highest prevalence of dental fluorosis, 87%, was found in Kamra village where the drinking water fluoride was in the range of 0.3 - 7.0 ppm.

Ray *et al* (36) reported 28.21% dental fluorosis in a rural area of Varanasi. Desai *et al* (37) observed 35.3% dental fluorosis among 4544 tribals from 24 villages. In a locality near Punjab, where water fluoride concentration was 9.7 ppm, 70.7% of the population had dental fluorosis (22). In the present survey the prevalence of dental fluorosis was 42% where the upper limit of fluoride in drinking water sources was 1.6 ppm, which is close to the upper permissible limit of 1.0 ppm (Table 1). Manji *et al* (38) found high prevalences and severity of dental fluorosis in Kenya at low water-fluoride levels 0.1-1.0 ppm. Smith and Hodge (39) reported that 2 ppm fluoride caused mottled enamel. Ray *et al* (36) observed dental fluorosis at 0.4 ppm fluoride in drinking water. Brouwer *et al* (26) have shown dental fluorosis in children in Senegal where water-fluoride ranged from 0.1 to 7.4 ppm, with mild dental fluorosis prevalence of 68.5% at 1.0 ppm. When fluoride content exceeded 4 ppm, dental fluorosis prevalence reached 100%.

### Skeletal fluorosis

The present survey revealed that overall prevalence of cases of skeletal fluorosis in the four villages, verified through 3 physical tests, was 27% (Table 2 and Figure). The results clearly showed (Table 1) that the incidence of skeletal fluorosis was dependent on fluoride concentrations in drinking water. These observations are in accordance with the observations made earlier by Jolly *et al* (22,23), who reported 2.4% skeletal fluorosis prevalence at drinking water fluoride concentrations of 0.9-2.5 ppm and 70.7% prevalence at concentrations of 6.0-16.2 ppm. Singh and Jolly (40), on the basis of extensive epidemiological surveys, found that crippling fluorosis resulted from continuous daily intake of 20-80 mg fluoride for 10-20 years. However, in India skeletal fluorosis has been reported at very low levels of fluoride intake for shorter duration (20,22). In some studies in tropical countries reviewed by the Royal College of Physicians (15,23,41) relatively marked osteofluorotic symptoms were found with drinking water fluoride levels of 1.0-3.0 ppm.

An Indian report in a 1970 WHO publication indicated that one can be afflicted with fluorosis by drinking water contaminated with 20 ppm fluoride for ten years (40). The fact remains that even 2 ppm fluoride contaminated water consumed for two years can cause crippling fluorosis, if the calcium content of the water is low and alkalinity is high (42). A recent epidemiological survey conducted by Teotia and Teotia (1990) showed that dental and skeletal fluorosis occur in rural areas of Uttar Pradesh where the fluoride content of drinking water is only 0.6 ppm. They also reported that consuming fluoride over a period of six months to one year is adequate for the onset of manifestations of skeletal fluorosis (43,44).

It can be concluded that fluoride in drinking water is an important disease factor. In fact, skeletal fluorosis and its associated manifestations can develop following ingestion of fluoride within "permissible" limits. This observation suggests that fluoride can enter the body from other sources besides drinking water and that the effects can be aggravated by other factors (15,20,41,43) *e.g.* low calcium, high water alkalinity, and dietary deficiency of calcium and vitamin C besides the hormonal profile.

### Non-skeletal manifestations

In the present survey the overall incidence of non-skeletal manifestations in the four villages was 41% (Table 2). The maximum prevalence was 67.9%, observed when the mean value of fluoride in drinking water was 3.7 ppm. A prevalence of 63.9% was observed when the mean water fluoride value was 3.2 ppm, and a prevalence of 26% at mean of 2.5 ppm. However, a 31.5% prevalence of non-skeletal manifestations was also observed at a mean water fluoride level of only 1.0 ppm (Table 1).

In Faridabad District, severe gastro-intestinal problems have been observed. The overall prevalence of non-ulcer dyspeptic symptoms in the four villages was 26%. The highest prevalence was found in one village which had 52.4% with gastrointestinal problems at fluoride levels ranging from 0.25 to 8.0 ppm. Waldbott (45) had also reported that 47% of fluorosis patients in Sicily were affected with gastrointestinal problems. The extent of fluoride absorption from the stomach has implications in that gastric acidity enhances both the absorption and the toxicity of fluoride (30). Susheela reported that gastro-intestinal complaints are an early warning sign of fluoride toxicity and fluorosis (46).

It has also been shown by Susheela and Das (47) that in rabbits subjected to oral administration of NaF at the dose of 10mg/kg body weight for a period of 24 months fluoride toxicity destroys the gastro-intestinal mucosa, *i.e.* causes loss of microvilli, loss of mucus, and surface abrasions due to epithelial cell degeneration. Susheela and Kumar (48) have also reported damage and abrasion of epithelial cells of mucosa of systems other than gastrointestinal, *viz.* vas deferens and ductuli efferentis of rabbits after oral administration of NaF at 10mg/kg body weight daily for varying time intervals. The authors also reported a significant reduction in mucus droplets in fluoride treated animal both in the gastrointestinal tract and vas deferens.

A recent study (32) on long term ingestion of fluoride by human patients from endemic areas and patients on sodium therapy for otosclerosis revealed non-ulcer dyspeptic symptoms in 70% of the subjects. In upper gastrointestinal endoscopy, petechiae, erosion, and erythema were seen in all patients compared to normal healthy controls. The biopsy material obtained, when examined under the scanning electron



microscope, revealed mucosal abnormalities. The observation suggests that gastrointestinal complaints comprising non-ulcer dyspeptic symptoms in endemic areas can be caused by ingestion of excess fluoride. We have observed that, when such patients revert to safe drinking water either in an endemic area or while in hospital, they are relieved of the dyspeptic symptoms and complaints within 2-3 weeks.

This is one of the first reports revealing the results of fluoride toxicity after a systematic epidemiological survey and water quality analysis. It provides evidence, supported by laboratory and other clinical investigations including endoscopy, which suggests that gastrointestinal complaints are early warning signs of fluoride toxicity and fluorosis in an endemic area.

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