DIAGNOSIS OF FLUOROSIS AND RECOVERY THROUGH EASY TO PRACTISE INTERVENTIONS

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ABSTRACT: The objectives of the present study were to highlight (i) how to diagnose fluorosis, due to fluoride ion (F) toxicity, in out-patient departments, by retrieving the history and through tests and (ii) the interventions available for recovery. In patients suspected of fluorosis, the F levels were investigated in samples of drinking water and body fluids (serum and urine), and fore-arm X-ray radiographs taken to assess the presence of interosseous membrane calcification. The haemoglobin (Hb) was tested for monitoring purposes. With the results obtained and the history retrieved, a correct diagnosis of fluorosis was arrived at. The meaning of any deviations in the results are discussed in three case studies. Recovery was obtained through two corrective measures: (i) diet editing and (ii) diet counselling. In diet editing F ingestion from all sources was withdrawn and in diet counselling an adequate consumption of essential nutrients, vitamins, antioxidants, and micronutrients through dietary sources was promoted. Pharmaceutical products were not recommended as recovery with them is slow. The first reassessment, scheduled at 4–6 weeks post-intervention, was a confidence building exercise for the patient. The disappearance of the health complaints and the subsequent recovery were related to the reduction in the F levels in the urine and a rise in the Hb. The study provides an overview of the clinical manifestations of fluorosis, diagnostic tests, differential diagnosis, interventions practised, monitoring, and recovery from the disease. The report highlights that kidney failure may occur in F toxicity and that diagnostic tests for fluorosis are helpful in the understanding of the occurrence of renal failure in association with fluorosis.

Key words: Case studies; Diagnosis; Fluorosis; Interventions; Monitoring; Recovery;

INTRODUCTION

Fluorosis is a public health problem caused by the consumption of fluoride ions (F) through water, food, and a variety of other sources.[1-3] It is a slow progressive crippling disease.[4] Every organ, tissue, and cell in the body may be affected and result in health complaints which have manifestations which overlap with those of other diseases.[5] The duration required for the clinical manifestations to appear varies depending on the susceptibility of an individual to F. The diagnosis of fluorosis may be made through X-ray radiographs revealing ligamental calcification, enhanced bone density, and increased bone mass. The diagnosis of fluorosis at the late stages of the condition is not helpful as the bone deformities cannot be reversed. Similarly, dental fluorosis can be recognized by the presence of overtly visible discoloured teeth, the occurrence of pitted, perforated, demineralized, and chipped teeth in children, and the loss of permanent teeth in adults.[6-9] The presence of dental fluorosis is an indication that there was exposure to F in intrauterine life or in childhood. The discolouration of the teeth cannot be removed except by masking. The extended effects of dental fluorosis remained unknown until recent years. [10] The medical and dental fraternity were dependent on the clinical manifestations of fluorosis for making the diagnosis. These two forms of fluorosis appeared at the advanced stages of F toxicity when there was very little scope for recovery or treatment. This led to

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the concept among ailing communities and clinicians until recent years that fluorosis was untreatable and that there is no mitigation available for the disease. Neglect of the disease loomed large. Changing to a focus on clinical symptoms to suspect the diagnosis of fluorosis based on soft tissue manifestations was a major break-through for addressing the disease.[11,12] The earliest symptoms of F toxicity manifest themselves in the form of (i) digestive tract symptoms with inflammatory bowel disease (IBD) / non-ulcer dyspepsia (NUD),[13,14] (ii) kidney problems resulting in polyuria and polydipsia, (iii) muscle weakness and loss of muscle power,[15,16] (iv) fatigue and tiredness with low Hb,[12] and (v) pain in the joints.

F toxicity may be suspected when the earliest symptoms suggestive of F toxicity occur and then be diagnosed with a battery of tests. When the diagnosis is made, the patient may be introduced to appropriate interventions allowing full recovery to occur and the patient's return to normal life. Due to an unawareness of the manifestations of F toxicity, the symptoms of toxicity have often considered to be 'non-specific' and disregarded leading to the disease remaining undiagnosed or misdiagnosed and the agony of the patient continuing. Temporary relief of joint pain may be obtained with treatment with analgesics but, as the ingestion of F continues, the fluorosis is aggravated resulting in restricted movements and rigidity of the joints.

The objectives of this communication are to give information in three areas: (i) how to suspect fluorosis while retrieving the history in out-patient departments (OPDs); (ii) how to diagnose the disease correctly, and (iii) how to introduce interventions and monitor for recovery.

MATERIALS AND METHODS

Patients with complaints suggestive of fluorosis were referred to the Fluorosis Foundation of India by a few clinicians in hospitals in Delhi and the neighbouring states who were aware of the developments in recent years in the understanding of fluorosis. Referrals also came from overseas patients through the internet who suspected that they had fluorosis. We reviewed the reports generated for 58 patients referred during 2012–2016. Thirty-three of the patients were males aged 17–72 years and 25 were females aged 26–61 years. Two of the case studies described are of patients from earlier years who were monitored for a longer duration (3–5.5 yr).

A. DIAGNOSTIC PROCEDURE FOR THE EARLY DETECTION OF FLUOROSIS:

Retrieval of history: The history of the patients was retrieved using a pre-coded proforma in which a record was made of the place of residence, village / town, district, state, duration of stay, occupation, source of drinking and cooking water, and health complaints. The responses of the patients to the questions about the presence of a particular health complaint pertaining to non-skeletal fluorosis, skeletal fluorosis, and dental fluorosis, and dietary status regarding F consumption, were recorded on the pre-coded proforma with 1–3 ticks or a minus symbol ($\sqrt{}\sqrt{}\sqrt{}$ = severe, $\sqrt{}\sqrt{}$ = mild, $\sqrt{}$ = less, – = none) (Table 1).

Table 1. The pre-coded proforma used for the retrieval of the patients' history and dietary habits

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1. Personal information:

    (i)   Name: ––––––––––––––––––––––––––––––––– Age: –––––– Yrs. Sex: M / F

    (ii)   Place of Birth: (a) Village / Town: –––––––––––––– (b) District: ––––––– (c) State: ––––––––––

    (iii)   Duration of stay (in yr.): –––––––––––––– (iv) Occupation: ––––––––––––––––––––––––

    (v)   Source of drinking water (Tick √): Hand pump □ Open well □ Tube well □

                 Bore well □ River water □ Supply water □ RO Filter □ Others (specify, if any): –––––––––

2. Health Complaints pertaining to   Tick (√), if YES

   A. Non-Skeletal Fluorosis:

		Baseline*	Monitoring*
(i)	Pain in the stomach	□ □ □	□ □ □
(ii)	Gas formation	□ □ □	□ □ □
(iii)	Nausea / Loss of appetite	□ □ □	□ □ □
(iv)	Constipation with intermittent diarrhea	□ □ □	□ □ □
(v)	Polyuria (Frequent tendency to urinate)	□ □ □	□ □ □
(vi)	Polydipsia (Excessive thirst)	□ □ □	□ □ □
(vii)	Fatigue / Tiredness / muscle weakness	□ □ □	□ □ □
(viii)	Anemia / low Hb	□ □ □	□ □ □
(ix)	Headache	□ □ □	□ □ □
(x)	Allergic reaction on the skin	□ □ □	□ □ □
(xi)	Repeated abortion / still birth (in case of female)	□	□
(xii)	Infertility in case male	□	□

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* Severe: √√√   Mild: √√   Less: √   None: (−)

Table 1 (continued). The pre-coded proforma used for the retrieval of the patients' history and dietary habits

2. Health Complaints pertaining to		Tick (√), if YES	
B. Skeletal manifestations (Pain in major joints / stiffness):		Baseline*	Monitoring*
(i)	Neck	□ □ □	□ □ □
(ii)	Shoulder	□ □ □	□ □ □
(iii)	Elbow	□ □ □	□ □ □
(iv)	Back (Lumber spine)	□ □ □	□ □ □
(v)	Hip (Pelvic region)	□ □ □	□ □ □
(vi)	Knee	□ □ □	□ □ □
(vii)	Restricted movements	□ □ □	□ □ □
(viii)	Unable to stand straight or walk normally	□ □ □	□ □ □
(ix)	Gait & Posture - Abnormal	□ □ □	□ □ □
(x)	Inability to squat	□ □ □	□ □ □
C. Dental Fluorosis (Discoloration on the enamel surface):			
(i)	Horizontal streaks / spots, away from the gums and bilaterally symmetrical	□ □ □	□ □ □
(ii)	Pitting and perforation on enamel surface	□ □ □	□ □ □
(iii)	Chipping off teeth	□ □ □	□ □ □
(iv)	Loss of teeth in adults	□ □ □	□ □ □
3. Dietary status of the patient (whether consuming / using the following items):			
(i)	Black Rock Salt / Sandha Namak / Vrat Ka Namak / Lahori Namak	□	□
(ii)	Pickles, snacks, street foods, spices, ayurvedic tablets (Churans) laced with black rock salt	□	□
(iii)	Black tea / lemon tea / Canned fruit juice / soft drinks	□	□
(iv)	Chewing of Tobacco / Areca nut (Supari)	□	□
(v)	Fluoridated toothpaste	□	□
(vi)	F containing drugs	□	□

* Severe: √√√   Mild: √√   Less: √   None: (−)

Diagnostic tests: After collecting the history of the patient, five essential laboratory tests were carried out:

(i) Fluoride level in the blood (serum):[17] Approximately 4–5 mL of blood was drawn and allowed to clot for 30 min at room temperature followed by centrifugation at 3000 rpm for 10 min, and the separation of the serum into a plastic vial.

(ii) Fluoride level in the urine:[18] The spot urine sample was drawn at any time during the day.

(iii) Fluoride level in the drinking water:[19]

The body fluid and drinking water samples were collected in plastic bottles only, not in glass bottles. The labelling was done on adhesive tape stuck on the bottle with the details written using pencil rather than not ink to avoid smudging. The F level was estimated by the potentiometric method using Ion Selective Electrode (ISE) technology. The results were reported in mg F/L.

(iv) Forearm X-ray radiograph: This was taken in a hospital when a patient complained of joint pain. If a forearm X-ray radiograph not taken as part of the routine hospital procedures, a special request for this investigation was made. The patient was advised to keep the forearm with 5 fingers flat on the table with the palm facing towards the source of the X-ray exposure. It is a requirement that the radius and the ulna remain parallel to one another. Ectopic calcification is suggested if interosseous membrane calcification is present along the margin of the two bones and it may vary in shape from a wavy outline to thorny projections. The presence of calcification of the forearm interosseous membrane is useful for the differential diagnosis of fluorosis from other bone disorders such as arthritis, osteoporosis, osteomalacia, and ankylosing spondylitis, where ectopic calcification is not known to occur.

(v) Haemoglobin (Hb) level: A finger was pricked using a sterile lancet and the Hb was estimated from a drop of blood (the 3rd drop preferably) using a portable battery-operated Haemoglobinometer.[20,21] The Hb was used for assessing the absorption of nutrients from the diet following the practising of the interventions. The results were expressed in g of Hb/dL.

B. MITIGATION OF THE DISEASE THROUGH INTERVENTIONS:

After the laboratory tests were carried out, a report was generated. The patient was informed of the results and the F levels in her/his body fluids and drinking water were explained. The normal reference range for the F content in serum (SFL) and urine (UFL) were considered to be 0.02–0.05 mg/L and 0.1–1.0 mg/L, respectively, while for drinking water (DWFL) the upper limit was considered to be 1.0 mg F/L with the proviso of the less the better as F is injurious to health (as per the Bureau of Indian Standards [BIS] guidelines). The normal Hb levels for men and women were ≥13.0 g/dL and ≥12.0 g/dL, respectively. The patients were advised to practise two interventions for recovery from the disease. The two interventions introduced were the following:

(i) Intervention 1—diet editing: Diet editing was introduced to avoid further consumption of F, so that the disease progression was stopped.

(a) If on reviewing the data on the F content in the drinking water, the F level was found to be beyond the normal safe limits (i.e., the level was >1.0 mg/L), the patient was informed and asked to bring additional water samples for testing so that they could shift to an existing safe source of water from their neighbourhood for drinking and cooking purposes. If no safe source could be found, the patient was advised to install a Reverse Osmosis Filter (RO) in their supply line.

(b) If the drinking water F was below 1.0 mg/L (and therefore safe) but the urinary F was high (above 1.0 mg/L), the source(s) of F was traced through retrieving information on the diet and the dietary habits in order to find the food item(s) being consumed which contained a high F content. Sources known to contain a high level of F, such as black rock salt[22] were also edited to eliminate them as a source of entry of the poison.

(c) Editing the use of toothpaste was also required.[23] The patient was advised to use an ayurvedic or a herbal paste with the least amount of F and to squeeze a very small amount (size of a pea) on to the brush for brushing. Children are known to eat the paste and adults can absorb F instantaneously through the sublingual blood vessels, which is a route of drug administration, leading to an enhanced F level in the systematic circulation.

(ii) Intervention 2—diet counselling: Diet counselling was offered for promoting the intake of essential nutrients (viz,. calcium, iron, vitamin C, vitamin E, vitamin $B_{12,}$ other anti-oxidants, and micronutrients) through the consumption of dairy products, vegetables, and fruits.[24]

The diet editing and diet counselling were reinforced by providing a pictorial booklet illustrating the various food recommendations, viz., items that need to be eliminated due to F contamination and how to have a intake, where possible, of larger portions of fruits, vegetables, and dairy products, including through the use of juices, salads, and soups.[25] The guidance and counselling offered helped an understanding of the role of diet in recovery from the ailment to be achieved by a variety of families: rural and urban, poor and rich, and educated and uneducated. The interventions resulted in changes in food consumption and life style.

Patient care and management: After commencing the practice of the two interventions, the patients were monitored closely for 4–6 weeks, and twice more after a longer interval, by measuring the UFL and the Hb. When the interventions were practised well, the UFL reduced below the normal reference range and the Hb content in blood was enhanced. The Hb level was an excellent indicator for assessing the health improvement.[26] The health complaints initially recorded in Table 1, and those that disappeared after the practice of the interventions were noted in an adjacent column together with the duration of the monitoring. Most of the patients came on their own for the first monitoring but some needed to be reminded. Some patients reported again to us after a couple of years due to recurrence of the health complaints.

RESULTS AND DISCUSSION

After receiving the test reports, they were reviewed critically and the levels of F in the body fluids and drinking water were explained to the patient. Most importantly, when the levels were elevated, it was made abundantly clear that F was a poisonous chemical when consumed for a long period of time and that the levels were raised

beyond the normal range. F-induced ectopic calcification was confirmed when calcification was found in interosseous membrane between the radius and ulna in the forearm.   A wavy outline of the calcification in the interosseous membrane was present at the early stages of the calcification. In some, when the exposure to F was for a longer duration, the radius and ulna showed enhanced bone mass and density, When these test results confirmed that the disease was fluorosis, the values of the SFL and the UFL in the test report were shown to the patient and it was explained that the SFL and the UFL levels need to be reduced for recovery.   The patient was advised that way forward was to practise the interventions to reduce the F towards the lower limit of the reference range, as the lesser the amount of the poison in body fluids, the better it would be for their recovery. The importance and significance of the practice of the interventions, which focused on the elimination F and the promotion of nutrients through dietary sources, were therefore explained to the patient.
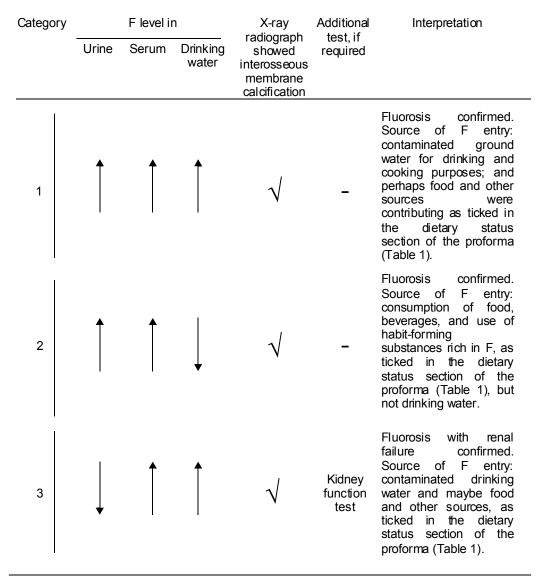
For a better understanding of the management-cum-monitoring based on the source of the F entry, the patients classified and summarised under three categories (Table 2).

The patients attending the OPDs were likely to fall under any one of the three categories, whichever hospital they visited. In all three categories of patients, some patients were sensitive to health complaints and hospital intervention was sought quite early. At this juncture, it was of paramount importance not to miss a case of F toxicity/fluorosis and we carefully followed the procedure reported in this communication, including recording personal information, the health complaints, and the dietary status (Table 1). The three categories were further substantiated with data obtained from testing the blood, urine, and drinking water samples and forearm X-Ray radiographs. In categories 1 and 2, the body fluids had a high F level and were suggestive of a high consumption of F. The drinking water F level was raised in category 1 but not in category 2. This led, for category 2 patients, to the suspicion that the F entry was through other sources. Determining these other sources was assisted by referring to the record of the patients' dietary history (Table 1). Reducing the F intake from the identified sources was dealt with during the diet editing and diet counselling. In the category 3 patients with fluorosis, the reduced level of F in the urine along with a raised level of F in serum, was suggestive of non-functional kidney tubules. To confirm this diagnosis of fluorosis with kidney failure, a renal function test was done.

Case studies:

The three case studies reported reveal the nature of the disease. The variation in the F levels in the body fluids depended upon a variety of factors, including hormonal status, body physiology, and nutritional status. The interpretation of the results of the three case studies can best be discussed by using the information provided in Table 2. In diagnosing fluorosis, testing the F level in three samples, serum, urine and drinking water, has a great advantage over testing for F in only the two samples of urine and drinking water. Testing on all three samples allowed information on other linked diseases, such as renal failure, to emerge.

Table 2. The three categories into which the patients were classified
and the interpretation of the test results

Category	F level in			X-ray radiograph showed interosseous membrane calcification	Additional test, if required	Interpretation
	Urine	Serum	Drinking water			
1	↑	↑	↑	√	–	Fluorosis confirmed. Source of F entry: contaminated ground water for drinking and cooking purposes; and perhaps food and other sources were contributing as ticked in the dietary status section of the proforma (Table 1).
2	↑	↑	↓	√	–	Fluorosis confirmed. Source of F entry: consumption of food, beverages, and use of habit-forming substances rich in F, as ticked in the dietary status section of the proforma (Table 1), but not drinking water.
3	↓	↑	↑	√	Kidney function test	Fluorosis with renal failure confirmed. Source of F entry: contaminated drinking water and maybe food and other sources, as ticked in the dietary status section of the proforma (Table 1).

Normal reference range of F:
      urine: = 0.1– 1.0 mg/L;
      serum: = 0.02– 0.05 mg/L;
      drinking water: permissible limit for F in drinking water = up to 1.0 mg/L, with the
            proviso that the less the better as F is injurious to health (as per
            Bureau of Indian Standards, 2012).

↑ = increase

↓ = decrease

√   = calcification of the forearm interosseous membrane present

The test results obtained during the baseline investigations allowed the diagnosis of fluorosis to be made and thereafter, during the post-intervention phase, they assisted with the monitoring of the disease. The protocol followed in the case studies of obtaining a history and making investigations removed the possibility that a patient might be undiagnosed or misdiagnosed. If an analgesic (pain killer) prescription was given to a patient reporting to hospital with severe joint pain without testing samples for F there would be a risk that some further adverse effects of fluorosis would become evident later. An analgesic prescription might be given for a short interval of a few days to a patient with severe joint pain while the results of the blood and urinary F tests were awaited to clarify whether the patient was suffering from F toxicity / fluorosis or any other bone disease. If a diagnosis of fluorosis was made, intervention with diet editing and diet counselling was commenced to withdraw the sources of F in order to prevent the disease progressing with the development of restricted movements, rigidity, and further pain, which might be irreversible.

The interventions practised were not laid down not as a uniform protocol but were patient specific. This is an important aspect of patient recovery.

Case study 1: A 44-yr-old housewife, from a village in Haryana state in India, responded with "YES" to a number of the health complaints in Table 1. The serum and urinary fluoride levels (SFL and UFL, respectively) were both raised (Table 3).

Table 3. Baseline and post-intervention follow-up data for the serum fluoride level (SFL), the urinary fluoride level (UFL), and the drinking water fluoride level (DWFL), together with the result of the forearm radiograph, for case study 1 of a 44-yr-old female consuming bore well water (BW), canal water (CW), and water from a reverse osmosis (RO) filter

Data for case study 1 of a 44-yr-old female				
	SFL (mg/L)	UFL (mg/L)	Forearm X-ray radiograph	DWFL (mg/L)
Baseline data:				
	0.08	8.0	Interosseous membrane calcified	3.00 (BW)
Monitoring (re-testing) data after the practice of the interventions:				
After 1 month (mo)	0.03	4.5		0.27 (CW)
After 4 mo	0.02	1.6		
After 6 mo	0.02	0.6		
After 4 yr 6 mo	0.086	2.66		2.39 (CW + BW)
After 5 yr 6 mo	0.161	2.49		0.210 (RO filter)
Normal reference range of F	0.02–0.05	0.1–1.0		Up to 1.0 mg/L, the less the better

BW = bore well; CW = canal water; RO = reverse osmosis

The source of the F entry was through the consumption of contaminated ground water, with 3.0 mg F/L, from a bore well. She was shifted to using canal water (safe water), and consequently, within 6 months, the SFL, the UFL and the health complaints reduced significantly compared to the baseline values. However, after 4½ years the patient returned with a recurrence of the health complaints, a SFL of 0.086 mg/L (normal range: 0.02–0.05 mg/L), and a UFL of 2.66 mg/L (normal range 0.1–1.0 mg/L) due to consumption of contaminated water with a high F content of 2.39 mg/L (normal range: up to 1.0 mg/L with the proviso of the less the better as F is injurious to health). The canal water was mixed with bore well water due to a water shortage and supplied through a pipeline by the Public Water Supply Dept. She was then advised to acquire a reverse osmosis (RO) filter and fix it to the supply line. This supplied safe water with a fluoride content of 0.210 mg/L (normal range: up to 1.0 mg/L with the proviso of the less the better). With her having suffered and recovered from F poisoning earlier, she knew what to do and further diet editing and diet counselling were not required for her recovery from the second episode of F toxicity.

Case study 2: A 49-yr-old businessman from Delhi ticked for several of the health complaints in Table 1. The baseline data for the SFL, 0.120 mg/L (normal range: 0.02–0.05 mg/L), and the UFL, 2.65 mg/L (normal range 0.1–1.0 mg/L), were high (Table 4).

Table 4. Baseline and post-intervention follow-up data for the serum fluoride level (SFL), the urinary fluoride level (UFL), the drinking water fluoride level (DWFL), and the blood haemoglobin (Hb) level, together with the result of the forearm radiograph, for case study 2 of a 49-yr-old male consuming water from a reverse osmosis (RO) filter and dietary items with a high F content

	SFL (mg/L)	UFL (mg/L)	Forearm X-ray radiograph	DWFL (mg/L)	Hb (g/dL)
Data for case study 2 of a 49-yr-old male					
Baseline data:					
	0.120	2.65	Interosseous membrane calcified	0.201 (RO filter)	13.7
Monitoring (re-testing) data after the practice of the interventions:					
After 2 months (mo)	–	1.25		–	13.0
After 1 yr 4 mo	–	0.531		–	13.7
After 2 yr	–	0.363		–	14.4
After 3 yr	–	0.835		–	15.4
Normal reference range of F	0.02–0.05	0.1–1.0		Up to 1.0 mg/L, the less the better	≥13.0

RO = reverse osmosis

An X-ray radiograph of the forearm revealed interosseous membrane calcification. His drinking water source was a RO filter which gave water with a F level of 0.201 mg F/L (normal range: up to 1.0 mg/L with the proviso of the less the better). The source of the F entry was traced to dietary sources (Table 1) involving the consumption of street food, the use of black rock salt in cooking, and chewing churans. Eating these high F dietary items led to his experiencing health problems. Upon the practice of the interventions, the UFL reduced from 2.65 mg/L to 0.835 mg/L (normal range 0.1–1.0 mg/L) and the Hb level increased from 13.7 to 15.4 g/dL (normal range: ≥13.0 g/dL). His consuming nutritious food yielded better results than would have been achieved with using drugs. Simultaneously with the withdrawal of the dietary F, the consumption of nutritious food was promoted. The improved nutrition allowed the damage caused by F to be repaired. One of the positive changes that occurred was the regeneration of the microvilli in the gastrointestinal tract allowing a return to normal function. With the patient's Hb rising to beyond 15.0 g/dL (normal range: ≥13.0 g/dL), case study 2 is an excellent example of the improvement that may occur in health when F toxicity is treated.

Case study 3: A 40-yr-old female patient from a highly endemic village for fluorosis in Villupuram District, Tamil Nadu, was admitted to a teaching hospital in Puducherry (South India). When F poisoning was suspected, the Department. of Clinical Immunology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Puducherry, referred her to the Foundation for disease confirmation. The F levels in the serum, the urine and the drinking water were tested. The Foundation received a scan of a forearm X-ray radiograph from Puducherry (Table 5).

Table 5. Baseline data for the serum fluoride level (SFL), the urinary fluoride level (UFL), and the drinking water fluoride level (DWFL), together with the result of the forearm radiograph, for case study 3 of a 40-yr-old female, with chronic renal failure, consuming untreated ground water (UGW) and dietary items with a high fluoride content

	Data for case study 3 of a 40-yr-old female			
	SFL (mg/L)	UFL (mg/L)	Forearm X-ray radiograph	DWFL (mg/L)
Baseline data:				
	0.37	0.96	Interosseous membrane calcified	2.31 (UGW)
Normal reference range of F	0.02–0.05	0.1–1.0		Up to 1.0 mg/L, the less the better

UGW= untreated ground water

Her SFL was high 0.37 mg/L (normal range: 0.02–0.05 mg/L), while her UFL, 0.96 mg/L was within the normal range (0.1–1.0 mg/L). Having a normal UFL together with an elevated SFL is unusual in a case of fluorosis. The forearm interosseous

membrane was calcified. Two sources were identified for the F entry consisting of untreated ground water in the endemic village and high F containing food. Further enquiry regarding the patient's kidney function revealed that patient was suffering from chronic renal failure. We thus confirmed that diagnosis for the patient was fluorosis with renal failure.

CONCLUSIONS

In conclusion, this communication reports the developments which have taken place in India over many decades on the development of a reliable, meaningful approach to making a correct diagnosis of fluorosis when suspicion of this arises from the history retrieved from a patient and how recovery may be achieved through the practice of interventions. We have emphasized the need for and importance of using a pre-coded proforma for the history retrieval. Three categories of patients were highlighted as the patients presenting at hospital OPDs may belong to any of the three the categories. As well as presenting the three case studies, we have described the type of tests done, the relative merits of each test, and the manner in which the body fluids (serum and urine) and the Hb changed during monitoring. A careful study of this report may help with how fluorosis can be dealt with in a hospital environment and enable recovery from fluorosis to begin after only a few days when diet editing and diet counselling are effectively executed. The diagnosis of fluorosis can be made in the laboratory medicine department or the clinical pathology laboratory of any hospital where routine diagnostic testing is done. The only equipment required for measuring F levels is an ion meter with a fluoride ion sensitive electrode as well as a reference electrode. Alternatively, a combination electrode may be a better option for testing the F level when the sample volume is less, as with a serum sample. A technician may need a few days training on the machine to be able to conduct the tests and report reliable results.

ACKNOWLEDGEMENTS

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