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A Review on Endemic Fluorosis with Special Reference to Its Medical Implications

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Abstract

Fluoride is one of the most potent anabolic agents for bone and has been extensively used in the prevention of skeletal caries. Its role in the management of osteoporosis is still debatable. Skeletal fluorosis is symptomatic only in later stages of natural history of disease. This research paper presented the endemic fluorosis diagnosis by using various techniques for accuracy in treatment like Radiological treatment including Radionuclide skeletal imaging, Magnetic Resonance Imaging for Diagnosis of skeletal fluorosis. No satisfactory treatment is presently available for the fully established symptoms of such skeletal fluorosis disease. Presently, the provision of fluoride free water seems to be only solution several defluoridation techniques are available.

Fluoride is an essential trace element that plays an important role in bone and dental health when present within permissible limits. However, prolonged exposure to excessive fluoride, particularly through drinking water, can lead to a serious public health condition known as endemic fluorosis. Skeletal fluorosis is a chronic metabolic bone disease characterized by progressive changes in bone structure, mineralization, and joint mobility. In many regions of India and other Southeast Asian countries, high fluoride concentrations in groundwater have resulted in widespread incidence of fluorosis affecting millions of people.

Keywords: *skeletal Fluorosis, Histopathology, Histomorphometry, Radiological and defluoridation techniques*

Introduction

Fluoride is a trace element and is essential for many enzymatic functions. It is widely distributed in nature Fluoride is almost completely absorbed in the stomach and intestine. Gastric pH, dietary calcium, plasma fluoride levels and other dietary cations determine rate of absorption. The main retention sites of fluoride are bone, cartilage and dental tissue. Almost 50% of the fluoride is excreted through kidneys; there is a positive correlation of creatinine and fluoride clearance. Plasma fluoride levels indicate ongoing fluoride exposure while urinary fluoride reflects past fluoride exposure, since fluoride released during bone remodeling continue to be eliminated though the renal route long after fluoride exposure has ceased.

Retention of fluoride in skeletal tissue, teeth and cartilage the major component in the metabolism. Significantly high concentrations of fluoride are found in trans- iliac bone biopsies obtained from subjects with skeletal fluorosis the bone fluoride concentration increases with duration and dose of fluoride contamination. There an active redisposition of fluoride released during bone modeling.

Prolonged ingestion of fluoride either through drinking water or other sources leads to crippling skeletal and neurological disability. It constitutes a major public health problem in India and other Southeast Asian countries.

Action of fluoride bone cells and remodeling:

Fluoride induces structural and functional changes in bong cells, matrix protein and mineral components

a. **Effect on osteoblasts:** Fluoride, in-vitro, increases the rate of proliferation of bone cells derived from embryonic chick calvarias, alkaline phosphatase contents and mineralization of embryonic bone. Further, fluoride induces differentiation of pre-osteogenic mesenchyme into osteoblast and deposit bone matrix.

The molecular mechanism whereby fluoride induces mitogenic effects on osteoblasts is still debatable. The tyrosine phosphatase hypothesis proposes fluoride enhances, in dose dependent concentrations, tyrosyl phosphorylation of certain intracellular proteins involved in cellular proliferation.

b. The Effects osteoclasts. Direct effect of fluoride on osteoclasts is poorly studied and understood. Though there are biochemical and histomorphometric evidences of increased bone reabsorption in fluorosis, in vitro studies with osteoclasts have shown a decrease in resorptive activity.

c. **Osteocyte:** Fluoride seems to have an effect on osteocytes, as evidenced by the presence of mottled peri-osteocytic lacunae surrounded by an eccentric hypo-mineralized zone as well as enlarged lacunae due to perilacunar resorption.

d. **Protein matrix:** Proteoglycans and their spatial arms, the glycosaminoglycans (GAG) are known to interact with hydroxyapatite and are considered to have a role in the regulation of mineralization. There is progressive inhibition of GAG adsorption to hydroxyapatite with graded increase in fluoride concentration.
e. **Bone mineralization:** Fluoride, being highly reactive, easily substitutes for [OH] during hydroxyapatite crystal nucleation and growth leading to formation of fluoro-hydroxyapatite. There is an improvement in crystallinity, reduction of crystal surface area and decreased solubility. Fluoride also delays initiation of matrix mineralization of newly formed periosteal bone matrix as well as decreases rate at which matrix, once nucleated, becomes fully mineralized.

Fluoride and non-skeletal tissue:
Fluoride affects several systems and tissue; however, these have been studied less extensively. Fluoride reduces insulin secretion in experimental rats on fluoride enriched water. In a prospective study, impaired glucose tolerance was observed in 10 of 25 subjects with endemic fluorosis; Patients with IGT had significantly higher fasting serum immunoreactive insulin, higher fasting serum fluoride and significantly lower fasting glucose to insulin ratio than patients with normal glucose tolerance. provision of drinking water with fluoride within permissible limits. Non-ulcer dyspepsia, abdominal pain, abnormal upper Gl endoscopy, and chronic atrophic gastritis have been reported in subjects with fluorosis.

Skeletal fluorosis:
Fluorosis is a term used to describe the syndrome resulting from chronic exposure to excess fluoride. This disorder principally affects the skeletal, cartilaginous, and dental tissue and other systems as well. The commonest source of fluoride intoxication continues to be the drinking water and in India alone, 25 million people is risk of the fluorosis
Clinical features: Skeletal involvement in fluorosis may be symptomatic or have mild non-specific musculoskeletal symptoms in early stages. Later subjects develop polyarthralgia and bone pains, joint stiffness and restricted movement of joints. Weight bearing joints are more commonly involved. There may be bowing of long bones leading of bow legs, knock knee, sabre shin, windswept deformities and spinal deformities e.g. kyphosis. Various neurological deficits due to radiculopathy and radiculomyelopathy further add to the disability. Mottling of teeth occurs above water fluoride levels of 1.5mg/l. Concentrations beyond 3-6 mg/I may be associated with symptoms of skeletal fluorosis and beyond 10 mg/l, skeletal fluorosis is usually crippling. In many parts of world including India, symptoms of fluorosis have been reported below these concentrations, perhaps because of factors such as climatic conditions, food habits, and malnutrition. In fact, a positive correlation between the degree of dental fluorosis and environmental temperature has been observed earlier.

Osteofluorosis has been also observed in children who are more prone due to rapid bone accretion. Bowing of long bones, genu varum, genu valgum and wind swept deformities are more commonly observed in children. The skeletal morbidity is further aggravated in children with poor calcium intake. Clinical evidences of rickets are occasionally noticed. Majority of children have dental fluorosis, i.e., chalky white opaque areas over enamel surface, marked attrition of surface, brown staining, and discrete or confluent pitting and enamel hypoplasia.

Histopathology and histomorphometry:

Histology of human iliac bone biopsy shows large trabeculae bordered by expanded and thick osteoid seams, linear formation defects and mottled periosteocytic lacunae with hypomineralized halos around mottled lacunae. Under polarized light the mineralized bone shows lamellar arrangement with only a few areas of woven bone. However, few workers have reported excessive proportion of woven bone. Bone fluoride contents in bone ash of fluorotic subjects are usually more than 3 times of upper limit found in normal subjects.
Histomorphometric: The analysis of bone from patients with osteosclerosis shows significant increase in cortical width and porosity without reduction in cortical bone mass and increased cancellous / trabecular bone volume. Parameters reflecting bone formation (trabecular osteoid volume, trabecular osteoid surface and thickness of osteoid seams) are markedly elevated. The increase in osteoid surface is significantly greater than that seen on trabecular surface. There is greater number of osteoblasts, although there are a greater proportion of flat osteoblasts as compared to plump osteoblasts. There is evidence of mineralization defects in the form of increased proportion of mottled peri osteocytic lacunae and increased volume of cancellous interstitial mineralization defects. Double tetracycline labeling shows decreased mineral opposition rate and increased mineralization lag time. Bone formation and adjusted apposition rate are significantly decreased Thus there is evidence of an unbalanced coupling in favor of bone formation.

Radiology:

Initial descriptions of radiological picture in skeletal fluorosis emphasize the presence of osteosclerosis, in particular of spine, pelvis and ribs. Calcification of interosseous membrane in the forearm has been regarded as sine qua non of the skeletal fluorosis. In addition, calcification of ligaments, in particular posterior spinal ligament, and muscular attachments, exostosis and irregular osteophytes are also observed in florid cases. In addition of osteosclerosis, axial osteosclerosis with distal osteopenia of long bones, coarse trabecular pattern, diffuse osteopenia, evidence of rickets/osteomalacia and hyperparathyroidism have been described.

Radionuclide skeletal imaging:

The skeletal scintigraphy in subjects with skeletal fluorosis revealed high bone turnover in the whole skeleton. There is an increased uptake in the axial and appendicular skeleton along with reduced soft tissue uptake and renal uptake, similar to "metabolic superscan". All the subjects had increased uptake irrespective of radiological picture; however, subjects with higher serum alkaline phosphatase seemed to have higher skeletal uptake than those with normal levels.

Magnetic Resonance imaging (MRI):

MRI of fluorotic subjects shows non-specific low signal intensity corresponding to sclerotic area on radiographs. In a small but significant study using MRI spin-echo and fast low angle shot sequence in four subjects with compressive

myelopathy, cord compression was demonstrated due to ossification of posterior longitudinal ligament and / or ligament flavum.

Diagnosis of skeletal fluorosis:
Diagnosis of skeletal fluorosis is made by the presence of typical clinical and radiological features. Epidemiological features like residence in endemic area and similar affliction of other family members and the members in the same locality contribute to the diagnosis of endemic fluorsisis. It is confirmed by demonstration of increased urinary fluoride levels.

Treatment:

No satisfactory treatment is presently available for the fully established syndrome of skeletal fluorosis. However, in experimental studies, calcium and/or magnesium supplementation ameliorated the toxic effect of chronic fluoride intake on collagen in rats. In an interesting study, Wa et al. found increased absorption of fluoride in subjects suffering from endemic fluorosis. Supplementation with calcium and magnesium absorption led to a decrease in intestinal absorption and increase in fecal exertion of fluoride. Other treatment proposed includes dihydromonocalcium phosphate for prevention and treatment, and acupuncture reflexotherapy. Their efficacy, however, is still unconfirmed. Presently, removing the source of fluoride, for example by providing fluoride free water, is the only option. Reversal of osteofluorosis has been reported in Occasional cases. In result in some reduction in aches and pains, although the restricted motility and deformities persists

Prevention:

Prevention of fluorosis is the only effective measure to escape the disease. For hydric fluorosis, change or modification of the water supply is the only long-term practical solution. Digging deep bore wells has been shown to provide lower but not appropriate fluoride contents. Defluoridation of water can also be performed by adding activated alumina or by Nalgonda technique (alkalization and chlorination followed by adding aluminum salts). However, these methods, though effective, require strong motivation and cooperation of local population administration. Pontie, et. al have proposed nano filtration as a cost effective defluoridation measure, which can be applied to field conditions in third world countries. Nano filtration (also called as low pressure reverse osmosis) is a membrane-based process, which can be selectively remove small molecules and ions including fluoride and can sterilize water. It is a low cost technique, is effective and requires a small amount of energy, which can he supplied by a photoelectric cell.

Conclusions:

Fluoride affects virtually all the components of bone metabolism. The pathogenesis of skeletal fluorosis is still not clear. The role of micronutrients and macronutrients still need to be well defined. Skeletal fluorosis is symptomatic only in later stages of natural history of disease. Presently, no effective treatment is available, provision of fluoride free water seems to be only solution several defluoridation techniques are available but has not attained wide acceptance for the want of public cooperation and bureaucratic apathy.

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Conflicts of interest
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