SKELETAL FLUOROSIS IN CHRONIC RENAL FAILURE - A CASE REPORT

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Abstract:
Skeletal fluorosis is a condition associated with prolonged accumulation of fluorideresulting in fragile bones having low tensile strength. It affects the joints as well as the bones. It is not easily recognizable till advanced stage. Our patient is a chronic renal failure presented to us for evaluation of cervical cord myelopathy. MRI and CT of cervical and lumbar spine showed diffuse osteosclerosis and calcification of interosseous ligaments. Plain radiographs of the spine (for osteosclerosis) and forearms (for periosteal bone formation and interosseous membrane ossification) are accurate enough for the diagnosis of early skeletal fluorosis. Proliferative changes at ligament and tendon insertions and periostitis appears. It is the combination of radiologic findings in fluorosis that is diagnostic.

Keyword: Skeletal fluorosis, Diffuse osteosclerosis, Calcification of interosseous membrane

INTRODUCTION
Skeletal fluorosis occurs in 2 stages.
EARLY STAGE: Sporadic pain; stiffness of joints; osteosclerosis of pelvis & vertebral column.
LATE STAGE: Chronic joint pain; arthritic symptoms; slight calcification of ligaments.

The initial symptoms usually were headache and weakness. These were followed by multiple joint pains, mostly in the feet, knees, and back. Spinal stiffness and kyphosis developed in a few patients.

Vague, diffuse aches and stiffness of joints with decreased range of motion are common initial symptoms. With disease progression, kyphosis with limited spinal mobility, flexion contracture of lower extremities, and restricted chest wall expansion occur.

Although the prevalence of this disease has decreased considerably, it still occurs in some parts of the world. Increasing numbers of people with carpal-tunnel syndrome, arthritic-like pains, osteoporosis may be due to the mass fluoridation of drinking water.
Fluorine is known to bind calcium in the body, causing ionic calcium to decrease; this, in turn, causes secondary hyperparathyroidism. It is thus clear that the clinical picture of fluorosis includes softening of the bones and osteoporosis as well as secondary hyperparathyroidism on a global basis.

CASE REPORT

HISTORY

65 year old male patient with chronic renal failure, presented with following complaints for past 8-10 months.Ø Fall from chair after an episode of giddiness & loss of consciousness.Ø Weakness & diminished sensation involving all 4 limbs.Ø Clinically diagnosed as Cervical cord myelopathy.

WORKUP

MRI cervical spine was done.

CT Cervical spine was done.

Plain X-ray of skull, dorsolumbar spine, both knees, both forearm bones, both wrists was done.

IMAGING FINDINGS

Since the patient presented with weakness & diminished sensation involving all 4 limbs & Cervical cord myelopathy was clinically suspected, MRI cervical spine was done initially.
DIFFERENTIAL DIAGNOSIS

Differential diagnosis for Diffuse Osteosclerosis:
- Metabolic disorders (such as hypothyroidism, hypoparathyroidism, renal osteodystrophy)
- Fluorosis
- Osteopetrosis
- Osteoblastic metastases
- Paget’s disease
- Lymphoma and
  - Myelofibrosis, Mastocytosis & Sickle cell anemia

Differential diagnosis for vertebral osteophytosis with soft-tissue calcification and ossification:
- Spondylitis deformans
- DISH
- Fluorosis
- Ankylosing spondylitis, Psoriasis, Reiter’s syndrome other spondyloarthropathies
- Acromegaly and Alkaptonuria

FINAL DIAGNOSIS
Calcification of interosseus membrane in forearm & interspinous ligaments of cervical and lumbar spine.

Diffuse osteosclerosis –both axial & appendicular skeleton

Both the above features are suggestive of SKELETAL FLUOROSIS.
DISCUSSION

Skeletal fluorosis caused by endemic fluoride poisoning was once thought to result merely in osteosclerosis, causing marblelike changes. Later, various radiologic features were found, including osteosclerosis, osteomalacia, and osteoporosis. Although this disorder has a wide variety of appearances, little attention has been given to the spectrum of radiologic appearances.

Radiologic features of fluorosis include increased bone density; blurring or haziness of trabeculae; compact bone thickening; periosteal bone formation; and ossification of the attachments of tendons, ligaments, and muscles. Involvement of the axial skeleton is characteristic, and changes are most marked in the spine, pelvis, and ribs.

In early fluorosis, the first changes are bone deposition and thickening at the junctions of trabeculae. This is seen as sanlike, granular, or particle-like bone structure on radiographs. In more advanced fluorosis, the trabeculae are more generally thickened because of new bone formation on the trabecular surface. At this stage, radiographs show thickening and condensation of trabeculae, with coarse reticulum or woven bone striations. If the trabeculae are fused, focal round densities are seen in the medullary bone.

The pathogenesis of these diverse radiologic appearances remains unclear. It is generally thought that several factors influence the type of bone change seen in fluoride intoxication. These include the nature, dose, and duration of fluoride exposure; nutritional status; hormonal responses; age; sex; type of bone affected (cortical or otherwise); and dietary habits. The different appearances of this disease probably represent different combinations of these variables.

Plain radiographic examination is the best method for diagnosing fluorosis, especially when the disease is in the asymptomatic phase. Plain radiographs of the spine (for osteosclerosis) and forearms (for periosteal bone formation and interosseous membrane ossification) are accurate enough for the diagnosis of early skeletal fluorosis in children. However, osteosclerosis alone is a nonspecific finding seen in a wide variety of disorders, including metastases, myelofibrosis, Paget's disease, hemoglobinopathies, renal osteodystrophy, and congenital disorders. Likewise, vertebral osteophytosis with soft-tissue calcification and ossification can occur in spondylitis deformans, diffuse idiopathic skeletal hyperostosis, ankylosing spondylitis, psoriasis, Reiter's syndrome, other spondyloarthropathies, acromegaly, neuropathy, and alkaptonuria and is not diagnostic when seen alone. Proliferative changes at ligament and tendon insertions and periostitis appear not only in fluorosis but also in diffuse idiopathic skeletal hyperostosis, hyperparathyroidism, X-linked hypophosphatemic osteomalacia, and plasma cell dyscrasia.

Gnandjean et al has suggested that characteristic ligament calcifications and a history of long-term heavy exposure to fluoride are very important in the diagnosis of skeletal fluorosis. A history of exposure is essential for early diagnosis. The results of our study show that skeletal fluorosis has various radiologic appearances, and none of them alone is characteristic. It is the combination of findings in fluorosis that is diagnostic.

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