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IMAGES

Fluorosis Masquerading as Compressive Myelopathy

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Abstract

A 60-year-old woman from Cuddalore presented with restricted neck movements, progressive difficulty in walking, and lower limb weakness, leading to bed confinement. Clinical examination revealed spastic paraparesis, brisk reflexes, and sensory deficits below the hips. Laboratory investigations indicated renal insufficiency and elevated urine fluoride levels. Imaging demonstrated calcifications of sacroiliac, sacrospinous ligaments, interosseous membrane, and posterior longitudinal ligament, with osteosclerosis and osteoporosis. Differential diagnoses included Ankylosing Spondylitis and Diffuse Idiopathic Skeletal Hyperostosis. However, the unique pattern of ligament calcification and elevated fluoride levels supported a diagnosis of fluorosis. This case underscores the importance of considering fluorosis in patients from endemic areas with spastic paraparesis and characteristic radiological findings. Posterior decompression via laminectomy was performed, emphasizing the need for awareness of fluorosis as a potential cause of cervical compressive myelopathy.

Keywords: Fluorosis, posterior longitudinal ligament calcification, interosseous membrane calcification, osteosclerosis, Diffuse idiopathic skeletal hyperostosis (DISH)

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A 60 year female, diabetic for 10 years, housewife from Cuddalore, presented with restricted neck movements for 1 year, difficulty in walking for 6 months- knee pain aggravating on movement, weakness of both lower limb for 1 month- difficulty in sitting up from lying down position, bed bound for 1 month, no movement of legs and loss of sensation over both lower limbs for 1 month. No bladder and bowel involvement. No involvement of upper limb. Similar complaints are reported in her parents. Higher mental function, cranial nerves normal. Bulk normal, increased tone in both lower limbs, lower limb power 1/5 across all joints. All deep tendon reflexes were brisk except ankle jerk, Hoffman and wartenberg reflex positive. All sensory modalities reduced below hip. Normal cerebellar and skull, spine examination. Provisional diagnosis considered was cervical compressive myelopathy. Complete hemogram normal, urea-95 mg/dl, creatinine-4.2 mg/dl, urine routine and microscopy normal and USG KUB showed bilateral shrunken kidney. Nerve conduction study showed Bilateral Sural sensory axonal neuropathy probably secondary to diabetes mellitus. Fundus normal. Urine fluoride levels measured to be 2.6 parts per million (Normal<1). Imaging showed calcification of sacroiliac, sacrospinous ligament (Figure 1A), calcification of the interosseus membrane (Figure 1B), increased bone density (osteosclerosis), osteoporosis, thickening of the compact bone, ossification of the tendon and ligament attachments and ossification of posterior longitudinal ligament (Figure 1C). Water fluoride levels were >1.2 ppm and patient few family members who share same water source had similar symptoms. We have notified the Public Works Department regarding the non-endemicity and to take necessary steps to check Water fluoride levels of that particular region and decide on need for defluoridation technique. Posterior decompression via laminectomy done. Post procedure patient was able to walk without support and symptomatically better at 3 months follow up. Differential diagnosis to be considered in this case were Ankylosing spondylitis (AS) and Diffuse idiopathic skeletal hyperostosis (DISH). AS will have sacroiliitis, romanus lesion, syndesmophytes, classical interspinous ligament calcification with reduced disc space. DISH will have syndesmophytes with classical anterior longitudinal ligament calcification with preserved disc space. The unique features of fluorosis are the classical posterior longitudinal ligament, interosseous membrane, sacrospinous and sacrotuberous ligament calcification with preserved disc space. Excessive fluoride concentration in drinking water causes activation of both osteoblasts and osteoclasts resulting in increased bone turnover by aberrantly activated osteoblasts, activates parathyroid hormone pathway leads to poorly woven bone matrix, periosseous tissue ossification and immature bone deposition. In endemic areas, sclerosed osteophytes/ thick ligaments due to fluorosis can cause compressive myelopathy [1,2,3] and cause restrictive neck movements, progressive spastic quadriparesis similar to our case. Non-skeletal features of fluorosis include increased thirst, depression, infertility, abdominal pain, constipation, bloating, loss of appetite and myopathy [1]. Fluorosis is endemic in geographical fluoride belt which includes 22 countries – India, China, Japan, Saudi Arabia, Pakistan, Middle east, Argentina, Mexica and North- East African countries [3]. Normally it gets incorporated into the enamel hydroxyapatite crystals and

reduces enamel demineralization and prevents caries formation. It is double edged sword as excess fluoride interferes with enamel mineralization, maturation leading to soft, pitted and pigmented enamel. Bony deposition of fluoride causes bony sclerosis, osteoporosis and ligament calcification. Absence of dental fluorosis does not exclude fluorosis as in our case.

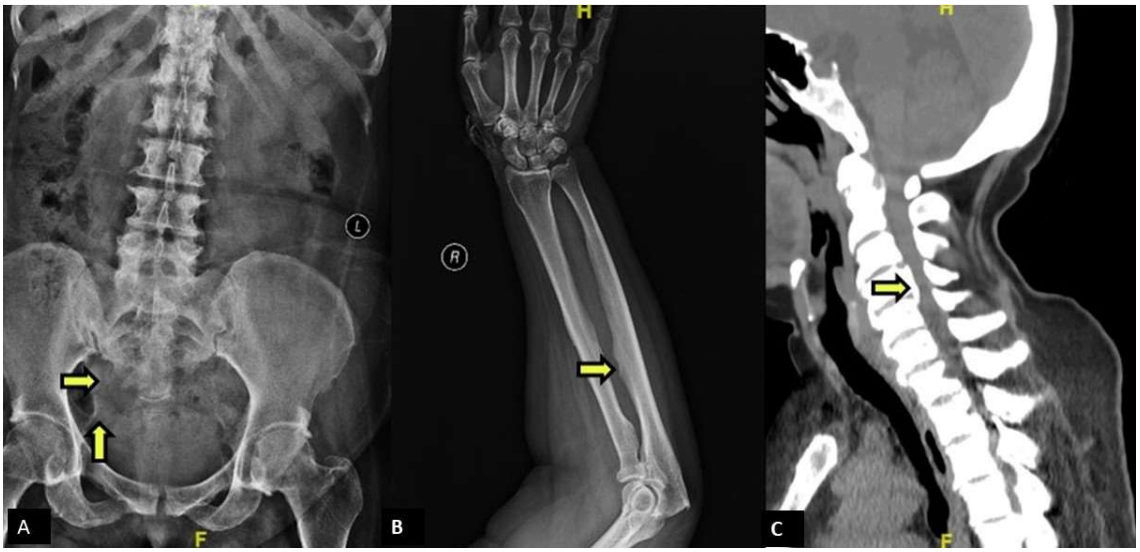

Figure 1. **1A** Xray pelvis AP view showing calcification of sacroiliac ligament (marked by arrow) and sacrospinous ligament (marked by arrow); **1B** Xray of right forearm anteroposterior view showing interosseous membrane calcification (marked by arrow); **1C** Computed Tomography spine (CT spine) showing ossification of posterior longitudinal ligament (Marked by arrow)

Learning points

In patients presenting with progressive spastic paraparesis or quadriparesis with progressive spastic paraparesis/ quadriparesis with neck pain, brisk reflexes, interosseus calcification in Xray forearm and posterior longitudinal ligament calcification- cervical compressive myelopathy-Fluorosis must be considered.

Statements and Declarations

Conflicts of interest

The authors declares that they do not have conflict of interest.

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