CERVICAL CORD COMPRESSION IN A PATIENT WITH FLUOROSIS

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ABSTRACT

A 61 year-old male patient from Chiangrai province (northern part of Thailand) who presented with cervical cord compression. Bone survey, CT and MR scan shows diffuse sclerosis of the vertebrae, ribs, and pelvic bones. Calcified ligaments at multiple sites are shown and ossified posterior longitudinal ligament as the cause of the cervical cord compression. Though the fluorine level in the blood of the patient was normal at the admission time, fluorosis is obvious by images.

INTRODUCTION

Fluorosis or chronic fluorine intoxication arises when the drinking water contains fluoride in concentrations higher than 4 parts per million (ppm).[1,2] It occurs as an endemic problem in certain regions of the world; in Thailand the patients usually come from the northern part. It was first described in the 1930's and sporadically in almost a worldwide distribution.[1,3] Industrial workers who are exposed to fluorine compounds over a period of years,[4] laboratory personnel who have inhaled fluorine vapors, patients who received medications containing high doses of fluorine [5] and individuals who habitually drink fluorine-containing wine [6] also developed this entity. Fluoride concentration in 1 ppm can reduce dental caries, in 2 ppm or more can lead to mottled enamel, 8 ppm can produce osteosclerosis in 10 per cent of individuals and greater than 100 ppm may induce growth disturbances, kidney damage, or death.[7]

Approximately 50 per cent of the absorbed fluoride is excreted mainly in the urine and approximately 99 per cent of the fluoride retained in the body is deposited in the calcified tissue.[8] The biologic half-life for bone fluoride is about 8 years, owing to the slow rate of turnover of skeletal tissues. With cessation of the exposure to fluoride and with continued, although slow, metabolism of bone tissue, excretion of fluoride can lead to an improvement in the pulmonary and skeletal manifestations of the disease. Radiographic improvement has been documented.[9]

Cervical cord compression could occur in this disease due to ossified / calcified posterior longitudinal ligament.

CASES REPORT

A 61-year-old man from Chiangrai province, developed progressive weakness of all extremities for 4 months. Physical examination was compatible with C6-7 cord compression. The patient consumed drinking water from a well and regulary chew the tea leaves. The urine fluoride was within normal limit. Renal function was mildly impaired.

Plain films of the bones showed diffuse

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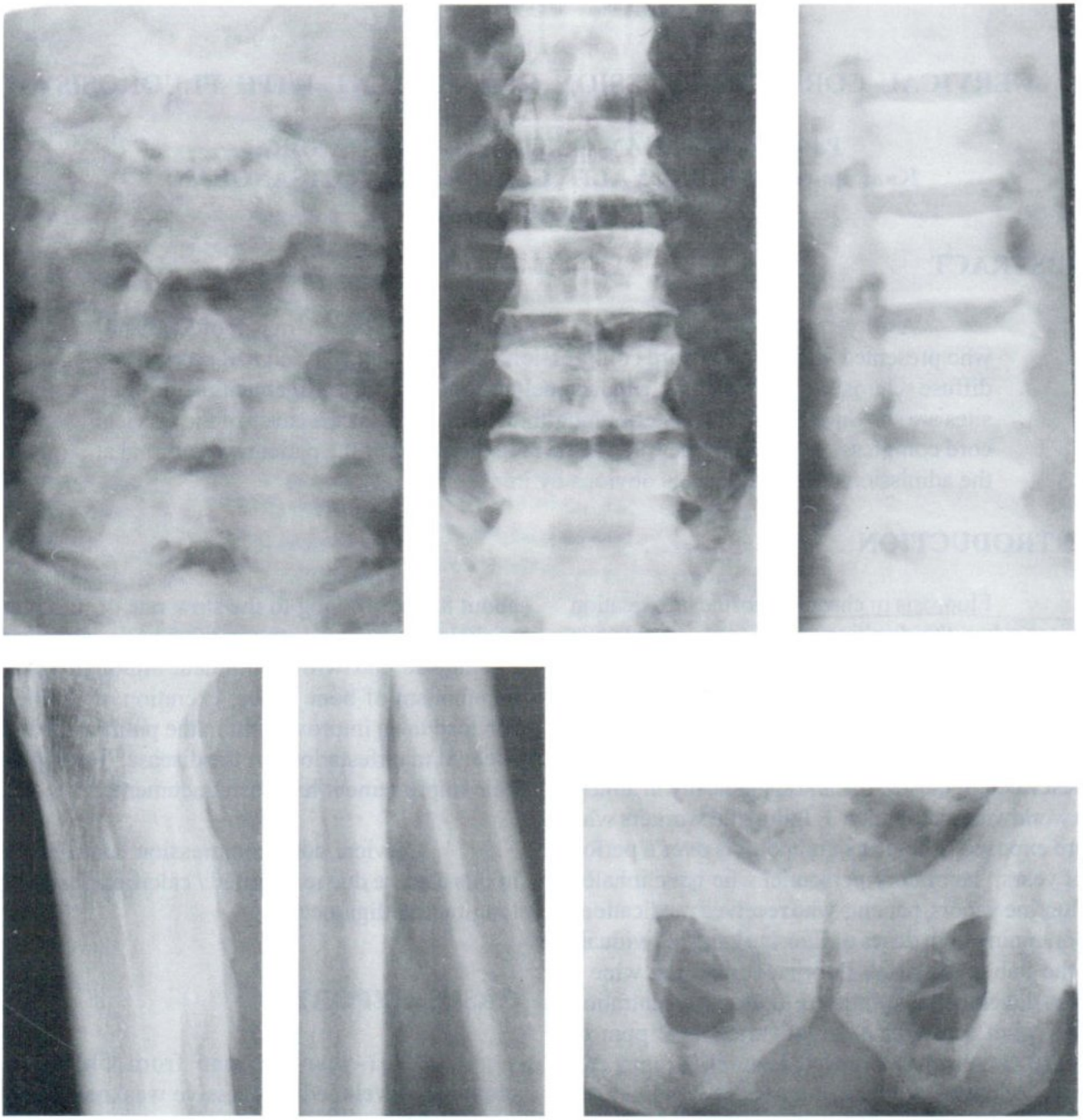

Fig. 1 Radiographic images of axial skeleton, ribs and pelvic bones and limbs which showed diffuse osteosclerosis, osteophytosis, periostitis and ligamental calcification.

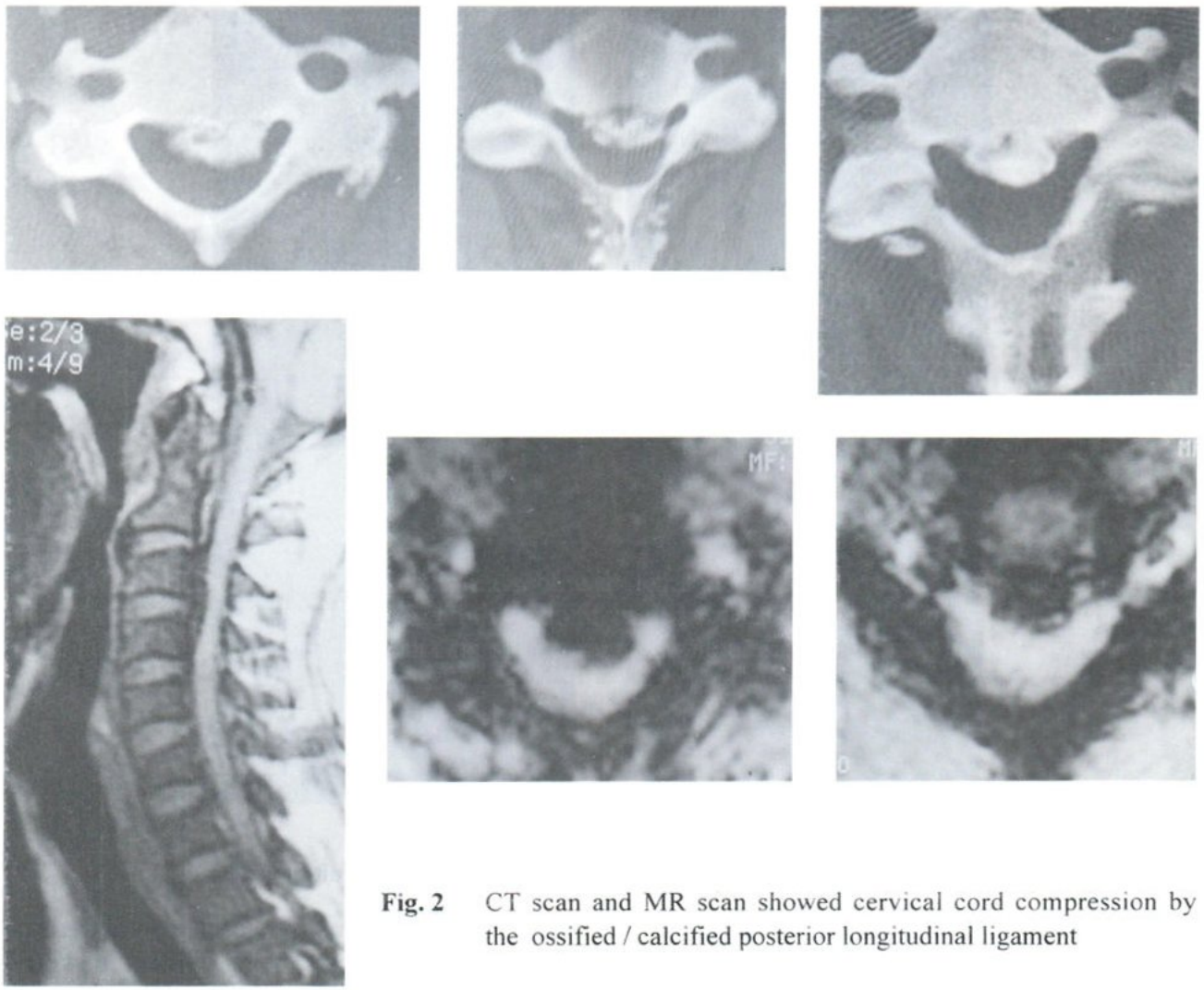

Fig. 2 CT scan and MR scan showed cervical cord compression by the ossified / calcified posterior longitudinal ligament

osteosclerosis of the axial skeleton, ribs and pelvic bones, vertebral osteophytosis, periostitis and multiple sites of ligamentous calcification (Fig.1). CT scan and MRI study showed ossified or calcified posterior longitudinal ligament with cord compression at C2-4 (Fig.2). Corpectomy of C3-4 and was performed. The 99m Tc-MDP bone scan revealed increased uptake along the axial skeleton, both shoulders, ribs and pelvic bones, left tip of the calcaneous indicating increased bone formation (Fig.3).

DISCUSSION

Clinical manifestations of chronic fluoride exposure include joint pain and restriction of motion, back stiffness, restriction of respiratory movements, functional dyspnea, dental alterations, paraplegia, and palpable thickening of the bones, including the clavicle, tibia and ulna.[8] Involvement of the axial skeleton is characteristic. Changes are most marked in the spine, the pelvis, and the ribs. Osteosclerosis usually appears first. Increasing trabecular condensation eventually creates a radiodense or chalky appearance throughout the thorax, vertebral column, and pelvis with obscuration of bony architecture. The skull and tubular bones of the appendicular skeleton are relatively spared in this sclerotic process.[1]

Vertebral osteophytosis can lead to encroachment on the spinal canal and intervertebral foramina. In the axial skeleton, hyperostosis and

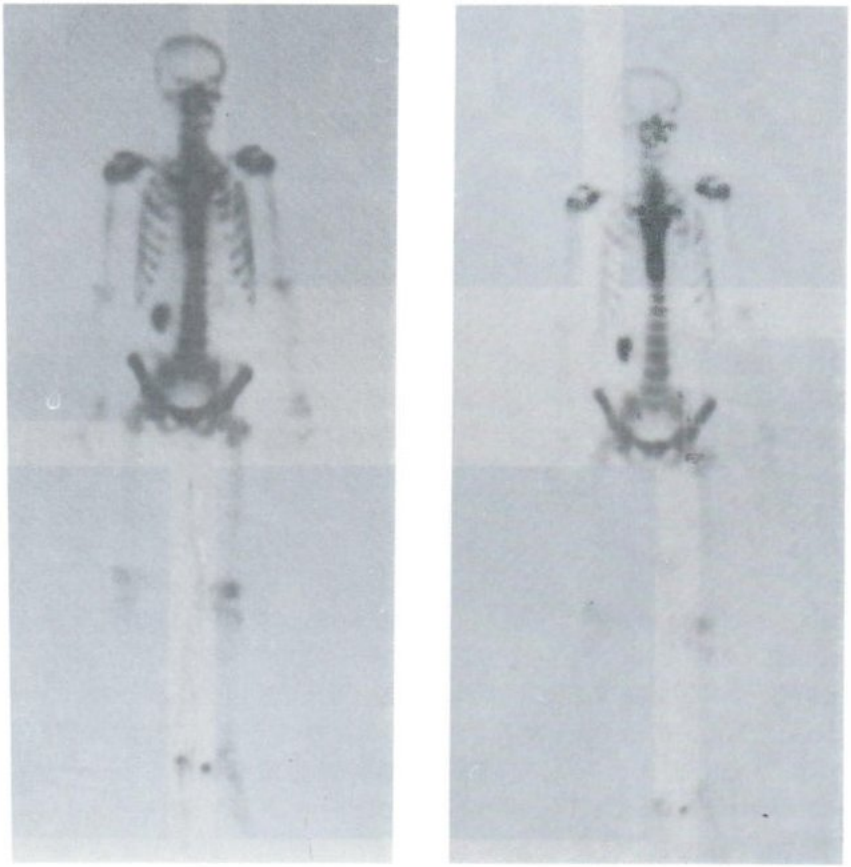

Fig. 3 Bone scintigraphy revealed increased uptake at multiple sites.

bony excrescences develop at sites of ligamentous attachment, especially in the iliac crests, ischial tuberosities, an inferior margins of the ribs. Calcification of paraspinal ligaments as well as sacrotuberous and iliolumbar ligaments can be noted.

In the appendicular skeleton, periosteal thickening, calcification of ligaments, and excrescences at ligamentous and muscular attachments to bone can be seen at one or more sites, particularly near the interosseous membranes of the forearm and leg, the calcaneus, the posterior surface of the femur, the tibial tuberosity and the proximal humerus. Soft tissue ossification resembling myositis ossificans and cartilaginous atrophy and ulceration have been noted .[11]

Osteosclerosis, osteophytosis, and ligamentous calcification represent a useful triad of abnormalities that are evident on pelvis and spine roentgenograms. Osteosclerosis alone is not diagnostic of fluorosis, being evident in skeletal metastasis, myelofibrosis, mastocytosis, certain hemoglobinopathies, renal osteodystrophy, Paget's disease, congenital disor-ders and other conditions. Likewise, vertebral osteo-phytosis or similar outgrowths can accompany many diseases, including fluorosis, spondylosis deformans, diffuse idiopathic skeletal hyperostosis, ankylo-sing spondylitis, the spondylitis of psoriasis, Reiter's syndrome, and inflammatory bowel disorders, acromegaly, neuroarthropathy, and alkaptonuria.[1] Proliferative changes at ligamen-tous and tendinous insertions in bones are apparent not only in fluorosis but also in diffuse idiopathic skeletal hyperostosis, hypoparathyroidism, X-linked hypophosphatemic osteomalacia and certain plasma cell dyscrasias. Periostitis similar to that which may be seen in fluorosis can be detected in hypertrophic osteoarthropathy,

pachydermoperiostosis, and thyroid acropathy. Thus, each individual radiographic finding of skeletal fluorosis can be apparent in other disorders as well: it is the combination of findings in fluorosis that is diagnostic.[1]

Ossification of the posterior longitudinal ligament (OPLL) is a dense, ossified strip or plaque of variable thickness along the posterior margins of the vertebral bodies and intervertebral disks.[12] It is most common in the midcervical (C3-5) and the midthoracic (T4-T7) spine. Multilevel involvement is characteristic. On NCE CT scans, the ossified strip that characterizes OPLL is typically separated from the vertebral body by a thin radiolucent zone. Sagittal T1- or proton-density weighted MR scans show increased signal intensity from the fatty marrow of thickly ossified lesions; axial imaging is necessary to demonstrate thinly ossified OPLL.[13] The differential diagnosis of OPLL includes calcified HNP and calcified meningioma, although the shape and multilevel involvement of OPLL are characteristic. Calcified/ ossified OPLL in this case was a part of disease process of fluorosis.

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NCE CT = Non-contrast enhancement CT.
HNP = Herniated Nucleus Pulposus.