Skeletal fluorosis mimicking seronegative arthritis

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Fluorosis is endemic in certain parts of the world, especially the Asian subcontinent (1). We report an unusual presentation of fluorosis mimicking seronegative spondyloarthritis. Although fluorosis is known to cause irritable bowel syndrome-like disorder and joint pain, this could be wrongly diagnosed as a case of seronegative arthritis.

Case report

A 35-year-old female presented with joint pain involving the lower back, both heels, and the knee for the past 3 years. She described early morning stiffness lasting for more than 30 min and swelling over both ankles. She denied any history of skin lesions, oral ulcers, uveitis, and genital ulcers. She also reported increased frequency of stools along with occasional pain abdomen. She had never had tenesmus or blood in the stool. She had been prescribed various anti-inflammatory drugs by a private practitioner, without much benefit. Physical examination revealed dental discoloration with brownish stains. Eye and skin examination was otherwise normal. Joint examination did not reveal any evidence of synovitis. Her Achilles’ tendon was swollen and tender on both the sides. Spine examination revealed tenderness at the lumbar region with mild restriction of movement. Her baseline investigations were normal with the exception of erythrocyte sedimentation rate (ESR) raised to 44 mm/h. Rheumatoid factor and ANA were also negative. Tissue typing for HLA B27 was negative. Considering the possibility of enteropathic arthritis, X-ray of the pelvis and ankle joint and flexible sigmoidoscopy was performed. Flexible sigmoidoscopy showed normal mucosal pattern with no growth or strictures. Biopsy from the colon showed mild non-specific colitis. X-ray of the pelvis did not show any features of sacroiliitis, but there was increased bone density along with calcification of ligaments and tendon (Figure 1A). X-ray of the ankle revealed a bilateral calcified Achilles’ tendon (Figure 1B). The patient was a resident of New Delhi, a state endemic for fluorosis. Considering the possibility of fluorosis, fluoride levels were analysed in our hospital in serum and urine samples from the patient and in the drinking water in her household. Fluoride levels were increased in her serum (0.05; normal < 0.02 ppm), urine (0.88; normal < 0.10 ppm), and drinking water from the hand pump (1.87; normal < 1 ppm). Similar analysis on her husband and other family members showed elevated fluoride levels.

Discussion

Endemic fluorosis is a chronic metabolic bone disease caused by ingestion of large amounts of fluoride through either water or food in a geographic area where high levels of fluoride occur naturally. Early bone fluorosis is not clinically obvious; often the only complaints of young adults are vague pains in the small joints of the hands, feet, and lower back. Such cases may be misdiagnosed as rheumatoid arthritis or anklylosing spondylitis (2, 3). Endemic skeletal fluorosis can have a wide variety of radiographic appearances, including
calcification and/or ossification of the attachments of soft-tissue structures to bone, osteosclerosis, osteoporosis, growth lines, and metaphyseal osteomalacic zones. In one study, 89% of the adults had some evidence of calcification and/or ossification of the attachments of ligaments, tendons, muscles, and interosseous membranes. Among the non-skeletal manifestations, gastrointestinal disturbances are well known and present with loss of appetite, nausea, abdominal pain, flatulence, constipation, and intermittent diarrhoea mimicking irritable bowel syndrome (5–7). The patient in our report presented with symptoms like those of enteropathic arthritis and the diagnosis of fluorosis could have been missed if attention had not been given to the increased density in the pelvic bones and ligamentous calcification. Moreover, tendon-Achilles calcification has not been reported in the literature to date.

This patient presented with chronic symmetrical arthralgia with accompanying gastrointestinal disturbance, raising the possibility of enteropathic arthritis. The diagnosis of skeletal fluorosis was surprising, with fluoride levels being high in body fluids and drinking water.

The minimum daily dose capable of producing the various stages of fluorosis is still poorly understood. Skeletal fluorosis in India and China has been reported to occur when the fluoride concentration in water exceeds 1 ppm, and has been found to occur in communities with only 0.7 ppm (8, 9). The Chinese government now considers any water supply containing over 1 ppm fluoride a risk for skeletal fluorosis (8). This is in contrast to the USA, were the maximum contaminant limit is established by the US Environmental Protection Agency as 4 ppm. This difference in limits of fluoride levels may be explained by the lower tendency to develop fluorosis in this population. Many case reports in the USA have documented skeletal fluorosis among people with kidney diseases at water fluoride levels as low as 1.7 ppm, and among heavy tea drinkers at water fluoride levels as low as 2.2–3.5 ppm.

Thus, in areas where fluorosis is endemic, skeletal fluorosis is a common mimic of seronegative arthritis and should be pursued with investigations for diagnosis of fluorosis with measurement of fluoride levels, wherever applicable.

References


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