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Diffuse idiopathic skeletal hyperostosis: A review

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Abstract
Diffuse Idiopathic Skeletal Hyperostosis (DISH) is a relatively common disorder of unknown etiology presenting in middle to late age with characteristic radiological changes. Etiology and pathophysiological mechanism are poorly understood. The purpose of this article is to review historical aspects, likely pathophysiology clinical and radiological features of DISH.

Keywords: diffuse idiopathic skeletal hyperostosis (dish), forestier-rotes-querol disease, ligament ossification, ankylosis

Introduction
Diffuse Idiopathic Skeletal Hyperostosis (DISH) is a relatively common yet insufficiently investigated and understood disorder. DISH is a systemic noninflammatory disorder characterized principally by calcification and ossification of spinal ligaments and entheses; seen commonly in middle aged and elderly population. The etiology of condition is poorly understood, although correlations with diabetes mellitus, obesity and age have been found. Although DISH is asymptomatic in most individuals, sometimes spinal and extraspinal ossifications can lead to pain, stiffness, Decreased range of motion and increased risk of spinal fractures. The aim of this article is to review pathogenesis, clinical features and diagnosis of this condition.

History
DISH has been described under various names before getting its present name in 1976. Knaggs [1] and Openheimer [2] described the illness as Spondylitis ossificans. Oppenheimer [2] reported 282 cases with calcification or ossification of vertebral ligaments. Spondylosis hyperostotica was introduced by Ott.[3] Physiologic vertebral ligamentous calcification was described by Smith et al. [4] Forestier [5] and Rotes-Querol [5] gave description of disease with clinic-o-radiological description of nine cases including necropsy findings of two cases by Forestier and Rotes-Querol. Disease came to known as Forestier-Rotes-Querol Disease. The disease was labelled as senile ankylosing hyperostosis of the spine. The term Diffuse Idiopathic Skeletal Hyperostosis (DISH) was coined by Resnick et al [6] in 1976 in case series of 215 cadaverics spines and 100 patients with the disease.

Epidemiology
DISH is more common in men than women. The incidence varies by population and increases with age. In a population-based study of individuals over age 30 in Finland, the incidence was 0.7 and 0.4 per 100 person-years in men and women, respectively [7]. In another Finnish study, the prevalence in people over 40 years of age was 3.8 and 2.6 percent in men and women, respectively [8]. Among those over age 70, the prevalence was 10.1 and 6.8 percent. In a study in Hungary, the prevalence in those over 50 years was 4.9 percent in men and 1.4 percent in women [9]. Hiyama et al [10] in an hospital based study reported prevalence of DISH based on whole spine CT at 19.5%.
A South African study of hospitalized patients 40 years of age and older found that the prevalence of DISH in African blacks was 3.8% in men and 4.2% in women; the prevalence rose with increasing age, from 1% in the 40-49 year age group to 13.6% in those over 70 years [11].

Etiopathogenesis
While the cause of diffuse idiopathic skeletal hyperostosis (DISH) remains unknown, mechanical factors, dietary contributions, drugs, environmental exposures, and metabolic conditions have been hypothesized to be important. It is thought that the bone formation that is distinctive of DISH results from abnormal osteoblastic differentiation and activity at the enthesis.
Natural inhibitors of bone formation, such as matrix Gla protein and Dickkopf-1, may be deficient in DISH patients, contributing to hyperostosis [12]. An appropriate genetic background, as yet unidentified, external factors, or local or systemic factors may stimulate the abnormal osteoblastic differentiation. Metabolic factors including obesity, a high waist circumference ration, hypertension, diabetes mellitus, hyperinsulinemia, dyslipidemia, elevated Growth Hormone levels, elevated Insulin like Growth Factor(IGF)– 1, hyperuricemia, use of retinoids, and genetic factors have been associated with increased incidence of DISH. Owing to these

metabolic derangements, patients with DISH have an increased likelihood of being affected by metabolic syndrome and an increased risk for the development of coronary artery disease and stroke [13].

Clinical features

Clinical manifestations of DISH can be from asymptomatic disease to dorsolumbar pain with morning stiffness and reduced range of motion. Patients may have pain involving large and small joints and peripheral entheses such as heel, Achilles tendon, shoulder, patella and olecranon. Ossification and large osteophytes may result in spinal stenosis and spinal stiffening increasing risk of fractures. Severe complications in the form of dysphagia, hoarseness and myelopathy may occur due to involvement of cervical spine.

Thoracic spine involvement is characteristic which differentiates it from spinal osteoarthritis in which thoracic spine involvement is uncommon or late. Preserved intervertebral height is also uncommon in osteoarthritis as seen in DISH.

Radiological features

The hallmarks of DISH are radiographic abnormalities. DISH is characterized on imaging by the ossification of paravertebral ligaments and peripheral entheses. The characteristic finding in thoracic spine is flowing linear calcification and ossification along the anterolateral aspects of the vertebral bodies that is continuous across the disc space. (Image 1) This type of flowing calcification is less commonly observed in other areas of spine. Ossification of posterior spinal ligament is unusual in DISH.

Extra spinal involvement of entheses is commonly seen in DISH. Radiographic changes in the appendicular skeleton are often symmetric. The following findings have been noted:

- Pelvic and hip involvement includes bony proliferation (also called whiskering), ligament ossification, and periarticular osteophytes. (Image 2)
- Knees can be involved at the patella, at the insertion of the ligaments, and at the tibial tuberosity. (Image 3)
- Ankles and feet can have bony excrescences over the dorsal surface of the talus, the dorsal and medial tarsal navicular, the lateral and plantar aspects of the cuboid, and the base of the fifth metatarsal.
- Shoulder involvement includes bony irregularity along the deltoid tuberosity, the inferior glenoid, and the inferior distal clavicle, as well as calcification and ossification of the coracoclavicular ligament.
- Hand changes include broadening and arrow heading of the phalangeal tufts, enlarged sesamoid bones, thickened cortical width of tubular bones, exostoses, new bone in the joint capsule, and striking proximal phalangeal enthesopathy.
- Bone density is increased in DISH due to the artifact resulting from measurement in areas with substantial osteophytosis.

Laboratory findings

There are no consistent laboratory abnormalities that are associated with DISH. The following are either normal or nondiagnostic: complete blood counts; erythrocyte sedimentation rate; serum levels of calcium, phosphorus, growth hormone, liver enzymes, parathyroid hormone, and fluoride; and the serum protein electrophoresis. The frequency of human leukocyte antigen (HLA)-B27 is not increased.

Diagnostic criteria

Criteria of Resnick and Niwayama [6]
1. The presence of flowing calcification and ossification along the anterolateral aspect of at least four contiguous vertebral bodies with or without associated localized pointed excrescences at the intervening vertebral body-intervertebral disc junctions.
2. The presence of relative preservation of intervertebral disc height in the involved vertebral segment and the absence of extensive radiographic changes of "degenerative" disc disease, including vacuum phenomena and vertebral body marginal sclerosis.
3. The absence of apophyseal joint bony ankylosis and sacroiliac joint erosion, sclerosis, or intraarticular osseous fusion.
Criteria of Utsinger [14]
1. Continuous ossification along the anterolateral aspect of at least four contiguous vertebral bodies, primarily in the thoracolumbar spine. Ossification begins as a fine, ribbon-like wave of bone but commonly develops into a broad, bumpy, buttress-like band of bone.
2. Continuous ossification along the anterolateral aspect of at least two contiguous vertebral bodies.
3. Symmetrical and peripheral enthesopathy involving the posterior heel, superior patella, or olecranon, with the entheseal new bone having a well-defined cortical margin.
Exclusions:
i) Abnormal disc space height in the involved areas
ii) Apophyseal joint ankylosis
Categories of DISH according to the Utsinger criteria are:
Definite = criterion 1
Probable = criteria 2 and 3

Differential Diagnosis

Ankylosing Spondylitis

AS is an inflammatory disorder of the axial skeleton involving the sacroiliac joints, the diskovertebral junction, the apophyseal joints, and the costovertebral and costo- transverse joints. Symptoms usually start in the second and third decades of life and rarely after age 40. The axial distinctive radiographic findings of disease evolve over many years, with the earliest, most characteristic findings seen in the sacroiliac joint. AS and DISH are two different diseases that could usually be differentiated for several clinical features. Symptoms of AS begin at a young age, usually late adolescence and early adulthood, and consist of inflammatory spinal pain and stiffness and decreasing range of spinal motion. After many years, the illness can result in characteristic postural abnormalities (eg, Bechterew stoop). In contrast, DISH affects middle-aged and elderly persons and is often asymptomatic, or is associated with mild dorsolumbar pain and some restriction of spinal mobility [15].

Treatment

Therapy of diffuse idiopathic skeletal hyperostosis (DISH) is symptomatic and generally similar to the treatment of mild chronic low back pain, including physical therapy, exercise, and symptomatic pain management. No treatments have been documented to alter the disease course, and there have been no randomized trials examining the effectiveness of any interventions for the condition.

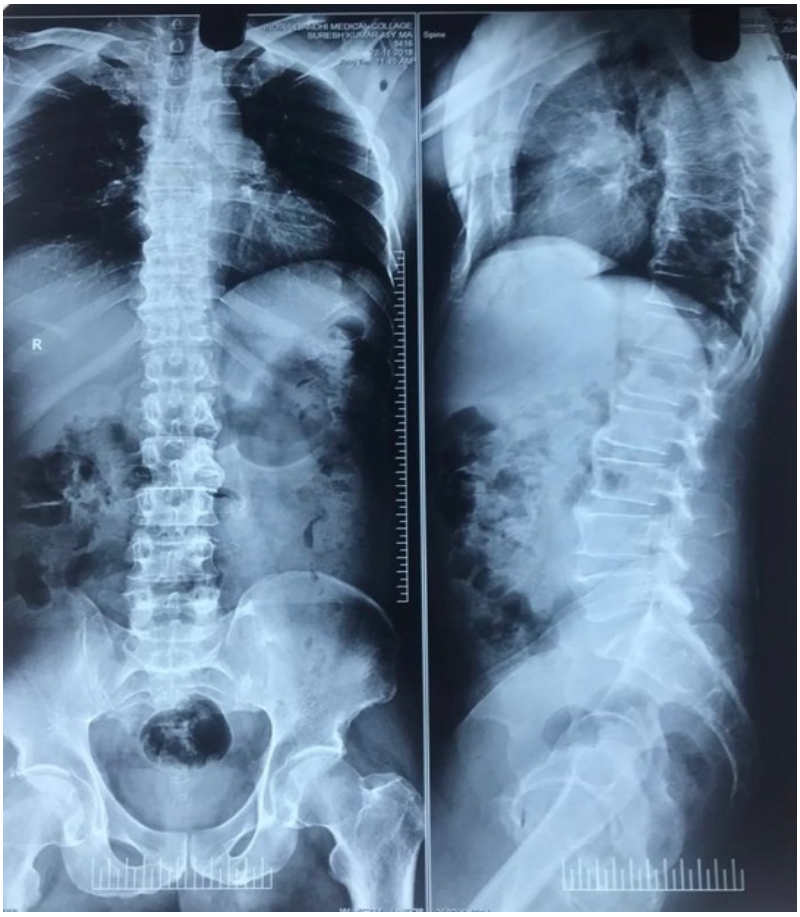

Fig 1: AP and Lateral view of spine showing flowing ligamentous calcifications ESP in dorsal spine.

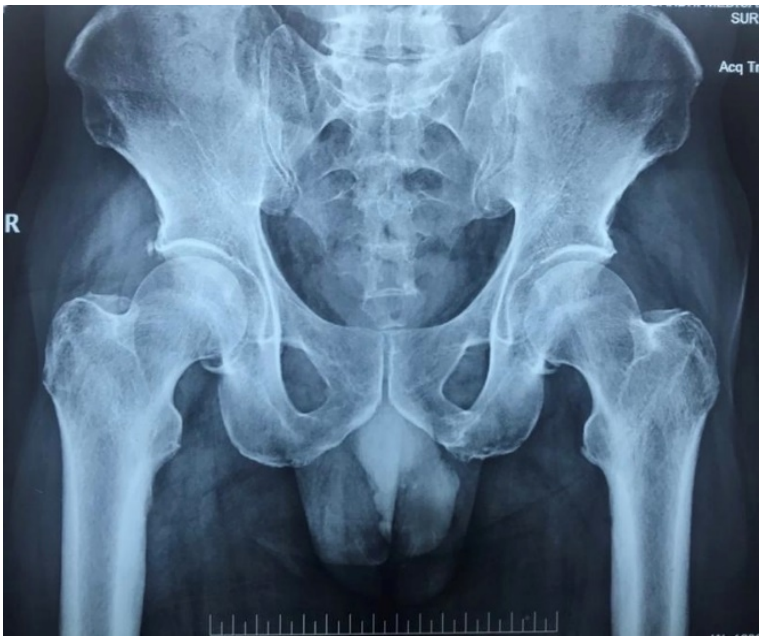

Fig 2: AP Pelvis and bilateral hip showing bony proliferation (also called whiskering), ligament ossification, and periarticular osteophytes.

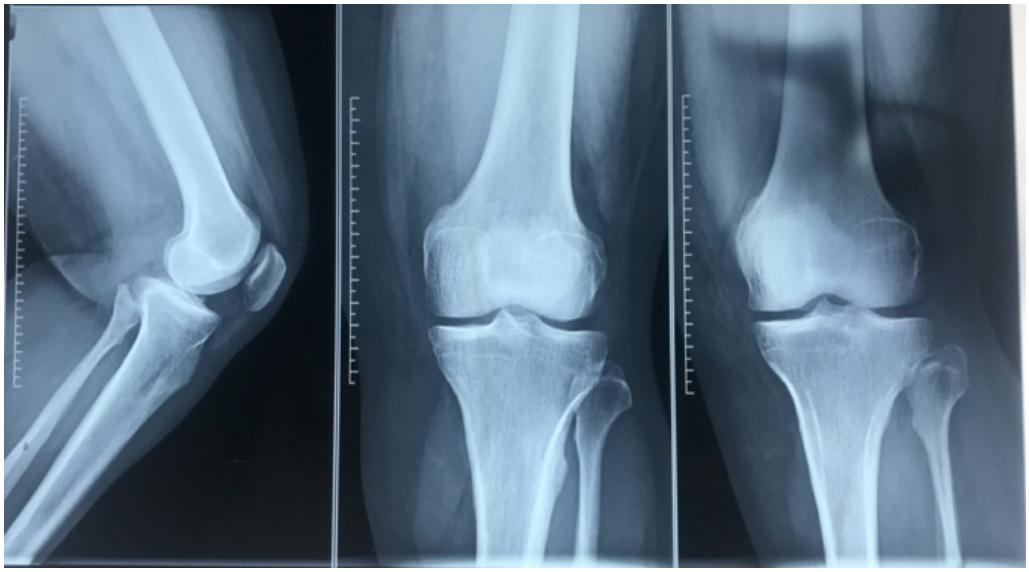

Fig 3: AP and lateral view of knee showing ossification at at the patella, at the insertion of the ligaments, and at the tibial tuberosity

References

1. Knaggs RL: Spondylitis deformans. Br J Surg,1925:I2:524-546.
2. Oppenheimer A: Calcification and ossification of vertebral ligaments. Roentgen study of pathogenesis and clinical significance. Radiology,1942:38:160-173.
3. Ott VR: Uber die spondylosis hyperostotica. Schweiz Med Wochenschr,1953:83:79-799.
4. Smith Smith CF, Pugh DG, Polley HF. Physiologic vertebral ligamentous calcification: An aging process. Am J Roentgen01,1955:74:1049-1058.
5. Forestier J, Rotes-Querol J. Senile ankylosing hyperostosis of the spine. Ann Rheum Dis,1950:9:321-30.10.1136/ard.9.4.321
6. Resnick D, Niwayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). Radiology,1976:119:559-68.
7. Julkunen H, Knekt P, Aromaa A. Spondylosis deformans and diffuse idiopathic skeletal hyperostosis (DISH) in Finland. Scand J Rheumatol,1981:10(3):193.
8. Julkunen H, Heinonen OP, Knekt P, Maatela J. The epidemiology of hyperostosis of the spine together with its symptoms and related mortality in a general population. Scand J Rheumatol,1975:4(1):23.
9. Kiss C, O'Neill TW, Mituszova M, Szilágyi M, Poór G. The prevalence of diffuse idiopathic skeletal hyperostosis in a population-based study in Hungary. Scand J Rheumatol,2002:31(4):226.
10. Hiyama A, Katoh H. Prevalence of diffuse idiopathic hyperostosis (DISH) assessed with whole spine CT. BMC Musculoskelet Disord,2018:19:178.
11. Cassim B, Mody GM, Rubin DL. The prevalence of diffuse idiopathic skeletal hyperostosis in African blacks. Br J Rheumatol,1990:29(2):131-2.
12. Senolt L, Hulejova H. Low circulating Dickkopf-1 and its link with severity of spinal involvement in diffuse idiopathic skeletal hyperostosis. Ann Rheum Dis,2012:71(1):71.
13. Miyazawa N, Akiyama I. Diffuse idiopathic skeletal hyperostosis associated with risk factors for stroke. Spine,2006:31:E225–E229
14. Utsinger PD. Diffuse idiopathic skeletal hyperostosis. Clin Rheum Dis,1985:11:325.
15. Ignazio Olivieri. Diffuse idiopathic skeletal hyperostosis: differentiation from ankylosing spondylitis. Curr Rheumatol Rep,2009:11(5):321-8.