



4^ο ΔΙΑΠΑΝΕΠΙΣΤΗΜΙΑΚΟ ΠΡΟΓΡΑΜΜΑ ΕΚΠΑΙΔΕΥΣΗΣ ΣΤΗ ΡΕΥΜΑΤΟΛΟΓΙΑ 2022-24

DISH & Haemochromatosis

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DISH

- Diffuse idiopathic skeletal hyperostosis (DISH) is bone forming disease characterized by enthesal ossification and/or calcification involving mainly the thoracic spine

- Peripheral joints and adjacent entheses can also be involved

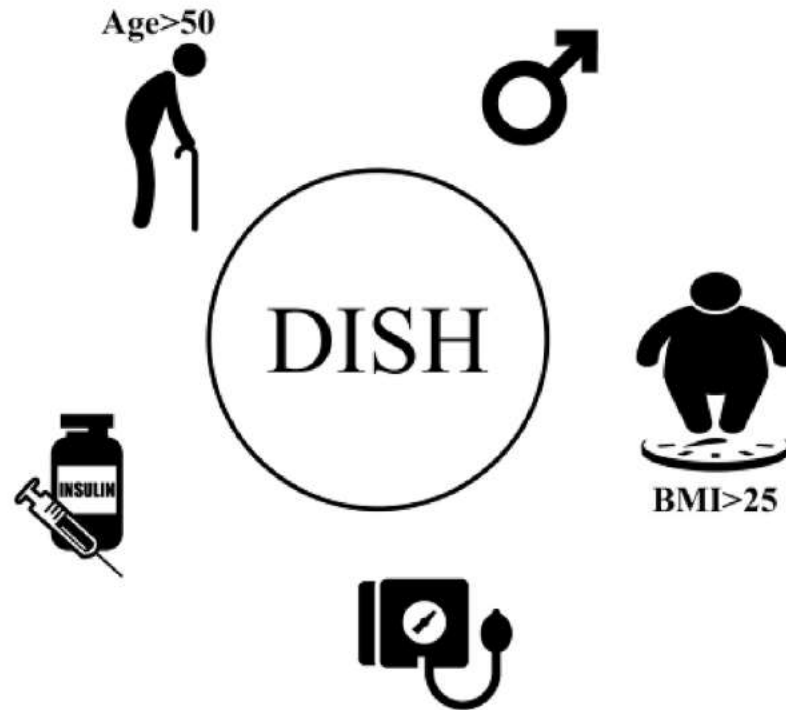


Fig. 2. DISH is associated with older age, male sex, obesity, hypertension, and diabetes mellitus.

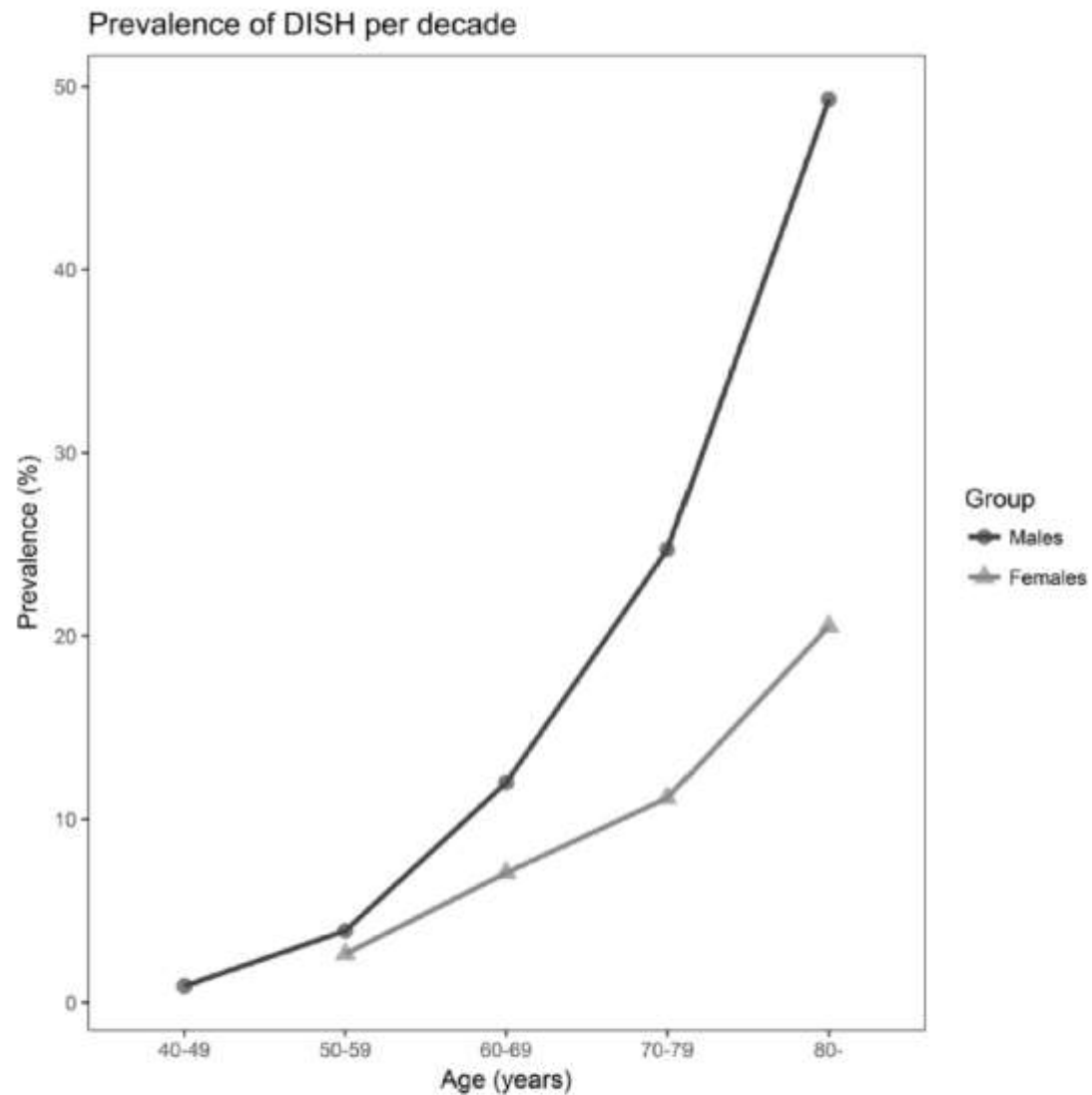


Fig. 3. Prevalence of DISH based on the data of studies describing the prevalence of DISH. The prevalence was calculated using data from seven different studies. Five studies presented their findings separate for men and women and two studies described only one sex (Julkunen 1971, Bloom 1984, Cassim 1990, Kiss 2002, Pappone 2005, Holton 2011, and Hirasawa 2016). Most authors used Resnick criteria to diagnose DISH on conventional imaging and studies were performed in Finland, Israel, South Africa, Hungary, Italy, USA, and Japan.

Pathophysiology of DISH

Differentiation of mesenchymal cells into bone-forming cells

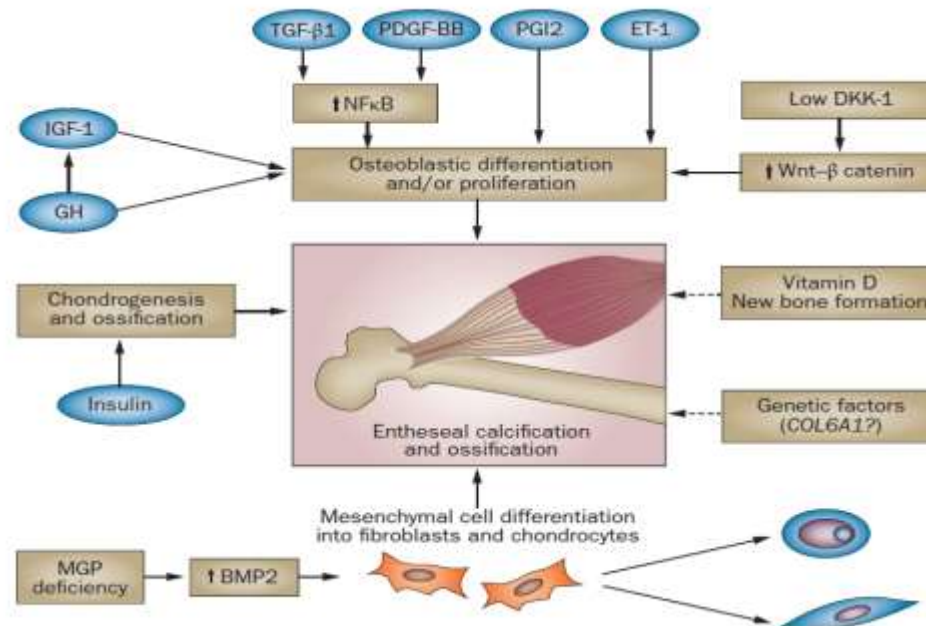


Figure 3 | Suggested factors that promote enteseal calcification and ossification in DISH. GH promotes osteoblastic differentiation and/or proliferation either directly, or indirectly by promoting IGF-1 production. NFκB activity is stimulated by PDGF-BB and TGF-β1 and promotes soft tissue ossification. ET-1 and PGI2 may promote osteoblastic differentiation. Low DKK-1 levels enhance the activity of the Wnt-β-catenin pathway, which induces osteoblastic differentiation and/or proliferation. MGP deficiency increases BMP2 activity, leading to the differentiation of mesenchymal cells into chondrocytes and fibroblasts, which may calcify. Insulin induces chondrogenesis and ossification. Abbreviations: BMP2, bone morphogenetic protein 2; DISH, diffuse idiopathic skeletal hyperostosis; DKK-1, Dickkopf-related protein 1; ET-1, endothelin 1; GH, growth hormone; IGF-1, insulin-like growth factor 1; MGP, matrix Gla protein; NFκB, nuclear factor κB; PDGF-BB, platelet-derived growth factor BB; PGI2, prostaglandin I2; TGF-β1, transforming growth factor β1.

Table 1 | Suggested main diagnostic features of DISH

Definition	Number of vertebrae connected by bony bridges	Peripheral enthesopathies	SIJ involvement
Resnick and Niwayama ³	4 in the thoracic spine	Not required	Not involved
Arlet and Mazieres ⁹³	3 in the lower thoracic spine	Not required	Ossification in the vicinity of the SIJ allowed
Utsinger ²⁸			Involvement of the SIJ is not an exclusion criterion
Definite DISH	4 in the thoracolumbar spine	Not required	
Probable DISH	2 in the thoracolumbar spine	Bilateral enthesopathies	
Possible DISH	2 in the thoracolumbar spine None	Not required Symmetrical enthesopathies preferably in >2 anatomical sites	
Rogers and Waldron ¹⁴	3 in the thoracic spine	Peripheral calcification or ossification of ligaments and/or entheses	No reference

Abbreviations: DISH, diffuse idiopathic skeletal hyperostosis; SIJ, sacroiliac joint.

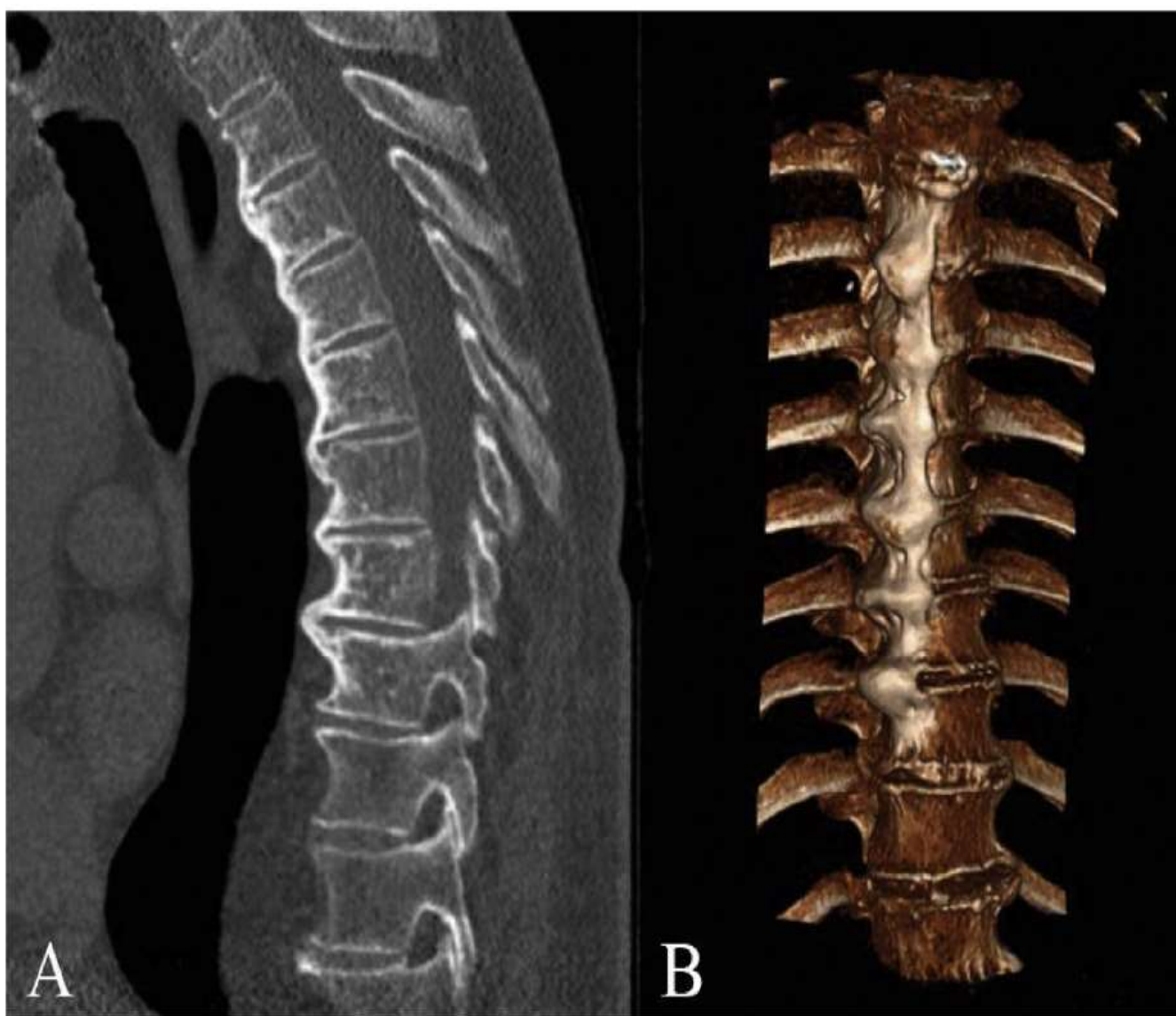
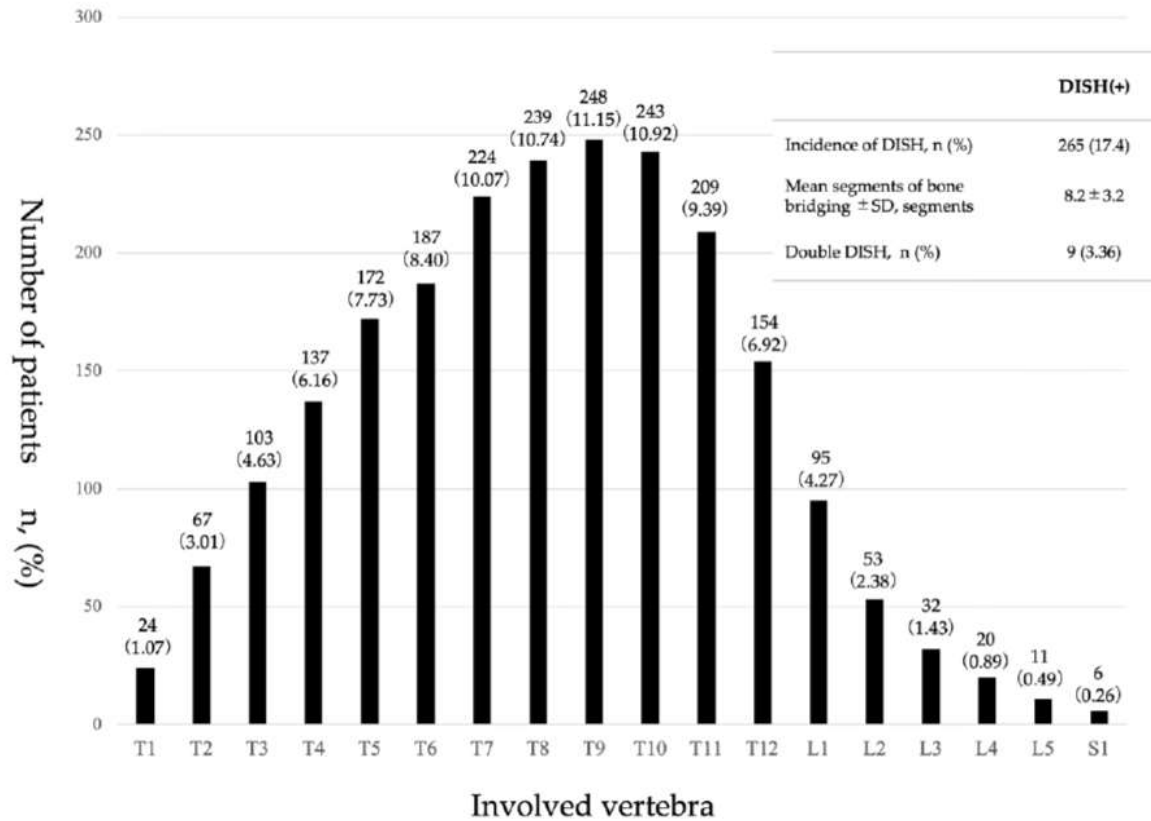
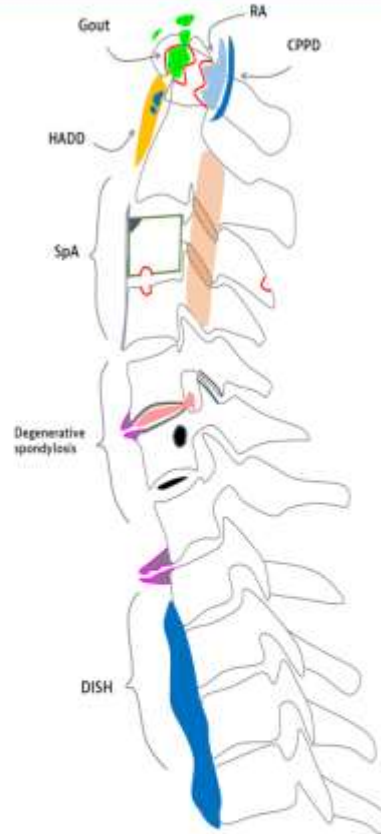
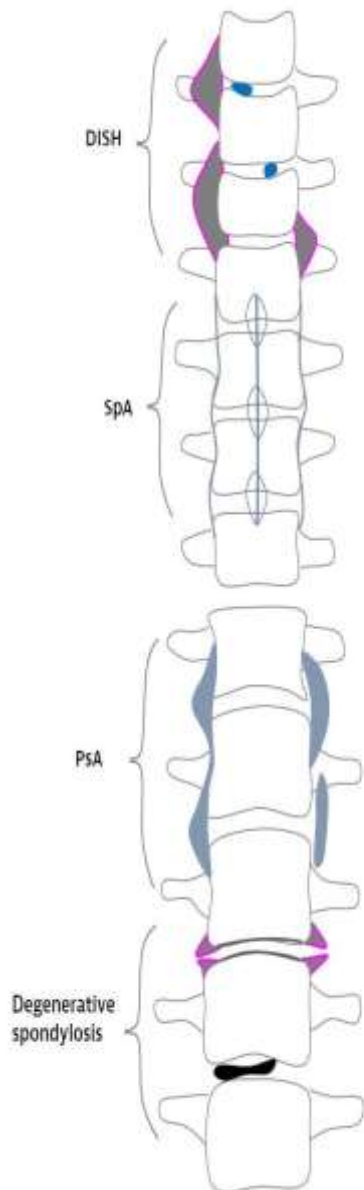


Fig. 1. Computed tomography scan of a 70-year-old male patient. In A, the sagittal reconstruction is shown, clearly demonstrating bridges of bone over more than four vertebral bodies. In B, the three-dimensional reconstruction demonstrates the flowing cortical new bone.

Distribution of DISH





RA	Rheumatoid arthritis. Erosion of the dens and synovitis.
Gout	Erosions of C1-C2 and monosodium urate deposition.
CPPD	Calcium pyrophosphate deposition disease. Calcification in the transverse ligament of the atlas.
DISH	Diffuse idiopathic skeletal hyperostosis. Coarse osteophytes, thick flowing ligament ossification and disc calcification.
Degenerative	Bone sclerosis of the endplates and facet joint surface; disc protrusion; narrowing of intervertebral space; osteophytes; bone cyst and vacuum phenomenon and joint space narrowing.
HADD	Hydroxyapatite deposition disease. Calcification in the longus colli muscle tendon and muscle edema.
SpA	Spondyloarthritis. Syndesmophytes (ossification of annulus fibrosus), ossification of interspinous and anterior longitudinal ligaments, erosions of the endplate and spinous process, square vertebra, sclerosis of anterior vertebral corner, and ankylosis.

1. DISH frequently involves the thoracic spine, which is not usually involved in spondylosis until late stages
2. The intervertebral disc height is usually preserved in patients with DISH, whereas it is reduced in individuals with spondylosis
3. In spondylosis, the main target is the cartilage of the intervertebral discs, whereas in DISH the target is the enthesis (with sparing of the intervertebral discs)
4. The osteophytes in spondylosis are usually transverse, whereas the osteophytes in DISH are coarse, vertical and bridging

Table 1. Main differences between SpA and DISH.

	SpA	DISH
Clinical Characteristics	<ul style="list-style-type: none">• Adolescents, young adults• Hx of psoriasis, IBD, uveitis, dactylitis• Family history• Hx of back pain before the age of 45• Inflammatory back pain or stiffness• Postural abnormalities• Response to NSAIDs	<ul style="list-style-type: none">• Elderly, but can be found in patients under 45 yo• Usually asymptomatic• Limitation of spinal mobility• Kyphosis, rarely dorsolumbar pain• dysphagia, hoarseness and stridor in some cases• strong association with DM and obesity
Laboratory	<ul style="list-style-type: none">• High CRP levels• HLA-B27	<ul style="list-style-type: none">• Metabolic syndrome associated features
Radiographic findings	<ul style="list-style-type: none">• Primarily affects the anulus fibrosus of the intervertebral discs• Continuous spine lesions• Shiny corner sign• Bamboo spine• Vertebral body squaring• Syndesmophytes• Sacroiliitis: Joint space stenosis, subchondral sclerosis and ankylosis of the lower one third (synovial) part of the SI joint	<ul style="list-style-type: none">• Continuous bulky calcification of the anterior longitudinal ligament• Osteophytic bridging of at least 4 continuous vertebrae, typically of the thoracic spine• Whiskering enthesopathy of the greater and lesser trochanters, ischial tuberosities and iliac crests• Periarticular hyperostosis of the hands, knees, elbows and quadriceps tendon insertion



Figure 1. Imaging features in diffuse idiopathic skeletal hyperostosis and in spondyloarthropathies. Left: diffuse idiopathic skeletal hyperostosis – CT scan of thoracic spine broad arrows showing coarse intervertebral bone formation and ossification. Right: ankylosing spondylitis – x-ray of lateral thoracic spine, thin arrow showing syndesmophytes.

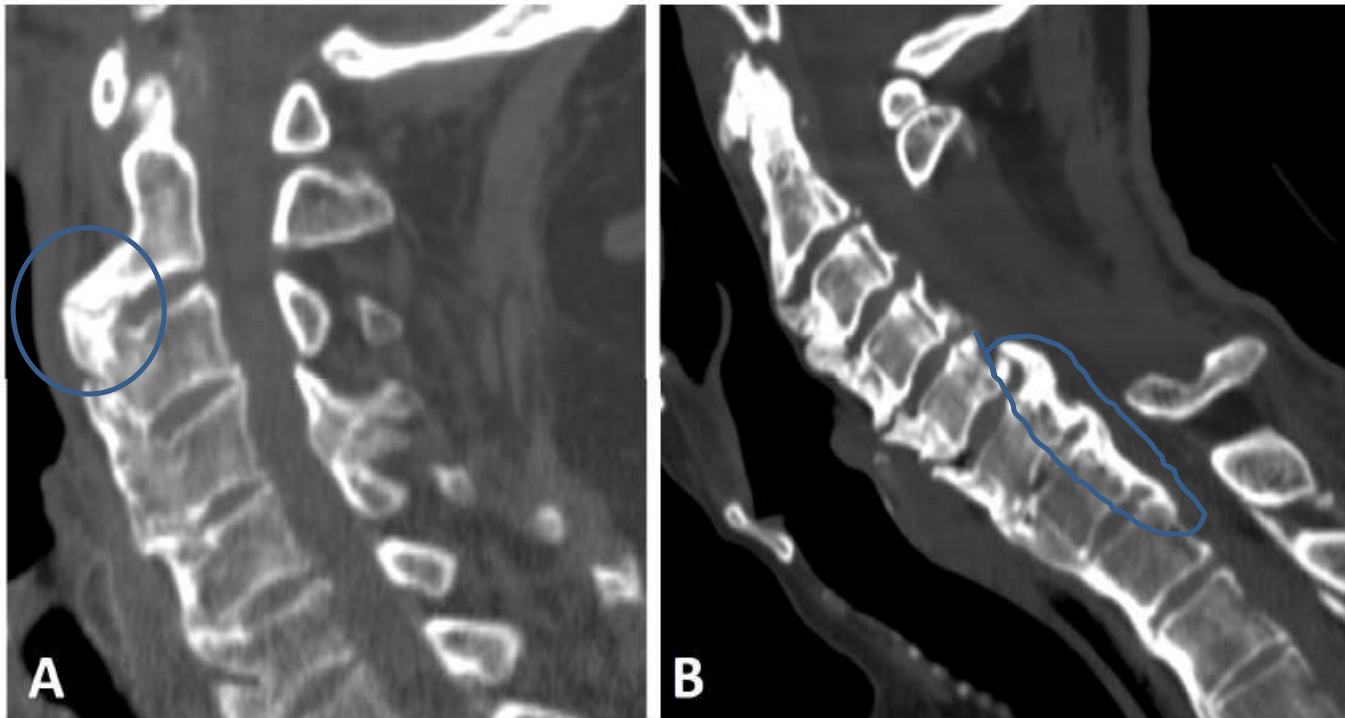
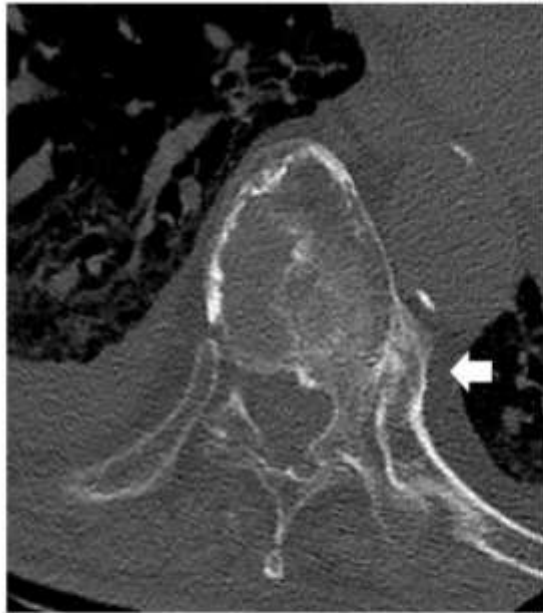


Figure 2. Sagittal CT reconstruction images of two patients with thoracic (not shown) and cervical DISH. (A) Flowing chunky osteophytes are located anteriorly to the vertebrae, forming a “candle flame” or “parrot-beak” image. (B) Thick, not-yet-flowing anterior osteophytes and thick posterior osteophytes at the C5–6 level.



(a)



(b)

Figure 2. Evaluation of hyperostosis around the costovertebral joint. The cross-sectional CT image passing through the costovertebral joint from T1 vertebra to the T12 vertebra was used to evaluate the hyperostosis around the costovertebral joint. (a) There is ankylosis and excrescence in the left costovertebral joint (white arrow). (b) There is bone bridging in the right costovertebral joint (black arrow).

1. There is no ankylosis of the facet joints in DISH. Only hypertrophic changes with capsular ossification.
2. On the contrary costovertebral joints could be ankylosed
3. Involvement of CV joints in DISH s approximately 40-50%

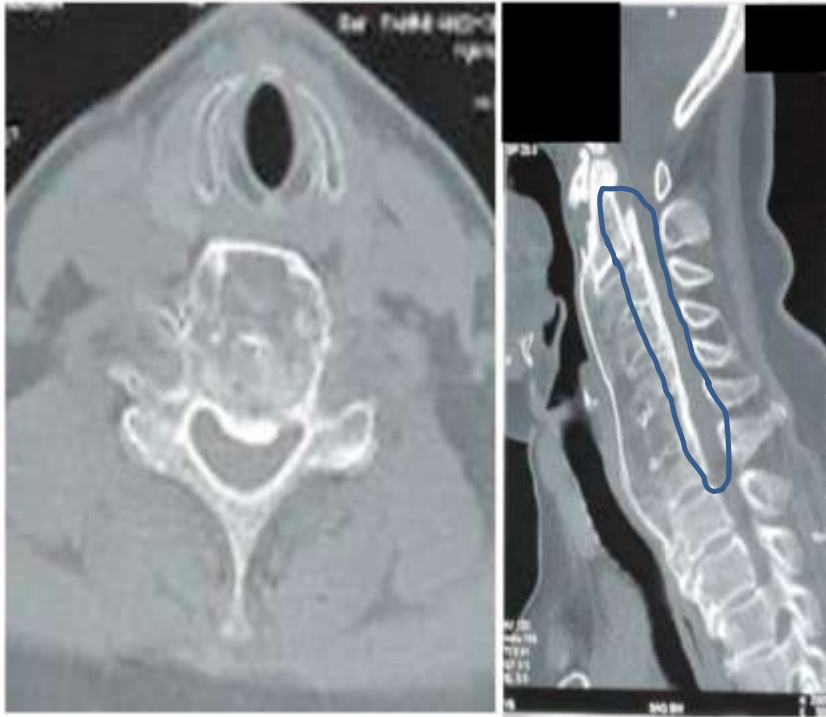


Fig. 3. CT Cervical. Axial and sagittal view showed diffuse ossification of the corpus of vertebra cervical 2 to 7 and anterior thickening of the OPLL.

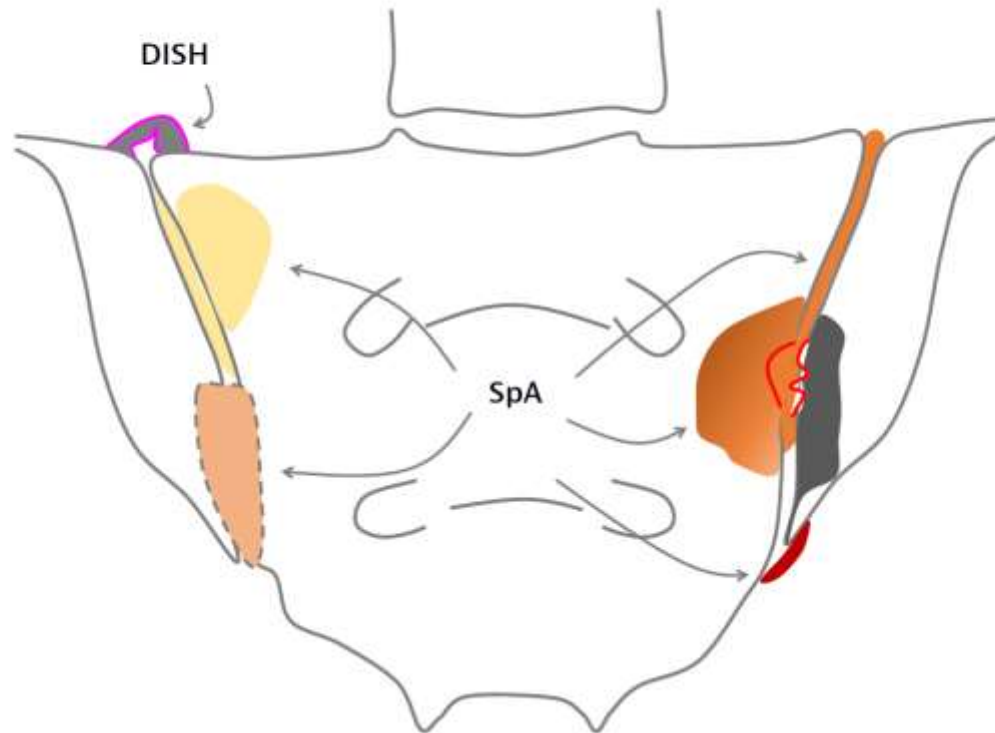
1. Prevalence of DISH in patients with cervical OPLL is approximately 50 % with older age as significant correlating factor.
2. OPLL can cause myelopathy
3. Correlation with DM
4. Common in Asian populations with DISH



Figure 2. Lateral cervical spine radiograph. The extensive ossification of the stylohyoid ligaments, identified by asterisks () extends from the skull base to the hyoid bone. A pseudoarticulation between the abnormal ossification and the hyoid bone is evident. Also observed is an 8 mm thick vertically oriented shield of ossification adjacent to the anterior vertebral bodies and disc spaces from C2 through C7 typical of DISH. The intervertebral disc spaces are typically preserved and the facet joints are normal in DISH as illustrated in this case. Of note, there is a 1 cm circular calcified mass located anterior to the C6-7 level most likely representing a benign thyroid cyst.*

- elongation of the styloid process, which results from enthesal calcification and ossification of the stylohyoid ligament
- In rare cases, this elongation has been reported to cause craniofacial or cervical pain, termed Eagle syndrome

Sacroiliac joint



- | | |
|---------------|---|
| SpA | Spondyloarthritis. Bone marrow edema, capsulitis, subchondral erosion, joint space inflammation, sclerosis, fat metaplasia, backfill and ankylosis. |
| OA | Osteoarthritis. Osteophytes, bone sclerosis and vacuum phenomenon in the joint space. |
| DISH | Diffuse idiopathic skeletal hyperostosis. Coarse bridging osteophytes across the anterior joint capsule. |
| SAPHO | Synovitis, acne, pustulosis, hyperostosis, osteitis syndrome. Hyperostosis and joint erosions. |
| Septic | Bone marrow edema, erosion, joint effusion, soft tissue inflammation and osteoporosis. |

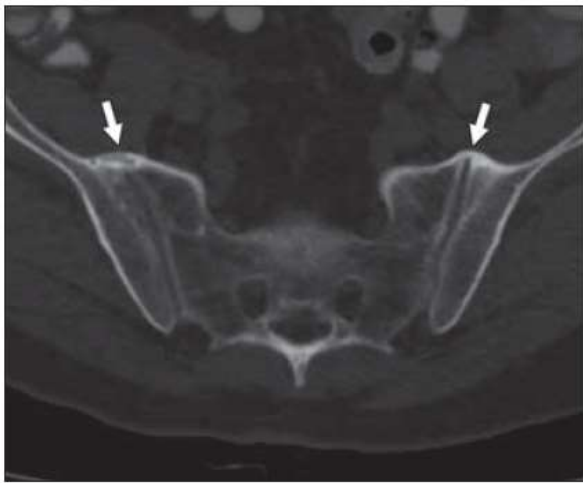


Fig. 1—75-year-old man with diffuse idiopathic skeletal hyperostosis. Axial CT image of sacroiliac joints (SIJs) shows bilateral anterior bridging (arrows). There is some iliac cortical irregularity with subchondral cysts in right SIJ and no evidence of intraarticular abnormality in left SIJ.



Fig. 2—74-year-old man with diffuse idiopathic skeletal hyperostosis. Axial CT image of sacroiliac joints (SIJs) shows bilateral enthesal bridging and ankylosis (arrowheads). Anterior bridging of right SIJ can also be detected. There is no evidence for intraarticular SIJ abnormality.

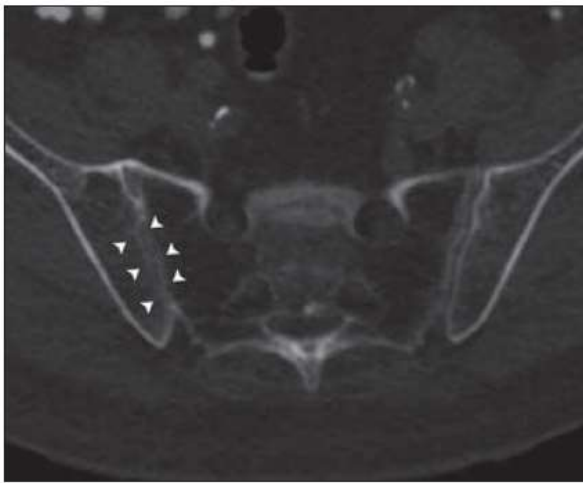
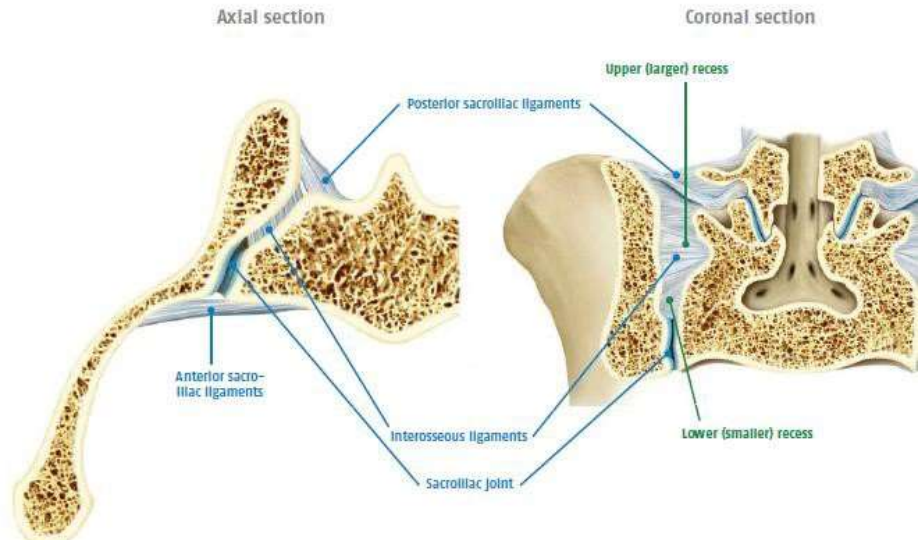
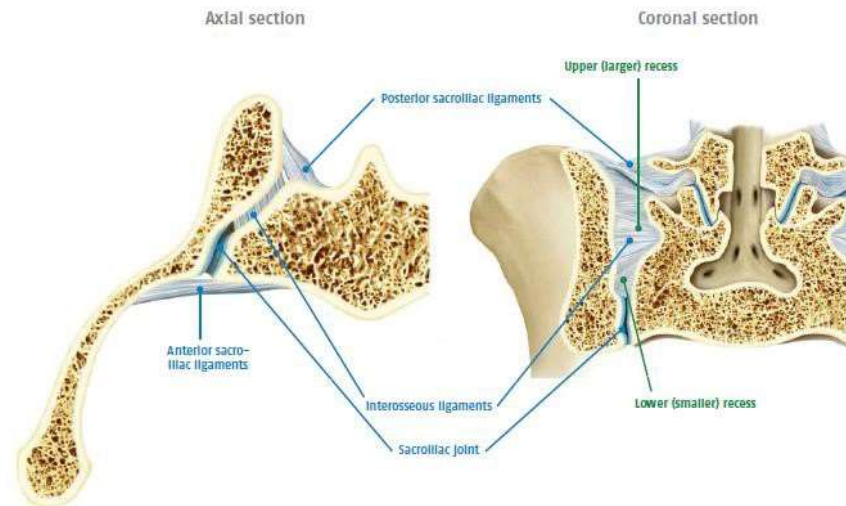
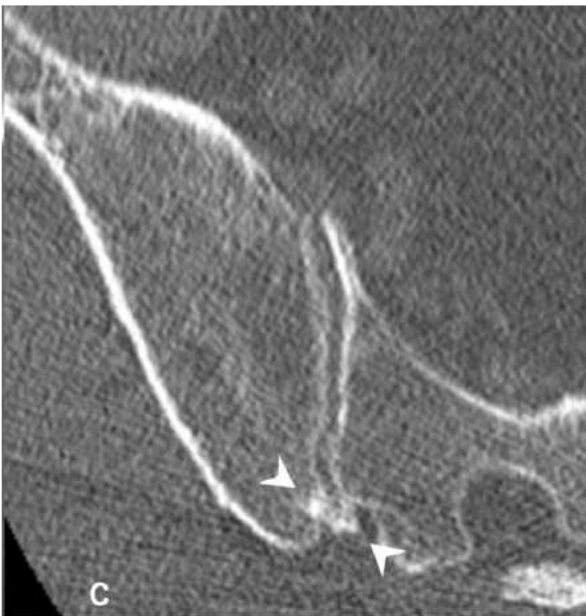
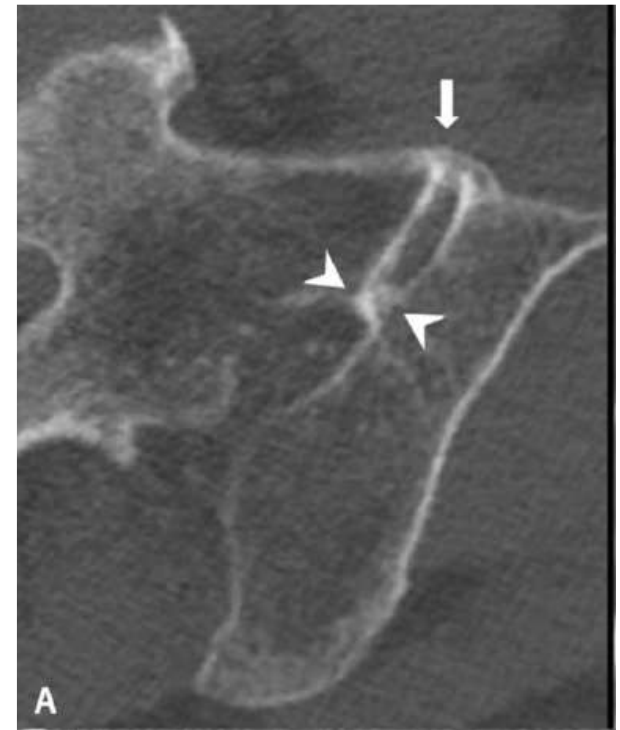
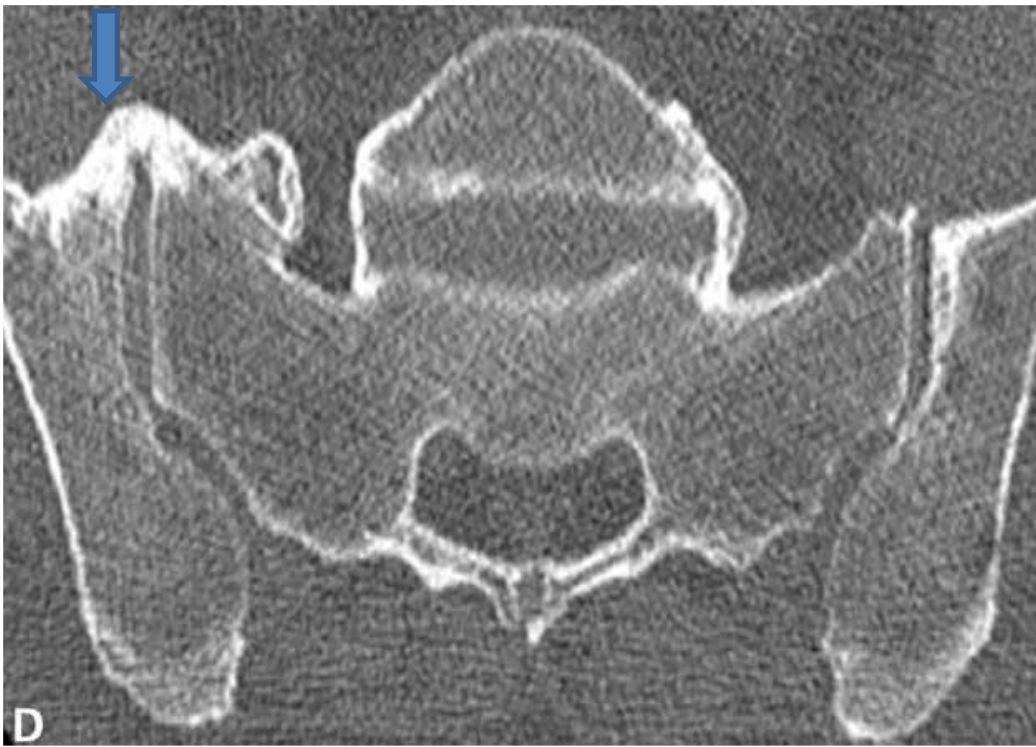


Fig. 3—84-year-old man with diffuse idiopathic skeletal hyperostosis. Axial CT image of sacroiliac joints (SIJs) shows right-sided intraarticular SIJ ankylosis (arrowheads) as well as anterior bridging. Left SIJ is intact.



1. anterior bridging (commonest)
2. posterior bridging
3. enthesal bridging



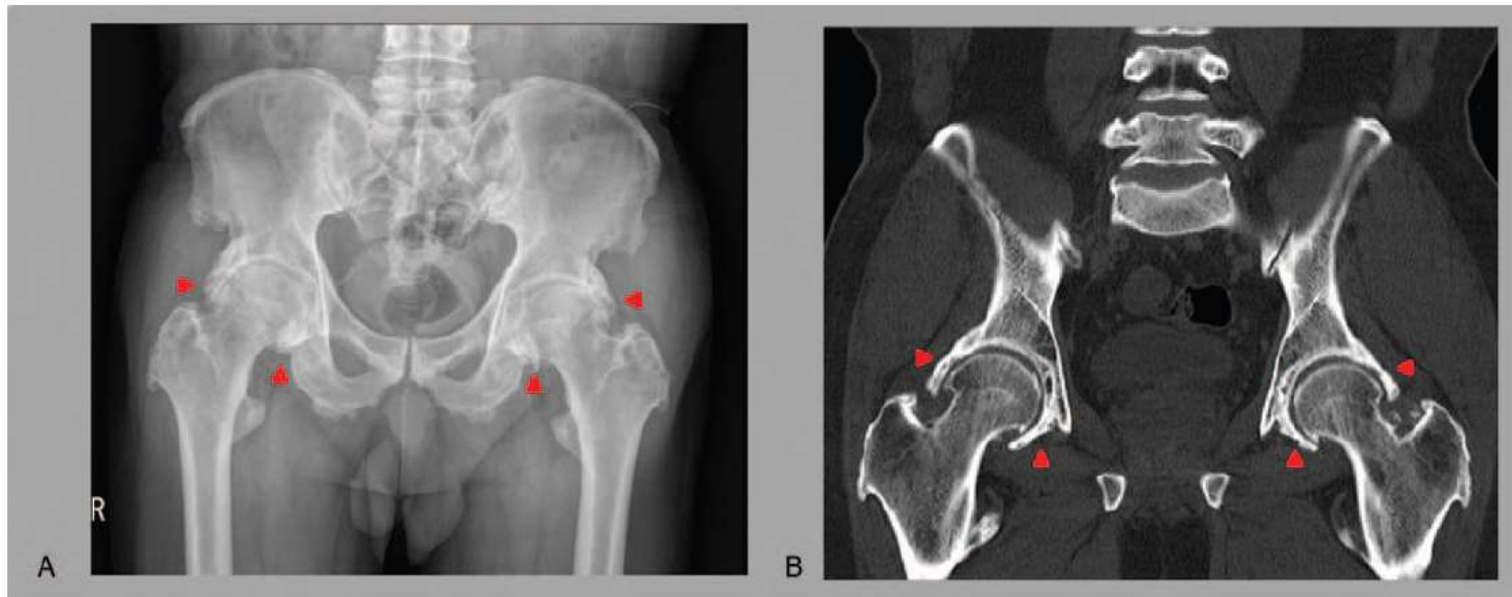


Figure 2. On anteroposterior pelvic X-ray (A) and coronal pelvic computed tomography (B), severe and diffuse hyperostosis was observed around the bilateral hip joints (red arrow heads).

AS vs DISH. The two sides of the same coin ?



- Inflammatory changes are common on the spine MRI of symptomatic DISH patients, and may fulfill the ASAS definition of a spine MRI suggestive of axial spondyloarthritis (axSpA).
- Similar rate of new bone formation and radiographic progression in both diseases

But!

- By contrast, bone marrow edema lesions are scarce on sacroiliac joint (SIJ) MRI of symptomatic DISH patients.
- Erosions, both at the spine and the SIJ level, were infrequent and could help discriminate DISH from axSpA in the elderly.

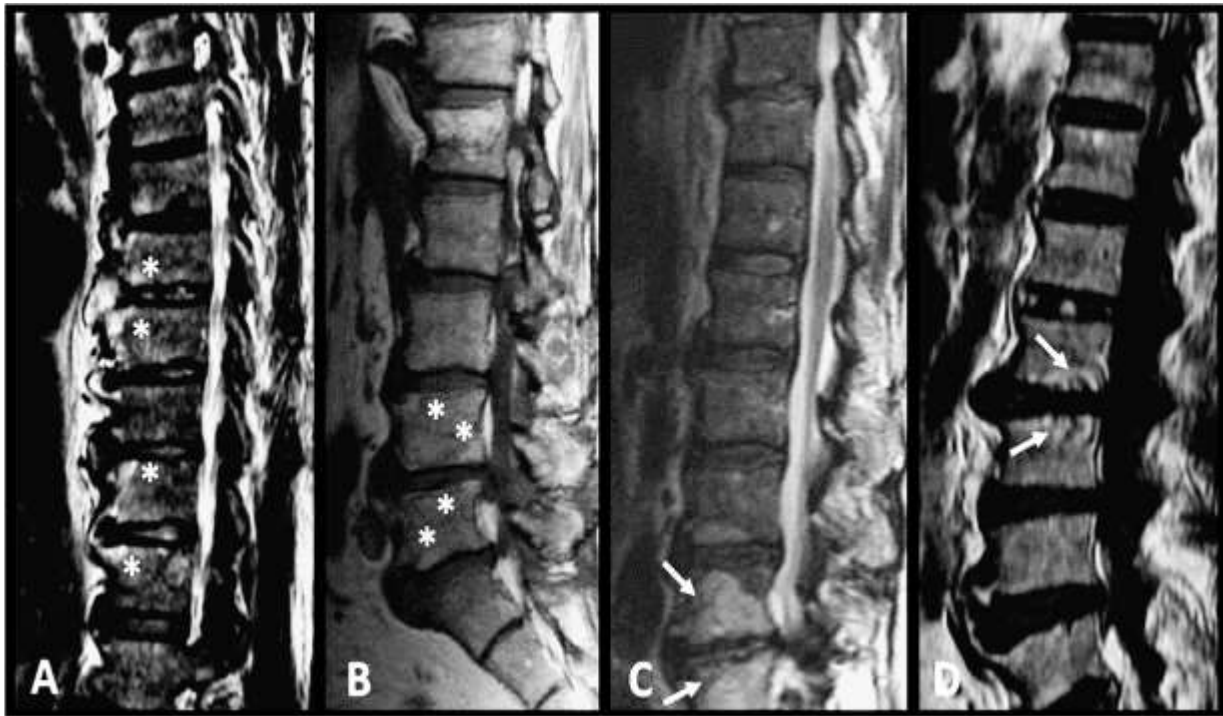


Fig. 1 Active inflammatory and chronic fatty lesions in the vertebral corners and endplates of patients with DISH: a STIR image of the thoracic spine with multiple anterior spondylitis lesions (*). b T1w image of the lumbar spine with multiple anterior and posterior corner fat deposition lesions (*) c STIR image of the lumbar spine showing inflammatory spondylodiscitis at L5-S1 and d T1w image showing endplate fat deposition at L2–3. flowing osteophytes can be detected in a and d

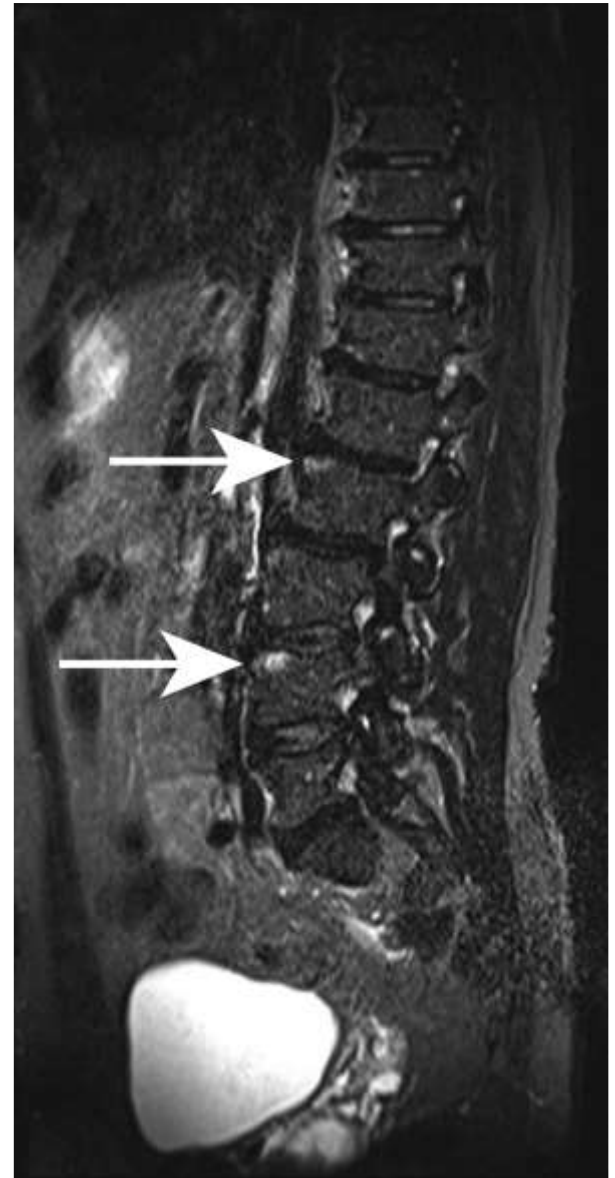
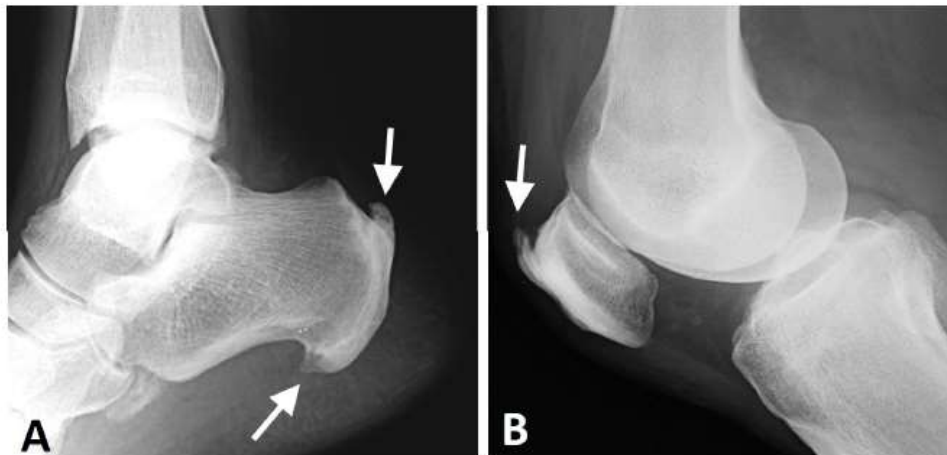


Figure 4. Diffuse idiopathic skeletal hyperostosis, short-tau inversion recovery sequence. Arrows indicate bone marrow edema on the upper-anterior vertebral corners

Extraspinal manifestation of DISH



FIG. 2. Elbow enthesopathies involving the olecranon (1) and both epicondyles (2).



1. involvement of joints usually unaffected by primary OA
2. increased hypertrophic changes compared with primary OA
3. prominent enthesopathies at various sites adjacent to peripheral joints
4. calcification and ossification of entheses in sites other than joints.

Iris Eshed. Diagnostics 2023

Reuven Mader et al. Rheumatology 2009

Fig. 1 CT scan of a male patient with morphological characteristics of both AS and DISH



In AS, the ankylosis involves the annulus fibrosus of the intervertebral discs

In DISH, the ALL is the primary anatomical site of ossification

The coronal (A) and sagittal (B) CT image of an 86-year-old male with radiographic characteristics of both AS and DISH. In the coronal image, the fused SI joint (AS) is demonstrated with the bilateral fusion of the vertebral bodies in the thoracic region (bamboo spine). In the sagittal image, abundant new bone is bridging the vertebral bodies of the thoracic and upper lumbar spine, typical for DISH.

Complications of DISH

- dysphagia-esophageal obstruction
- myelopathy-radiculopathy-spinal stenosis
- aspiration pneumonia
- stridor and hoarseness
- thoracic outlet syndrome.
- spinal fractures with instability and secondary neurological deficits
- heterotopic ossification
- enthesophytes can also make endotracheal and endoscopic procedures difficult.

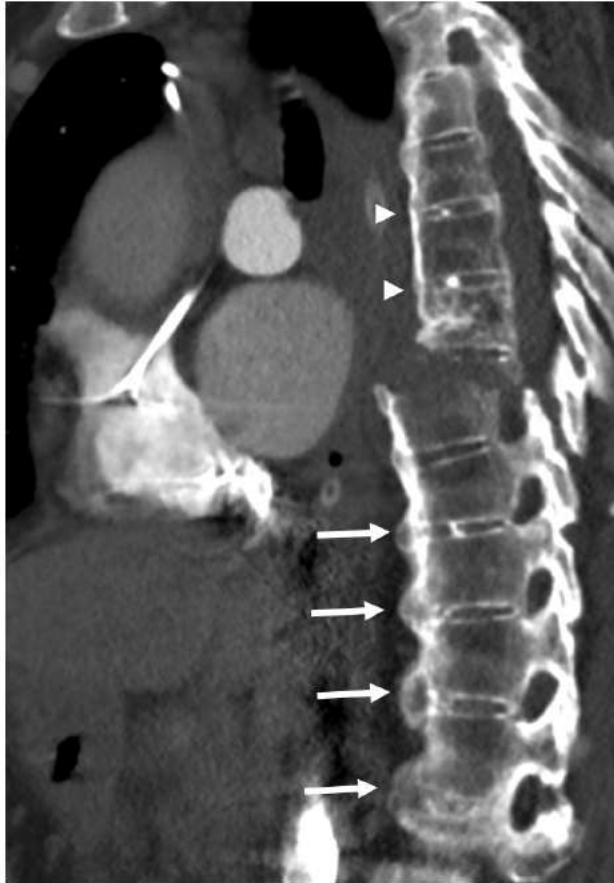


Figure 6. Sagittal CT reconstruction of the thoracic spine of a 78-year-old patient with thoracic DISH after low-energy trauma, showing an unstable extension-type fracture of the mid-thoracic spine. In the given slice, there are two vertically oriented bony bridges in the upper thoracic spine (arrowheads) that may lead to the misconception that this is a patient with ankylosing spondylitis; however, there are clearly many flowing osteophytes compatible with DISH in the lower part of the thoracic spine (arrows).

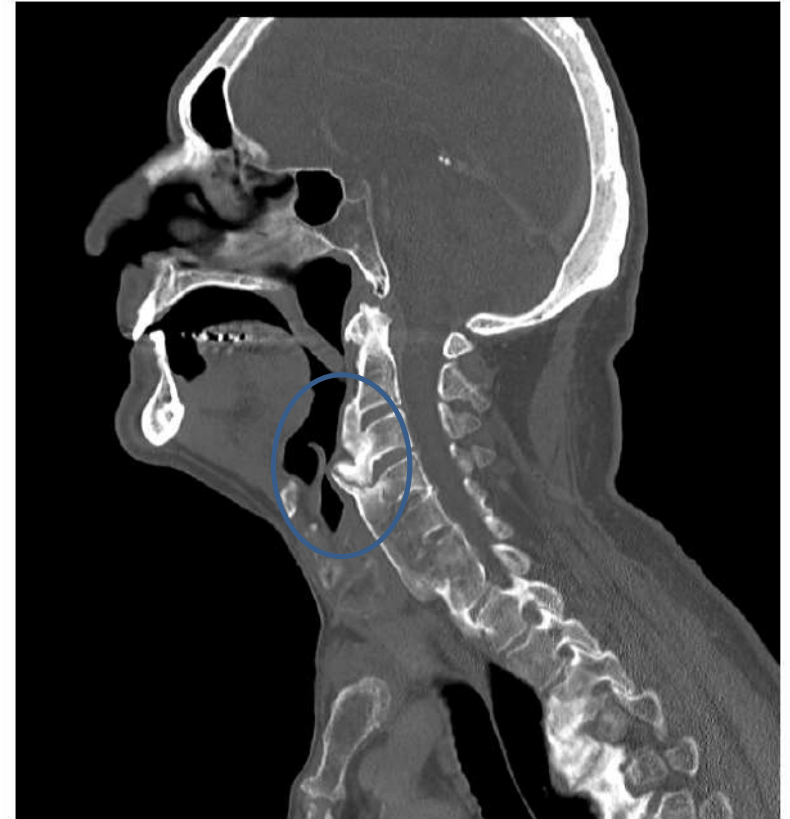


FIGURE 2 | CT: sagittal scan showing anterior osteophytes and ossification of the anterior longitudinal ligament.

Box 1 | Suggested therapeutic interventions in DISH

- Weight reduction and physical activity
- Physical therapy
- Diet low in saturated fat and carbohydrate
- Instruction in joint-protection techniques
- Enthesal protection and local corticosteroid infiltration
- Local application of NSAIDs or capsaicin
- Analgesics
- Systemic NSAIDs
- Avoidance of thiazide diuretics and β -adrenergic antagonists
- Prevention of accidental falls and aspiration pneumonia
- Prevention of post-surgical heterotopic ossification
- Surgical resection of cervical osteophytes (in extreme cases)

Abbreviation: DISH, diffuse idiopathic skeletal hyperostosis.



Prevention of complications

Precaution in patients undergoing endotracheal intubation or upper GI endoscopy
Prevention of heterotopic ossification following orthopaedic surgical procedures: anti-vitamin K, NSAIDs and irradiation

Future perspectives

Interventions at molecular level to inhibit factors that might promote mesenchymal differentiation into osteoblasts:
NF- κ B, PDGF-BB, TGF- β 1, PGI₂ and BMP-2

Practice points

- Do not look only thoracic spine
- Look pelvic entheses and trochanters
- SI joints can also be involved but without erosions and BME
- If you see extensive enthesal ossification and new bone formation especially in the joints which are not commonly involved in OA think about DISH
- Be aware regarding complications
- MRI of thoracic-lumbar spine is not helpful especially in painful DISH

Haemochromatosis



- Comprises a group of inherited disorders that can cause iron overload, which primarily affects **the liver and joints** and results from a failure in the regulation of the key liver-derived iron regulatory hormone hepcidin to respond to increasing iron stores
- The cause of 95% of cases of hemochromatosis is a homozygous mutation in *HFE* (hemostatic iron regulator; chromosome 6p22.2, exon 4)
- This disorder affects approximately 1 in every 150 to 220 persons of northern European descent

Table 1. Classification of Hemochromatosis According to the Molecular Target.*

Variable	HFE Hemochromatosis	Non-HFE Hemochromatosis	
Molecular target	<i>HFE</i>	Genes encoding HJV, hepcidin, or Tfr2	Gene encoding ferroportin
Frequency	Common; often due to p.C282Y homozygosity; in rare cases, due to compound heterozygosity	Very rare	Very rare
Population at risk	Northern European origin	Any population	Any population
Age group at clinical risk	Adults	Persons <30 yr of age (with HJV or hepcidin as target), or adults (with Tfr2 as target)	Adults
Mechanism	Loss of function of target	Loss of function of target	Gain of function of target, resistance to hepcidin
Hepcidin production	Reduced	Reduced	Increased

* The classification is from Girelli et al.¹ HFE hemochromatosis is caused by mutation of *HFE* (most often the homozygous p.C282Y mutation), whereas non-HFE hemochromatosis is caused by mutation of the genes encoding hemojuvelin (HJV), hepcidin, transferrin receptor 2 (Tfr2), or ferroportin.

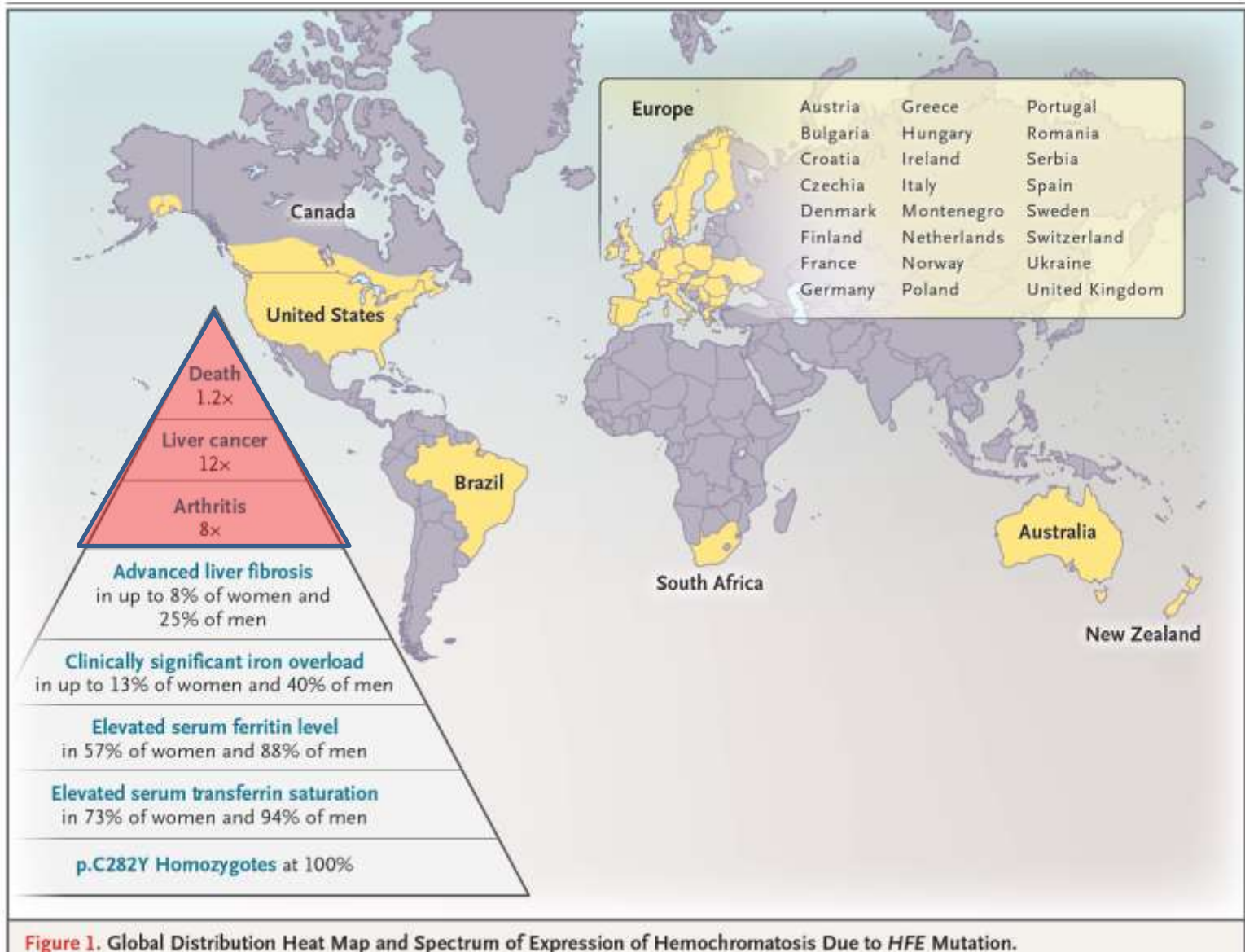


Figure 1. Global Distribution Heat Map and Spectrum of Expression of Hemochromatosis Due to *HFE* Mutation.

A**Suspected Hemochromatosis**

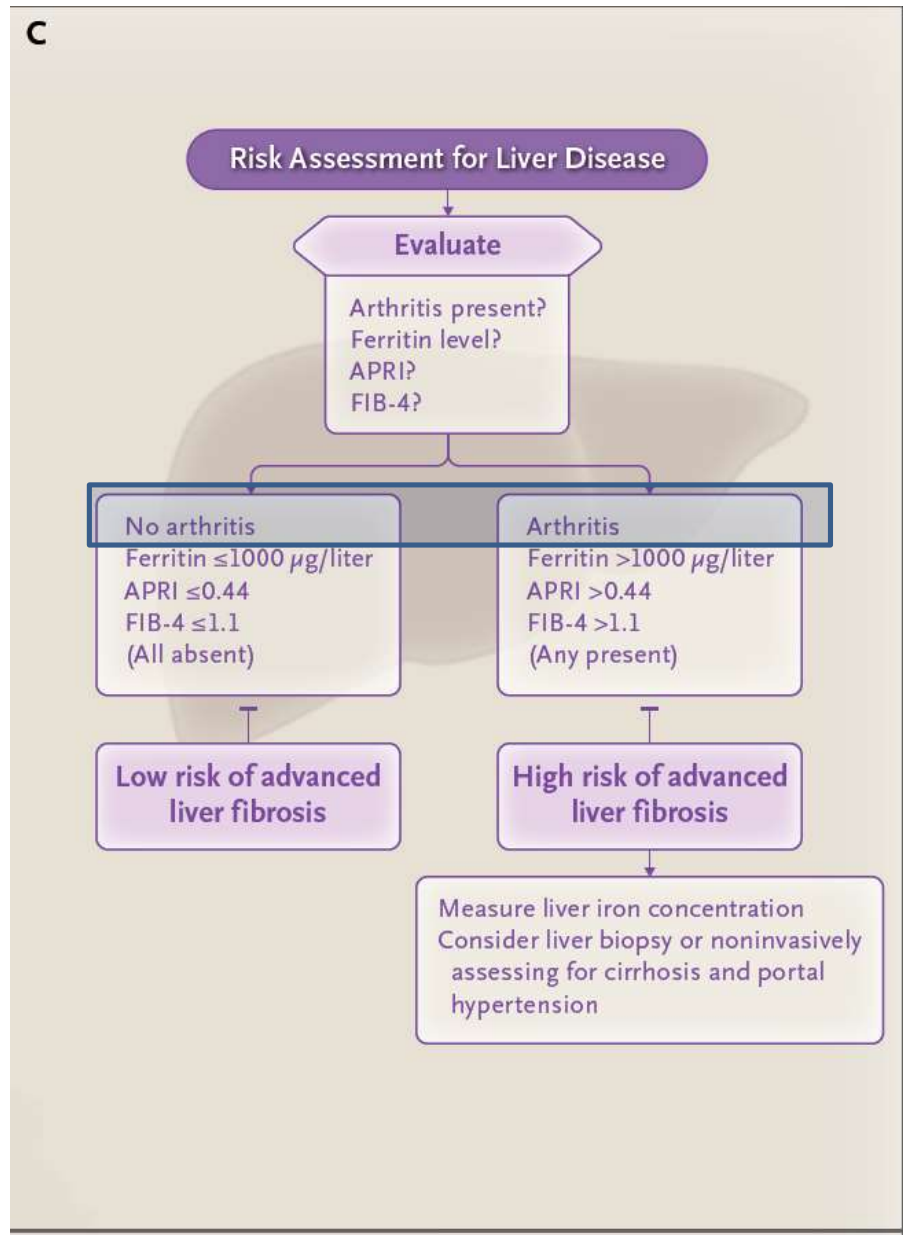
Family history

Biochemical triggerElevated ferritin level
Transferrin saturation >45%**Hematologic trigger**

Mean red-cell volume >94 fl

Clinical triggerArthritis or liver disease
(hepatomegaly or abnormal serum
aminotransferases)
Another compatible clinical feature**B****Diagnosis****Northern European origin**Assessment for *HFE* mutation
Serum transferrin saturation and ferritin level**Other origin**Serum transferrin saturation and ferritin level
Genetic testing for known mutations only in highly
selected cases after proof of iron overload

C



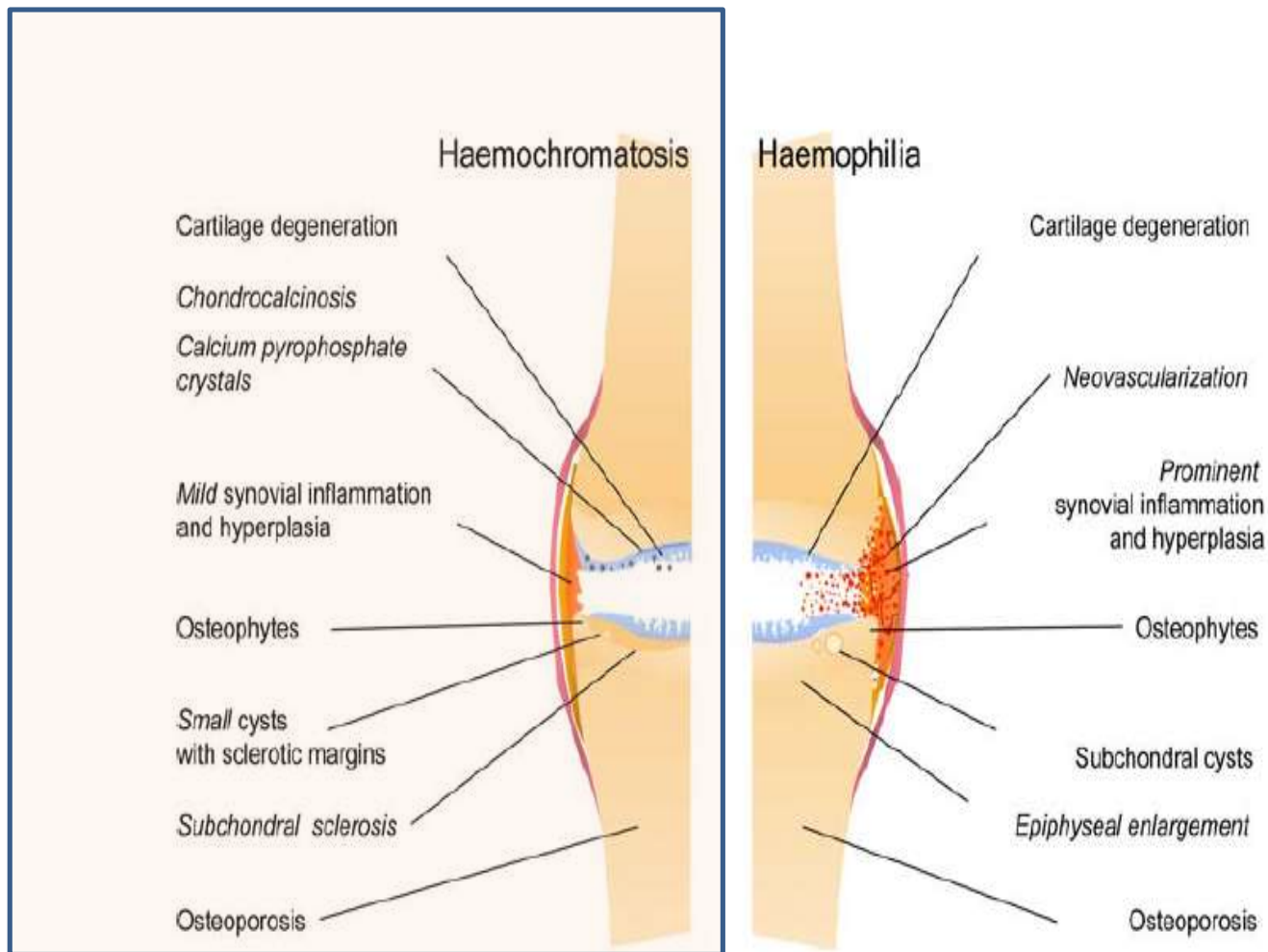
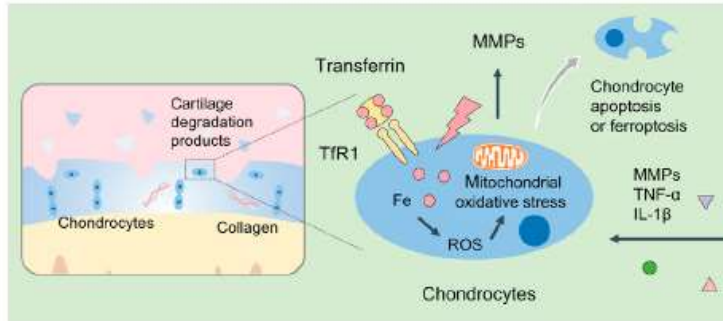
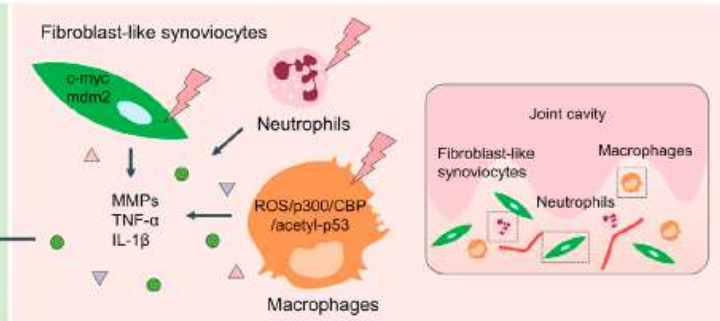


Figure 1 Schematic representation of arthropathy in haemochromatosis versus haemophilia. The structural changes in arthropathy in haemochromatosis are shown on the left, and those in haemophilia on the right. Note the differences indicated in italics.

A Effects of iron overload on chondrocyte dysfunction and cartilage degradation



C Effects of iron overload on manifestations of synovial lesions



B Effects of iron overload on subchondral bone remodeling

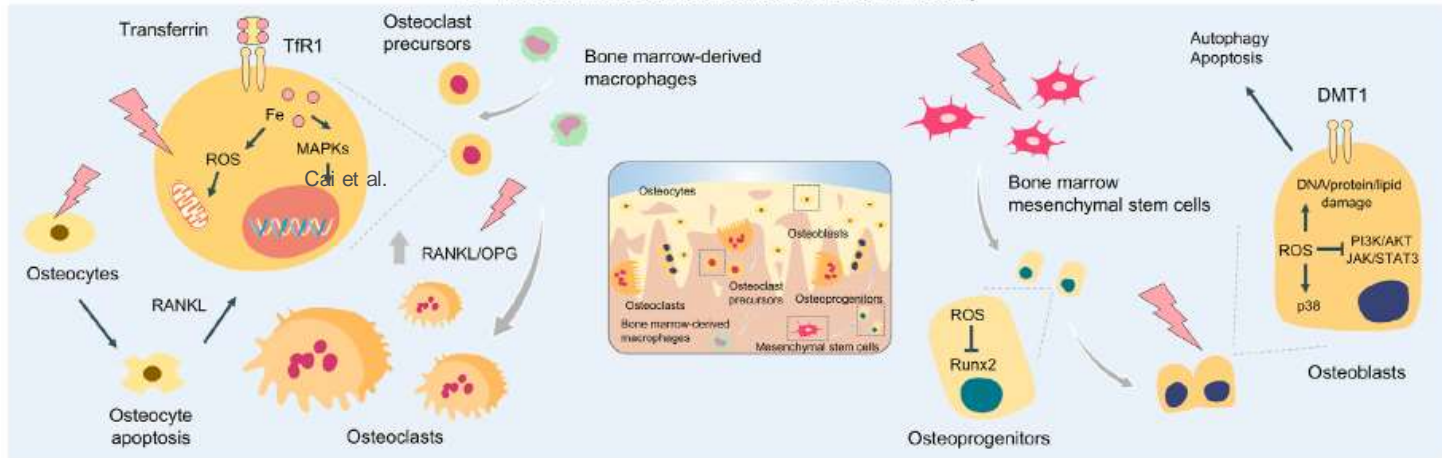


FIGURE 2 | Cellular and molecular mechanisms of iron overload involved in OA. Cellular and molecular mechanisms of iron overload involved in OA include the effects of iron overload on chondrocyte dysfunction and cartilage degradation **(A)** subchondral bone remodeling process which osteoclasts and osteoblasts coordinately regulate **(B)**, and manifestations of synovial lesions mediated by macrophages, fibroblast-like synoviocytes, and neutrophils **(C)**.



Figure 1. Photograph of the bilateral hands demonstrating joint enlargement, most notable in the second and third metacarpophalangeal joints, and proximal interphalangeal joints.

Risk factors for arthritis include:

1. increased age
2. advanced liver fibrosis
3. serum ferritin levels exceeding 1000 μg per liter
4. serum transferrin saturation above 50% for at least 6 years
5. persons with hemochromatosis and arthritis have a significantly higher mean red-cell



Fig. 3. Haemochromatosis arthritis of 2nd and 3rd MCP with hook-shaped osteophyte of the 3rd metacarpal head.

Table 2. Clinical and biochemical features of 93 patients with HH subdivided according to presence or not of radiographic MCP2–3 arthropathy.

	Radiographic arthropathy of MCP2–3		<i>p</i> -value
	Present (<i>n</i> =35)	Absent (<i>n</i> =58)	
Age, years	67.1 (5.3)	55.7 (11.6)	<0.001
Gender, women	9 (25.7)	30 (51.7)	0.014
Body Mass Index, kg/m ²	28.9 (5.8)	26.5 (5.0)	0.032
Disease duration, years	9.0 (7.0)	8.2 (4.9)	0.54
HFE gene status, C282Y/C282Y	22 (62.9)	25 (43.1)	0.065
Ferritin ≥1000 ng/ml at diagnosis ¹	16 (48.5)	12 (23.1)	0.015
Diabetes	7 (20.0)	3 (5.2)	0.037
Hepatic cirrhosis	7 (20.0)	1 (1.7)	0.004

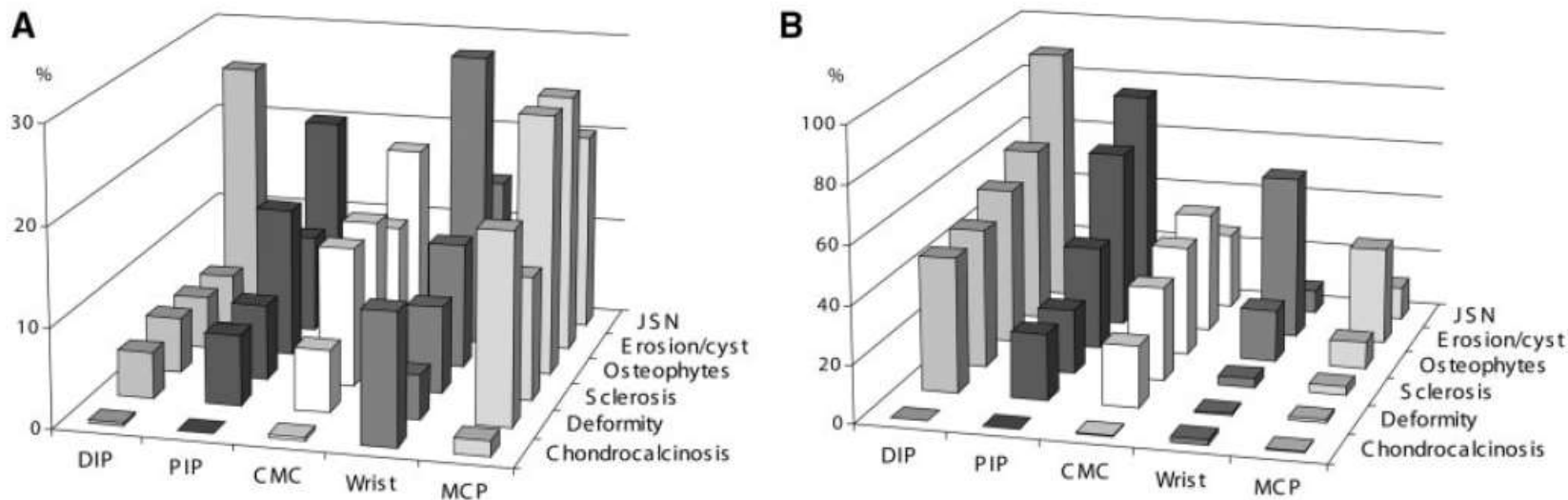
Values are expressed as mean (standard deviation) or numbers (percentage).
¹Eight missing values (two patients with radiographic arthropathy of MCP2–3).

Table 2 Comparison of the common characteristics of primary generalised osteoarthritis and the accelerated osteoarthritis phenotype of haemochromatosis arthritis

	Primary generalised osteoarthritis	Haemochromatosis arthropathy
Gender prevalence	Female > Male (knee, hand)	Male > Female
Average age of onset	>50 years	40–55 years
Preceding joint injury/deformity	Common (hip, knee)	Unusual
Common affected joints	Hip, knee, first CMC, PIP, DIP	Hip, knee, first CMC, PIP, DIP, MCP 2 and 3, ankle
Osteophytes	Present	Exuberant
Subchondral cysts/bone marrow lesions (MRI)	Present	Larger and more numerous
Progression to arthroplasty	Usually slow	Higher prevalence and can be rapid

CMC: carpometacarpal joint; DIP: distal interphalangeal joint; MCP: metacarpophalangeal joint; PIP: proximal interphalangeal joint

Fig. 1 Prevalence of radiographic features in hand joints of patients with **(A)** HH arthropathy and **(B)** primary HOA.



JSN: joint space narrowing.



Fig. 5. Centered glenohumeral osteoarthritis in haemochromatosis.

Ankle arthropathy



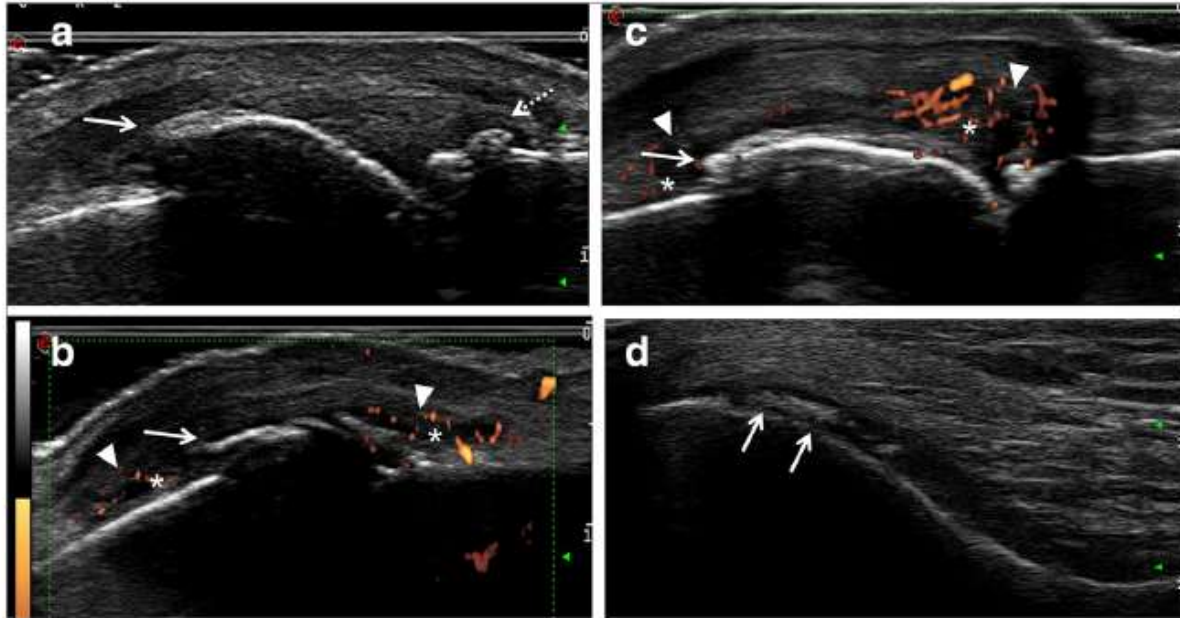


Fig. 1 Ultrasound image examples of patients with hereditary haemochromatosis. **a** Longitudinal dorsal scan of a metacarpophalangeal (MCP) joint (left = proximal) indicating an extensive proximal osteophyte (arrow) and a smaller osteophyte in the distal part of the joint (broken arrow). **b, c** Longitudinal dorsal scan of MCP joints indicating a proximal osteophyte (arrow), grey scale synovitis (arrow heads) and power Doppler signals (asterix). **d** Transverse suprapatellar scan of a knee indicating calcium pyrophosphate deposition in the hyaline cartilage (arrow)

Synovitis and CPPD with ultrasound in thalassaemia / S. Ermurat et al.

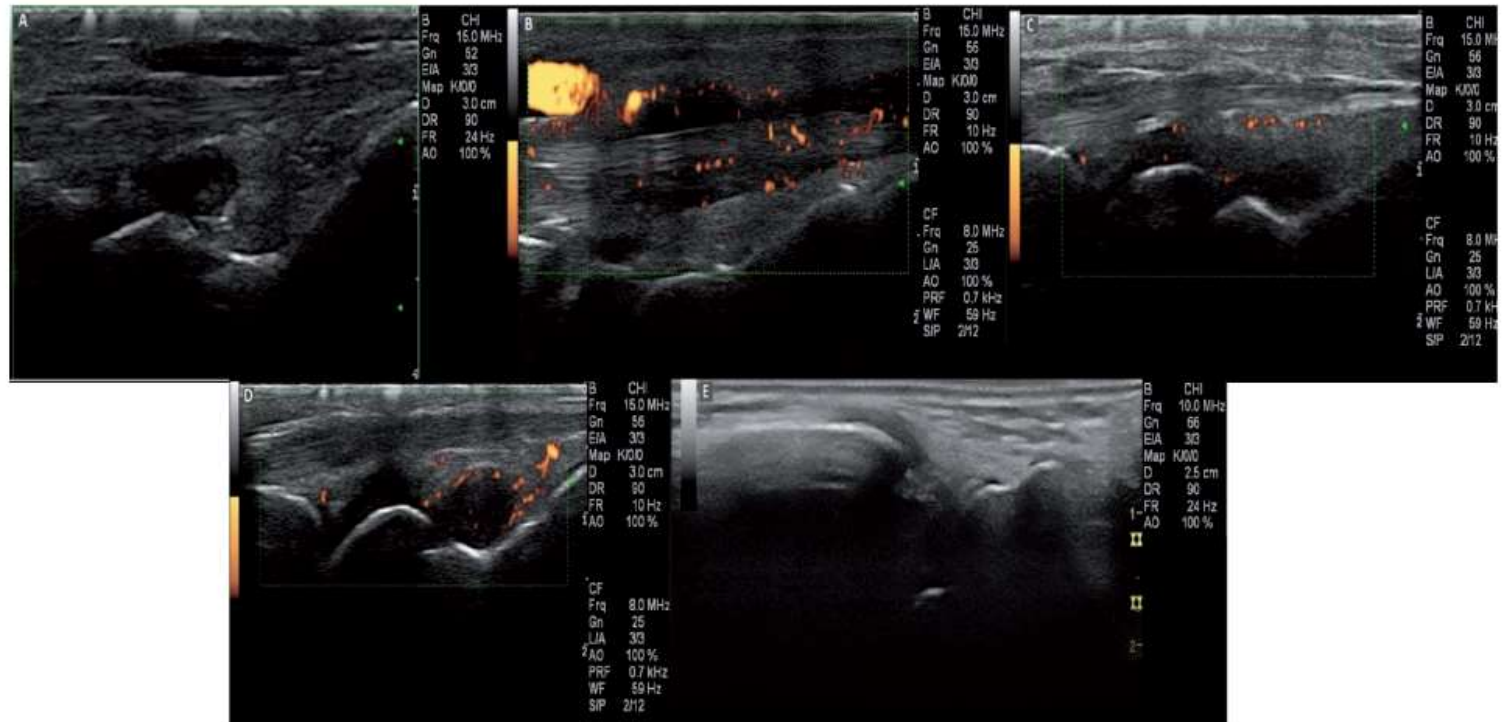


Fig. 1. US images of thalassaemia patients.

A: US image of a patient with synovial hypertrophy; B: US image of a patient with tenosynovitis; C: US image of a patient with Grade 2 synovitis; D: US image of a patient with Grade 3 synovitis; E: US image of a patient with a TCC cartilage calcification.

Treatment of arthropathy

- It has independent course (no improvement with phlebotomy)
- Mainly symptomatic treatment
- Earlier joint prosthesis in comparison with general population
- Difficulties on cDMARDs initiation due to liver involvement
- In oligoarticular involvement steroid injections could be used
- HCQ could be possible treatment as well as colchicine
- No experience with any of biologics but IL-1 inhibitor could be a possible choice in resistant cases

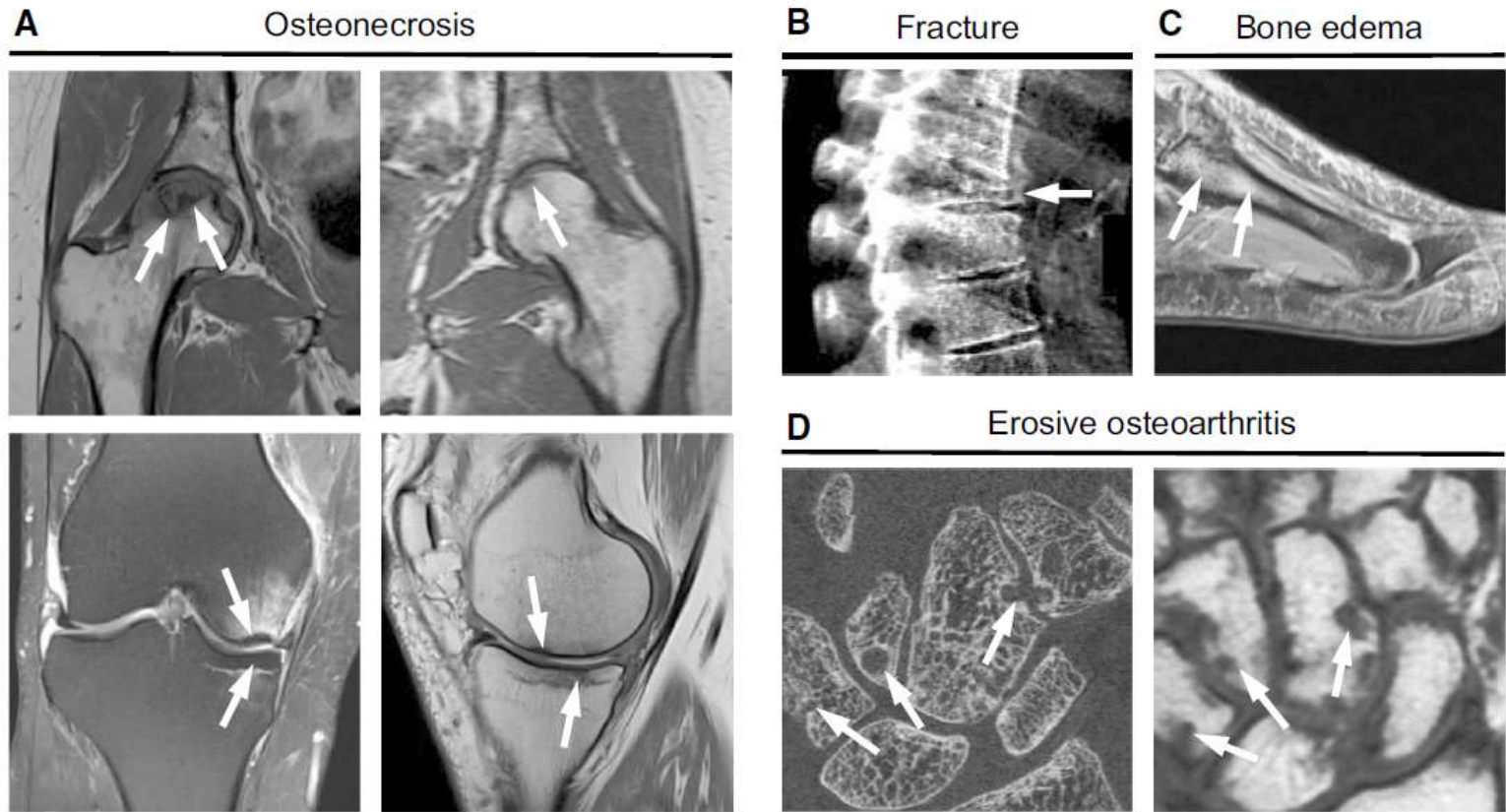


Fig. 1 Representative examples of different bone affections in HHC. **a** Osteonecrosis in MRI T1- or PD-weighted, **b** vertebral fracture in lateral vertebral fracture assessment (VFA) using DXA, **c** bone mar-

row edema in PD-weighted MRI sequence, **d** erosive osteoarthritis in HR-pQCT of the carpal bones (left) and T1-weighted MR images (right)

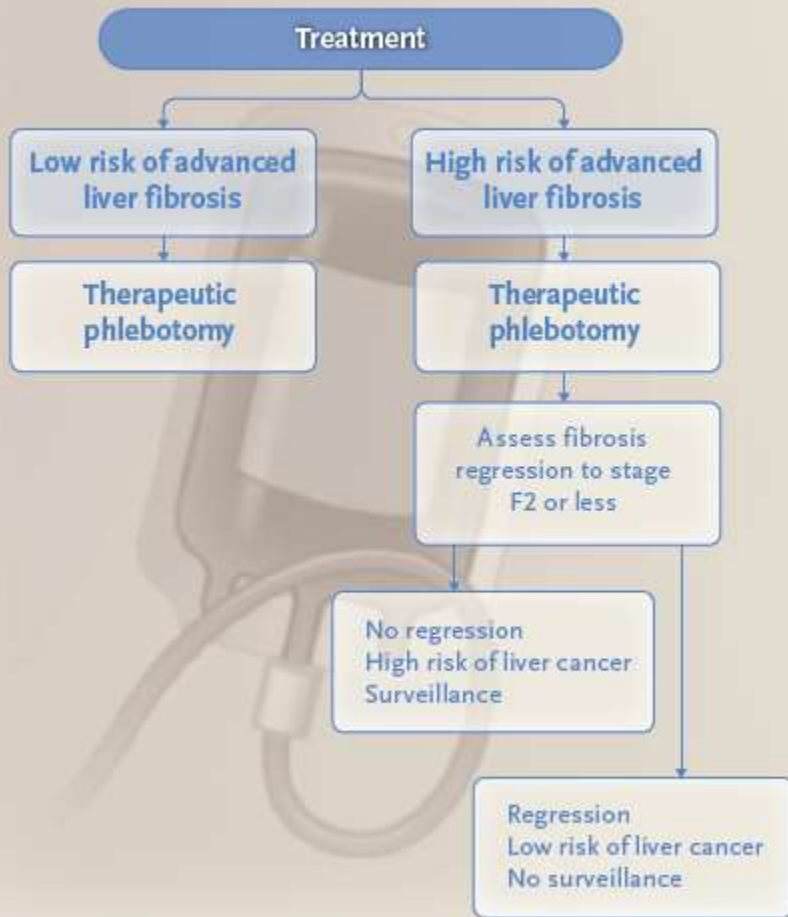
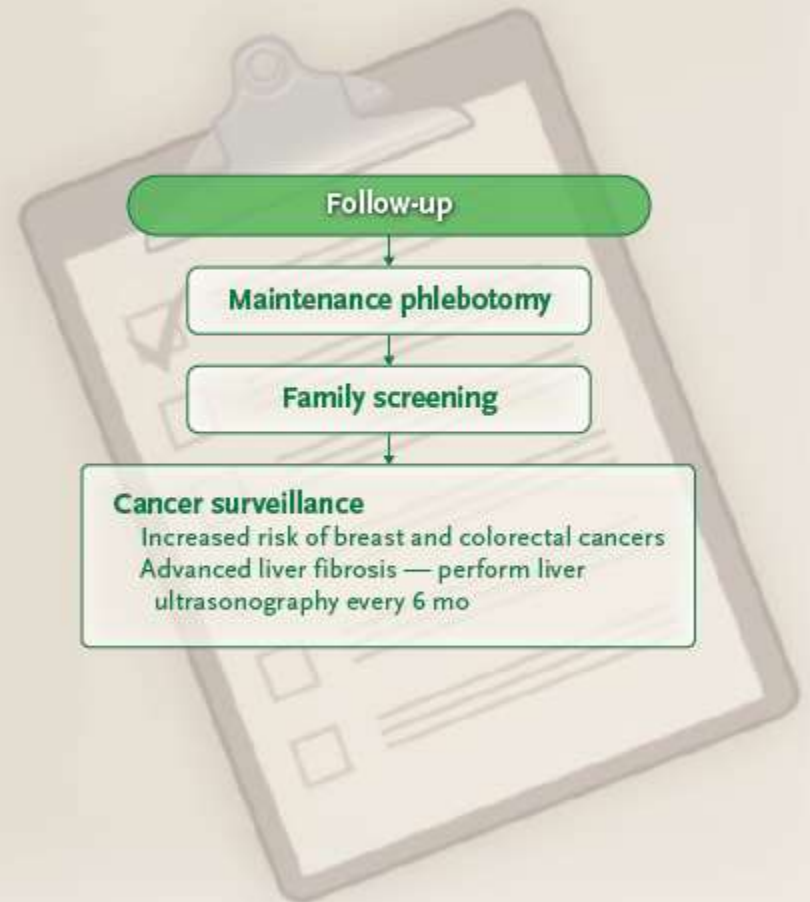
Osteoporosis in haemochromatosis

- The prevalence of osteoporosis ranges from **23.3% to 34.2%** compared to **4.6%** in a sex and age matched healthy comparison population
- **Iron overload accelerate the bone mass loss**
- The prevalence of fractures **is estimated to be 20%**, preferentially affect vertebrae and are especially likely in those who had **a peak ferritin >1000 µg/L**.
- The occurrence of an osteoporotic fracture **at an unexpectedly young age without traditional risk factors should raise the suspicion of HH**.
- The management of osteoporosis **does not differ** from that of the non-haemochromatosis population. We treat according to national guidelines



L. Valenti et al. Osteoporosis Int 2009

Richette P , et al. J Rheumatol 2010

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Practice points

- In the case of unexplained joint pain or arthropathy resembling osteoarthritis, chondrocalcinosis or CPPD, haemochromatosis should be suspected, especially in people younger than in the usual presentation of these diseases or in case of unusual joint location.
- Haemochromatosis arthropathy is associated with a higher rate of joint prosthesis replacement than in the general population and in younger people.
- Osteoporosis is a complication of iron overload and haemochromatosis. It should be assessed particularly in patients with severe overload and other risk factors for osteoporosis or with previous low energy fractures.

