




Is there a place for magnetic resonance imaging in diffuse idiopathic skeletal hyperostosis?

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Abstract

Diffuse idiopathic skeletal hyperostosis (DISH) is a systemic condition characterized by the new bone formation and enthesopathies of the axial and peripheral skeleton. The diagnosis of DISH currently relies upon the end-stage radiographic criteria of Resnick and Niwayama, in which bridging osteophytes are present over at least four thoracic vertebrae. The pathogenesis of DISH is not well understood, and it is currently considered a non-inflammatory condition with an underlying metabolic derangement. However, an inflammatory component was suggested due to the similarities between DISH and spondyloarthritis (SpA) in spinal and peripheral enthesal new bone formation. Magnetic resonance imaging (MRI) is the imaging modality of choice in the diagnostic work-up and follow-up of patients with SpA, as well as in understanding its pathogenesis. The aims of the current review were to evaluate the current and future role of MRI in imaging DISH.

Keywords

Diffuse idiopathic skeletal hyperostosis, magnetic resonance imaging, spine, sacroiliac joints, entheses, osteophytes

Introduction

Diffuse idiopathic skeletal hyperostosis (DISH) is a bone-producing disease characterized by excessive new bone formation in the axial and peripheral skeleton. It usually affects the thoracic spine, while new bone formation involves enthesal sites, mainly those of the pelvis [1–3]. Although initially considered a radiographic rather than a clinical entity, various clinical symptoms have been reported in patients with DISH, including back pain, spinal stiffness [4], and dysphagia [5].

The diagnosis of DISH relies upon the radiographic criteria of Resnick and Niwayama, in which bridging osteophytes along the anterolateral aspect of the spine extend over at least four contiguous vertebral bodies in the absence of degenerative disc disease and inflammatory sacroiliac or facet joint changes [1]. These criteria represent a late, ankylosing, and end stage of DISH and although several alternative criteria have been



suggested over the years, the Resnick and Niwayama criteria continue to be the ones most commonly used in current practice [6].

The pathogenesis of DISH is not well understood, and it is currently considered a non-inflammatory condition with an underlying metabolic derangement that results in new bone formation [7, 8]. However, an inflammatory component has also been suggested due to similarities between DISH and spondyloarthritis (SpA), which represents a group of rheumatic diseases of which ankylosing spondylitis (AS) is the prototype and which shares a tendency towards spinal and peripheral enthesal new bone formation similar to that of DISH [9]. The clinical presentation of DISH may mimic long-standing advanced AS with severe limitation of spinal mobility and postural abnormalities [10]. Moreover, individuals with DISH are similarly susceptible to unstable spinal fractures [11].

Magnetic resonance imaging (MRI) has been widely used in the diagnostic work-up and therapeutic follow-up of patients with SpA. Inflammation and its resultant structural damage can be readily identified in MRI studies of the sacroiliac joints (SIJ) and the spine, making MRI the current imaging modality of choice for the diagnosis and evaluation of SpA [12, 13]. Furthermore, imaging findings on MRI were employed for formulating several etiopathogenetic theories related to SpA [14, 15], as well as for deciphering the nature of the spectrum starting from inflammation and extending to structural progression [16–19].

It is thus reasonable to assume that MRI may also add valuable input to establishing the diagnosis and to conducting of research on DISH. Therefore, the aims of the current review were to evaluate the role of MRI in the imaging of DISH and to investigate the extent and nature of its contribution. The use of anonymized images was approved by the local ethics committee (9916-22-SMC). Patient consent was waived by the Sheba Medical Center's institutional ethics committee due to the retrospective nature of the analysis.

Clinical role of MRI in DISH

Although many subjects with DISH are asymptomatic, back pain and spinal stiffness are frequently reported [20]. Whether spinal ankylosis is of itself the source of pain, however, is open to question. While one study reported that the prevalence of back pain was similar for DISH and non-DISH [21], another study reported a comparatively lower prevalence among patients with DISH, and those authors subsequently suggested that the relatively increased spinal stability in DISH limits rather than induces the extent of pain [22]. Potential causes of pain due to DISH may be osteophytic pressure on neighboring organs, such as the anterior cervical spine, causing both pain and dysphagia in that region (Figure 1) [23].

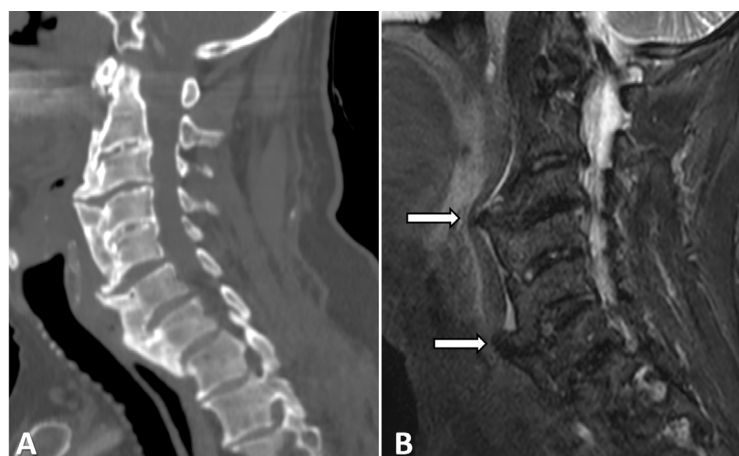


Figure 1. A 76-year-old male with DISH and dysphagia. A coarse and thick anterior osteophyte can be detected on both (A) the sagittal cervical spine computed tomography (CT) and on (B) sort tau inversion recovery (STIR) and is seen to be placing pressure upon the esophagus (arrows)

In the upper cervical spine, the protruding osteophytes and new bone formation in DISH subjects were reported to cause spinal cord compression and myelopathy [24, 25], while a combination of bridging osteophytes with an ossified posterior longitudinal ligament (OPLL) was reported to cause myelopathy of

cervical (Figure 2) as well as thoracic spines [26–28]. Therefore, MRI may succeed in identifying the cause of back pain when other imaging modalities have failed.

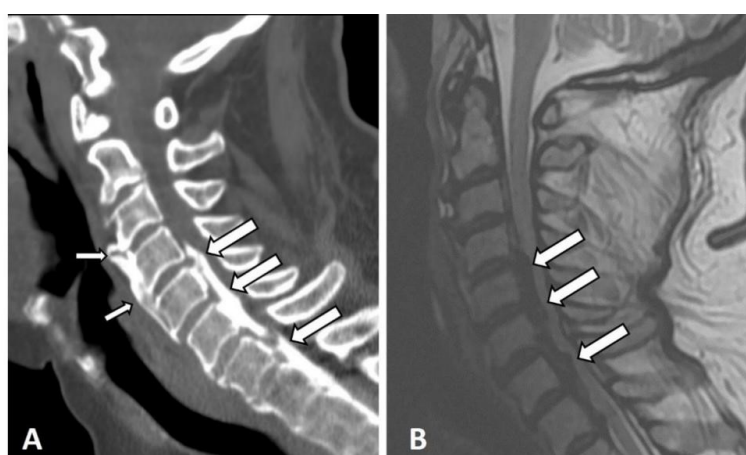


Figure 2. Sagittal cervical spine CT and T2-weighted (T2-W) MRI images of a 67-year-old male with DISH in the thoracic spine. Anterior flowing osteophytes between C3–5 (thin arrows) and an OPLL (thick arrows) causing stenosis of the spinal canal and myelopathy are depicted. Notice the difficulty in detecting the flowing osteophytes on MRI

It is worth noting that it is mostly the structural causes of nerve root compromise that are currently detected by MRI. Moreover, MRI is reportedly not sensitive enough for the detection of nonstructural causes for nerve root compromise and radiculopathy [29].

The rigid ankylotic spine of subjects with DISH is vulnerable to fracture even after seemingly low-energy trauma [30, 31]. There is a four-fold increased risk for fractures in an ankylosed spine compared to a non-ankylosed one, while associated spinal cord injury is seen in up to 58% of subjects with an ankylosed spine [32]. Hyperextension and displacement-type fractures are the ones most frequently observed in DISH, and they characteristically pass through the vertebral body [32, 33]. These fractures are associated with greater instability and several more complications that may be caused by a delay in diagnosis [32]. However, the interpretation of radiographic images can be challenging, and the detection of fracture was reportedly 65% at best with low inter-observer rates [34]. Consequently, both CT and MRI are often recommended for post-traumatic subjects with DISH [11], and MRI is specifically indicated when a neurological deficit is present (Figure 3) [35, 36].

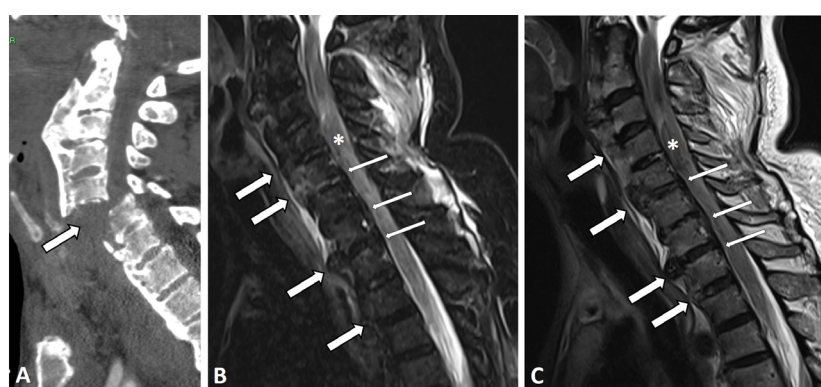


Figure 3. Sagittal CT image of the cervical spine of an 84-year-old male with DISH. (A) Displayed is a devastating extension dislocation-type fracture through C5–6 (thick arrows); (B, C) sagittal STIR and T2-W MRI images of the cervical spine of a 72-year-old male with upper limb parasthesia and weakness. Anterior flowing osteophytes (thick arrows) and OPLL (thin arrows) are depicted in both sequences, as is a thickened non-homogenous cord with blood residue contained within it (asterisk)

MRI as a potential tool for differentiating between DISH and SpA

Both DISH and AS are bone-producing ankylosing diseases, and differentiating between them is usually based upon distinct clinical features, such as age and genetic predisposition as well as characteristic imaging

features, e.g., vertical, symmetrical, and marginal slender intervertebral syndesmophytes in AS compared to chunky, thick, and horizontal osteophytes in DISH [9, 37]. However, the diagnosis of DISH is not always straightforward and a differential diagnosis of AS is sometimes suggested. This is probably due to the fact that both types of bone formation (syndesmophytes and bridging osteophytes) can be detected simultaneously both in DISH (Figure 4) and in AS, albeit with different ratios, with the majority being syndesmophytes in AS and the majority being osteophytes in DISH [37]. Moreover, the simultaneous occurrence of both conditions in the same patient has been reported [38].

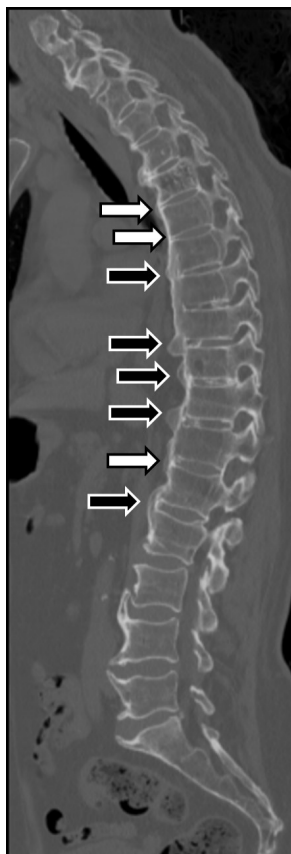


Figure 4. Sagittal thoracic and lumbar spine CT image of a 74-year-old male with DISH. Both thick horizontal osteophytes (black arrows), as well as thin vertical syndesmophytes (white arrows), are present, although in different frequencies

The MRI characteristics of the involved spine and SIJ in SpA have been well documented. They include acute lesions, such as anterior spondylitis inflammatory lesions, and periarticular osteitis in the SIJ, as well as structural lesions, such as erosions, fat metaplasia, sclerosis, and ankylosis in the vertebral corners or the SIJ [39, 40].

The presence of periarticular bone marrow edema (BME), or osteitis, which is highly suggestive of sacroiliitis with or without characteristic structural lesions, is part of the Assessment in SpondyloArthritis International Society classification criteria for axial SpA. It is now known that periarticular SIJ BME is not pathognomonic to sacroiliitis and that its detection alone does not necessarily imply that diagnosis [41]. Indeed, BME has also been described in the MRI examinations of patients with SIJ-related mechanical degeneration [42, 43].

There are two studies that demonstrated the presence of known SpA-related active and structural MRI lesions in the spine and in the SIJ of subjects with DISH [44, 45]. Both studies describe some degree of BME in their SIJ, however, BME highly suggestive of sacroiliitis was rarely seen [44], and when it was present, it was located mainly in the upper parts of the joint [45]. It is also our clinical impression that BME in DISH is seldom seen and that it is not similar in either amount or distribution to that appearing in SpA (Figure 5), thus allowing for the distinction between the two pathological entities based upon a characteristic appearance of SpA [46, 47]. This clinical impression warrants further confirmation.



Figure 5. MRI of the SIJ of a 60-year-old male with DISH. (A) Coronal oblique T2-W and (B) ultra-short echo-time-weight (USTE-W) images of the SIJ. An anterior bridging osteophyte is seen on the left sacroiliac joint (white arrows) as is some very minor BME on the right sacral and left iliacal parts of the joints (black arrows)

In the spine, anterior vertebral BME corners (anterior spondylitis) as well as fat metaplasia in the corners of the vertebrae have been described in subjects with DISH (Figure 6) [44, 45, 48]. Their frequency in DISH, however, was much lower compared to that of SpA [45].

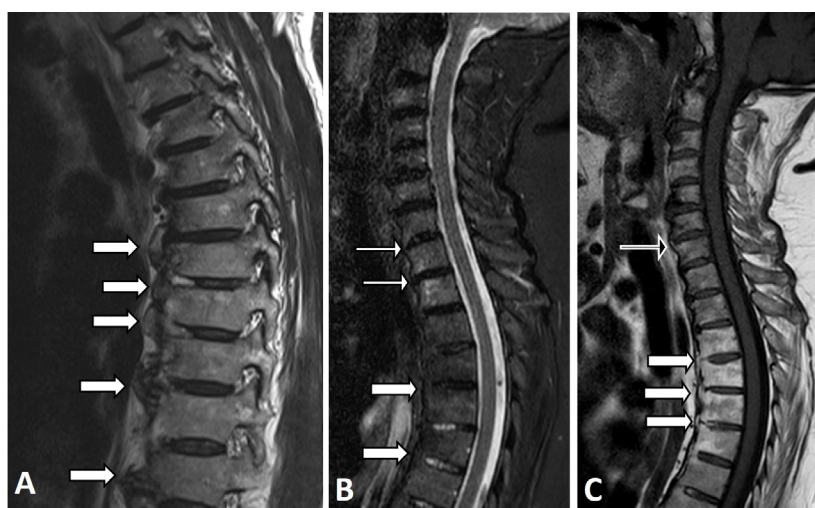


Figure 6. Thoracic spine MRI images of two patients with DISH. (A) Sagittal T1-W MRI image showing the most common presentation of flowing osteophytes with a regular bone marrow signal (thick white arrows); (B) sagittal STIR and (C) T1-W MRI images of another patient in which the fatty normal marrow can be seen in the upper thoracic vertebrae (thick white arrows), while some mild BME can be seen in the endplates of the cervical vertebral corners (thin white arrows in Figure 6B), and a corner containing mild fat metaplasia can be detected at the base of an osteophyte of C7 (black arrow in Figure 6C)

These findings are commonly seen in the elderly population, and they may be attributed to mechanical changes and advanced age and not necessarily to DISH [49, 50]. Either way, the presence of only a few vertebral BME and fat corners on an MRI should neither establish the diagnosis nor differentiate between SpA or DISH, while the presence of many vertebral BME corners (above 3 BME corners and 5 fat corners) would be more strongly suggestive of SpA [40].

Contrast enhancement is a characteristic finding on MRIs of enthesopathy and inflammation. Since both DISH and SpA are enthesopathies, albeit with different pathogenesis, it is possible that contrast injection may help in differentiating between them. However, the role of MRI contrast material injection in differentiating between DISH and SpA has not yet been evaluated.

In a study that compared whole-body MRI findings between subjects with DISH and those with AS, lesions located in the posterior corners of the thoracic spinal vertebrae (either BME or fatty lesions) as well as intervertebral and facet joint ankylosis were rarely found in the ones with DISH [45]. The depiction of those lesions highlights the most promising feature of the future role of MRI in differentiating between the two entities.

Erosions of the SIJ are quite specific to SpA and were not characteristically detected on CT in subjects with DISH [51]. Thus, the presence of erosions on MRI may help in ruling out a diagnosis of DISH. Indeed,

CT is currently the gold standard imaging modality for the detection of erosions, although new erosion-sensitive sequences on MRI show comparable ability for its detection [52, 53].

Bridging osteophytes appear in subjects with DISH not only along the spine but anterior to the SIJ as well [51, 54, 55]. Nevertheless, the identification of osteophytes or syndesmophytes on MRI is difficult and easy to miss, both in the spine and in the SIJ (Figure 7) [56]. Therefore, CT rather than MRI would be the imaging modality of choice for their detection.

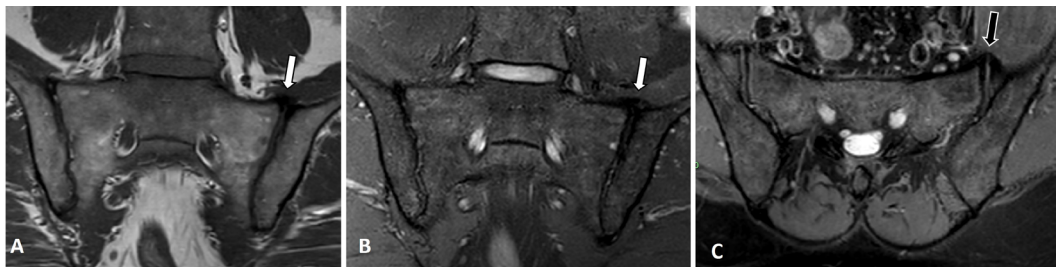


Figure 7. MRI images of the sacroiliac joints of a patient with DISH. (A) Coronal oblique T1-W and (B) T2-W with fat suppression and (C) an axial oblique T2-W with fat suppression MRI sequences. An anterior bridging osteophyte can be seen although not easily detected in the left SIJ on the semi coronal plane (white arrows), while it is somewhat easier to detect on the axial oblique plane (black arrow)

MRI characteristics differentiating between DISH, SpA, and degenerative spinal disease are summarized (Table 1).

Table 1. MRI characteristics of the SIJ and the spine in patients with DISH vs. SpA and degenerative disease

MRI lesions		DISH	SpA	Degenerative changes
SIJ	Periarticular BME	Rare	Common	Rare
	Suggestive of SpA	Upper part of SIJ	Along entire SIJ	Upper part of SIJ
	Erosions	Rare	Common	Rare
	Bone production	Bridging osteophytes anterior to SIJ	Intra-articular bridging osteophytes	Small periarticular osteophytes
	Periarticular fat metaplasia	Rare	Common	Rare
Spine	Bone production	Thick, bridging horizontal osteophytes	Thin, slender, vertical syndesmophytes	Horizontal osteophytes
	BME corners	Present but uncommon	Common	Present but uncommon
	Fat corners	Present, not rare	Common	Present, not rare

Future role of MRI in deciphering etiopathogenesis of DISH

DISH mainly affects the elderly (men more than women) and its prevalence increases with age [20, 57]. Thus, differentiating between DISH and degenerative disc disease as a cause of clinical presentation is a common diagnostic challenge.

The classification criteria of Resnick and Niwayama aim at differentiating between DISH and degenerative and inflammatory diseases of the spine [1]. The first criterion of four or more flowing osteophytes differentiates DISH from spondylitis deformans in which spinal osteophytes are increasingly more prevalent with age due to degenerative disc disease [58]. The second criterion relates to disc height preservation and the absence of degenerative disc-related changes, and it aims to differentiate DISH from vertebral osteochondrosis thought to result from age-related disc dehydration [58]. However, the Resnick and Niwayama criteria establish the diagnosis of DISH in its end stage when intervention is of little use [59]. The rate of new bone formation in DISH and AS was reported to be quite similar, with approximately 10 years of progression for a single bridging osteophyte to develop [37, 60]. That means that using the current Resnick and Niwayama classification criteria will delay the identification of DISH by at least 10 years. Other classification criteria were suggested for the purpose of earlier diagnosis of DISH [61–63], however, there is currently no consensus on which diagnostic criteria should be applied [61]. It was suggested that MRI

may be of assistance in identifying earlier signs of DISH and, indeed, it is our impression that in some patients with DISH, as in those with extra-spinal enthesitis [64], inflammation can already be detected at the base of an osteophyte or in its soft tissue surrounding. However, there are currently no published reports on early MRI signs of DISH. Prospective studies targeting a population at risk of developing DISH, such as patients with the metabolic disease [7], are warranted and may help in characterizing early, preliminary stages of DISH as well as in deciphering its etiopathogenesis.

Conclusions

MRI is currently the recommended imaging modality for the clinical evaluation of symptomatic painful or post-traumatic patients with DISH. MRI may assist in differentiating between DISH and AS when other imaging modalities fail. Establishing a correlation between imaging characteristics of DISH with its clinical manifestations may facilitate early diagnosis of DISH as well as promote the understanding of the pathogenesis of DISH.

Abbreviations

AS: ankylosing spondylitis

BME: bone marrow edema

CT: computed tomography

DISH: diffuse idiopathic skeletal hyperostosis

MRI: magnetic resonance imaging

OPLL: ossified posterior longitudinal ligament

SIJ: sacroiliac joints

SpA: spondyloarthritis

Declarations

Author contributions

IE: Conceptualization, Investigation, Writing—original draft, Writing—review & editing.

Conflicts of interest

The author declares that she has no conflicts of interest.

Ethical approval

The use of anonymized images was approved by the local ethics committee (9916-22-SMC) and this study complied with the Declaration of Helsinki.

Consent to participate

Patient's consent was waived by the Sheba Medical Center's institutional ethics committee due to the retrospective nature of the manuscript.

Consent to publication

Not applicable.

Availability of data and materials

Not applicable.

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References

1. Resnick D, Niwayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). *Radiology*. 1976;119:559–68.
2. Resnick D, Niwayama G. Entheses and enthesopathy. Anatomical, pathological, and radiological correlation. *Radiology*. 1983;146:1–9.
3. Slonimsky E, Leibushor N, Aharoni D, Lidar M, Eshed I. Pelvic enthesopathy on CT is significantly more prevalent in patients with diffuse idiopathic skeletal hyperostosis (DISH) compared with matched control patients. *Clin Rheumatol*. 2016;35:1823–7.
4. Mader R, Verlaan JJ, Buskila D. Diffuse idiopathic skeletal hyperostosis: clinical features and pathogenic mechanisms. *Nat Rev Rheumatol*. 2013;9:741–50.
5. Harlianto NI, Kuperus JS, Mohamed Hoesein FAA, de Jong PA, de Ru JA, Oner FC, et al. Diffuse idiopathic skeletal hyperostosis of the cervical spine causing dysphagia and airway obstruction: an updated systematic review. *Spine J*. 2022;22:1490–503.
6. Kuperus JS, Mohamed Hoesein FAA, de Jong PA, Verlaan JJ. Diffuse idiopathic skeletal hyperostosis: etiology and clinical relevance. *Best Pract Res Clin Rheumatol*. 2020;34:101527.
7. Mader R, Novofestovski I, Adawi M, Lavi I. Metabolic syndrome and cardiovascular risk in patients with diffuse idiopathic skeletal hyperostosis. *Semin Arthritis Rheum*. 2009;38:361–5.
8. Pariente-Rodrigo E, Sgaramella GA, Olmos-Martinez JM, Pini-Valdivieso SF, Landeras-Alvaro R, Hernandez-Hernandez JL. Relationship between diffuse idiopathic skeletal hyperostosis, abdominal aortic calcification and associated metabolic disorders: data from the Camargo Cohort. *Med Clin (Barc)*. 2017;149:196–202.
9. Mader R, Pappone N, Baraliakos X, Eshed I, Sarzi-Puttini P, Atzeni F, et al. Diffuse idiopathic skeletal hyperostosis (DISH) and a possible inflammatory component. *Curr Rheumatol Rep*. 2021;23:6.
10. Olivieri I, D'Angelo S, Cutro MS, Padula A, Peruz G, Montaruli M, et al. Diffuse idiopathic skeletal hyperostosis may give the typical postural abnormalities of advanced ankylosing spondylitis. *Rheumatology (Oxford)*. 2007;46:1709–11.
11. Dan Lantsman C, Barkay G, Friedlander A, Barbi M, Stern M, Eshed I. Whole spine CT scan for the detection of acute spinal fractures in diffuse idiopathic skeletal hyperostosis patients who sustained low-energy trauma. *Spine (Phila Pa 1976)*. 2020;45:1348–53.
12. Lambert RG, Bakker PA, van der Heijde D, Weber U, Rudwaleit M, Hermann KG, et al. Defining active sacroiliitis on MRI for classification of axial spondyloarthritis: update by the ASAS MRI working group. *Ann Rheum Dis*. 2016;75:1958–63.
13. Rudwaleit M, van der Heijde D, Landewe R, Akkoc N, Brandt J, Chou CT, et al. The assessment of spondyloarthritis international society classification criteria for peripheral spondyloarthritis and for spondyloarthritis in general. *Ann Rheum Dis*. 2011;70:25–31.
14. McGonagle D, Marzo-Ortega H, O'Connor P, Gibbon W, Pease C, Reece R, et al. The role of biomechanical factors and HLA-B27 in magnetic resonance imaging-determined bone changes in plantar fascia enthesopathy. *Arthritis Rheum*. 2002;46:489–93.
15. Benjamin M, McGonagle D. Histopathologic changes at “synovio-entheseal complexes” suggesting a novel mechanism for synovitis in osteoarthritis and spondylarthritis. *Arthritis Rheum*. 2007;56:3601–9.
16. Aouad K, Ziade N, Baraliakos X. Structural progression in axial spondyloarthritis. *Joint Bone Spine*. 2020;87:131–6.

17. van der Heijde D, Ostergaard M, Reveille JD, Baraliakos X, Kronbergs A, Sandoval DM, et al. Spinal radiographic progression and predictors of progression in patients with radiographic axial spondyloarthritis receiving ixekizumab over 2 years. *J Rheumatol*. 2022;49:265–73.
18. Baraliakos X, Heldmann F, Callhoff J, Listing J, Appelboom T, Brandt J, et al. Which spinal lesions are associated with new bone formation in patients with ankylosing spondylitis treated with anti-TNF agents? A long-term observational study using MRI and conventional radiography. *Ann Rheum Dis*. 2014;73:1819–25.
19. Maksymowych WP, Chiowchanwisawakit P, Clare T, Pedersen SJ, Ostergaard M, Lambert RG. Inflammatory lesions of the spine on magnetic resonance imaging predict the development of new syndesmophytes in ankylosing spondylitis: evidence of a relationship between inflammation and new bone formation. *Arthritis Rheum*. 2009;60:93–102.
20. Utsinger PD. Diffuse idiopathic skeletal hyperostosis. *Clin Rheum Dis*. 1985;11:325–51.
21. Schlapbach P, Beyeler C, Gerber NJ, van der Linden S, Burgi U, Fuchs WA, et al. Diffuse idiopathic skeletal hyperostosis (DISH) of the spine: a cause of back pain? A controlled study. *Br J Rheumatol*. 1989;28:299–303.
22. Holton KF, Denard PJ, Yoo JU, Kado DM, Barrett-Connor E, Marshall LM. Diffuse idiopathic skeletal hyperostosis and its relation to back pain among older men: the MrOS Study. *Semin Arthritis Rheum*. 2011;41:131–8.
23. Kaur J, Virk JS. Dysphagia due to DISH-related anterior osteophytes: DISHphagia!! *BMJ Case Rep*. 2017;2017:bcr-2017-222512.
24. Storch MJ, Hubbe U, Glocker FX. Cervical myelopathy caused by soft-tissue mass in diffuse idiopathic skeletal hyperostosis. *Eur Spine J*. 2008;17 Suppl 2:S243–7.
25. Pascal-Moussellard H, Drossard G, Cursolles JC, Catonne Y, Smadja D. Myelopathy by lesions of the craniocervical junction in a patient with forestier disease. *Spine (Phila Pa 1976)*. 2006;31:E557–60.
26. Griffiths ID, Fitzjohn TP. Cervical myelopathy, ossification of the posterior longitudinal ligament, and diffuse idiopathic skeletal hyperostosis: problems in investigation. *Ann Rheum Dis*. 1987;46:166–8.
27. Reisner A, Stiles RG, Tindall SC. Diffuse idiopathic skeletal hyperostosis causing acute thoracic myelopathy: a case report and discussion. *Neurosurgery*. 1990;26:507–11.
28. Guo Q, Ni B, Yang J, Zhu Z, Yang J. Simultaneous ossification of the posterior longitudinal ligament and ossification of the ligamentum flavum causing upper thoracic myelopathy in DISH: case report and literature review. *Eur Spine J*. 2011;20 Suppl 2:S195–201.
29. Tawa N, Rhoda A, Diener I. Accuracy of magnetic resonance imaging in detecting lumbo-sacral nerve root compromise: a systematic literature review. *BMC Musculoskelet Disord*. 2016;17:386.
30. Rustagi T, Drazin D, Oner C, York J, Schroeder GD, Vaccaro AR, et al. Fractures in spinal ankylosing disorders: a narrative review of disease and injury types, treatment techniques, and outcomes. *J Orthop Trauma*. 2017;31 Suppl 4:S57–74.
31. Caron T, Bransford R, Nguyen Q, Agel J, Chapman J, Bellabarba C. Spine fractures in patients with ankylosing spinal disorders. *Spine (Phila Pa 1976)*. 2010;35:E458–64.
32. Westerveld LA, van Bommel JC, Dhert WJ, Oner FC, Verlaan JJ. Clinical outcome after traumatic spinal fractures in patients with ankylosing spinal disorders compared with control patients. *Spine J*. 2014;14:729–40.
33. Balling H, Weckbach A. Hyperextension injuries of the thoracolumbar spine in diffuse idiopathic skeletal hyperostosis. *Spine (Phila Pa 1976)*. 2015;40:E61–7.
34. Barkay G, Dan Lantsman C, Menachem S, Shtewee A, Ackshota N, Caspi I, et al. Limitations of plain film radiography in identification of hyperextension fractures in patients with ankylosing spinal disorders. *Global Spine J*. 2022;12:24–8.

35. Campagna R, Pessis E, Feydy A, Guerini H, Thevenin F, Chevrot A, et al. Fractures of the ankylosed spine: MDCT and MRI with emphasis on individual anatomic spinal structures. *AJR Am J Roentgenol*. 2009;192:987–95.
36. Reinhold M, Knop C, Kneitz C, Disch A. Spine fractures in ankylosing diseases: recommendations of the spine section of the German society for orthopaedics and trauma (DGOU). *Global Spine J*. 2018;8:56S–68S.
37. Baraliakos X, Listing J, Buschmann J, von der Recke A, Braun J. A comparison of new bone formation in patients with ankylosing spondylitis and patients with diffuse idiopathic skeletal hyperostosis: a retrospective cohort study over six years. *Arthritis Rheum*. 2012;64:1127–33.
38. Kuperus JS, Waalwijk JF, Regan EA, van der Horst-Bruinsma IE, Oner FC, de Jong PA, et al. Simultaneous occurrence of ankylosing spondylitis and diffuse idiopathic skeletal hyperostosis: a systematic review. *Rheumatology (Oxford)*. 2018;57:2120–8.
39. Maksymowych WP, Lambert RG, Ostergaard M, Pedersen SJ, Machado PM, Weber U, et al. MRI lesions in the sacroiliac joints of patients with spondyloarthritis: an update of definitions and validation by the ASAS MRI working group. *Ann Rheum Dis*. 2019;78:1550–8.
40. Hermann KG, Baraliakos X, van der Heijde DM, Jurik AG, Landewe R, Marzo-Ortega H, et al. Descriptions of spinal MRI lesions and definition of a positive MRI of the spine in axial spondyloarthritis: a consensual approach by the ASAS/OMERACT MRI study group. *Ann Rheum Dis*. 2012;71:1278–88.
41. Campos-Correia D, Sudol-Szopinska I, Diana Afonso P. Are we overcalling sacroiliitis on MRI? Differential diagnosis that every rheumatologist should know - part I. *Acta Reumatol Port*. 2019;44:29–41.
42. Arnbak B, Jensen TS, Manniche C, Zejden A, Egund N, Jurik AG. Spondyloarthritis-related and degenerative MRI changes in the axial skeleton-an inter- and intra-observer agreement study. *BMC Musculoskelet Disord*. 2013;14:274.
43. Arnbak B, Jensen RK, Manniche C, Hendricks O, Kent P, Jurik AG, et al. Identification of subgroups of inflammatory and degenerative MRI findings in the spine and sacroiliac joints: a latent class analysis of 1037 patients with persistent low back pain. *Arthritis Res Ther*. 2016;18:237.
44. Latourte A, Charlon S, Etcheto A, Feydy A, Allanore Y, Dougados M, et al. Imaging findings suggestive of axial spondyloarthritis in diffuse idiopathic skeletal hyperostosis. *Arthritis Care Res (Hoboken)*. 2018;70:145–52.
45. Weiss BG, Bachmann LM, Pfirrmann CW, Kissling RO, Zubler V. Whole body magnetic resonance imaging features in diffuse idiopathic skeletal hyperostosis in conjunction with clinical variables to whole body MRI and clinical variables in ankylosing spondylitis. *J Rheumatol*. 2016;43:335–42.
46. Slobodin G, Lidar M, Eshed I. Clinical and imaging mimickers of axial spondyloarthritis. *Semin Arthritis Rheum*. 2017;47:361–8.
47. Eshed I, Lidar M. MRI findings of the sacroiliac joints in patients with low back pain: alternative diagnosis to inflammatory sacroiliitis. *Isr Med Assoc J*. 2017;19:666–9.
48. Arad U, Elkayam O, Eshed I. Magnetic resonance imaging in diffuse idiopathic skeletal hyperostosis: similarities to axial spondyloarthritis. *Clin Rheumatol*. 2017;36:1545–9.
49. Baraliakos X, Richter A, Feldmann D, Ott A, Buelow R, Schmidt CO, et al. Frequency of MRI changes suggestive of axial spondyloarthritis in the axial skeleton in a large population-based cohort of individuals aged < 45 years. *Ann Rheum Dis*. 2020;79:186–92.
50. Weber U, Zubler V, Zhao Z, Lambert RG, Chan SM, Pedersen SJ, et al. Does spinal MRI add incremental diagnostic value to MRI of the sacroiliac joints alone in patients with non-radiographic axial spondyloarthritis? *Ann Rheum Dis*. 2015;74:985–92.
51. Leibushor N, Slonimsky E, Aharoni D, Lidar M, Eshed I. CT abnormalities in the sacroiliac joints of patients with diffuse idiopathic skeletal hyperostosis. *AJR Am J Roentgenol*. 2017;208:834–7.

52. Ye L, Liu Y, Xiao Q, Dong L, Wen C, Zhang Z, et al. MRI compared with low-dose CT scanning in the diagnosis of axial spondyloarthritis. *Clin Rheumatol*. 2020;39:1295–303.
53. Deppe D, Hermann KG, Proft F, Poddubnyy D, Radny F, Protopopov M, et al. CT-like images of the sacroiliac joint generated from MRI using susceptibility-weighted imaging (SWI) in patients with axial spondyloarthritis. *RMD Open*. 2021;7:e001656.
54. Jans L, Van Praet L, Elewaut D, Van den Bosch F, Carron P, Jaremko JL, et al. MRI of the SI joints commonly shows non-inflammatory disease in patients clinically suspected of sacroiliitis. *Eur J Radiol*. 2014;83:179–84.
55. Mader R, Baraliakos X, Eshed I, Novofastovski I, Bieber A, Verlaan JJ, et al. Imaging of diffuse idiopathic skeletal hyperostosis (DISH). *RMD Open*. 2020;6:e001151.
56. Braun J, Baraliakos X, Golder W, Hermann KG, Listing J, Brandt J, et al. Analysing chronic spinal changes in ankylosing spondylitis: a systematic comparison of conventional x rays with magnetic resonance imaging using established and new scoring systems. *Ann Rheum Dis*. 2004;63:1046–55.
57. Weinfeld RM, Olson PN, Maki DD, Griffiths HJ. The prevalence of diffuse idiopathic skeletal hyperostosis (DISH) in two large American Midwest metropolitan hospital populations. *Skeletal Radiol*. 1997;26:222–5.
58. Resnick D. Degenerative diseases of the vertebral column. *Radiology*. 1985;156:3–14.
59. Mader R, Verlaan JJ, Eshed I, Bruges-Armas J, Puttini PS, Atzeni F, et al. Diffuse idiopathic skeletal hyperostosis (DISH): where we are now and where to go next. *RMD Open*. 2017;3:e000472. Erratum in: *RMD Open*. 2017;3:e000472corr1.
60. Yaniv G, Bader S, Lidar M, Herman A, Shazar N, Aharoni D, et al. The natural course of bridging osteophyte formation in diffuse idiopathic skeletal hyperostosis: retrospective analysis of consecutive CT examinations over 10 years. *Rheumatology (Oxford)*. 2014;53:1951–7.
61. Kuperus JS, de Gendt EEA, Oner FC, de Jong PA, Buckens S, van der Merwe AE, et al. Classification criteria for diffuse idiopathic skeletal hyperostosis: a lack of consensus. *Rheumatology (Oxford)*. 2017;56:1123–34.
62. Utsinger PD, Resnick D, Shapiro R. Diffuse skeletal abnormalities in forestier disease. *Arch Intern Med*. 1976;136:763–8.
63. Kuperus JS, Oudkerk SF, Foppen W, Mohamed Hoesein FA, Gielis WP, Waalwijk J, et al. Criteria for early-phase diffuse idiopathic skeletal hyperostosis: development and validation. *Radiology*. 2019;291:420–6.
64. Eshed I, Bollow M, McGonagle DG, Tan AL, Althoff CE, Asbach P, et al. MRI of enthesitis of the appendicular skeleton in spondyloarthritis. *Ann Rheum Dis*. 2007;66:1553–9.