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CASE REPORTS

Diffuse Idiopathic Skeletal Hyperostosis Presenting as Cervical Radiculopathy in Metabolic Syndrome: A Case Report

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Background

DISH is common. It affects about 12% of the general population and a quarter of men over 50. The pathophysiology is metabolic. Circulating insulin phosphorylates IRS-1 in osteoblasts at entheseal insertion points, which activates PI3K/Akt and MAPK cascades and ultimately drives ectopic bone formation along spinal ligaments. Once enough of the spine has fused, the whole column behaves as a long bone. It can no longer dissipate force across mobile segments. Harlianto et al. reported vertebral fractures in 22.6% of DISH patients compared with 15.2% in ankylosing spondylitis, and Caron et al. reported 51% mortality with conservative fracture management versus 23% with surgery. Despite these numbers, primary care physicians rarely consider the diagnosis.

Case

We treated a man in his fifties who woke from sleeping in a chair with severe posterior cervical pain in a C2-C3 distribution, radiating to the left ear and scalp. His history was notable for uncontrolled type 2 diabetes (hemoglobin A1c 9.2%), class I obesity, coronary artery disease, and paroxysmal atrial fibrillation. On examination he had posterior cervical rigidity and suboccipital tenderness. Neurologic examination was normal. Lateral cervical films showed flowing anterior osteophytes from C3 through C7 with preserved disc spaces, facet hypertrophy at C4-C5 and C5-C6, and no fracture. We prescribed gabapentin and methocarbamol, sent him to physical therapy with orders to avoid manipulation, and spent time on fracture risk education. His pain went from 10/10 to 3/10 over two weeks.

Conclusions

DISH can look like cervical radiculopathy, occipital neuralgia, or simple mechanical neck pain. The same hyperinsulinemia that produces hepatic steatosis and accelerates atherosclerosis also drives pathologic bone formation at spinal entheses. A lateral cervical radiograph is all that is needed for diagnosis. Flowing anterior osteophytes with preserved disc spaces look nothing like the syndesmophytes of ankylosing spondylitis or the irregular osteophytes of degenerative spondylosis. What matters most clinically is the fracture risk. These patients have a rigid spine that cannot absorb force from even a minor

fall. In rural practice, where a neurosurgeon may be hours away, identifying DISH and teaching the patient when to go to an emergency department may be the most important thing we do for them.

BRIEF BACKGROUND

Diffuse idiopathic skeletal hyperostosis (DISH) is a systemic metabolic disorder characterized by flowing ossification of spinal ligaments and entheses. Flowing refers to the uninterrupted candle wax contour of bone that spans adjacent vertebral bodies along the anterior longitudinal ligament, distinct from the discrete osteophytes of spondylosis. First described by Forestier in 1950,[1] the condition affects 11.9% of the general population,[2] and prevalence reaches 25% in males over age 50 years.[3] DISH demonstrates strong association with metabolic syndrome components: type 2 diabetes mellitus, obesity, dyslipidemia, and hypertension.[4]

The pathophysiology centers on hyperinsulinemia. Insulin and insulin-like growth factor-1 activate insulin receptor substrate pathways in osteoblasts and drive mesenchymal progenitor differentiation at entheseal sites.[4] Bone morphogenetic protein-2 and transforming growth factor-beta amplify this process through endochondral ossification. New bone forms through a cartilage intermediate, not through direct calcium deposition.[4]

Anterior osteophytic overgrowth compresses adjacent structures and produces dysphagia or nerve root irritation. Spinal ankylosis converts the vertebral column into a rigid lever arm that concentrates stress at sites of potential fracture. Vertebral fractures occur in 22.6% of DISH patients.[5] Most primary care physicians do not recognize the condition.[4]

CASE REPORT

A man in his 50s presented to a rural clinic with severe posterior cervical pain radiating to the left ear and scalp in a C2-C3 distribution. Pain began abruptly after sleeping in a chair. He rated intensity as 10 of 10. Symptoms worsened with neck flexion and Valsalva maneuvers. He denied focal neurologic deficits.

Medical history included type 2 diabetes mellitus (hemoglobin A1c 9.2% [77 mmol/mol]), class I obesity (body mass index 32.4 kg/m^2), hyperlipidemia, coronary artery disease with prior stenting, hypertension, and paroxysmal atrial fibrillation on rivaroxaban (Xarelto). Current medications included tirzepatide (Mounjaro) 15 mg weekly, metformin 1000 mg twice daily, empagliflozin (Jardiance) 25 mg daily, and rosuvastatin. He reported chronic shoulder problems including impingement and rotator cuff tear.

Physical examination revealed blood pressure 138/82 mmHg, heart rate 78 beats per minute. Cervical examination showed preserved range of motion but marked posterior rigidity and suboccipital tenderness. Spurling maneuver was negative. Neurologic examination was intact: cranial nerves II-XII normal, motor strength 5/5, sensation intact, reflexes 2+ and symmetric.

Differential diagnosis included occipital neuralgia from C2-C3 nerve root compression, cervical strain, facet arthropathy, and occult fracture. We obtained lateral cervical radiographs at the initial visit.

Lateral cervical radiographs demonstrated flowing anterior osteophytes spanning C3-C7 with preserved disc spaces. The radiologist diagnosed DISH based on these findings, which meet the numerical Resnick criteria (four or more contiguous vertebrae), though the criteria were developed for thoracic spine imaging.[6] Moderate facet hypertrophy at C4-C5 and C5-C6 was noted. No fracture or instability was present.

Management included gabapentin 300 mg three times daily and methocarbamol 500 mg four times daily for nerve pain and muscle spasm. Physical therapy focused on gentle range-of-motion exercises, with explicit avoidance of manipulation. We counseled the patient on fracture risk and reviewed fall prevention strategies.

At two-week follow-up, pain decreased from 10 of 10 to 3 of 10 at rest and 5 of 10 with movement. He resumed normal activities.

DISCUSSION

The Metabolic-Skeletal Axis

DISH represents metabolic dysfunction manifest in bone. Hyperinsulinemia drives insulin receptor substrate-1 phosphorylation in osteoblasts. In turn, PI3K/Akt (phosphoinositide 3-kinase/protein kinase B) and MAPK (mitogen-activated protein kinase) pathways are activated which promote cellular proliferation and matrix synthesis.[7] At entheseal sites, where mechanical stress concentrates, sustained signaling creates conditions favorable to ectopic ossification. The patient's metabolic syndrome phenotype (diabetes, obesity, dyslipidemia, hypertension) provided the substrate for DISH development. His chronic shoulder impingement and rotator cuff pathology represent peripheral enthesopathy, an extraskeletal manifestation consistent with systemic DISH.

DISH must be distinguished from ankylosing spondylitis, which also produces spinal fusion but through a very different mechanism. Ankylosing spondylitis is an autoimmune inflammatory condition that affects young adults positive for human leukocyte antigen B27 (HLA-B27). Clinical manifestations include inflammatory back pain, elevated inflammatory markers, and sacroiliac joint erosion. On imaging, ankylosing spondylitis

shows vertical syndesmophytes that give rise to the classic bamboo spine. DISH instead shows flowing anterior osteophytes with preserved disc spaces and no sacroiliac involvement. Peripheral enthesopathy, exhibited in our patient, is absent in purely axial ankylosing spondylitis.

Tirzepatide, a dual glucose-dependent insulinotropic polypeptide and glucagon-like peptide-1 receptor agonist, addresses the fundamental metabolic derangement. The SYNERGY-NASH trial demonstrated that tirzepatide achieves metabolic dysfunction-associated steatohepatitis resolution in 44% to 62% of patients at 52 weeks.[8] Whether improved insulin sensitivity modifies DISH progression requires prospective study.

The Long Lever Arm

Spinal ankylosis transforms biomechanics. A normal spine dissipates trauma across mobile segments. Each disc and facet absorbs energy. In DISH, this protection vanishes. The fused column acts as a long bone and concentrates stress at potential fracture sites. Minor trauma, a ground-level fall or a low-speed collision, can produce unstable three-column fractures involving the anterior column (the anterior vertebral body), the middle column (the posterior vertebral body wall), and the posterior column (pedicles, laminae, and facets).[5,9]

Harlianto et al. reported a pooled vertebral fracture prevalence of 22.6% in DISH versus 15.2% in ankylosing spondylitis.[5] In a retrospective review of spine fractures in patients with ankylosing spinal disorders, Caron et al. found 32% overall mortality, which correlated with age over 70, number of comorbidities, and low-energy mechanism of injury.[10] In that series, 19% of patients had a delayed fracture diagnosis, and 81% of those sustained neurologic compromise. A family physician who recognizes DISH and counsels the patient on fracture risk moves that patient toward urgent imaging after even minor trauma, and that decision separates a survivable injury from a fatal one. Surgical mortality remains substantial given the comorbidity burden in this population.[9]

Diagnostic Mimicry

Cervical DISH may present identically to primary occipital neuralgia. Hypertrophic osteophytes and facet changes produce C2-C3 nerve root irritation with pain radiating to the occiput, vertex, and temporal regions. The presentation also mimics cervical strain, disc herniation, and facet arthropathy. What separates DISH from these diagnoses is the combination of chronicity, metabolic context, and radiographic pattern.

The diagnosis rests on identifying the metabolic phenotype. Patients over 50 years with diabetes, obesity, and neck pain should prompt consideration of lateral cervical radiographs. Flowing anterior osteophytes spanning multiple contiguous levels suggest DISH. Chest radiographs carry 77% sensitivity

and 97% specificity for DISH.[11] Cervical films show symmetric anterior ossification distinct from the candle wax pattern of thoracic DISH.[12] Thoracic imaging strengthens diagnostic certainty but was deferred in our patient because it would not have altered management.

Recognition is important for family physicians in rural settings due to the limited resources available locally. Advanced imaging is often delayed, and subspecialty referral is limited by both socioeconomic and geographic factors. Plain radiographs suffice for initial diagnosis. Patient education about fracture risk and fall prevention can become a life-saving intervention.

Clinical Implications

Three actions emerge.

- Obtain lateral cervical radiographs in metabolic syndrome patients who present with neck pain.
- Educate DISH patients that significant trauma to the head, neck, or spine, particularly falls, motor vehicle collisions, or direct blows, warrants urgent evaluation.
- Coordinate care with physical therapy with a focus on gentle mobilization while avoiding manipulation of the ankylosed spine.

AI Disclosure

The authors used OpenEvidence, DoximityGPT, and UpToDate AI for literature search, clinical reference, literature organization, and citation formatting. Grammarly AI was used for grammar and sentence structure refinement. All content was critically reviewed and verified by the authors against primary sources. The authors retain full responsibility for the scientific content and clinical conclusions of this manuscript.

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REFERENCES

1. Forestier J, Rotes-Querol J. Senile ankylosing hyperostosis of the spine. *Ann Rheum Dis*. 1950;9(4):321-330. doi:10.1136/ard.9.4.321

2. Weng R, Guo H, Ma L, et al. Global prevalence estimates of diffuse idiopathic skeletal hyperostosis: a systematic review and meta-analysis. *Front Endocrinol*. 2025;16:1517168. doi:10.3389/fendo.2025.1517168

3. 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(4):222-225. doi:10.1007/s002560050225

4. Mader R, Verlaan JJ, Buskila D. Diffuse idiopathic skeletal hyperostosis: clinical features and pathogenic mechanisms. *Nat Rev Rheumatol*. 2013;9(12):731-742. doi:10.1038/nrrheum.2013.165

5. Harlianto NI, Ezzafzafi S, Foppen W, et al. The prevalence of vertebral fractures in diffuse idiopathic skeletal hyperostosis and ankylosing spondylitis: a systematic review and meta-analysis. *N Am Spine Soc J*. 2024;17:100312. doi:10.1016/j.xnsj.2024.100312

6. Resnick D, Niwayama G. Radiographic and pathologic features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). *Radiology*. 1976;119(3):559-568. doi:10.1148/119.3.559

7. Fulzele K, Riddle RC, DiGirolamo DJ, et al. Insulin receptor signaling in osteoblasts regulates postnatal bone acquisition and body composition. *Cell*. 2010;142(2):309-319. doi:10.1016/j.cell.2010.06.002

8. Loomba R, Hartman ML, Lawitz EJ, et al. Tirzepatide for metabolic dysfunction-associated steatohepatitis with liver fibrosis. *N Engl J Med*. 2024;391(4):299-310. doi:10.1056/NEJMoa2401943

9. Westerveld LA, Verlaan JJ, Oner FC. Spinal fractures in patients with ankylosing spinal disorders: a systematic review of the literature on treatment, neurological status and complications. *Eur Spine J*. 2009;18(2):145-156. doi:10.1007/s00586-008-0764-0

10. Caron T, Bransford R, Nguyen Q, et al. Spine fractures in patients with ankylosing spinal disorders. *Spine (Phila Pa 1976)*. 2010;35(11):E458-E464. doi:10.1097/BRS.0b013e3181cc764f

11. Mata S, Hill RO, Joseph L, et al. Chest radiographs as a screening test for diffuse idiopathic skeletal hyperostosis. *J Rheumatol*. 1993;20(11):1905-1910.

12. Bakker JT, Kuperus JS, Kuijf HJ, et al. Morphological characteristics of diffuse idiopathic skeletal hyperostosis in the cervical spine. *PLoS One*. 2017;12(11):e0188414. doi:10.1371/journal.pone.0188414

SUPPLEMENTARY MATERIALS

Lateral Cervical Radiograph

Download: https://tfmrj.scholasticahq.com/article/164252-diffuse-idiopathic-skeletal-hyperostosis-presenting-as-cervical-radiculopathy-in-metabolic-syndrome-a-case-report/attachment/351613.png?auth_token=jdOb55v7zd1lULmHOrsT

Anteriorposterior cervical radiograph

Download: https://tfmrj.scholasticahq.com/article/164252-diffuse-idiopathic-skeletal-hyperostosis-presenting-as-cervical-radiculopathy-in-metabolic-syndrome-a-case-report/attachment/351614.png?auth_token=jdOb55v7zd1lULmHOrsT