ISSN: 2755-015X

Journal of Surgery & Anesthesia Research

SCIENTIFIC Research and Community

Case Report

Open Access

Hydroxyapatite Crystal Deposition Disease with Multiple Joint Involvement Resulting in Severe Lumbar Spinal Canal Stenosis: A Case Report

Agnis Saulitis[2*], Rihards Lulens[1], Egils Pukitis[1] and Julija Dolgopolova[1,2]

[1]Pauls Stradins Clinical University Hospital, Department of Neurosurgery, Riga, Latvia

[2]Riga Stradins University, Faculty of Medicine, Riga, Latvia

ABSTRACT

Objectives: To describe a rare complication of hydroxyapatite (HA) crystal deposition disease (HADD) – spinal canal stenosis. To discuss clinical features and surgical management of this entity.

Background: HADD is a crystal-induced arthropathy caused by para-articular and/or intra-articular deposition of HA crystals. Etiology is uncertain, predisposing factors include microtrauma, local ischemia, end-stage renal disease. Commonly, glenohumeral joint is affected, less often - elbow, wrist, hand, hip, knee, ankle, foot and spine. Rarely – deposition of crystals is associated with spinal canal stenosis.

Methods: We report a case of 34-year-old male who was admitted to the hospital with recurrent fever and elevated inflammatory markers for the past few months. Patient had a history of stage 5 chronic kidney disease treated with hemodialysis, anabolic-androgenic steroid use, arterial hypertension, tertiary hyperparathyroidism and multiple calcium deposits along glenohumeral, iliofemoral joints bilaterally, left foot and lumbar spine. Computed-tomography showed hyperdense calcifications in soft tissue along lumbar spine with minimal intraspinal involvement. Over the course of a few weeks' symptoms worsened, the patient presented with lower back pain radiating in both legs, progressive lower paraparesis, walking difficulties. Magnetic-resonance imaging showed hypointense periarticular lesions with fluid levels on both T1 and T2-weighted images from L1 to L3 extending intraspinally with severe spinal canal stenosis, medullary cone compression. Surgical resection of the pathological tissue was indicated.

Results: The patient underwent L1-L3 laminectomy and resection of the lumbar soft tissue and intraspinal extradural calcified depositions. Histopathological examination confirmed diagnosis of HADD associated spinal canal stenosis. Physical rehabilitation followed, progressive recovery was seen, with almost complete resolution of motor deficits 6 months post-surgery.

Conclusions: HADD, affecting lumbar spine, can lead to inflammation, swelling and stenosis of the spinal canal resulting in severe back pain and progressive neurological deficits. Surgical treatment in such cases can be curative and lead to good outcomes.

***Corresponding author**

Agnis Saulitis, Riga Stradins University, Faculty of Medicine, Riga, Latvia.

Received: January 02, 2024; **Accepted:** January 09, 2024; **Published:** January 16, 2024

Keywords: Hydroxyapatite Crystal Deposition Disease, Lumbar Spine, Lumbar Stenosis, Spinal Canal Stenosis

Introduction

Hydroxyapatite (HA) crystal deposition disease (HADD) is a crystal-induced arthropathy caused by para-articular and/or intra-articular deposition of HA crystals. Etiology is uncertain, predisposing factors include microtrauma, local ischemia, end-stage renal disease. Commonly, glenohumeral joint is affected, less often - elbow, wrist, hand, hip, knee, ankle, foot and spine. Rarely – deposition of crystals is associated with spinal canal stenosis. Our objectives was to describe a rare complication of HADD– spinal canal stenosis and discuss clinical features and surgical management of this entity [1, 2].

Case Description

We report a case of 34-year-old male who was admitted to the hospital with recurrent fever and elevated inflammatory markers for the past few months. Patient had a history of stage 5 chronic kidney disease treated with hemodialysis, anabolic-androgenic steroid use, arterial hypertension, tertiary hyperparathyroidism and multiple calcium deposits along glenohumeral, iliofemoral joints bilaterally, left foot and lumbar spine. Computed-tomography (CT) showed hyperdense calcifications in soft tissue along lumbar spine with minimal intraspinal involvement. (Figure 1)

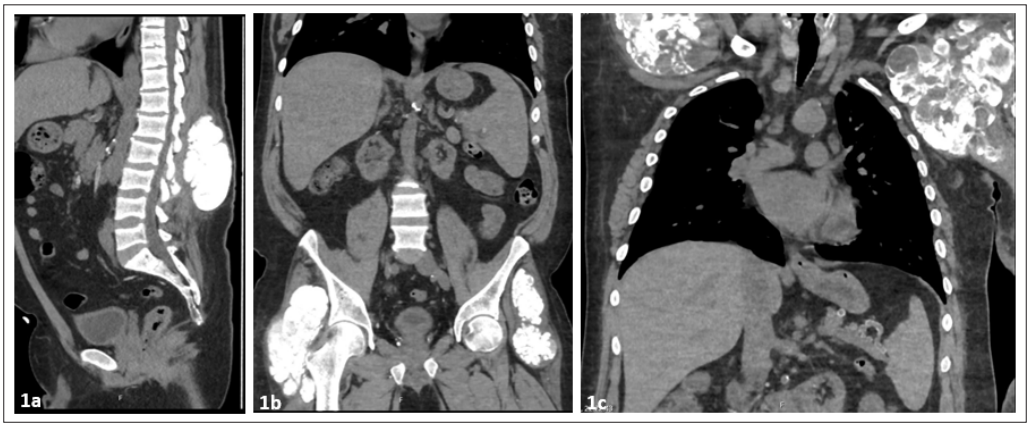

Figure 1: Saggital (1a), Coronal (1b) Abdominal, Coronal (1c) Thoracic CT Scans Demonstrating Massive Crystal Depositions in Surrounding Soft Tissue of Lumbar Spine, Iliofemoral Joints Bilaterally and Glenohumeral Joints Bilaterally.

Over the course of a few weeks' symptoms worsened, the patient presented with lower back pain radiating in both legs, progressive lower paraparesis, walking difficulties. Magnetic-resonance imaging (MRI) showed hypointense periarticular lesions with fluid levels on both T1 and T2-weighted images from L1 to L3 extending intraspinally with severe spinal canal stenosis, medullary cone compression. (Figure 2)

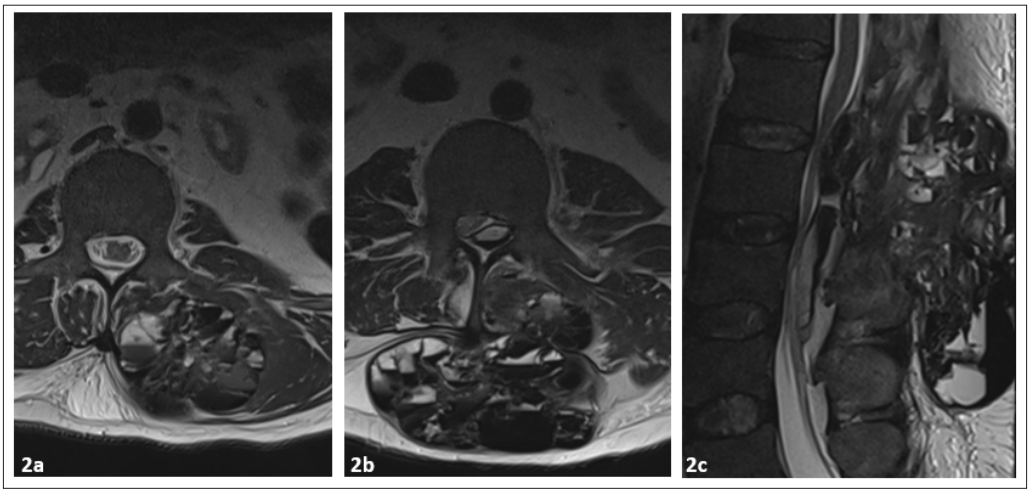

Figure 2: Lumbar axial (2a) T2W MRI Showing L1 Periarticular Lesions with Fluid-Filled Levels, Axial (2b) Image Showing Spinal Canal Stenosis at the Level of L3. Saggital (2c) Lumbar T2W MRI Demonstrating Severe Spinal Canal Stenosis from L1 to L3 Level.

Surgical resection of the pathological tissue was indicated. The patient underwent L1-L3 laminectomy and resection of the lumbar soft tissue and intraspinal extradural calcified depositions. (Figure 3)

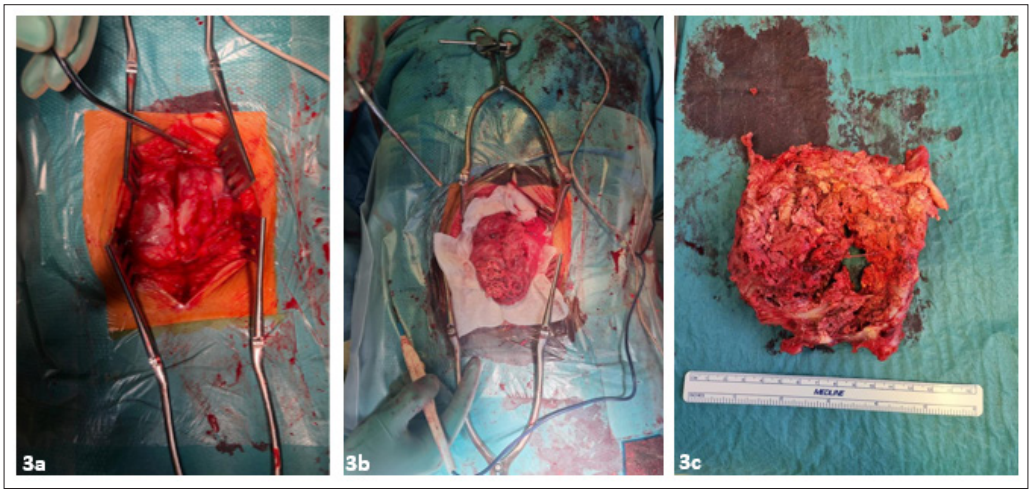

Figure 3: Intraoperative View of Calcified Depositions (3a-3c)

Histopathological Examination Confirmed Diagnosis of HADD Associated Spinal Canal Stenosis. Physical Rehabilitation Followed, Progressive Recovery was Seen, With Almost Complete Resolution of Motor Deficits 6 Months Post Surgery.

Discussion

The presented case highlights a rare complication of HADD – spinal canal stenosis. This condition predominantly affects the glenohumeral joint, and less commonly the elbow, wrist, hand, hip, knee, ankle, foot, and spine - in rare instances leading to spinal canal stenosis, as observed in our case [1]. Patient's presentation with recurrent fever, elevated inflammatory markers points out the systemic impact of HADD. HADD's association with conditions such as chronic kidney disease and anabolic-androgenic steroid use suggests potential predisposing factors in this case [2-5].

Imaging played a crucial role in diagnostics, assesing the severity of the spinal involvement. CT revelaed hyperdense calcifications along the lumbar spine, wile magnetic resonance imaging provided detailed insights into the extent of the pathology. The identification of hypointense periarticular lesions with fluid levels on both T1 and T2-weighted images, along with severe spinal canal stenosis and medullary cone compression, guided the decision for the intervention [6-8].

The surgical approach involved L1-L3 laminectomy and resection of the lumbar soft tissue and intraspinal extradural calcified depositions. Histopathological examination confirmed the diagnosis of HADD-associated spinal canal stenosis. The postoperative course demonstrated progressive recovery, with almost complete resolution of motor deficits six months post-surgery. This positive outcome emphasizes the importance of timely surgical management in cases of HADD-related spinal canal stenosis to prevent irreversible neurological damage.

In conclusion, this case report sheds light on the rare but serious complication of spinal canal stenosis associated with HADD. Clinicians should be vigilant in patients with HADD, particularly those with predisposing factors, to identify and manage potential complications promptly. The successful surgical intervention in this case underscores the therapeutic potential of timely and targeted approaches in mitigating the impact of HADD on spinal structures.

References

1. Glenn M Garcia, Gary C McCord, Rajendra Kumar (2003) Hydroxyapatite crystal deposition disease. Seminars in Musculoskeletal Radiology 7: 187-194.
2. Paik NC (2014) Acute calcific tendinitis of the gluteus medius: an uncommon source for back, buttock, and thigh pain. Semin Arthritis Rheum 43: 824-829.
3. Kerl-Skurka A, Flueckiger B, Ali S, Kilgus M (2014) Severe bursitis in shoulder arthritis – a manifestation of hydroxyapatite crystal deposition disease. Case Reports in Clinical Pathology 2.
4. Best JA, Shapiro RD, Kalmar J, Westesson PL (1997) Hydroxyapatite deposition disease of the temporomandibular joint in a patient with renal failure. J Oral Maxillofac Surg 55: 1316-1322.
5. Freire V, Moser TP, Lepage-Saucier M (2018) Radiological identification and analysis of soft tissue musculoskeletal calcifications. Insights Imaging 9: 477-492.
6. Hegazi T (2023) Hydroxyapatite Deposition Disease: A Comprehensive Review of Pathogenesis, Radiological Findings, and Treatment Strategies. Diagnostics (Basel) 13: 2678.
7. Hongsmatip P, Cheng KY, Kim C, Lawrence DA, Rivera R (2019) Smitaman E. Calcium hydroxyapatite deposition disease: Imaging features and presentations mimicking other pathologies. Eur J Radiol 120: 108653.
8. Davani-Davari D, Karimzadeh I, Khalili H (2019) The potential effects of anabolic-androgenic steroids and growth hormone as commonly used sport supplements on the kidney: a systematic review. BMC Nephrol 20: 198.