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NOT ALWAYS CRYSTAL CLEAR: PSEUDOGOUT AS A CAUSE OF LUMBAR RADICULAR PAIN—A CASE REPORT

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Background: Calcium pyrophosphate crystal deposition (CPPD) also known as "pseudogout" is a lesser-known cause of radicular pain that can occur in the spine after surgical intervention. Crystals may deposit extradurally in the ligamentum flavum, facet joints, and intervertebral discs causing symptoms ranging from mild-to-intractable radicular pain or cauda equina syndrome.

Case Report: A 60-year-old man with a history of multiple lumbar surgeries and recent right L2-L3 hemilaminectomy/facetectomy presented with severe, radicular pain. Physical exam demonstrated decreased left L5 dermatome sensation. Postop imaging confirmed worsening foraminal encroachment at L5-S1 compared to preop imaging. The patient failed steroid taper, neuropathic agents, nonsteroidal anti-inflammatory drugs, physical therapy, and 3 L5-S1 epidural steroid injections. He ultimately underwent L5-S1 microdiscectomy where numerous crystalline deposits were identified and sent for pathology, revealing CPPD with foamy histiocytes.

Conclusions: Clinicians should consider CPPD in at-risk patients with radicular pain following surgery to expedite appropriate workup and rheumatologic management.

Key words: Case report, pseudogout, CPPD, lumbar radiculopathy

BACKGROUND

Lumbar radiculopathy is a common complaint, currently affecting an estimated 3% to 5% of the population. Age is a primary risk factor for radiculopathy, as it can develop secondary to the degenerative processes that may affect several structures surrounding the exiting spinal nerve roots. Some of the most common degenerative etiologies include bony hypertrophy of the facet joints, spondylosis, spondylolisthesis, and intervertebral disc herniation. Depending on the underlying etiology of radiculopathy, treatment options are generally thought to be surgical or symptom-based conservative medical management. Beyond the many known causes of radicular pain, a lesser-known cause is calcium pyrophosphate crystal deposition (CPPD), also known as pseudogout, which typically arises from degenerative, inflammatory (autoimmune), or traumatic processes, but can occur acutely following surgical disruption of the tissues. At present, the true prevalence of pseudogout is unknown, existing literature estimates that it may range anywhere from 4% to 40% (1,2). CPPD exacerbations may require surgical decompression and may have some response to conservative management. However, proper diagnosis of CPPD is key as effective

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pain management may be achieved with other rheumatologic medications and patients may be at higher risk of recurrence. This case highlights a clinical presentation in a 60-year-old man with risk factors for CPPD who experiences progressive worsening of lumbar radiculopathy immediately following recent surgery.

CASE PRESENTATION

A 60-year-old man with a past medical history of degenerative joint disease, multiple prior lumbar spine surgeries, and recent right L2-L3 hemilaminectomy with medial facetectomy presented to our clinic complaining of 2 months of progressive, severe, electric-like left buttock pain radiating down to his posterior left leg with initial onset of pain occurring the day after his surgery. Upon physical exam, the patient demonstrated baseline 4/5 knee extension in manual muscle strength testing, stable 0/2 patellar reflex, and new onset decreased sensation to the L5 dermatome on the left. Postop magnetic resonance imaging (MRI) was obtained and findings were consistent with interval worsening of foraminal encroachment at the left L5-S1 level when compared with MRI from 2 months prior to surgery. The patient trialed conservative management with a methylprednisolone dose pack, neuropathic agents, nonsteroidal anti-inflammatory drugs, a full course of physical therapy, and 3 left-sided L5-S1 transforaminal epidural steroid injections all with little-to-mild relief of his symptoms. He ultimately underwent a left-sided L5-S1 microdiscectomy, during which time crystalline deposits within the spine were identified. These were sent for pathology and found to be calcium pyrophosphate (CPP) with foamy histiocytes, consistent with CPPD. The patient had initial pain relief after surgery with gradual recurrence of pain, which was refractory to neuropathic and anti-inflammatory medications, an unexpected outcome. He was subsequently referred to Rheumatology where he was prescribed colchicine in addition to continuing care with pain management. The patient was eventually able to achieve pain relief once his CPPD was well controlled with colchicine.

CONCLUSIONS

Most patients affected by acute CPPD arthritis are men over the age of 65, with 30% to 50% of patients presenting over the age of 85 years and clinically significant presentation under age 60 being incredibly rare (1,3). CPPD remains an idiopathic condition; however, several studies have shown it to have a high positive association with hyperparathyroidism and less strongly with gout, osteoarthritis, rheumatoid arthritis, and hemochromatosis (3,4,5). Other associated comorbidities include osteoporosis, hypomagnesemia, chronic kidney disease, and calcium supplementation (4,6-8). The pathophysiology of CPPD is attributed to an imbalance between the production of pyrophosphate and the elevated levels of pyrophosphatases, which are present in diseased cartilage. As pyrophosphate deposits in the synovium and adjacent tissues, it can combine with calcium to form CPP crystals. CPPD tends to affect predisposed joints, usually caused by inflammatory conditions (i.e., rheumatoid arthritis, systemic lupus erythematosus), degeneration, or trauma with peripheral joints being the most commonly involved (1).

This case is unique because it evidences that, though rare, CPPD can occur in the spinal column and may or may not be seen with peripheral joint involvement as well. Typically, the cervical region is most commonly affected, followed by the lumbar spine, and rarer still, the thoracic spine (9). Calcium crystal deposition in the lumbar spine has been noted in the extradural space, ligamentum flavum, facet joints, synovial cysts, and intervertebral discs of the lumbar spine. Prior histological studies and autopsy examination specifically of lumbar intervertebral discs suggest that trauma and previous lumbar surgery may be predisposing factors to CPPD (2,5,13). While the pathogenesis of CPPD following surgery is not completely known, some studies (10) suggest the disruption and subsequent degeneration of elastic fiber bundles in the ligamentum flavum serve as a nidus for calcium crystal deposition by hypertrophic chondrocytes.

Clinically, patient presentation can vary in both acuity and severity, ranging from mild-to-intractable radicular pain, with rare occurrences of cauda equina syndrome. Acute presentations can present as warm, erythematous, and edematous joints with mild leukocytosis and elevated erythrocyte sedimentation rate/C-reactive protein, often mimicking gout or infection. The gold standard for diagnosis is histological confirmation of tissue biopsy; however, joint aspiration, if possible, can also be used. Diagnosis of CPPD in the spine can become more difficult in terms of obtaining tissue or aspirate, especially considering that CPP crystals can also coexist with monosodium urate crystals seen in gout (11,12). Advanced imaging is usually obtained in the workup of acute, severe, or progressive presentations. Numerous case reports (13,14) have shown that CPPD

can commonly be falsely interpreted as osteomyelitis or discitis. Older studies (15-17) have demonstrated that gradient-echo MRI sequences can be helpful and have higher sensitivity for detecting CPPD in joints; however, if not possible, computed tomography would be the next best imaging option. Clinician correlation of clinical, radiographic, laboratory, and pathological findings is key. Once diagnosed, these patients can benefit from rheumatologic care to decrease the recurrence of flares and symptom management with colchicine and interleukin-1-receptor-antagonist medications. The strengths of this case report include relevant teaching points in a typical patient presentation, guidance based on existing literature for diagnosing spinal CPPD if suspected, and it also provides stronger evidence to the existing body of literature and prevalence. Limitations of this case report are lack of a control patient to compare intervention, eventual loss of a patient to follow-up, and no initial lab testing to help support the diagnosis, though this is not the standard of care in a pain management clinic setting.

CONCLUSION

Clinicians should be aware of CPPD as a possible diagnosis with varying acuity and severity in a patient presentation. Although CPPD involvement in the lumbar spine is rare, it should be suspected in elderly patients with risk factors of male gender, osteoarthritis, prior or recent spine surgery, hyperparathyroidism, and chronic kidney disease as well as other weaker associations. Diagnosis requires correlation of clinical, lab, histologic, and imaging findings. Advanced imaging of the lumbar spine in cases of CPPD is often misdiagnosed as osteomyelitis or discitis on MRI. CPPD with spinal involvement may not respond to conservative management. Patients can be referred to Rheumatology for disease management for both symptom management and to reduce risk of recurrence. Rheumatologic treatment options may also consist of colchicine and interleukin-1-receptor-antagonist medications.

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