



LETTER TO EDITOR

CPPD Causing RA Like Tendon Ruptures in Hand- Highlighting the Known Facts for Clinical Update

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I read with great interest the article entitled “A Case of Extensive Hand Extensors Tendon Rupture Due to Pseudo-rheumatoid Arthritis - A Great Mimicker of Rheumatoid Arthritis by Young Min Cho, et al. in your esteemed journal in October’2022”. Joint & soft tissue problems seen with these crystals often are mistaken for gout and other conditions like RA etc. as appropriately highlighted by the author.

For the benefit of the readers, it is important to mention that about the complex clinical classification of ‘pseudogout’ (type A), ‘pseudo-rheumatoid arthritis’ (type B), ‘pseudo-osteoarthritis’ (with acute attacks, type C; without inflammation, type D), ‘lanthanic or asymptomatic’ (type E) and ‘pseudoneuropathic’ (type F), to which other forms were later added [1].

The EULAR CPPD Task Force has suggested ‘calcium pyrophosphate deposition’ (CPPD) as the umbrella term for all instances of CPP crystal deposition which includes asymptomatic CPPD, OA with CPPD, acute CPP crystal arthritis and chronic CPP crystal inflammatory arthritis. It was felt that terms prefixed by ‘pseudo’ should be abandoned because they do not specify the causative crystal, are probably not discrete clinical subsets, are a source of potential confusion for patients, and intimate that CPP crystals are of secondary importance and interest compared to sodium urate. The term chondrocalcinosis (CC) is retained for cartilage calcification, which is most commonly due to CPPD. It is appreciated that introduction of new terminology may prove inconvenient in the short term, but would

be beneficial in the long term for research, education and clinical practice [2].

Here, author has not mentioned about the family history as the disease is frequently genetic in nature. Molecular genetics studies have identified two genetic locations for familial CC involving a mutation in the long arm of chromosome 8 (8q), and the other resulting from a mutation on the short arm of chromosome 5 (5p) [3]. An autosomal dominant mode of inheritance has been suggested in several reports [4,5].

Clinicians should be made aware that knee is the most commonly involved joint, followed by the wrist, metacarpophalangeal, hips, shoulder, elbow and ankle joints [6,7]. But all joints are susceptible, including the first MTP joint [8].

To add to the report extra-articular deposition of calcium pyrophosphate dihydrate (CPPD) crystals has been described in rare cases, especially in the ligamentum flavum also, which may manifest with back and neck related neurological issues [9].

It is important to do iron studies, including measurement of magnesium, iron, transferrin, ferritin levels and parathyroid hormone levels, not mentioned here. Also, Parenteral administration of granulocyte colony-stimulating factor (G-CSF) and of bisphosphonates which are commonly used in the management of osteoporosis etc. can also trigger pseudogout, the former likely by ignition of smoldering subclinical intra-articular inflammation, the latter

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theoretically via pyrophosphatase inhibition, because bisphosphonates are nonhydrolyzable analogues of PP_i [10].

It is still controversial whether it leads to calcification of articular fibrocartilage or hyaline cartilage [11]. As clinically, both tissues can be affected.

Literature shows that these crystals contain mainly calcium pyrophosphate dihydrate and basic calcium phosphate crystals, the latter being related to hydroxyapatite, carbonate-substituted apatite, and octacalcium phosphate [12].

Author could have done the Gram stain of collected fluid can be done to show the presence of calcium pyrophosphate dihydrate crystals. Histological examination has also not been done here as staining by haematoxylin and eosin and by von Kossa stain can reveal calcium phosphate deposits and is likely to show a non-specific scar remodelling of the synovium with gelatinous deposits and area of CPPD crystal deposits [13].

The crystals are weakly positively birefringent on polarized microscopy and have a rhomboid or rod shape. Author has not shown the shape of the crystals here, which is paramount for the diagnosis of CPPD.

In these patient's hand X-rays usually shows hook like projections arising from radial aspect of second and third metacarpal heads with scapholunate advanced collapse. There can be linear or punctate calcification of the hyaline and fibrocartilage. The X-rays can show flecks of linear calcification in the region of the tendon sheath [14]. This has not been shown here in the published report.

Linear hyperechoic bands positioned along the major axis of the tendon from sonography refer to the presence of calcium pyrophosphate dihydrate crystals, which helps in diagnosing this condition too [15].

Recently, Raman spectroscopy is a chemical analysis technique that is 100% specific in fingerprinting species based on the identification of chemical bonds unique to each material has been used as a diagnostic modality [16,17].

MRI is able to identify tendon and ligament abnormalities associated with arthropathy, including tendon rupture, tenosynovitis, and changes within tendons and ligaments in association with enthesitis. MRI picture does not show the rupture as highlighted in the article & even not showing any changes in the 3rd & 5th digits which raises serious questions on the diagnosis.

For management point of view, data support the use of hydroxychloroquine in patients with CPPD disease [18].

Also, a recent randomized, controlled trial involving

similar patients who were assigned to methotrexate, administered subcutaneously at a dose of 15 mg per week, or placebo showed no difference between the drug and placebo [19]. However, Methotrexate is associated with few side effects in these patients and remains an option for selected patients in whom other therapies have failed. There are some latest anecdotal evidence supports the use of interleukin-1 β inhibitors in patients with chronic CPP crystal arthritis.

References

- McCarty DJ (1976) Calcium pyrophosphate dihydrate crystal deposition disease--1975. *Arthritis Rheum* 19: 275-285.
- Zhang W, Doherty M, Bardin T, Barskova V, Guerne P-A, et al. (2011) European league against rheumatism recommendations for calcium pyrophosphate deposition. Part I: Terminology and diagnosis. *Ann Rheum Dis* 70: 563-570.
- Netter P, Bardin T, Bianchi A, Richette P, Loeuille D (2004) The ANKH gene and familial calcium pyrophosphate dihydrate deposition disease. *Joint Bone Spine* 71: 365-368.
- Van der Korst JK, Geerards J, Driessens FC (1974) A hereditary type of idiopathic articular chondrocalcinosis. Survey of a pedigree. *Am J Med* 56: 307-314.
- Ahn JK, Kim HJ, Kim EH, Jeon CH, Cha HS, et al. (2003) Idiopathic calcium pyrophosphate dihydrate (CPPD) crystal deposition disease in a young male patient: a case report. *J Korean Med Sci* 18: 917-920.
- Doherty M, Dieppe P (1988) Clinical aspects of calcium pyrophosphate dihydrate crystal deposition. *Rheum Dis Clin North Am* 14: 395-414.
- Whelan B, O'Shea F, McCarthy G (2008) Pseudoneuropathic CPPD arthropathy: Magnesium matters. *Rheumatology* 47: 551-552.
- Rowdon G, Taylor D (2008) Arthritic, metabolic, and vascular disorders. In: *Baxter's the foot and ankle in sport*. (2nd edn), Elsevier, 241-249.
- Ellman MH, Vazquez T, Ferguson L, Mandel N (1978) Calcium pyrophosphate deposition in ligamentum flavum. *Arthritis Rheum* 21: 611-613.
- (2017) Calcium crystal disease-robert terkeltaub. In: Firestein GS, Budd RC, Gabriel SE, McInnes IB, O'Dell JR, Kelley and Firestein's textbook of rheumatology. (10th edn), Elsevier.
- Brower AC, Flemming DJ (2012) *Arthritis in black and white*. (3rd edn), Elsevier health sciences.
- Ea HK, Lioté F (2004) Calcium pyrophosphate dihydrate and basic calcium phosphate crystal-induced arthropathies: Update on pathogenesis, clinical features, and therapy. *Curr Rheumatol Rep* 6: 221-227.
- Yoshida H (2016) Tenosynovitis due to pseudogout. *N Z Med J* 129: 84-85.
- Dalbeth N, McQueen FM (2009) Use of imaging to evaluate gout and other crystal deposition disorders. *Curr Opin Rheumatol* 21: 124-131.
- Ho K-Y, Liang JN (2019) Calcium pyrophosphate deposition disease in the achilles tendon. *J Belg Soc Radiol* 103: 79.
- Li B, Singer NG, Yeni YN, Haggins DG, Barboym E, et al.

- (2016) A point-of-care Raman spectroscopy-based device for the diagnosis of gout and pseudogout: comparison with the clinical standard microscopy. *Rheumatol Int* 68: 1751-1757.
17. Wu Y, Chen K, Terkeltaub R (2016) Systematic review and quality analysis of emerging diagnostic measures for calcium pyrophosphate crystal deposition disease. *RMD Open* 2: e000339.
18. Rothschild B, Yakubov LE (1997) Prospective 6-month, double-blind trial of hydroxychloroquine treatment of CPDD. *Compr Ther* 23: 327-331.
19. Finckh A, McCarthy GM, Madigan A, Linthoudt DV, Weber M, et al. (2014) Methotrexate in chronic-recurrent calcium pyrophosphate deposition disease: no significant effect in a randomized crossover trial. *Arthritis Res Ther* 16: 458.