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【 EDITORIAL 】

Acute CPP Crystal Arthritis Causing Carpal Tunnel Syndrome

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Calcium pyrophosphate dihydrate crystal deposition (CPPD) disease is an inflammatory arthritis produced by the deposition of calcium pyrophosphate (CPP) crystals in articular and periarticular soft tissues. The clinical presentation associated with CPPD varies and is now divided into the following four phenotypes according the European League Against Rheumatism (EULAR) recommendations: asymptomatic CPPD, acute CPP crystal arthritis (also known as pseudogout), osteoarthritis with CPPD, and chronic CPP crystal inflammatory arthritis (1).

In the Internal Medicine, Itagaki presented a 92-year-old man with acute CPP crystal arthritis causing carpal tunnel syndrome (CTS) (2). This patient presented to the emergency department with the rapid development of severe inflammation in the left wrist with a swollen hand (Picture 1) and acute compression neuropathy of the median nerve, i.e., finger dysesthesias and positive Tinel's sign (referred to as "distal tingling on percussion"). The etiology of the CTS was suggested to be acute CPP crystal arthritis, since radiographic cartilage calcification (CC) was detected in the wrists (Picture 2), and distention in the palmar bursae due to synovitis was indicated by the cystic lesion observed on CT (Picture 3, 4). The presence of CPP crystals and inflammatory cell infiltration was proved by a synovial fluid examination, leading to the definitive diagnosis.

CPPD is common, especially in the elderly (>65 years of age), as the prevalence of CC has been estimated to be 4% to 7% in the adult populations of Europe and the United States (3, 4), and the risk doubles every decade between 45 and 85 years (odds ratio=2.25, 95% confidence interval= 1.79-2.82), independent of other risk factors (5). Among the EULAR clinical classifications, acute CPP crystal arthritis is characterized by self-limiting, acute attacks of arthritis (1). The traditional term pseudogout underlines its clinical resemblance to arthritis attacks of urate gout. Both crystal-induced diseases typically present as acute monoarticular or oligoarticular arthritides; however, acute CPP crystal arthritis more frequently affects large joints, such as the knees, wrists, and ankles, while gout commonly involves the first metatarsophalangeal joint. The distinction between these diseases is essentially made based upon the detection of characteristic urate or CPP crystals in the synovial fluid, which are virtually phagocytosed within polymorphonuclear leukocytes during acute attacks. In contrast to the needle-shaped, strongly, and negatively birefringent monosodium urate crystals in gout, CPP crystals are rhomboidal or parallelepiped and show weakly positive birefringence under polarized light microscopy. On radiography, CPP crystal deposits appears as punctate and linear radiodensities in articular cartilage, whereas gout arthritis appears as radiolucent bone erosions around the joints. The radiographic evidence of CC strengthens the diagnosis of CPPD, but its absence does not rule it out. Ultrasonography has been shown to be a useful modality for diagnosing CC (6).

Interleukin-1β (IL-1β) plays a central role in crystal-induced inflammation (7). CPP crystals activates the NALP3 inflammasome, which converts pro-IL-1β into mature IL-1β via caspase-1 activation. IL-1β-mediated acute inflammation within the palmar bursae, induced by CPP crystal deposition, may have caused the median nerve compression in the carpal tunnel observed in the case presented by Itagaki. At present, treatments for acute CPP crystal arthritis include rest, local application of ice or cool packs, joint aspiration, non-steroidal anti-inflammatory drugs, colchicine, and/or intra-articular glucocorticosteroid injection (once infection is excluded) (8). There has been increasing interest in the use of IL-1β-targeting therapy to treat crystal-induced arthritides, such as gout and CPPD diseases.

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