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Literature Review

Magnesium disorders can cause calcium pyrophosphate deposition disease: A case report and literature review

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Abstract

Calcium pyrophosphate deposition (CPPD) disease, also known as pseudogout, is one of the most common forms of inflammatory arthritis. A variety of comorbidities and metabolic conditions have been recognized to predispose to CPPD. We describe here a patient with chronic CPP arthritis due to hypomagnesemia, which is one of the metabolic etiologies associated with CPPD, especially in younger patients. We also performed a literature search and reviewed all reported cases of CPPD disease associated with hypomagnesemia. All cases of hypomagnesemia and its etiologies leading to CPP arthropathy identified in the literature by this systematic search are summarized in this paper.

Keywords: Magnesium, CPPD, pseudogout, calcium pyrophosphate deposition disease, hypomagnesemia, chondrocalcinosis

Introduction

Calcium pyrophosphate deposition (CPPD) disease, also known as pseudogout, is one of the most common forms of inflammatory arthritis. Chondrocalcinosis, defined as the calcification of the cartilage and identified by radiological or histological examinations, is a common feature of CPPD. Calcification of the articular cartilage was reported in the literature as early as the 1920s (1). Zitnan et al. (2) described radiographic chondrocalcinosis associated with joint disease in the 1950s. However, it was not until 1962 that McCarty et al. (3) identified CPPD crystals in the synovial fluid.

Historically, various nomenclatures such as pseudorheumatoid arthritis, pseudoankylosing spondylitis, articular chondrocalcinosis, and pseudogout have been employed to describe CPPD disease (2, 4). In 2011, the European League Against Rheumatism task force redefined the terminology into the subsets of asymptomatic CPPD, osteoarthritis (OA) with CPPD, acute CPP crystal arthritis, and chronic CPP crystal inflammatory arthritis to illustrate the entire spectrum of CPPD disease (5).

A variety of comorbidities and metabolic conditions have been recognized to predispose to CPPD (Table 1). We describe here a patient with chronic CPP arthritis due to hypomagnesemia, which is one of the metabolic etiologies associated with CPPD, especially in younger patients. We also performed a literature search and reviewed all reported cases of CPPD disease associated with hypomagnesemia. We used the following keywords to search the MEDLINE database: (a) "magnesium," (b) "chondrocalcinosis," (c) "CPPD," and (d) "pseudogout." Searching for (b), (c), or (d) produced 1,936 articles, and combining these titles with the keyword "magnesium" resulted in 60 articles. All cases of hypomagnesemia leading to CPP arthritis identified by this systematic search are summarized in Table 2.

Table 1. Risk factors associated with CPPD disease

1. Increasing age
2. Known osteoarthritis of the joints
3. Previous joint injury
4. History of knee meniscectomy
5. Metabolic diseases
   a. Hypomagnesemia
   b. Hemochromatosis
   c. Hyperparathyroidism
   d. Hypophosphatasia

CPPD: calcium pyrophosphate deposition

Case Report

A 32-year-old Caucasian female presented with acute onset of left ankle pain and swelling. Arthrocentesis revealed cloudy yellowish fluid with a white blood cell (WBC) count of 32,300 cells/mm^3, red blood cell (RBC) count of 2,100 cells/mm^3, but no crystals. She underwent irrigation and debridement of the joint and was treated with intravenous antibiotics and nonsteroidal anti-inflammatory drugs (NSAIDs). Two weeks later, she again presented with acute right knee pain and swell-

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Table 2. All cases of CPP arthritis due to hypomagnesemia identified in the literature

Reference	Age/Sex	Primary Diagnosis	Mg Level	Presentation
Hahn et al. (24)	60/M	SBS	0.2 mmol/L	Arthralgias with CC
Whelan et al. (30)	56/M	Thiazide-induced hypomagnesemia	0.48 µg/L	A-CP
Richetite et al. (31)	56/F	SBS	0.56 mmol/L	A-CP
	73/F	SBS and radiation enteritis	0.62 mmol/L	CC, A-NCP
	45/M	SBS	0.41 mmol/L	CC, A-NCP
Hang-Korng et al. (32)	53/M	Gitelman syndrome	0.54 mmol/L	CC, A-NCP
Perez-Ruiz et al. (20)	64/F	Liver transplant for chronic liver disease	1.36 mg/dL	A-CP, CC
	48/F	Liver transplant for chronic liver disease	1.39 mg/dL	A-CP
Melvin et al. (21)	65/F	Hyperparathyroidism	0.8 meq/L	A-NCP, CC
Gonzalez Dominguez et al. (33)	40/M	Hypomagnesemia - unknown cause	1.4 mg/dL	A-NCP, CC
	43/M	Hypomagnesemia - unknown cause	1.6 mg/dL	A-NCP, CC
Waitman et al. (34)	60/M	Bartter syndrome	0.75 mg/dL	A-CP, CC
Milazzo et al. (35)	40/F	Genetic defect of Mg metabolism	0.46 mmol/L	A-CP, CC
Resnick et al. (36)	30/M	Renal dysfunction causing low Mg	0.5 mgm/dL	A-NCP, CC
	52/M	Renal dysfunction causing low Mg	1.8 mg/dL	A-CP, CC
Salvarani et al. (37)	38/M	Bartter syndrome	0.8 mg/dL	A-CP, CC
Bauer et al. (38)	24/M	Bartter syndrome	0.4 mmol/L	CC
	40/F	Bartter syndrome	0.53 mmol/L	CC
De Bruyne et al. (25)	32/M	Bartter syndrome	0.7 meq/L	CC
Goulon et al. (39)	37/F	Bartter syndrome	16 mg/L	CC
	27/M	Bartter syndrome	14 mg/L	CC
Hurault de Ligny et al. (40)	24/F	Bartter syndrome	0.46 mmol/L	CC
Schwarz et al. (41)	32/M	Bartter syndrome	0.29 meq/L	CC
Punzi et al. (42)	54/F	Gitelman syndrome	0.44 mmol/L	A-CP, CC
	27/M	Gitelman syndrome	0.6 mmol/L	A-CP, CC
	35/M	Gitelman syndrome	0.59 mmol/L	CC
	37/F	Gitelman syndrome	0.35 mmol/L	A-CP, CC
Cimbeck et al. (43)	17/M	FHHNC	1.1 mg/dL	A-NCP
Hisakawa et al. (44)	45/F	Bartter syndrome	1.2 meq/L	A-NCP, CC
Mayoux-Beuhamou et al. (45)	26/F	Bartter syndrome	0.55 mmol/L	CC
	56/F	Bartter syndrome	0.35 mmol/L	CC
Dupond et al. (46)	38/F	Bartter syndrome	0.48 mmol/L	CC
De Heide et al. (47)	44/F	Bartter syndrome	0.65 mmol/L	CC
Jones et al. (48)	49/F	Bartter syndrome	0.5 mmol/L	CC
Smilde et al. (49)	39/F	Bartter syndrome	0.56 mmol/L	CC
	56/M	Bartter syndrome	0.60 mmol/L	CC
	59/M	Bartter syndrome	0.60 mmol/L	CC
	41/F	Bartter syndrome	0.49 mmol/L	CC
	37/M	Bartter syndrome	0.54 mmol/L	CC
	43/M	Bartter syndrome	0.49 mmol/L	CC
	44/M	Bartter syndrome	0.52 mmol/L	CC
Munoz-Fernandez et al. (50)	65/M	Bartter syndrome	1.2 mmol/L	CC

A-CP: arthritis-crystal proven; A-NCP: arthritis-non-crystal proven; CC: chondrocalcinosis; SBS: short bowel syndrome; Mg: magnesium

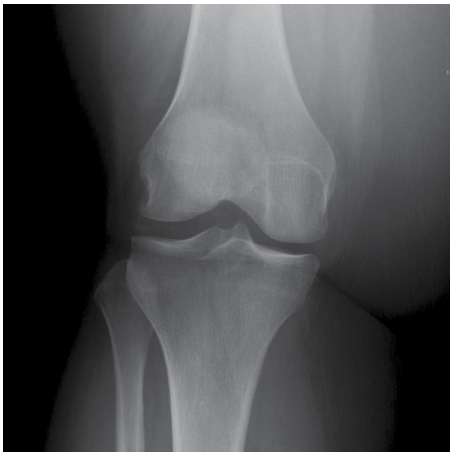

Figure 1. Anteroposterior radiograph of the right knee demonstrating radiodense material between the articular surfaces suggesting meniscal calcification

ing. Arthrocentesis of the right knee revealed slightly cloudy yellowish synovial fluid with WBC count of 2,400 cells/mm^3, RBC count of 400 cells/mm^3, and CPP crystals. Radiography of the right knee (Figure 1) revealed significant chondrocalcinosis. Additional investigations were performed to screen for conditions associated with CPPD disease, which revealed low levels of magnesium (Mg) (0.9 mmol/L) and ionized Mg (0.21 mmol/L; normal range: 0.43-0.61 mmol/L). She had normal ferritin, iron saturation, total iron-binding capacity, parathyroid hormone levels, and negative rheumatoid factor and anti-cyclic citrullinated peptide antibodies. C-reactive protein level was elevated at 5.0 mg/dL (normal: 0.0-0.5 mg/dL). Hypomagnesemia-induced CPPD disease was diagnosed, and the patient was given intra-articular steroid injection followed by oral prednisone 10 mg daily, colchicine 0.6 mg twice daily, and hydroxychloroquine 200 mg twice daily. The joint swelling and pain resolved in 6 weeks, and the patient was maintained on hydroxychloroquine and colchicine. Twenty-four-hour urine studies demonstrated low urinary excretion of potassium and calcium and high urinary excretion of Mg at 4.7% (>2% is significant in the setting of hypomagnesemia), indicating Mg wasting. In the setting of these findings and normal blood pressure, a diagnosis of Gitelman syndrome was made. Besides Mg supplements, the patient was also started on amiloride at 5 mg daily, with the dose eventually titrated to 30 mg daily.

Pathogenesis of CPPD Disease

Calcium pyrophosphate crystal formation is principally dependent on the presence of extracellular inorganic pyrophosphate (PPi) mostly produced in the joints by chondrocytes. Extracellular PPi is primarily produced by two mechanisms:

1. Transportation of the intracellular PPi across the chondrocyte membrane by ankylosing protein, a membrane transporter
2. Degradation of ATP into AMP and PPi by nucleoside triphosphate pyrophosphohydrolase ectoenzymes found on the plasma membranes of chondrocytes and on matrix vesicles found in the cartilage matrix

Various factors have been found to modify the production of extracellular PPi by chondrocytes. One of the strongest stimulators of extracellular PPi is the transforming growth factor-β (TGF-β) (6, 7). Intracellular growth factor-1 (IGF-1) antagonistically inhibits the production of extracellular PPi induced by TGF-β (8, 9).

Cartilage intermediate layer protein, which restricts IGF-1 from inhibiting TGF-β, has been found to increase in the cartilage tissue with increasing age (10). Patients with OA may have increased PPi levels due to the insensitivity of chondrocytes to IGF-1, leading to CPPD deposition in the joints damaged with OA (11). PPi is hydrolyzed to inorganic phosphate by alkaline phosphatase (ALP) and inorganic pyrophosphatases. In hypophosphatasia, a congenital condition due to a mutation in *ALPL*, decreased ALP levels lead to excess accumulation of inorganic pyrophosphatases (12). Excess iron in hemochromatosis acts as an inhibitor of ALP, thus leading to excess inorganic pyrophosphatase. Similarly, Mg acts as a cofactor for ALP enzyme, and its deficiency results in excess accumulation of inorganic pyrophosphatase.

Magnesium Disorders

Several conditions causing hypomagnesemia have been attributed in the literature to cause CPPD disease (Table 3). In Gitelman syndrome, an autosomal recessive disorder, mutations in *SLC12A3*, which encodes for thiazide-sensitive sodium chloride cotransporter in the distal convoluted tubule, lead to urinary Mg wasting, hypomagnesemia, hypokalemia, hypocalciuria, and metabolic alkalosis (13, 14). Patients with Bartter syndrome, an autosomal recessive salt-wasting tubulopathy, characteristically present with hypokalemia, metabolic alkalosis, and secondary hyperaldosteronism with normal to low blood pressure. The Gitelman variant of Bartter syndrome is frequently linked with hypomagnesemia, and it is suggested that most cases of chondrocalcinosis and CPPD disease reported in Bartter syndrome most likely had the Gitelman variant rather than Bartter syndrome per se (15, 16). Hypomagnesemia occurs in short bowel syndrome (SBS) through various mechanisms. Bacterial fermentation of carbohydrates and high intake of dietary fat can increase the concentration of fatty acids that bind Mg and prevent its absorption in the gut (17). SBS itself can cause diarrhea, but sometimes Mg supplementation increases intestinal motility, leading to further diarrhea, which in turn increases the loss of water and electrolytes and leads to secondary hyperaldosteronism and further renal Mg wasting (18). Familial hypomagnesemia with hypercalciuria and nephrocalcinosis, a rare autosomal recessive disorder, is characterized by severe renal Mg and calcium wasting (19). Acute CPP arthritis due to hypomagnesemia has also been described in patients with liver transplant, in whom it has been suspected to occur from a combination of Mg depletion before transplant and tacrolimus use, which induces renal Mg leakage (20). The relationship between hyperparathyroidism and hypomagnesemia is complex. Hyperparathyroidism is an independent risk factor for CPPD disease. However, extreme hypomagnesemia immediately after parathyroidectomy and precipitation of acute CPP arthritis in this setting have also been described (21). It has been proposed that hypomagnesemia in the period after parathyroidectomy for hyperparathyroidism is related to the hungry bone syndrome and Mg being driven from the extracellular to the intracellular space (22).

Table 3. Major disorders described in the literature that cause hypomagnesemia with the presence of CPPD disease

1.	Gitelman syndrome
2.	Bartter syndrome
3.	Short bowel syndrome
4.	Renal dysfunction leading to magnesium wasting
5.	Familial hypomagnesemia with hypercalciuria and nephrocalcinosis
6.	Liver transplant for chronic liver disease
7.	Thiazide diuretics-induced hypomagnesemia

CPPD: calcium pyrophosphate deposition

Management

Treatment of the underlying disorder, correction of hypomagnesemia, and treatment of joint inflammation (with NSAIDs, prednisone or colchicine) were simultaneously used in most of the patients described. Both Bartter and Gitelman syndrome are treated with medications that block the distal tubule sodium-potassium exchange. In addition, NSAIDs are also administered for Bartter syndrome, in which renal prostaglandin E2 (PGE2) is often markedly increased. Potassium and Mg replacement should be performed for both Gitelman and Bartter syndromes. In a 6-month duration dou-

ble-blinded placebo-controlled trial, Doherty et al. (23) reported improvement in symptoms of CPPD in patients treated with Mg.

The main reason for hypomagnesemia in SBS is primarily malabsorption due to various factors previously described (18, 24). The mainstay of therapy therefore constitutes replacement therapy, often requiring parenteral supplementation in severe cases, followed by long-term oral supplementation. In patients with diuretic-induced hypomagnesemia, apart from replacing Mg, switching to a different form of therapy or diuretic should be considered. In patients with hyperparathyroidism, undergoing parathyroidectomy treatment with parenteral Mg replacements to prevent acute CPP arthritis in the post-surgery period should be attempted (22, 25).

Besides the treatment of the underlying disorder causing hypomagnesemia and appropriate Mg replacement, the treatment of inflammatory arthritis should be pursued as in any other crystal arthritides. Acute CPP arthritis can be treated with intra-articular or oral steroids, colchicine, or NSAIDs. Treatment of chronic CPP arthritis can be challenging and mainly consists of periodic intra-articular steroid injections for mono or oligoarthritis, colchicine 0.6-1.2 mg daily, daily NSAIDs, low-dose oral prednisone, or any combination of these. Treatment with interleukin-1β inhibitors, albeit very expensive, is also an option in the management of CPP arthritis (26). Hydroxychloroquine has been described in chronic CPP arthritis to have some benefits (27). The efficacy of methotrexate in chronic CPP arthritis is controversial; however, it is a reasonable option in patients who fail the standard treatment options (28, 29).

Conclusion

Hypomagnesemia is a rare but treatable cause of CPPD disease. Magnesium levels should be measured as part of diagnostic work-up of CPPD disease, especially in younger patients.

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