Calcium Pyrophosphate Dihydrate Crystal Deposition: A Clinical and Laboratory Analysis of 91 Thai Patients

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Abstract

The clinical features and laboratory findings of 91 Thai patients (33 males and 58 females) with CPPD crystal deposition disease were studied. Their average age was 71.54 years. Acute monoarthritis and oligoarthritis were the two most common forms of presentation and were seen in 89 per cent of cases. The knee, wrist and ankle were the three most common joints involved. Associated diseases were common and included hypertension (30 cases), renal insufficiency (23 cases), chronic obstructive pulmonary disease (17 cases), coronary heart disease (13 cases) and diabetes mellitus (12 cases). Eleven patients had malignancies. Five patients had concomitant gout and CPPD crystal deposition disease. The knee and the wrist were the two most common sites of chondrocalcinosis. Of 67 patients who had thyroid function tested, 2 had hyperthyroidism and 5 had hypothyroidism. Hypomagnesemia was seen in 19 per cent. None had hypercalcemia, hypophosphatasia, hemochromatosis or hyperparathyroidism. In contrast to the western series, acute arthritis in our series responded well to oral colchicine alone.

Key word : Crystal-induced Arthropathy, Calcium Pyrophosphate Dihydrate (CPPD), Pseudogout, Acute Arthritis, Clinical and Laboratory Analysis

The clinical syndromes associated with the deposition of calcium pyrophosphate dihydrate (CPPD) crystals have received increasing attention during the past few years. In 1962, McCarty et al(1) first described an association of CPPD crystals in synovial fluid (SF) or articular cartilage in patients who presented with acute arthritis, and suggested the name "pseudo-gout", since the clinical manifestations of this syndrome frequently simulated gout. Subsequent reports have shown that this crystal-induced arthropathy can have wide clinical presentations including those asymptomatic, or associated

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with acute and chronic arthropathy. They mimick many well defined rheumatic diseases such as gout, rheumatoid arthritis, osteoarthritis, neuropathic arthropathy and ankylosing spondylitis[2]. Tophaceous deposit similar to that seen in gout has also been described[3,4]. CPPD crystal deposition disease might have been the second most common form of crystal-induced synovitis besides gout.

Although the clinical features of CPPD deposition disease in western countries have been well recognized, they have been rarely described in Southeast Asia[1,5-12]. Only one study of this crystal-induced arthropathy has been described in Thailand[13]. In this study we report the clinical and laboratory findings in Thai patients with CPPD crystal deposition disease seen in a university hospital over an 8-year period.

MATERIAL AND METHOD

A retrospective chart review was carried out for in- and out-patients with the diagnosis of CPPD crystal deposition disease and seen between July 1, 1990 and September 31, 1998 at the Division of Rheumatology, Department of Medicine, Faculty of Medicine, Chiang Mai University. The diagnosis of CPPD crystal deposition disease included the presence of linear or punctate calcific deposit within hyaline cartilage or fibrocartilage in bone and joint radiographs and the presence of typical monoclinic or triclinic weakly birefringence with positive elongation crystals in SF[14].

Full history and physical examination were done in all patients, with special attention paid to the musculoskeletal system, history of arthritis, and any past or present medical or surgical illness. Endocrine or metabolic diseases associated with CPPD crystal deposition disease were identified. The number, site and duration of arthritis were recorded. Appropriate radiographs of the clinically involved joints were taken for evidence of chondrocalcinosis. A routine blood count, urine analysis and blood chemistries including blood sugar, serum uric acid, calcium, phosphorus, magnesium, renal and liver functions were taken as indicated clinically. SF was performed promptly after arthrocenthesis and conventional compensated polarized light microscopy was used to identify the crystals. Thyroid hormones were determined in cases with a suspicion of possible thyroid disease. Parathyroid hormone assay was not available at our institution.

The clinical patterns of CPPD crystal deposition disease followed McCarty's description[2]. The outcome of treatment was classified as good, fair and poor depending on the degree of arthritis improved within 48 hours after being treated in more than 50, 30-50, and less than 30 per cent respectively.

RESULTS

During the study period, 102 patients (38 males and 64 females) presented with peripheral arthritis and had CPPD crystals in their SFs. Ninety-one of these patients (33 males and 58 females) had typical chondrocalcinosis in their bone and joint radiographs and met the diagnostic criteria for CPPD crystal deposition disease. These 91 patients were included in this analysis. Their mean $\pm$ SD age was 71.5 $\pm$ 9.1 years (range 35-97 years). There was no statistical difference in mean age between male and female patients. Twenty-five patients had arthritis for the first time with a mean duration of arthritis of 4.8 $\pm$ 6.4 days. Sixty-six patients had recurrent arthritis, with their mean duration of disease and duration of the current episode being 4.3 $\pm$ 2.9 years and 4.5 $\pm$ 4.9 days respectively. Forty-seven patients had fever during their attack of arthritis.

The clinical arthritic patterns of the patients studied are shown in Table 1. Monoarthritis and oligoarthritis were the two most common forms of presentation seen in patients with both their first attack and recurrent attack, which occurred in 89 per cent of cases. Pseudo-gout was the most prevalent form of acute arthritis seen in patients with their first attack, while pseudo-gout and pseudo-osteoarthritis (pseudo-OA) with acute exacerbation were commonly seen in patients with a recurrent attack.

Table 2 shows the location of the joints involved in patients with their first attack and those with a recurrent attack. The knee, ankle and wrist were the three most commonly affected joints in both groups. Acute arthritis of the first metatarsophalangeal joint, or podagra, which mimicked gouty arthritis, could be seen during the first and recurrent attack, but this was not usual. Involvement of the small joints of the hands was usually seen in patients with pseudo-rheumatoid arthritis (pseudo-RA).

Provocative factors and associated diseases seen in this group of patients are shown in Table 3. Medical illness, especially infections, was the most common precipitating cause, followed by ischemic

heart disease and cerebrovascular accident. The arthritis developed after surgical procedure in 3 cases, which were aneurysmectomy of abdominal aortic aneurysm, closure of peptic ulcer perforation and corneal surgery in each one. Three patients developed acute arthritis after trauma. Hypertension, renal insufficiency, chronic obstructive pulmonary disease, ischemic heart disease, diabetes mellitus and

Table 1. Type and clinical patterns of arthritis.

	First attack (n = 25)	Recurrent attack (n = 66)	Total (n = 91)
Type of arthritis			
Monoarthritis	12	31	43
Oligoarthritis	12	26	38
Polyarthritis	1	9	10
Clinical patterns of arthritis			
Pseudo-gout	22	29	51
Pseudo-OA with acute flare	1	30	31
Pseudo-RA	2	7	9

Table 2. Location of the joint involved.

Joints	First attack (n = 25)	Recurrent attack (n = 66)	
		Current attack	Previous attack
Shoulder	1	5	5
Elbow	3	11	12
Wrist	9	14	11
Metacarpophalangeal	1	7	8
Proximal interphalangeal	0	5	4
Hip	0	0	1
Knee	17	60	65
Ankle	7	17	23
First metatarsophalangeal	2	6	8
Metatarsophalangeal	1	4	4

Table 3. Provocative factors and associated diseases.

Provocative disease	No. of patients	Associated conditions*	No. of patients
Medical illness		Hypertension	30
Infection	30	Renal insufficiency	23
Gastrointestinal	9	COPD	17
Genitourinary	8	CAHD	13
Pulmonary	8	Diabetes mellitus	12
Soft tissue and skin	5	Cancer	11
CAHD	15	Hyperlipidemia	9
CVA	9	Goiter	7
Upper GI hemorrhage	6	Gout	5
Others	3	CVA	4
Surgery	3	Cirrhosis	3
Trauma	3	Others	7

* each patient might have more than one disease; CAHD = coronary heart disease; CVA= cerebrovascular accident; COPD = chronic obstructive pulmonary disease

hyperlipidemia were common associated diseases. Eleven patients had malignancies. Except for carcinoma of the lung, which was seen in 2 cases, there were individual cases of carcinoma of the ovary, uterus, cervix, lip, larynx, nasopharynx, colon, liver and lymphoma. Five patients had co-existing gout. Other associated diseases included HIV infection (2 cases), and individual cases of thalassemia, epilepsy, Parkinson's disease, myasthenia gravis and myoclonus.

Sixty-five of 91 patients had enough SFs for complete analysis. One of these SFs was serosanguinous. The mean SF white blood count was 29,827 ± 29,757 cell/mm^3, with a mean per cent of polymorphonuclear cells and mononuclear cells of 83.0 ± 12.0 and 17.0 ± 11.6 per cent, respectively. Five SFs also contained monosodium urate crystals. There was no significant difference in mean WBC and differential count between patients with acute pseudo-gout, pseudo-OA with acute exacerbation and pseudo-RA.

Radiograph of the knee was done in all patients and chondrocalcinosis was found in 87 (95.6%). The calcification was frequently seen bilaterally in the fibrocartilage (Fig. 1). Calcification in the fibrocartilage of the wrist joint was not uncommon. Chondrocalcinosis at the pubic symphysis, shoulder, hip, ankle and metacarpophalangeal joint was occasionally seen. As bone and joint radiographs were not done on joints other than the knee joint in every patient, the actual incidence of chondrocalcinosis of those joints was not determinable.

Twenty-one patients were anemic (hematocrit less than 30 vol%). One was due to thalassemia. In 5 cases, the anemia was related to severe chronic renal failure. Chronic diseases and gastrointestinal loss contributed to anemia in the remaining patients. Other laboratory investigation results are shown in Table 4. Twenty-six of 60 patients who had blood sugar determination had fasting blood sugar of more than 110 mg/dL; 12 of these patients had diabetes mellitus. Twenty-three of 83 patients who had renal function determination had renal insufficiency (BUN > 24 mg/dL); 5 of these patients had severe chronic renal failure. Six patients had renal calculi. Sixteen of 51 patients who had serum uric acid determination were hyperuricemic; none had taken drugs that significantly increase the serum uric acid level. Five of these hyperuricemic patients also had gout, proven by the presence of monosodium urate crystals in their SFs. These 5 patients had a mean serum uric acid of 10.1 ± 1.8 mg/dL; 3 of these 5 patients had a mild degree of renal insufficiency. Two patients had hypercalcemia with serum calcium of 10.6 and 10.8 mg/dL, respectively; none had clinical and radiological evidence of hyperparathyoroidsm. Hypomagnesemia was seen in 14 of 73 cases that had been tested. Thyroid functions were determined in 67 patients and the results were euthyroid, hyperthyroid and hypothyroid in 60, 2 and 5 cases, respectively. All hypothyroid patients were subclinical. All patients with pseudo-RA had negative tests for rheumatoid factor.

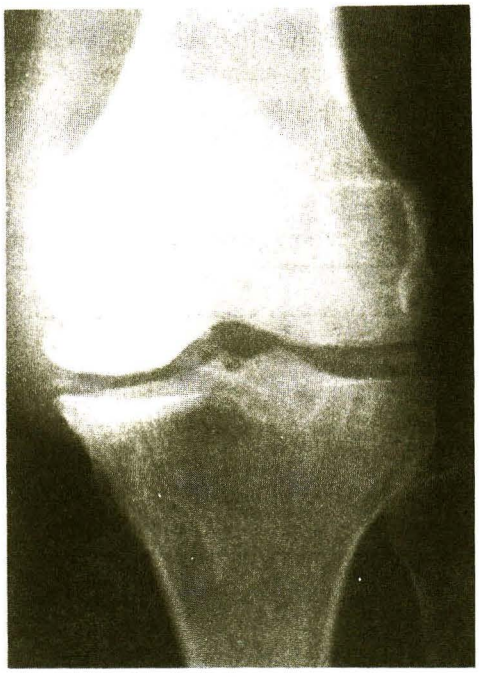

Fig. 1. Radiograph of the knee showing calcific density in both menisci which is typical of chondrocalcinosis.

Thorough joint aspirations were done daily in all peripheral joints effusions whenever possible. Treatment and outcome of the arthritis are shown in Table 5. Oral colchicine, at the dosage of 0.6-1.8 mg/day, was the main drug used in this study. A majority of patients showed a good response within 48-72 hours after therapy. Two patients who showed a poor response further improved when non-steroidal anti-inflammatory drugs (NSAIDs) were added to the treatment. Five patients received NSAIDs at a therapeutic dosage, and 4 showed a good response.

Table 4. Results of laboratory investigations.

Tests [N] (normal value)	Mean ± SD	No. with abnormal tests (n1,n2)
Fasting blood sugar [60] (70-110 mg/dL)	110.46 ± 34.11	3,26
Blood urea nitrogen [83] (7-24 mg/dL)	21.56 ± 11.23	2,23
Creatinine [81] (0.6-1.5 mg/dL)	1.37 ± 0.86	0,21
Uric acid [51] (3.0-7.0 mg/dL)	5.96 ± 3.10	9,16
Calcium [82] (7.0-10.5 mg/dL)	8.50 ± 0.84	1,2
Phosphorus [82] (2.5-4.5 mg/dL)	3.72 ± 1.41	6,10
Magnesium [73] (1.5-2.2 mg/dL)	1.72 ± 0.35	14,5
Alkaline phosphatase [71] (21-128 U/L)	97.64 ± 63.51	0,12

N = number being tested; n1= number with value less than normal; n2 = number with value greater than normal

Table 5. Outcome of treatment.

Treatment (No. of patients)	Outcome		
	Good	Fair	Poor
Colchicine (76)	62	12	2
Colchicine + NSAIDs (5)	4	1	-
NSAIDs (9)	6	3	-
Acetaminophen (1)	-	1	-

None of these patients developed serious gastrointestinal or renal side effects. Acetaminophen was used in one patient with pseudo-OA who had mild symptoms. The outcome in this study was only short term, as a majority of the patients failed to follow-up after being discharged from hospital or once the arthritis had disappeared.

DISCUSSION

In this study, we found that the CPPD crystal deposition disease occurred mainly in elderly patients with the mean of 70 years. Both acute and subacute arthritis were the two most common forms of presentation. The knee was the most commonly affected joint as well as having chondrocalcinosis in the radiographs. These findings were similar to those that have been previously described[1,5-13]. Females were predominant in this study and some of the others[6-8,12,13], while males were found in the others[5,9,10,15]. Eleven patients who did not have radiographic evidence of chondrocalcinosis were excluded, as they did not meet the criteria for the diagnosis of CPPD crystal deposition disease described by Ryan[14]. However, cases of CPPD crystal deposition disease without radiographic chondrocalcinosis have been described[12,16,17].

Pseudo-gout and pseudo-OA with acute exacerbation, in which monoarthritis and oligoarthritis were the common form of presentation and seen mainly in patients with both first attack and recurrent attack in this study. Although 9 patients presented with pseudo-RA, none had typical radiographic changes of rheumatoid arthritis or a positive rheumatoid factor test. Moreover, the arthritis disappeared shortly after treatment with colchicine or NSAIDs. This made the diagnosis of concomitant rheumatoid arthritis and CPPD crystal deposition disease less likely in the patients. Concomitant CPPD crystal deposition disease and rheumatoid arthritis have been reported with an incidence of 1-25 per cent[2,8,10,12]. No case of pseudo-OA, pseudo-neuropathic, or pseudo-ankylosing spondylitis was found in this study. Patients with osteoarthritis (pseudo-OA), chronic low back pain, or a milder degree of acute arthritis were usually taken care of by orthopedists in our institution. This should be kept in mind when interpreting the clinical features of CPPD crystal deposition disease of this study.

Provocative factors as well as associated diseases were commonly found. Acute arthritis of CPPD crystal deposition disease after acute medical and surgical illness is well recognized[9]. In this study, medical compared to surgical illness was much more common. This might be because these patients were elderly and had underlying problems or association with many medical conditions; making it necessary for them to be consulted or admitted to the medical service when they presented with active arthritis. Although many endocrine and metabolic diseases including diabetes mellitus, hypertension, hyperlipidemia, hemochromatosis, hypothyroidism, hyperparathyroidism, hypercalcemia and hypomagnesemia have been well recognized as associated diseases[18]; they did not differ significantly from those of osteoarthritis controls in some studies[10,15]. Severe hypothyroidism has been reported to be associated with CPPD crystal deposition disease[19], but recent studies failed to confirm this association[20,21]. Despite hyperparathyroidism has been found in patients with CPPD crystal deposition disease with an incidence of 5.5-10.7 per cent[9,10]; it was not found in this study or others[6-8,11-13], which suggested that the association of these 2 diseases was uncommon. It should be noted that 19 per cent of our patients had hypomagnesemia. Magnesium is a co-factor of alkaline phosphatase, which hydrolyses inorganic pyrophosphate to inorganic phosphate thus preventing crystallization of the CPPD crystal[18]. Unfortunately, the cause of hypomagnesemia was not determined in this study.

Five of our patients (5.5 per cent) had coexisting gout and CPPD crystal deposition disease. The concomitance of these 2 diseases was reported to be 1.9-38.9 per cent[6,7,10,12]. Hyperuricemia was found in 11.1-31.6 per cent of patients with CPPD crystal deposition disease[6,11,15]. The presence of these 2 crystals has been suggested for possible underlying hyperparathyroidism[6]. None of these 5 patients had clinical features of hyperparathyroidism. The association of these 2 crystal diseases in our patients was probably a co-incidental finding in elderly patients.

Twenty-five per cent of our patients had some degree of renal insufficiency. The causes of renal failure in these patients were not clear. Hypertension, diabetes mellitus, and chronic use of NSAIDs for arthritis might have contributed. The high incidence of chronic obstructive pulmonary disease might have been related to the smoking habits of elderly northern Thais. Eleven patients had malignancies. Again, this might be related to the advanced age of this group of patients and the tertiary care center of our institution, rather than that a direct causal relationship between malignancies and the CPPD crystal deposition.

SF analysis in these patients showed an inflammatory type. One patient had serosanguinous SF. Hemorrhagic SF has also been described in patients with CPPD crystals deposition disease[22]. SF white blood cell count was reported to be higher during the first 2 days of acute arthritis than by the end of the first week[7].

Treatment of acute arthritis in CPPD crystal deposition has included thorough aspiration, NSAIDs, intra-articular corticosteroids, and colchicine[1,6,9,12,13,23-25]. Intra-articular corticosteroids has been shown to be very effective. Intravenous colchicine has shown promising results[23-25], but oral colchicine has given inconsistent results [1,6,12,13]. A majority of our patients responded well to oral colchicine alone. This differed from the western series. Intravenous colchicine is not available in Thailand. In this study, colchicine was prefered to NSAIDs because a majority of the patients were elderly and had underlying medical diseases, and there was a risk of developing side effects from NSAIDs therapy. None of our patients developed serious gastrointestinal side effects from either colchicine or NSAIDs.

In conclusion, the clinical features and laboratory findings of Thai patients with CPPD crystal deposition disease were similar to those in western countries. However, the good response to oral colchicine alone in this study clearly differed from them. A significant number of patients were found to have hypomagnesemia. Further studies of the causes of hypomagnesemia in these patients will be of interest.

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โรคข้อจากผลึกเกลือแคลเซี่ยม ไพโรฟอสเฟต ไดฮัยเดรท: การศึกษาลักษณะทางคลินิกและห้องปฏิบัติการในผู้ป่วยไทย 91 ราย

วรวิทย์ เลาห์เรณู, พ.บ., วราพร สุขิตาวุธ, วท.บ.**

ได้ทำการศึกษาลักษณะทางคลินิกและห้องปฏิบัติการในผู้ป่วยไทยที่เป็นโรคข้ออักเสบจากผลึกเกลือแคลเซี่ยม ไพโรฟอสเฟต ไดฮัยเดรทจำนวน 91 ราย เป็นเพศชาย 33 รายและเพศหญิง 58 ราย อายุเฉลี่ยเท่ากับ 71.54 ปี ข้ออักเสบเฉียบพลันชนิดข้อเดี่ยวหรือ 2-3 ข้อเป็นอาการนำที่พบได้บ่อยที่สุดโดยพบได้สูงถึงร้อยละ 89 ข้อเข่า ข้อมือและข้อเท้าเป็นข้อที่มีการอักเสบบ่อยที่สุด โรคร่วมที่พบได้บ่อยได้แก่ โรคความดันโลหิตสูง (30 ราย), ไตทำงานบกพร่อง (23 ราย), โรคปอดอุดกั้นเรื้อรัง (17 ราย), โรคเส้นเลือดหัวใจอุดตัน (13 ราย) และโรคเบาหวาน (12 ราย) ผู้ป่วย 11 รายเป็นมะเร็งร่วม พบผู้ป่วย 5 รายที่เป็นโรคเก๊าท์ร่วมจากการตรวจพบผลึกเกลือยูเรตในน้ำไขข้อ ข้อเข่าและข้อมือเป็นข้อที่ตรวจพบหินปูนจับบริเวณกระดูกอ่อนที่พบได้บ่อยทางภาพรังสี จากการตรวจการทำงานของต่อมไทรอยด์ในผู้ป่วย 67 ราย พบผู้ป่วย hyperthyroidism 2 ราย และ hypothyroidism 5 ราย พบภาวะแมกนีเซียมในเลือดต่ำร้อยละ 19 แต่ไม่พบผู้ป่วยที่มีภาวะแคลเซียมในเลือดสูง, hypophosphatasia, hyperparathyroidism หรือเป็นโรค hemochromatosis ข้ออักเสบในผู้ป่วยส่วนใหญ่ตอบสนองดีต่อยาโคลชิซินชนิดรับประทาน ซึ่งผลการรักษานี้ต่างไปจากรายงานจากประเทศทางตะวันตก

คำสำคัญ : โรคข้อจากผลึกเกลือ, แคลเซี่ยม ไพโรฟอสเฟต ไดฮัยเดรท, เก๊าท์เทียม, ข้ออักเสบเฉียบพลัน, การวิเคราะห์ทางคลินิกและห้องปฏิบัติการ

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