A Clinical Study of Crystal-Proven Gouty Arthritis in a University Hospital†

WORAWIT LOUTHRENOO, MD*,
WARAPORN SUKITAWUT, BSc*,
NUNTANA KASITANON, MD*,
RAMJAI WICHAINUN, BSc*

Abstract

The clinical features of 567 patients with crystal proven gout (489 males, 78 females) seen in a University Hospital in northern Thailand was reviewed. The mean age at onset and mean duration of disease was 60.0 ± 11.7 years and 5.2 ± 4.8 years, respectively. Recurrent attacks accounted for 94 per cent. The knee and ankle were the 2 most common joints affected during the first attack and each one was seen in 55.6 per cent of cases. During a recurrent attack, the ankle, knee and first metatarsophalangeal joint were the 3 most common joints affected and were seen in 94.5 per cent, 81.2 per cent and 80.2 per cent of cases, respectively. Thirty-six per cent of the patients had tophi. Hypertension, hyperlipidemia, diabetes mellitus and ischemic heart disease were commonly associated diseases. Thirty-five per cent had renal calculi, and fifty-four per cent had renal insufficiency. Of 59 patients who tested with normal renal function, twelve per cent were hyperexcretor. The clinical features of gout seen in the university hospital in northern Thailand were similar to those reported in Bangkok, but with a higher incidence of tophaceous gout, renal failure and renal calculi.

Key word : Gout, Hyperuricemia, Uric Acid, Treatment, Outcome, Monosodium Urate, Crystal-Induced Arthritis

LOUTHRENOO W, KASITANON N, SUKITAWUT W, WICHAINUN R
J Med Assoc Thai 2003; 86: 868-875

* Division of Rheumatology, Department of Medicine, Faculty of Medicine, Chiang Mai University, Chiang Mai 50200, Thailand.

† Present in part at the 10th Asia Pacific League of Association for Rheumatology Meeting, 1-6 December 2002, Bangkok, Thailand.

Gout is a disease caused by deposition of monosodium urate (MSU) crystals in the tissues of the body, especially, in and around the joint, and kidney. It is a consequence of chronic or sustained hyperuricemia, a biochemical disorder of purine metabolism. The disease has a worldwide distribution, and might be the most common cause of acute arthritis. The clinical features of gout have included males of 40 years or older, intermittent severe arthritis involving the lower extremity joints, subcutaneous tophaceous deposits, and renal diseases (urate nephropathy and uric acid stones). These clinical features have been well described from Western countries[1].

There is evidence that the prevalence and incidence of gout is higher in Filipino, rural Javanese, Malayo-polynesian, and Mongoloid Chinese populations when compared with Caucasians[2,3]. Many studies in Asia have shown that complicated gout, e.g. cases with tophi or renal insufficiency, are not uncommon. Tophaceous gout was found in 15-40 per cent of cases in studies from Indonesia and Singapore[4-6]. Renal stone was found in 42 per cent, 25 per cent and 11 per cent of cases in studies from Malaysia[7], north Sulawesi, Indonesia[8], and Singapore[4], respectively.

In Thailand, the prevalence of gout was reported to be 0.16 per cent of the general population [9]. Although gout is a very common disease, clinical studies in Thailand are limited[10,11]. The authors, herein, report our experience of crystal proven gout seen in a university hospital over a 16-year period.

MATERIAL AND METHOD

A chart review was carried out for all in- and out-patients with the diagnosis of gout seen at the Division of Rheumatology, Department of Medicine, Faculty of Medicine, Chiang Mai University, between January 1986 and December 2001. Only cases with crystal-proven gout were included in the study. The diagnosis of gout was made when typical MSU crystals (strongly birefringent crystals with negative elongation), identified by compensated polarized light microscopy (Olympus, with 540 nm red compensator), were demonstrated in synovial fluids (SF) of acutely inflamed joints or from a tophus. It is the authors' routine practice to perform arthrocentesis in all patients who present with acute arthritis or have synovial effusions. The SFs were proceeded for cell counts, differential counts, and stained smears for microorganism, and crystal identification. In those whom the MSU crystals were not able to demonstrate in SFs and had a clinical suggestion of gout, a repeated arthrocentesis was performed at the next episode of acute arthritis until the crystals were identified.

A full history, with special attention paid to pain of the musculoskeletal system, past or present medical illnesses, renal calculi, and associated diseases were recorded. History of arthritis, the number of joints involved, site and duration of arthritis were noted. Physical examination included the number and location of the joints involved, and the location and amount of tophi. The amount of tophi was classified as minimal (< 1 cm^3), moderate (1-5 cm^3) and severe (> 5 cm^3). Laboratory investigations including a routine blood count, urine analysis, blood chemistry that included fasting plasma glucose, serum uric acid, renal and liver functions, and serum lipid profiles were noted.

RESULTS

During the study period, there was a total of 3,027 patients, in which 567 (18.7%) had crystal proven gout. There were 489 males and 78 females, giving a male to female ratio of 6.3 : 1. Their mean ± standard deviation (SD) age at onset and mean duration of disease was 60.0 ± 11.7 years and 5.2 ± 4.8 years, respectively. Thirty-six patients (6.3%) had the first attack. Five hundred and thirty-one patients

Table 1. Details of clinical presentation of patients with gouty arthritis.

	First attack (n = 36)	%	Recurrent attack* (n = 526)	%
Monoarthritis	16	44.4	112	21.3
Oligoarthritis	15	41.7	238	45.2
Polyarthritis	5	13.9	176	33.5

* Five patients presented with chronic tophaceous gout without acute arthritis at the time of presentation

Table 2. Location of joint involved.

	First attack (n = 36)	%	Recurrent attack			
			First attack (n = 424)*	%	Past and present attack (n = 531)	%
First metatarsophalangeal	11	30.6	123	29.0	426	80.2
2-5 metatarsophalangeal	3	8.3	9	2.12	284	53.5
Ankle	20	55.6	211	49.8	502	94.5
Knee	20	55.6	129	30.4	431	81.2
Wrist	5	13.9	14	3.3	243	45.8
Metacarpophalangeal	1	2.8	2	0.5	128	24.1
Interphalangeal	2	5.6	7	1.6	76	14.3
Elbow	6	16.7	6	1.4	171	32.2
Shoulder	1	2.8	2	0.5	53	10.0
Sternoclavicular	-	-	-	-	2	0.4

* Number of patients who recalled their first attack

(93.7%) who had recurrent attacks had duration of diseases of 5.2 ± 4.8 years (range 3 months - 38 years). Details of the clinical presentation of acute arthritis and the location of the joint involved in relation to the first or recurrent attacks are shown in Table 1 and 2, respectively. Among those with the first attack, acute mono- and oligoarthritis were common presentations that contributed to 44.4 per cent and 41.4 per cent of the cases, respectively. Those who had recurrent arthritis usually presented with oligo- and polyarthritis, which contributed to 45.2 per cent and 33.5 per cent of the cases, respectively. Those with acute arthritis had had it for 3.9 ± 3.2 days before they were seen by the authors. Among those with a first attack, the knee and ankle were the 2 most common joints affected and each one was presented in 55.6 per cent of the cases. In those with recurrent attacks, the ankle, knee and first metatarsophalangeal (MTP) were the 3 most common joints affected and were seen in 94.5 per cent, 81.2 per cent and 80.2 per cent of cases, respectively. One hundred and five patients (34.7%) had fever at the time of presentation, with a mean body temperature of 38.3 ± 0.5°C. A history of passing urinary tract stone was documented in 139 of 539 cases (25.8%).

Two hundred and seven patients (36.5%) had tophi. The mean duration of tophi in 164 cases, who could be recalled, was 2.6 ±2.4 years (range 0.5-20.0 years), or 5.1 ± 4.7 years after the first episode (range 0.5-28 years). The tophi were minimal, moderate and severe in 117 (56.5%), 64 (30.9) and 26 (12.6%) cases, respectively. Details of tophi locations are shown in Table 3. The malleoli, toes, dorsum of the feet and fingers were the common locations of tophaceous deposits. Deposit at the pinna was seen in 10 cases (4.8%). Miliary deposit of the skin was seen in 2 cases (1.0%). The mean serum uric acid of the patients studied was 9.0 ± 2.4 mg/dL. The mean ± SD serum uric acid in those who had normal renal function (serum creatinine < 1.6 mg/dL) was significantly lower than in those with impaired renal function (serum creatinine ≥ 1.6 mg/dL) (9.0 ± 2.3 *vs* 10.4 ± 2.8, $p < 0.01$). There was no significant difference in mean serum uric acid between male and female patients (9.0 ± 2.3 *vs* 8.9 ± 2.5 mg/dL, $p = 0.8$). There was a significant correlation between the amount of tophi and serum uric acid ($r^2 = 0.2$, $p < 0.01$) and the duration of the disease ($r^2 = 0.4$, $p < 0.01$). A mean ± SD of a 24 hour urine uric acid determined in 59 cases with normal renal function was 434.73 ± 255.5 mg, in which 52 (88.1%) patients were hypoexcretor (< 700 mg/day) and 7 (11.9%) hyperexcretor (≥ 700 mg/day).

Associated diseases with gout are shown in Table 4. Hypertension, hyperlipidemia, ischemic heart disease and diabetes mellitus were commonly associated diseases and seen in 54.3 per cent, 33.5 per cent, 14.5 and 12.9 per cent of cases, respectively. Other diseases with a frequency of less than 10 cases (not shown in table), included choledocholithiasis in 9, Guillan Barre syndrome in 8, aortic aneurysm in 5, Parkinsonism in 3, leprosy in 3, HIV infection in 3, epilepsy in 2, chronic hepatitis in 2, renal transplantation in 2, systemic lupus erythematosus in 2, mylodysplastic syndrome in 2, and one each in hypothy-

Table 3. Location of tophi.

Location	No. of cases (n = 207)	%
First MTP and toes	86	41.5
Dorsum of feet	71	34.3
Malleoli	95	45.9
Knees	26	12.6
Elbows	60	29.0
Wrists	44	21.3
Fingers	71	34.3
Pinna	10	4.8
Achilles tendon	15	7.2
Shoulder	1	0.5
Miliary deposit of the skin	2	1.0

Table 4. Associated diseases.

	N = 567	%
Hypertension	308	54.3
Hyperlipidemia	190	33.5
Diabetes mellitus	73	12.9
Ischemic heart disease	82	14.5
Chronic obstructive pulmonary disease	65	11.5
Cerebrovascular disease	46	8.1
Congestive heart failure	43	7.6
Tuberculosis	31	5.5
Malignancies	23	4.1
Thalassemia	15	2.6
Rheumatic heart disease	13	2.3
Cirrhosis	12	2.1

Table 5. Provocative factors for acute attack.

	N = 567	%
Medical illness		
Infection	94	15.8
Upper gastrointestinal hemorrhage	63	11.1
Cerebrovascular disease	26	4.6
Ischemic heart disease/myocardial infarction	21	3.7
Congestive heart failure	16	2.8
Hepatic encephalopathy	10	1.8
Surgical illness		
Eye surgery	15	2.6
Abdominal surgery	7	1.2
Kidney surgery	5	0.9
Cardiac surgery/catheterization	3	0.5
Spinal surgery	1	0.2
Consumption of alcohol	125	22.0
High purine diet	90	15.9

toidism, liver abscess, psoriasis, systemic sclerosis, immune hemolytic anemia, and factor VIII inhibitor. Among 23 cases with malignancies, there were carcinoma of the lung in 6, hepatoma in 3, leukemia in 3, carcinoma of the colon in 2, carcinoma of the larynx in 2, carcinoma of the penis in 2, and one in each of the stomach, pancreas, tongue, multiple myeloma and non Hodgkin's lymphoma. Thirty six patients (6.3%) presented with Cushing syndrome secondary to corticosteroid abuse.

Provocative factors for acute attack are shown in Table 5. Acute arthritis occurred after medical and surgical illness in 268 (47.3%) and 31 (5.5%) cases, respectively, with the mean duration of an acute attack of arthritis of 5.9 ± 4.4 and 6.9 ± 5.1 days after the onset of medical and surgical illnesses, respectively. Acute attacks were also frequent after the consumption of alcohol and high purine meals. Among the medical illnesses, infection was the most common provocative factor, which was seen in 94 cases (15.8%). The infections were urinary tract infection in 29, pneumonia and/or pleural effusion in 37, infectious diarrhea in 11, sepsis in 6, cellulitis or infected tophi in 8, and meningitis in 3. Other uncommon medical illnesses that provoked the attacks were seizures in 5, Guillian-Barre syndrome in 2, and electrolyte imbalance in 5 (not shown in table).

SF analysis was carried out in 555 patients, with mean SF cell counts of 29,104 ± 37,659 cells/mm^3. All of these SFs contained MSU crystals. Five SFs also contained calcium pyrophosphate dihydrate (CPPD) crystals. These five patients had typical chon-

drocalcinosis in the radiographs. The remaining 12 patients, whose SF analysis was not carried out had MSU crystals in their tophi.

One hundred and sixty-four patients were anemic (hematocrit < 30.0 vol%), of which 101 had renal insufficiency (creatinine ≥ 1.6 mg/dL). One hundred and seventeen of 339 cases (34.5%), who had plain radiograph of the kidney performed, showed opaque stones. Among 139 patients who had a history of passing urinary stones, 100 cases had opaque stone (71.9%) in the plain radiographs. Three hundred and four patients (53.6%) had renal insufficiency (serum creatinine ≥ 1.6 mg/dL) (mean ± SD serum creatinine of 3.8 ± 3.4 mg/dL) (range 1.6 - 22.0 mg/dL).

Details of the treatment and outcome are shown in Table 6. Colchicine was the most common form of monotherapy. Three hundred and twenty-two patients who were treated with colchicine alone showed a good response. Only 17 cases (4.2%) had diarrhea. A good response was also seen in those who received nonsteroidal antiinflammatory drugs (NSAIDs), corticosteroids, or a combination therapy.

DISCUSSION

In the present study, the incidence of crystal proven gout in Chiang Mai University Hospital was 18.7 per cent, indicating that gout was a common disease.

The clinical features of gout in the present series shared many similarities to those of Western countries[1,12,13]. There was a male preponderance; severe acute monoarthritis, predominantly in the lower extremity joints; and an association with many medical illnesses including hypertension, dyslipidemia, and diabetes mellitus. However, there were several clinical features of patients in the present study that differed from those of Western countries. The male to female ratio of 6.3 : 1 was lower than the 20 : 1 ratio in Western countries[13,14]. The mean ± SD age at onset of 60 years old was higher than that in western countries, which was usually lower than 50 years[1, 13-15]. Moreover, mon- and oligoarthritis presented in the first attack, and involving the ankle and knee, which differed from Western countries where monoarthritis involved the first MTP joint[1,12,13]. Interestingly, the mean ± SD duration of 5.1 ± 4.7 years between the first attack and presence of tophi in the presented patients was shorter than in Western countries, where tophaceous deposits usually occurred more than 10 years after the onset of gout[1,12,16]. Surprisingly, a report from Japan showed that only 9.2 per cent of patients had tophaceous deposits with a follow-up duration of 19 years[17].

Almost half of the patients (47.3%) had an acute attack after medical illnesses, while acute attacks occurred after surgical illnesses in only 5.5 per cent of cases. Acute gouty attack after medical and surgical illnesses was reported to be 20.3 per cent and 8.3 per cent, respectively[18]. Infection and upper gastrointestinal hemorrhage were the 2 most common provocative medical illnesses. The reason for infection contributing to an acute attack was not clear. However, it might be related to changes in serum electrolytes and serum uric acid level, which provoked acute attack. Upper gastrointestinal hemorrhage was usually caused by the complication of NSAIDs therapy. The rather low number of acute attacks after surgical illnesses might be because the present study was performed in the medical department, and cases with acute arthritis that developed in the surgical departments were not consulted to the authors. Consumption of alcohol and a high purine diet were also common provocative factors for an acute attack. Surprisingly, although infection was the most common provocative medical illness, no case of concomitant bacterial arthritis and gout was noted. Only one case of

Table 6. Treatment of gouty arthritis and outcome (n = 562).*

Drugs (N)	Good	%	Fair	%	Poor	%
Colchicine (404)	322	79.7	78	19.3	4	1.0
Nonsteroidal antiinflammatory drugs (NSAIDs) (43)	42	97.6	1	2.3	-	-
Corticosteroid (18)	16	88.9	2	11.1	-	-
Combination (97)						-
Colchicine + NSAIDs (41)	40	97.6	1	2.4	-	-
Colchicine + corticosteroids (53)	48	90.6	5	9.4	-	-
NSAIDs + corticosteroids (3)	3	100.0	-	-	-	-

*Five patients presented with chronic tophaceous gout without acute arthritis at the time of presentation

tuberculous arthritis complicating gout was found and had already been reported[19].

Although renal disease is common in gout, it usually does not contribute to renal failure. Renal insufficiency in gout is usually associated with the presence of hypertension, ischemic heart disease, pre-existing renal disease and a very high serum uric acid level (> 13 mg/dL in men and > 10 mg/dL in women) [20-22]. It should be noted that up to 54 per cent of the presented patients had renal insufficiency. The causes of renal insufficiency in these patients were not clear. Inappropriate use of NSAIDs during an acute attack and failure to control recurrent arthritis with hypouricemic agents, rendered these patients to the development of recurrent attacks and the frequent use of NSAIDs was the most likely contributing factor. Co-morbidity such as hypertension and diabetes mellitus could be another contributing factor. Lastly, the high incidence of urinary calculi seen in the presented population (34%) could be further contributing factor. Renal calculi have been found in 22 per cent of patients with gout in a Western series[23]. A very high incidence of renal calculi (42%) was reported from Malaysia[7]. The reason for the high incidence of renal calculi in the presented population was not clearly understood. Renal calculi and chronic renal failure is a major problem in the northern part of Thailand. Twelve per cent of 59 patients with normal renal function who had a 24-hour urine uric acid determination were hyperexcretor. This was the same percentage as in the Western countries[1]. Therefore, it was unlikely that hyperexcretor could contribute to the development of renal calculi in the presented patients.

The majority of the presented patients was old, and had multiple medical illnesses, especially renal insufficiency, therefore, colchicine was the most common antiinflammatory drug used during an acute attack. It should be noted that 80 per cent of the patients showed a good response to the treatment, and only 4.2 per cent developed diarrhea. The low incidence of diarrhea might be because the authors did not use colchicine in the way it was recommended and quoted in the standard textbook (0.6 mg every 1-2 hours until the patient developed diarrhea or had clinical improvement)[1,24,25]. In patients who have normal renal function, the authors usually prescribe colchicine at 0.6 mg 3-4 times/day on the first day and decrease it to twice/day after that. In those with impaired renal function, the dosage of colchicine is decreased to 0.6-1.2 mg on the first day and 0.6 mg on the following days. With this regimen, it was found that colchicine is effective in controlling acute arthritis in gout and it rarely causes diarrhea.

In order to see the pattern of gout in the university hospital in Thailand, the authors compared the data with 2 other university hospitals in Bangkok, Chulalongkorn Hospital and Pramongkutklao Hospital (Table 7). A majority of the gout patients in the university were cases with recurrent attacks after their first one at the age of 60. They had multiple medical

Table 7. Comparison of clinical features in gouty arthritis among university hospitals in Thailand.

	Desomchok U[10]	Asavatanabodee P[11]	Present study
Hospital, city	Chulalongkorn Hospital, Bangkok	Pramongkutklao Hospital, Bangkok	Chiang Mai University Hospital, Chiang Mai
Period of study	1976-1985	1988-1997	1986-2001
No of patients	194	116	567
Male : female	7.8 : 1	5.0 : 1	6.3 : 1
Mean age at onset (Yrs)	Over 60 years in 40.2%	53.0	60.0
First attack	7.7	8.6	6.3
Recurrent attack	92.3	91.4	93.7
Most common joint involved			
First attack (%)	MTP (43.3)	MTP (45.0)%)	Ankle (37.2)
Recurrent attack (%)	Ankle (73.2)	Ankle (75.7)	Ankle (88.5)
Tophi (%)	29.9	33.6	36.5
Hypertension/CAHD (%)	54.1	66.3	54.3
Hyperlipidemia (%)	NA	62.9	33.5
Diabetes mellitus (%)	8.8	23.2	12.8
Chronic renal failure (%)	29.4	47.7	53.6
Renal calculi (%)	NA	22.9	34.5

problems, a high incidence of tophi and renal insufficiency. This could be explained by the trend of self-medication implemented by most of the Thai patients. In the early stage of gout, patients usually treated themselves with NSAIDs obtained over the drug store counter, or prescribed by primary physicians. Even when the arthritis became more frequent, NSAIDs were often prescribed without consideration of long-term therapy with hypouricemic drugs. The prolonged and recurrent use of NSAIDS in this elderly population might be an important factor that leads to renal impairment. Moreover, among the patients who were prescribed hypouricemic drugs, both primary physicians and the patients tended to stop the drugs shortly after the arthritis was controlled; resulting in the development of recurrent attacks, and the frequent use of NSAIDs therapy. When gout became more complicated or more severe, they visited a university hospital for better management. This created the high incidence of tophaceous gout with renal insufficiency in the university hospital series(10,11). Furthermore, the small number of fully-trained rheumatologists in Thailand has also limited the delivery of proper care to patients with complicated gout.

In conclusion, complicated gout is commonly seen in university hospitals. The concept that gout is a curable disease, but requires long-term treatment, should be emphasized to both primary physicians and patients. A good compliance with long-term hypouricemic therapy, and avoidance of the inappropriate use of NSAIDs, which will in turn prevent complicated gout and renal failure, should be highlighted.

(Received for publication on March 28, 2003)

REFERENCES

1. Wortmann RL, Kelley WN. Gout and hyperuricemia. In: Ruddy S, Harris ED, Sledge CB, editors. Kelley's Textbook of Rheumatology. 6th ed. Philadelphia: WB Saunders; 2001: 1339-76.
2. Zimmet PZ, Whitehouse S, Jackson L, Thoma K. High prevalence of hyperuricemia and gout in an urbanized micronesian population. Br Med J 1978; 1: 1237-9.
3. Darmawan J, Valkenburg HA, Muirden KD, Wigley RD. The epidemiology of gout and hyperuricemia in a rural population of Java. J Rheumatol 1992; 19: 1595-9.
4. Koh WH, Seah A, Chai P. Clinical presentation and disease associations of gout: a hospital-based study of 100 patients in Singapore. Ann Acad Med Singapore 1998; 27: 7-10.
5. Lai YL, Ng SC. Clinical profile of gouty arthritis. Abstract book of the 4th ASEAN Congress of Rheumatology Meeting, 1993 Oct 31-Nov 4; Singapore: Singapore; 2002: 77.
6. Tehupeoiory E, Faridin HM, Mubin UBH. Profile of hyperuricemia and gouty arthritis in Indonesia. Proceeding of the 10th Asia Pacific League of Association for Rheumatology Meeting; 2002 Dec 1-6; Bangkok, Thailand. Bangkok: Supjaroon Printing; 2002: 145-6.
7. Shahdan MS. Clinical spectrum of gout in Malaysia. Abstract book of the 3rd ASEAN Congress of Rheumatology Meeting; 1991 Nov 17-21; Bangkok, Thailand 1991: 197.
8. Karema-Kaparang AMC. Clinical pictures of hyperuricemia and gouty arthritis in Manado General Hospital (north Sulawesi). Abstract book of the 5th ASEAN Congress of Rheumatology Meeting; 1998 Jan 18-22; Manila: Philippines; 1998: 135.
9. Chaiamnuay P, Darmawan J, Muirden KD, Assawatanabodee P. Epidemiology of rheumatic disease in rural Thailand: a WHO-ILAR COPCORD study. Community Oriented Programme for the Control of Rheumatic Disease. J Rheumatol 1998; 25: 1382-7.
10. Deesomchok U, Tumrasvin T. Clinical features of gouty arthritis. Chula Med J 1988; 32: 537-46.
11. Assawatanabodee P, Intarasupht T, Chaiamnuay P. Clinical analysis of 116 gout patients at Pramongkutklao Hospital. Royal Thai Army Med J 1997; 50: 197-203.
12. Talbott JH, Yu TF. Gout and uric acid metabolism: Clinical description of primary gout. Stuttgart: Georg Thieme; 1976: 119-32.
13. Grahame R, Scott JT. Clinical survey of 354 patients with gout. Ann Rheum Dis 1970; 29: 461-8.
14. Talbott JH, Yu TF. Gout and uric acid metabolism: incidence. Stuttgard: Georg Thieme; 1976: 26-38.
15. Hall AP, Barry PE, Dawber TR, McNamara PM. Epidemiology of gout and hyperuricemia. Am J Med 1967; 42: 27-37.
16. Gutman AB. The past four decades of progress in the knowledge of gout with an assessment of the present status. Arthritis Rheum 1973; 16: 431-45.

17. Nishioka N, Mikanagi K. Clinical features of 4000 gouty subjects in Japan. Adv Exp Med Biol 1980; 122A: 47-54.
18. McCarty DJ. Diagnostic mimicry in arthritis: Pattern of joints involvement associated with calcium pyrophosphate dihydrate crystal deposition. Bull Rheum Dis 1975; 25: 804-9.
19. Louthrenoo W. Tuberculous arthritis complicating gout. Intern Med (Thailand) 1993; 9: 43-5.
20. Gibson T, Highton J, Potter C, Simmons HA. Renal impairment and gout. Ann Rheum Dis 1980; 39: 417-23.
21. Yu TF, Berger L. Renal function in gout. Its association with hypertensive vascular disease and intrinsic renal disease. Am J Med 1982; 72: 95-100.
22. Fessel WJ. Renal outcomes of gout and hyperuricemia. Am J Med 1979; 67: 74-82.
23. Yu TF, Gutman AB. Uric acid nephrolithiasis in gout: Predisposing factors. Ann Intern Med 1967; 67: 1133-48.
24. Takeltaub RA. Pathogenesis and treatment of crystal-induced inflammation. In: Koopman WJ, editor. Arthritis and allied conditions. 14th ed. Philadelphia: Lippincott Williams & Wilkins; 2001: 2329-47.
25. Wallace SL, Bernstein D, Diamond J. Diagnostic value of colchicine therapeutic trial. JAMA 1967; 199: 525-8.

การศึกษาลักษณะทางคลินิกผู้ป่วยโรคเกาต์ที่ตรวจพบผลึกเกลือในโรงพยาบาลของมหาวิทยาลัย†

วรวิทย์ เลาห์เรณู, พบ, นันทนา กสิตานนท์, พบ*,*
วราพร สุชิตาวุธ, วทบ, แรมใจ วิชัยนันท์, วทบ**

ได้ทำการศึกษาลักษณะทางคลินิกในผู้ป่วยโรคเกาต์ที่ตรวจพบผลึกเกลือในโรงพยาบาลคณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่ จำนวน 567 ราย (เพศชาย 489 ราย และเพศหญิง 78 ราย) ผู้ป่วยมีอายุเริ่มเป็นเฉลี่ยเท่ากับ 60.0 ± 11.7 ปี และมีระยะเวลาของการเป็นโรคมานานเฉลี่ย 5.2 ± 4.8 ปี ผู้ป่วยส่วนใหญ่ร้อยละ 94 เป็นชนิดเป็นกลับซ้ำ ข้อเข่าและข้อเท้าเป็นข้อที่พบการอักเสบได้บ่อยที่สุดในการเป็นครั้งแรก โดยพบได้ในแต่ละข้อร้อยละ 55.6 ในกลุ่มที่เป็นกลับซ้ำพบว่าข้อเท้า ข้อเข่า และข้อนิ้วหัวแม่เท้าเป็นข้อที่มีการอักเสบบ่อยที่สุด พบได้ร้อยละ 94.5, 81.2 และ 80.2 ตามลำดับ ตรวจพบปุ่มโทไฟร้อยละ 36 โรคร่วมที่พบได้บ่อยคือ ความดันโลหิตสูง ไขมันในโลหิตสูง เบาหวาน และโรคหลอดเลือดหัวใจตีบตัน พบนิ่วในทางเดินปัสสาวะร้อยละ 35 และผู้ป่วยร้อยละ 54 มีหน้าที่การทำงานของไตพกพร่อง ในจำนวนผู้ป่วย 59 รายที่มีหน้าที่การทำงานของไตปกติและได้รับการตรวจปริมาณการขับกรดยูริกออกทางไต พบว่าร้อยละ 12 มีการขับกรดยูริกออกทางไตมากกว่าปกติ ลักษณะทางคลินิกในผู้ป่วยโรคเกาต์ที่ทำการศึกษามีลักษณะใกล้เคียงกับการศึกษาในโรงพยาบาลของมหาวิทยาลัยในกรุงเทพ แต่ตรวจพบผู้ป่วยที่มีโทไฟ นิ่วในทางเดินปัสสาวะ และไตทำงานบกพร่อง มากกว่า

คำสำคัญ : โรคเกาต์, ภาวะกรดยูริกในเลือดสูง, กรดยูริก, การรักษา, ผลึกเกลือโมโนโซเดียมยูเรต, ข้ออักเสบจากผลึกเกลือ

วรวิทย์ เลาห์เรณู, นันทนา กสิตานนท์,
วราพร สุชิตาวุธ, แรมใจ วิชัยนันท์
จดหมายเหตุทางแพทย์ ฯ 2546; 86: 868–875

* หน่วยโรคข้อและรูมาติสซั่ม, ภาควิชาอายุรศาสตร์, คณะแพทยศาสตร์ มหาวิทยาลัยเชียงใหม่, เชียงใหม่ 50200

† บางส่วนของผลงานได้นำเสนอในการประชุม Asia Pacific League of Association for Rheumatology ครั้งที่ 10 ระหว่างวันที่ 1–6 ธันวาคม 2545