

The Management of Gout at an Academic Healthcare Center in Beijing: A Physician Survey

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ABSTRACT. *Objective.* Gout is a less commonly diagnosed rheumatic disease in China compared with Western countries, but its prevalence appears to be climbing. It is not known how Chinese physicians diagnose and treat their patients with gout, so we evaluated physician management of gout at a major academic healthcare center in Beijing, and investigated factors associated with better decision-making.

Methods. A 13-question anonymous survey was distributed and collected at a medical grand rounds and then at a rheumatology grand rounds at a major teaching hospital in Beijing. Physician demographic data including educational background, work experience, job titles, specialty or subspecialties, gout patient volume seen in a year, and continuing medical education (CME) in gout were also collected in the survey. Data were analyzed by multivariate regression models to identify factors associated with appropriate answers.

Results. Twenty-seven residents and general internists, 26 rheumatologists and fellows, and 28 physicians and fellows of other medical subspecialties from the Department of Medicine including visiting physicians responded to the survey. Among respondents, 78% think it is important for a definitive diagnosis of gout, but few actually perform aspiration of the affected joint fluid. Eighty-four percent report that they often follow the serum urate level of their patients with diagnosed gout. When treating acute gout in otherwise healthy patients, most physicians (77%) prefer oral colchicine, and in patients with renal impairment, about half of them (48%) choose corticosteroids or corticotropin as their first treatment. For longterm urate-lowering therapy, most physicians (87%) described a variety of indications that we consider less appropriate. They (86%) tend to initiate it early (< 2 weeks) after acute flares. When urate-lowering therapy is used, 80% of physicians sustain it less than 5 years. Further, only 12% of physicians use antiinflammatory prophylaxis during the initiation of urate-lowering treatment, and only 5% maintain it for an appropriate period of time. Logistic regression analysis of physician demographic data, educational background, and work experience found no consistent independent factors associated with better decision-making, other than CME, that were associated with establishing the definite diagnosis correctly. Specifically, the number of gout patients seen by physicians was not related to better decision-making.

Conclusion. The physicians' reported management of gout at this major academic healthcare center in Beijing was often inconsistent with current evidence. High quality CME is required to improve Chinese physician management of gout. (J Rheumatol 2006;33:2041–9)

Key Indexing Terms:

GOUT SYNOVIAL FLUID TEACHING CONTINUING MEDICAL EDUCATION

Gout is a heterogeneous disorder of urate metabolism characterized by the deposition of monosodium urate monohydrate in joints or other connective tissues. It is one of the most common forms of inflammatory joint disease, with an overall prevalence of less than 1% to 15.3%¹.

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The first 2 cases of gout in China were reported in 1948, and only 25 patients were identified in the Chinese literature in the following decade². For almost half a century it had been thought that the “disease of kings” was a rare rheumatic disorder in the Chinese population³, but recent studies suggest that the prevalence of gout in China is increasing, which might be explained by substantial improvement in standards of living in the past 20 years^{2,4–9}.

Although certain progress has been made in the management of gout, current therapies are based more on the clinician's preference than on evidence-based medicine^{10,11}. Variance in practice may also exist in Chinese physicians, but we found no research articles evaluating quality of care by searching the Chinese Medical Literature Database, Medline, Embase, or other electronic databases. As China is developing rapidly into a worldwide economic power, Chinese people are

more concerned about the status of their health and about the care provided. To clarify how Chinese physicians diagnose and treat their gout patients in general, we evaluated physicians' statements on management of gout by survey at a major academic healthcare center in Beijing, and investigated factors associated with better decision-making.

MATERIALS AND METHODS

Setting. The Peking Union Medical College (PUMC) Hospital is a major academic healthcare center of Western medicine in Beijing. Its rheumatology division is one of the oldest subspecialty programs in rheumatic diseases in China. Each year the division provides care for more than 36,000 patients with rheumatic diseases in clinics, as well as on a 50-bed subspecialty ward in the hospital.

Questionnaire. The original English questionnaire on gout was provided by Dr. R. Schumacher, University of Pennsylvania, and Dr. N. Schlesinger at the University of Medicine and Dentistry of New Jersey. It was translated into Chinese, and modified to conform to our circumstances. The questionnaire shown below comprises 4 groups of questions, focusing on diagnostic procedure and laboratory followup, treatment of acute gouty arthritis, urate-lowering therapy, and concurrent prophylaxis in hypouricemic therapy, respectively.

Questionnaire for physicians on the management of gout.

1. Do you think synovial fluid should be examined in a patient who has not previously had crystals identified but you suspect acute gout?
2. How often do you examine synovial fluid in such a patient?
3. How often do you order a serum uric acid investigation in a patient in whom you have diagnosed gout?
4. What is your drug of choice for an acute gout attack in an otherwise well patient?
5. What is your drug of choice for an acute gout attack in a patient with a creatinine of 2.2 mg/dl?
6. When do you prescribe a urate-lowering drug?
7. When initiating a hypouricemic drug, how often do you choose a xanthine oxidase inhibitor over a uricosuric drug?
8. How long after resolution of the acute gouty attack do you wait to initiate treatment with a urate-lowering drug?
9. Do you give prophylactic treatment to prevent acute gouty attacks when you begin a urate-lowering drug?
10. Which one do you usually prescribe as prophylaxis: colchicine, NSAID, or other?
11. For how long after achieving a normal uric acid do you give prophylactic treatment in a patient without evident tophi?
12. In your experience, how often do you see an acute attack occur while starting the patient on a xanthine oxidase inhibitor with and without prophylaxis?
13. For how long do you prescribe a urate-lowering drug?

We added 12 questions to ascertain the respondent's demographic characteristics, educational background, and work experience. Participants were specifically asked to note the length of their undergraduate medical education in medical school, whether they graduated from one of the Ministry of Health affiliated medical schools that are often thought to have better quality in recruited students and undergraduate medical education, their highest medical degree, length of work experience, work status and title, specialty or subspecialty, level of their hospital, patient volume seen in a year, their interest in gout, and whether they had any continuing medical education (CME) such as didactic lectures, grand rounds, or reading the literature on gout.

Subjects. The study subjects are internists working or being trained in the Department of Medicine at PUMC Hospital, including medical residents, general internists, subspecialists and fellows, and visiting physicians. We selected a medical grand rounds and then a rheumatology grand rounds without prior notification of the survey at PUMC Hospital in the year of 2004. The questionnaire was distributed as physicians attending the rounds entered the room. They were asked to finish it anonymously, and the questionnaires were

collected before they left the rounds. Those who attended both grand rounds were asked not to do the survey for a second time. To compare the management of gout specifically between physicians of different specialties as a secondary analysis, we classified respondents into 3 groups: medical residents and general internists, rheumatologists, and other medical subspecialists (each group includes fellows and visiting physicians of the specialty). We identified the number of respondents in each group, from the database of the Departments of Human Resource and Medical Education.

We classified the response to each question as appropriate if it was consistent with concurrent practice opinion in the literature, and as inappropriate if it was not¹¹⁻¹⁸, as follows:

Current practice for management of gout

1. Synovial fluid should be aspirated and examined to establish the diagnosis of gout in suspected acute gouty arthritis
2. The serum urate level should be checked periodically in patients with diagnosed gout
3. Colchicine or nonsteroidal antiinflammatory drugs (NSAID), but not corticosteroids or ACTH, are preferred choices for an acute gout attack in an otherwise well patient
4. Corticosteroids or ACTH, but not colchicine or NSAID, are appropriate treatment for an acute gout attack in a patient with significant renal insufficiency (serum creatinine \geq 2.0 mg/dl).
5. A urate-lowering drug should be prescribed for a patient with gout if acute attacks have occurred 2-4 times. The therapy should not be initiated for asymptomatic hyperuricemia, or after only one acute attack
6. The appropriate interval between resolution of the acute attack and initiation of urate-lowering therapy is more than 2 weeks
7. Prophylactic antiinflammatory treatment to prevent acute gouty attacks should be given concurrently with the urate-lowering therapy.
8. Prophylactic treatment should be sustained for 6 months to one year after the serum urate level reaches normal
9. Urate-lowering therapy should be maintained life-long.

Statistical analysis. Characteristics and answers to clinical questions were abstracted and entered in an Excel spreadsheet. We measured the characteristics of the participants and the distribution of responses to each question. In order to find factors associated with appropriate answers, for each question that interested us most, we defined the response as outcome variable, and the respondent's demographic characteristics, educational backgrounds, and work experience as predictor variables. The outcome was categorized as a dichotomous variable by classification criteria. Nominal and ordinal predictor variables were transformed into multiple dichotomous variables. Multicollinearity of predictor variables was assessed by Kendall's tau-b correlation analysis before variable transformation. Bivariate analysis was performed by the same method. Then conditional logistic regression models were constructed to identify factors that were independently associated with the appropriate response. Entries with missing data were deleted in the analysis. Predictor variables associated with the outcome at $p < 0.20$ in bivariate analysis were eligible for inclusion in the model. We used the forward selection technique for predictor variables to be included in the model at $p < 0.05$ and removed from it at $p > 0.10$. All analyses were carried out using the SPSS 11.5 software package. P values were 2-tailed.

RESULTS

Characteristics of respondents. The response rate at medical grand rounds was 70% (64/92), and at rheumatology grand rounds, 100% (29/29), while 5 rheumatologists who attended both grand rounds did not respond for the second time. Among the 93 questionnaires collected, 3 provided less than 20% of the respondent's background information, and 8 answered less than 20% of the clinical questions. These were excluded from further analysis. The remaining 82 respondents

missed less than 10% of all questions and were further analyzed as study subjects. For valid responses, we identified 27 residents and general internists, 26 rheumatologists, and 28 physicians of other medical subspecialties, each comprising one-third of the 82 respondents. According to the data provided by Human Resources and Medical Education, there were 82 residents and general internists, 37 rheumatologists, and 140 subspecialists working in the Department of Medicine when the survey was carried out. Each specialty or subspecialty group included trainees, faculty, and visiting physicians from other hospitals.

From the demographic data, we found that: 82% of the respondents were working in a tertiary academic healthcare center, 86% were younger than 40 years, 70% had less than 10 years' work experience, and 71% saw less than 10 gout patients in a year. While 85% of respondents stated that they had interest in gout, only half of them thought they had had CME of any kind in the disease. Other features of the respondent's educational background and work experience are listed in Table 1.

Using a simple correlation matrix we did not find serious multicollinearity between predictor variables. The correlation coefficients between age, professional appointment, and the length of appointment were between 0.6 and 0.8. Other coefficients were even lower.

Gout management

Diagnostic procedure and laboratory followup (Figure 1). In all, 64 (78%) respondents indicated that synovial fluid should be examined in a patient who had not previously had crystals identified when acute gout was suspected, but only 3 (4%) of the 82 respondents actually followed through with testing. In contrast, when asked about their followup of the serum urate level, 69 (84%) reported that they examined the serum urate level of their patients with diagnosed gout over 75% of the time. Our questionnaire did not ask about a specific target for serum urate.

For physicians' attitude towards synovial fluid examination, the logistic regression model identified 4 factors independently associated with the appropriate outcome (Table 2). Physicians who received their undergraduate medical education in a health ministry affiliated medical school or those who stated that they had had CME in gout were more likely to agree that synovial fluid should be examined in a patient with suspected gouty arthritis than those who did not, with adjusted odds ratio 4.88 (95% CI 1.17–20.41) and 10.31 (95% CI 1.78–58.82), respectively. However, some senior physicians were much less likely to think so. The odds ratio for those who had been working for 11–15 years was 0.08 (95% CI 0.01–0.43) as compared with those who had worked for < 5 years; for professors it was 0.04 (95% CI 0.003–0.52) as compared with residents, and no difference was found comparing other senior groups and the reference group.

For physicians' utilization of synovial fluid examination,

Table 1. Characteristics of respondents.

Characteristics ⁺	No. of Respondents (%)
Sex	
Male	28 (34)
Female	54 (66)
Age, yrs*	
20–29	29 (35)
30–39	42 (51)
≥ 40	10 (12)
Length of undergraduate medical education, yrs	
5 or 6	65 (79)
7	1 (1)
8	16 (20)
Ministry of Health affiliated medical school	
No	26 (32)
Yes	56 (68)
Highest medical degree**	
Bachelor	35 (43)
Master	19 (23)
Doctor	28 (34)
Length of work, yrs*	
< 5	23 (28)
5–10	34 (41)
11–15	12 (15)
> 15	12 (15)
Work title	
Resident physician	33 (40)
Attending physician	30 (37)
Associate professor	15 (18)
Professor	4 (5)
Specialty or subspecialty*	
Rheumatology	26 (32)
Internal medicine	27 (33)
Other internal subspecialties	28 (34)
Hospital	
Non-teaching hospital	15 (18)
Tertiary teaching hospital	67 (82)
Patient volume, per yr*	
< 5	42 (51)
5–9	16 (20)
10–19	7 (9)
≥ 20	15 (18)
Interest in gout*	
No	9 (11)
Yes	70 (85)
Continuous medical education in gout*	
No	41 (50)
Yes	40 (49)

* Data are missing where some physicians did not respond to the question.

+ The first group of each category is the reference group in multivariate logistic regression models. ** Any one of the 3 types of medical degrees is equivalent to MD in the US, as recognized by USMLE. The difference between them is in benchwork training.

we had 3 appropriate outcomes if we selected the cutoff point at 75% of the time; and the logistic regression model found no difference between any groups with different characteristics. When we moved the cutoff point to 50% of the time, we had 9 appropriate outcomes, and the logistic regression model showed that physicians with CME were more likely to per-

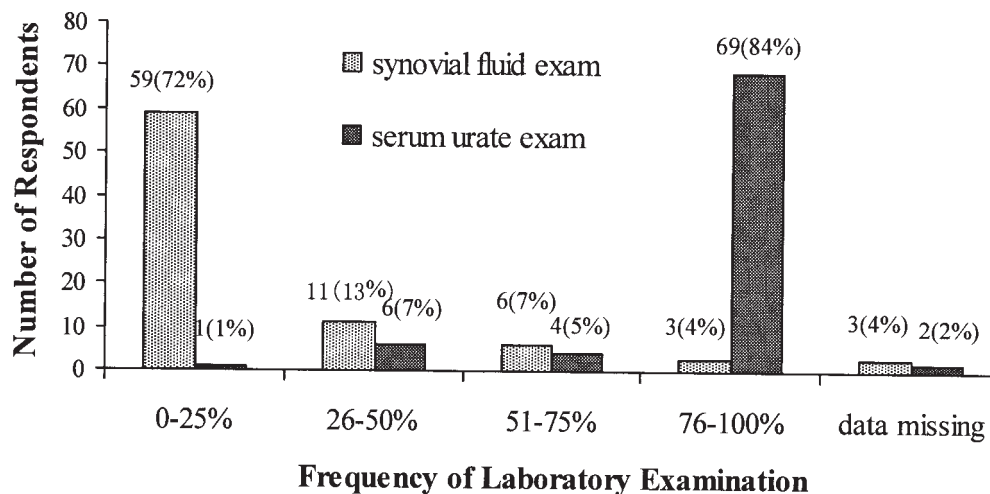


Figure 1. Diagnostic procedure and laboratory followup.

Table 2. The odds ratio of different attitude towards synovial fluid examination among physicians with different characteristics.

Characteristic	Synovial Fluid Examination		Adjusted OR	95% CI
	Should Be Done, N (%)	Should Not Be Done, N (%)		
Health ministry affiliated medical school				
No	17 (65.4)	9 (34.6)	1.0 (ref)	
Yes	47 (85.5)	8 (14.5)	4.88	1.17–20.41
Length of work, yrs				
< 5	21 (91.3)	2 (8.7)	1.0 (ref)	
11–15	5 (41.7)	7 (58.3)	0.08	0.01–0.43
Work title				
Resident physician	30 (90.9)	3 (9.1)	1.0 (ref)	
Professor	2 (50)	2 (50)	0.04	0.003–0.52
CME				
No	28 (68.3)	13 (31.7)	1.0 (ref)	
Yes	35 (89.7)	4 (10.3)	10.31	1.78–58.82

CME: Continuing medical education. Ref: reference value.

form the examination more than half of the time versus those who stated that they had not had CME in gout, adjusted odds ratio 12.66 (95% CI 1.42–111.11). We do not have data on actual utilization, which would also be of interest.

Further, using logistic multivariate analysis we found that CME was the only factor associated with physicians' followup of serum urate level in a patient with diagnosed gout over 75% of the time, adjusted odds ratio 11.63 (95% CI 2.71–50.00).

Treatment of acute gouty arthritis (Figure 2). When treating an acute gout attack in otherwise healthy patients, 63 (77%) physicians preferred oral colchicine, and 14 (17%) chose NSAID, with only 1 or 2 for intravenous colchicine or corticosteroids. For patients with a creatinine of 2.2 mg/dl, 40 (48%) physicians selected corticosteroids or corticotropin as their drug of choice. It is notable that 14 (17%) physicians

selected NSAID, and 21 (26%) still chose oral colchicine in such a situation, although we do not know whether they used it at a lower dosage.

For further analysis, we defined colchicine or NSAID, respectively, as the appropriate therapeutic choice for acute gout attacks in otherwise healthy patients, and steroid including oral prednisone, intraarticular corticosteroids, or intramuscular ACTH as appropriate for patients with renal insufficiency. Logistic regression models found that medical subspecialists other than rheumatologists were less likely to choose colchicine to treat acute gouty arthritis in otherwise healthy patients, odds ratio 0.19 (95% CI 0.06–0.60) versus rheumatologists (reference group), while the difference between general internists including residents and rheumatologists was not significant. No difference was found between any groups of

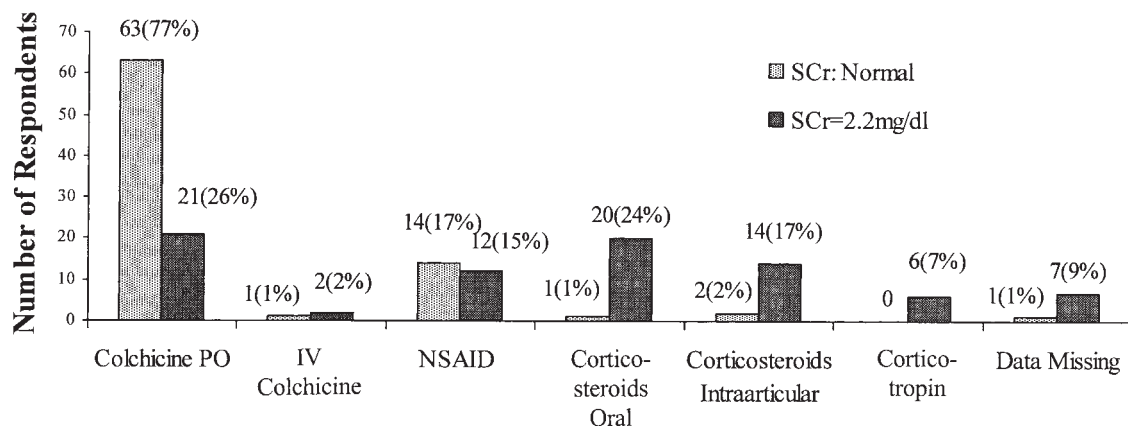


Figure 2. Drug of choice for acute attack.

physicians with different characteristics in choosing NSAID to treat otherwise healthy patients or in choosing steroid to treat patients with renal insufficiency.

Urate-lowering therapy. When they were asked about indications for urate-lowering therapy, 25 (30%) physicians responded that they would prescribe such a drug for asymptomatic hyperuricemia: 35 (43%) would do it after the first gouty attack, 10 (12%) after 2–4 attacks, 7 (9%) after even more. Counting the 4 physicians that chose multiple indications including “asymptomatic hyperuricemia” [other combined indications: “after first gouty attack” (3 physicians), “after 2–4 attacks” (1 physician)], the percentage for “asymptomatic hyperuricemia” reaches 35%. No one would wait until the patient developed tophi. We defined initiation of urate-lowering therapy after 2–4 attacks as the appropriate indication, and others grouped together as inappropriate indications. Unfortunately, the logistic regression model failed to find any characteristics associated with the appropriate indication.

When they were asked how long they would wait to initiate therapy after resolution of the acute gouty attack, 44 (54%) physicians said they would begin therapy in less than a week after resolution, 26 (32%) would begin in the second week, and only 9 (11%) after 2 weeks. It is notable that 30 (37%) physicians would start urate-lowering therapy right after the attack abated (0–2 days). We defined the appropriate time between resolution and initiation to be more than 2 weeks and by logistic regression analysis we found that physicians with a doctorate degree were more likely to choose the correct indication compared with the reference group who had only a bachelor degree, odds ratio 4.9 (95% CI 1.12–21.53).

When they were asked, for their hypouricemic drug, to choose between a xanthine oxidase inhibitor and a uricosuric drug, 38 (46%) physicians preferred a xanthine oxidase inhibitor more than 75% of the time, 26 (32%) preferred it 51–75% of the time, 15 (18%) preferred it 26–50% of the time, and 3 (4%) preferred it 0–25% of the time.

When they were asked how long they would prescribe a urate-lowering drug for a diagnosed gouty patient, 66 (80%)

of them said they would sustain therapy for less than 5 years, while only 7 (9%) thought it should be maintained life-long. We chose a loose definition, selecting 5 years but not being life-long as the cutoff point between appropriate and inappropriate duration of urate-lowering therapy, but still we could not find any characteristics associated with better choice in the model.

Concurrent antiinflammatory prophylaxis in hypouricemic therapy. When beginning a urate-lowering drug for a diagnosed gouty patient, only 10 (12%) physicians would give concurrent prophylactic treatment to prevent acute gouty attacks more than 75% of the time, 13 (16%) would give it 51–75% of the time, 22 (27%) 26–50% of the time, and 34 (41%) 0–25% of the time. We chose 75% of the time as the cutoff point to discriminate appropriate frequency of administering concurrent prophylaxis and inappropriate ones, but the multivariate model failed to find any characteristics associated with better outcome.

When asked to note the drug they usually prescribe for prophylaxis, 21% of respondents chose colchicine, 35% chose an NSAID, and 40% chose others that were not defined. Although 20–30% of physicians did not respond to the question about their experience of acute attack induced by initiating a xanthine oxidase inhibitor, there seems to be a trend that physicians observed acute gout attacks less in patients with prophylaxis than in those without it (Figure 3).

When physicians were asked about the length of prophylaxis they gave in a patient without evident tophi after achieving a normal serum urate, their answers varied: Most of them (65 individuals, 79%) prescribed it for less than half a year, and only 4 (5%) kept prophylaxis for 7–12 months (Figure 4). We defined 7–12 months as appropriate duration of prophylaxis although this might be controversial. The multivariate model failed to identify any characteristics associated with better decision-making.

Comparison of management between physicians of different specialties. There was no significant difference between rheumatologists, residents or general internists, and other

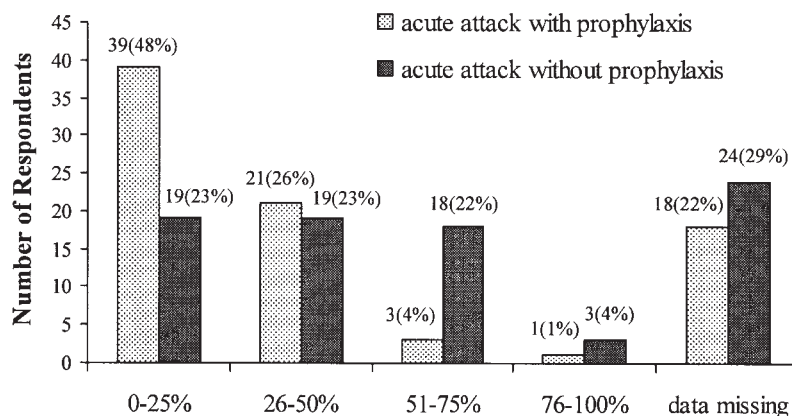


Figure 3. Frequency of acute attack with and without concurrent prophylaxis in hypouricemic therapy.

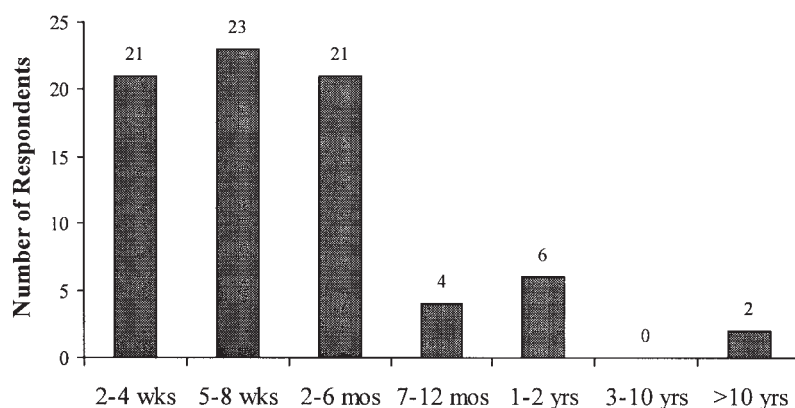


Figure 4. Length of concurrent prophylaxis in hypouricemic therapy.

medical subspecialists in the management of gout, with one exception that other medical subspecialists were less likely than rheumatologists to select colchicine for an acute gout attack in an otherwise healthy patient. The numbers and percentages of appropriate answers in each group, and unadjusted odds ratios of residents/internists and other medical subspecialists as compared with rheumatologists are listed in Table 3.

DISCUSSION

In the physician survey of gout management in China, we have several interesting findings. In establishing the diagnosis of gout, most physicians acknowledged the critical role of synovial fluid examination in a suspected patient with acute gouty arthritis, but actually only a few performed it in their practice. We did not explore the definite reasons for the discrepancy in the pre-structured questionnaire, but we hypothesize that the procedure had not been available in the hospital until the year before the survey; moreover, getting synovial fluid from the most frequently involved joints in the toes is technically difficult, and crystals can be missed or misinter-

preted by laboratories if they do not have experienced examiners¹⁹. On the other hand, about 1/5 respondents think it unnecessary in practice. Although the presumptive diagnosis of gout can be made when 6 of the 12 features in the preliminary criteria for the classification of acute gouty arthritis¹² are present, there are limitations when acute gouty arthritis presents an atypical clinical picture that is indistinguishable from acute septic arthritis and pseudogout, or even complicated with them, and the serum urate level may be normal in some acute attacks¹⁵. Aspiration of synovial fluid from the affected joint and analysis of the fluid by Gram stain, culture, and polarized light microscopic examination distinguishes them in most cases. The presence of intracellular needle-shaped crystals that have negative birefringence with compensated polarized light microscopy best establishes the definite diagnosis of gout^{20,21}.

Our multivariate analysis finds that physicians with better undergraduate medical education or CME in gout are more likely than others to have a clear conception in establishing the diagnosis of acute gouty arthritis, while some senior physicians are less likely to depend on the synovial fluid

Table 3. Comparison of answers to each question between physicians of different specialties.

N*	Answer	Specialty				
		Rheumatologists ⁺ N (%)	Residents/Internists N (%)	OR (95% CI)**	Other Medical Subspecialists N (%)	OR (95% CI)**
1	Yes	22 (84.6)	23 (85.2)	1.05 (0.23–4.71)	18 (66.7)	0.36 (0.10–1.38)
2	> 75% of time	2 (7.7)	1 (3.8)	0.48 (0.04–5.65)	0 (0)	0
3	> 75% of time	23 (88.5)	22 (84.6)	0.72 (0.14–3.58)	23 (85.2)	0.75 (0.15–3.73)
4	Colchicine	23 (92.0)	23 (85.2)	0.50 (0.08–3.00)	17 (60.7)	0.13 (0.03–0.69) ^{††}
	NSAID	2 (8.0)	4 (14.8)	2.00 (0.33–12.02)	8 (28.6)	4.60 (0.87–24.23)
5	Steroids	16 (64.0)	12 (52.2)	0.61 (0.19–1.95)	11 (42.3)	0.41 (0.13–1.27)
6	> 2–4 attacks	2 (8.0)	3 (11.1)	1.44 (0.22–9.41)	5 (18.5)	2.61 (0.46–14.90)
8	> 2 weeks	5 (20.0)	3 (11.1)	0.50 (0.11–2.36)	1 (3.8)	0.16 (0.02–1.48)
9	> 75% of time	2 (8.0)	2 (7.7)	0.96 (0.12–7.38)	5 (18.5)	2.61 (0.46–14.90)
11	6–12 mo	3 (12.5)	1 (3.8)	0.28 (0.03–2.90)	0 (0)	0
13	> 5 yrs	5 (20.0)	4 (15.4)	0.73 (0.17–3.09)	3 (11.5)	0.52 (0.11–2.46)

* Question number. ** Unadjusted odds ratio and 95% confidence interval. + Reference group. ^{††} $p = 0.016$. CI: confidence interval. NSAID: nonsteroidal antiinflammatory drugs.

analysis to confirm it. Although there were only 9 respondents who perform the procedure often (> 50% of time) in suspected patients, CME demonstrates a positive effect on physician behavior.

In treating acute gouty arthritis, both colchicine and NSAID are frequently used by physicians. Oral colchicine is the traditional treatment and has been confirmed to be more effective than placebo²², but now is less commonly used because many patients (about 80%) experience gastrointestinal side effects or toxicity before symptoms were relieved. Intravenous colchicine may induce life-threatening side effects¹⁷; therefore its clinical use is restricted to hospitalized patients who cannot take oral medications. All NSAID are generally effective compared with historical courses of untreated acute gout²³, but they may exacerbate renal insufficiency and occasionally cause interstitial nephritis or papillary necrosis¹⁷. Corticosteroids or ACTH have been demonstrated to be effective for the treatment of acute gout^{24–26}, but are usually reserved for patients in whom colchicine and NSAID are not tolerated or are contraindicated^{13,27}. Because there is limited evidence to compare the efficacy and side effects of different therapies for acute gouty arthritis, the decision in selecting potent agents is largely based on clinician preference, as colchicine is preferred to NSAID by French rheumatologists²⁸, while NSAID are the first choice of American physicians¹⁷. Our survey shows that Chinese doctors prefer colchicine in treating acute gout in otherwise normal patients, and rheumatologists may be the driving force to make the preference. In treating acute gouty arthritis in patients with renal insufficiency, oral colchicine may be given but the dose must be reduced. However, these patients are more susceptible to side effects of colchicine and NSAID^{11,13}, so in such circumstances corticosteroids or ACTH are reasonable alternatives. Our study finds less than half of respondents choose corticosteroids or ACTH to treat acute flares in patients with renal insufficiency, and no educational or professional factors contribute to better decision-making.

Although the incidences of gout and cardiovascular events are higher in patients with asymptomatic hyperuricemia than in healthy subjects^{11,29,30}, there is no evidence that urate-lowering therapy can reduce the risks effectively. The current opinions for treating asymptomatic hyperuricemia focus in identifying and correcting the cause of it¹⁶. When dietary and lifestyle factors are identified and appropriate changes have been made, the serum urate level may fall substantially, and acute gouty arthritis will not recur in many patients after a first attack¹⁰. One cost-effectiveness analysis of urate-lowering therapy concludes that it is cost-saving to initiate the therapy in patients who have 2 or more attacks in a year^{31,32}. Because urate-lowering therapy may induce an acute attack, most authorities concur that it should be initiated after the acute attack completely resolves, prophylactic drugs should be administered concurrently to prevent acute attacks of gout, and once initiated, the urate-lowering therapy should be life-long^{11,13,14}. Therefore, in the study we arbitrarily define “after 2–4 attacks” to be the appropriate indication and “more than 2 weeks after resolution of the acute attack” the appropriate time to start urate-lowering therapy.

Our results show that most respondents do not have clear recognition of the indication for urate-lowering therapy; they initiate it too early after the acute attack, and withdraw it much sooner than ideal, although the duration of urate-lowering therapy may be influenced by cost and availability of the drugs, and loss of patients in followup. Physicians in our survey used a xanthine oxidase inhibitor such as allopurinol more frequently than a uricosuric drug, probably because it is effective irrespective of the cause of hyperuricemia, less costly, and more easily available. Further, only 10% of respondents administer concurrent antiinflammatory prophylaxis for longterm treatment of gout, and only 5% sustain prophylaxis for a reasonable period of time. Multivariate analysis did not find any consistent factors independently associated with better decision-making.

We also find that when the choice of most respondents concurs with the current opinion, CME is strongly associated with better decision-making in multivariate analysis, but when most choices are not consistent with the appropriate one, they are diversified, and we cannot identify any of the educational or professional factors collected as contributing to better decision-making. In our study, the inconsistency appears much more obvious in longterm hypouricemic therapy and concurrent prophylaxis than diagnosis and management of acute gouty arthritis. We postulate that our fragmented and discontinuous healthcare system may have contributed to such poor quality of care: There are no primary care physicians in China to provide coordinated and continuous care for people; thus, residents, general internists, rheumatologists, and other medical subspecialists are all possible instant care-providers for patients with gout. However, the prevalent lack of high-quality continuing education, especially in a less commonly seen condition such as gout, may have played a greater role. In China, the management of gout is described in textbooks for medical students, but is not usually taught in didactic lectures as it is not felt to be a common disease. Students may learn about it during their clerkship or internship in clinical rotations, but most physicians see patients with gout after they graduate from medical schools and learn it by self-education, for example, by reading textbooks, communicating with experienced physicians, or keeping track of current literature. In our study, the question for CME simply asks, "Do you have any CME such as didactic lectures, grand rounds, or reading literature in gout?" but as far as we know, formal CME courses specifically devoted to gout in China are scarce, and Chinese literature in this area needs to be updated by being compared with current opinions. We therefore propose that the quality of CME be improved.

Our study has several limitations. First, it was carried out in a single medical center. Although we have visiting residents and fellows from other hospitals across the country, they are only a small proportion of respondents, and multivariate analysis does not show that visiting status made a difference. We postulate that their practice may have changed to be like ours and does not represent the situation in other hospitals. However, historically our hospital has had a great influence in the development of Western medicine in China, so it is hard for us to underestimate the challenges for the whole country. Second, many physicians of medical specialties other than rheumatology were absent from either of the 2 grand rounds, and most respondents indicated that they had interest in gout management, so we suspect that there is selection bias. We may have attracted more interested physicians who know more about gout than others, which may overestimate the quality of care in real practice. Third, our sample size is relatively small, which accounts for the large confidence interval of the odds ratios. Insufficient power may also explain why we cannot find factors significantly associated with better decision-making in certain areas. Fourth, we have challenges

in developing benchmarks to measure the quality of care for gout management. There are limited clinical studies of gout in the world, and current opinions are based more on physician preference than evidence^{10,11}. This may explain why the quality of care in gout is frequently suboptimal around the world^{33,34}. Only recently were indicators to benchmark quality of care in gout developed by systematically reviewing relevant articles in literature¹⁸. In China, we have even fewer studies in gout management and therefore have to base our criteria on evidence and current opinions obtained from Western countries. These are presumed to be applicable for the Chinese population: there appears to be no difference in the pathogenesis of gout between different populations. Better-designed multicenter studies are needed to assess the quality of care in China for patients with gout; it is even more important to design and carry out rigorous continuing medical education in this country to improve our management of gout.

In summary, physicians' reported management of gout at this major academic healthcare center of Western medicine in Beijing is often not consistent with current opinions in the world, especially concerning longterm hypouricemic therapy and concurrent prophylaxis. The findings are irrespective of the physician's specialty/subspecialties, work experience, or CME, etc. We conclude that high quality CME is required to improve the Chinese physician's management of gout.

REFERENCES

1. Chang HY, Pan WH, Yeh WT, Tsai KS. Hyperuricemia and gout in Taiwan: results from the Nutritional and Health Survey in Taiwan (1993-96). *J Rheumatol* 2001;28:1640-6.
2. Chen S, Du H, Wang Y, Xu L. The epidemiology study of hyperuricemia and gout in a community population of Huangpu District in Shanghai. *Chin Med J Engl* 1998;111:228-30.
3. Chang CJ. Gout and gouty arthritis. *Chin Med J* 1959;78:214-9.
4. Yang XY, Tang FL, Yin PD. The trend of proportion among inpatients with gout in 21 hospitals during the past 15 years. *Zhonghua Liu Xing Bing Xue Za Zhi* 1996;17:10-2.
5. Wang Q, Chen R, Du L, Zeng Q. An epidemiological and clinical study of primary gout. *Zhonghua Nei Ke Za Zhi* 2001;40:313-5.
6. Zeng Q, Wang Q, Chen R, Xiao Z, Huang S, Xu J. Primary gout in Shantou: a clinical and epidemiological study. *Chin Med J Engl* 2003;116:66-9.
7. Wigley RD, Zhang NZ, Zeng QY, et al. Rheumatic diseases in China: ILAR-China study comparing the prevalence of rheumatic symptoms in northern and southern rural populations. *J Rheumatol* 1994;21:1484-90.
8. Zeng Q, Huang S, Chen R. 10-year epidemiological study on rheumatic diseases in Shantou area. *Zhonghua Nei Ke Za Zhi* 1997;36:193-7.
9. Dai SM, Han XH, Zhao DB, Shi YQ, Liu Y, Meng JM. Prevalence of rheumatic symptoms, rheumatoid arthritis, ankylosing spondylitis, and gout in Shanghai, China: a COPCORD study. *J Rheumatol* 2003;30:2245-51.
10. Schlesinger N, Schumacher HR, Jr. Gout: Can management be improved? *Curr Opin Rheumatol* 2001;13:240-4.
11. Kim KY, Schumacher HR, Hunsche E, Wertheimer AI, Kong SX. A literature review of the epidemiology and treatment of acute gout. *Clin Ther* 2003;25:1593-617.
12. Wallace SL, Robinson H, Masi AT, Decker JL, McCarty DJ, Yu TF. Preliminary criteria for the classification of the acute arthritis of

- primary gout. *Arthritis Rheum* 1977;20:895-900.
13. Emmerson BT. The management of gout. *N Engl J Med* 1996;334:445-51.
 14. Terkeltaub RA. Clinical practice. Gout. *N Engl J Med* 2003;349:1647-55.
 15. Wortmann R, Kelley WN. Gout and hyperuricemia. In: Harris ED Jr, Ruddy S, Sledge CB, et al, editors. *Kelley's textbook of rheumatology*. Philadelphia: Elsevier Saunders; 2005:1402-29.
 16. Wortmann RL. Recent advances in the management of gout and hyperuricemia. *Curr Opin Rheumatol* 2005;17:319-24.
 17. Harris MD, Siegel LB, Alloway JA. Gout and hyperuricemia. *Am Fam Physician* 1999;59:925-34.
 18. Mikuls TR, MacLean CH, Olivieri J, et al. Quality of care indicators for gout management. *Arthritis Rheum* 2004;50:937-43.
 19. Schumacher HR Jr, Sieck MS, Rothfuss S, et al. Reproducibility of synovial fluid analyses. A study among four laboratories. *Arthritis Rheum* 1986;29:770-4.
 20. Nakayama DA, Barthelemy C, Carrera G, Lightfoot RW Jr, Wortmann RL. Tophaceous gout: a clinical and radiographic assessment. *Arthritis Rheum* 1984;27:468-71.
 21. Eisenberg JM, Schumacher HR, Davidson PK, Kaufmann L. Usefulness of synovial fluid analysis in the evaluation of joint effusions. Use of threshold analysis and likelihood ratios to assess a diagnostic test. *Arch Intern Med* 1984;144:715-9.
 22. Ahern MJ, Reid C, Gordon TP, McCredie M, Brooks PM, Jones M. Does colchicine work? The results of the first controlled study in acute gout. *Aust NZ J Med* 1987;17:301-4.
 23. Arnold MH, Preston SJ, Buchanan WW. Comparison of the natural history of untreated acute gouty arthritis vs acute gouty arthritis treated with non-steroidal-anti-inflammatory drugs. *Br J Clin Pharmacol* 1988;26:488-9.
 24. Groff GD, Franck WA, Raddatz DA. Systemic steroid therapy for acute gout: a clinical trial and review of the literature. *Semin Arthritis Rheum* 1990;19:329-36.
 25. Axelrod D, Preston S. Comparison of parenteral adrenocorticotrophic hormone with oral indomethacin in the treatment of acute gout. *Arthritis Rheum* 1988;31:803-5.
 26. Ritter J, Kerr LD, Valeriano-Marcet J, Spiera H. ACTH revisited: effective treatment for acute crystal induced synovitis in patients with multiple medical problems. *J Rheumatol* 1994;21:696-9.
 27. Taylor CT, Brooks NC, Kelley KW. Corticotropin for acute management of gout. *Ann Pharmacother* 2001;35:365-8.
 28. Rozenberg S, Lang T, Laatar A, Koeper AC, Orcel P, Bourgeois P. Diversity of opinions on the management of gout in France. A survey of 750 rheumatologists. *Rev Rhum Engl Ed* 1996;63:255-61.
 29. Tomita M, Mizuno S, Yamanaka H, et al. Does hyperuricemia affect mortality? A prospective cohort study of Japanese male workers. *J Epidemiol* 2000;10:403-9.
 30. Sundstrom J, Sullivan L, D'Agostino RB, Levy D, Kannel WB, Vasan RS. Relations of serum uric acid to longitudinal blood pressure tracking and hypertension incidence. *Hypertension* 2005;45:28-33.
 31. Fam AG. Should patients with interval gout be treated with urate lowering drugs? *J Rheumatol* 1995;22:1621-3.
 32. Ferraz MB, O'Brien B. A cost effectiveness analysis of urate lowering drugs in nontophaceous recurrent gouty arthritis. *J Rheumatol* 1995;22:908-14.
 33. Mikuls TR, Saag KG. Gout treatment: What is evidence-based and how do we determine and promote optimized clinical care? *Curr Rheumatol Rep* 2005;7:242-9.
 34. Mikuls TR, Farrar JT, Bilker WB, Fernandes S, Saag KG. Suboptimal physician adherence to quality indicators for the management of gout and asymptomatic hyperuricaemia: results from the UK General Practice Research Database (GPRD). *Rheumatology Oxford* 2005;44:1038-42.