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Gout in the Hmong in the United States: A Retrospective Study

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Abstract

Objective—To compare characteristics of gout in Hmong patients versus Caucasians and examine if Hmong ethnicity is associated with risk of tophaceous gout.

Methods—A retrospective chart review of Hmong and Caucasian patients with gout in a large health care system (HealthPartners) in St Paul, Minnesota, from 1/2001 to 3/2008, to compare clinical characteristics and risk factors for gout. Multivariable-adjusted hierarchical logistic regressions examined the association of Hmong ethnicity with risk of tophaceous gout, adjusting for age, sex, hypertension, diuretic use and kidney function.

Results—The analytic dataset consisted of 89 Hmong patients and 84 Caucasian controls, all of whom had ethnicity confirmed, an ICD-9 code for gout and had at least two physician-documented diagnoses of gout. The Hmong group was younger (58.3 vs. 66.3 y, $p = 0.04$), had an earlier onset of symptoms (37.4 vs. 55.0 y, $p < 0.001$) and higher mean serum uric acid levels during follow-up (9.1 vs. 7.6 mg/dl, $p = 0.001$). Hmong had higher rates of tophaceous gout (31.5% vs. 10.7%, $p = 0.001$), including hand tophi (21.3% vs. 3.6%, $p < 0.001$). In multivariable analyses that adjusted for age, sex, hypertension, diuretic use and kidney function, Hmong ethnicity was significantly associated with risk of tophaceous gout, with odds ratio 4.3 (95% CI 1.5, 12.2).

Conclusion—Hmong patients have an earlier onset of gout symptoms. Hmong race is an independent risk factor for tophaceous gout. Future studies need to examine whether genetic or other co-morbid factors predict this higher risk of more severe gout in Hmong.

Indexing Terms

Hmong; Gouty Arthritis; Tophaceous Gout

INTRODUCTION

Gout has been reported to be more prevalent in certain ethnicities, including the Maori in New Zealand [1] and aboriginal Chinese in Taiwan [2]. These groups have higher serum uric acid (SUA) levels as compared to controls and gouty arthritis is more prevalent in these ethnic groups compared to other populations [2,3]. The prevalence of other risk factors does

not seem to differ much from controls. Because hyperuricemia has been shown to be the most important risk factor leading to gout in these groups, there needs to be more awareness of the importance of treating gout in these populations [3,4].

Hmong are an ethnic group originally from southern China that later migrated to the northern regions of Laos, Vietnam and Thailand. Since the 1970s, large numbers of Hmong have come to the United States as refugees [5]. It is estimated that there are 190,000 Hmong in the U.S., with Minnesota having the second largest Hmong population after California [5]. The estimates of Hmong in Minnesota vary from 42,000 to 65,000 with the Twin Cities area having the largest metropolitan concentration of Hmong in the country [5,6]. Many physicians suspect that Hmong have more tophaceous gout as compared to Caucasians [7]. Hmong have increased risk for uric acid stones compared to controls [8]. The Hmong in the U.S. have an increased risk and earlier development of obesity, hypertension and diabetes compared to Caucasians, factors that increase the risk of hyperuricemia and possibly gout [9,10].

There are no previous large studies of gout in the Hmong, or whether the risk factors for gout in Hmong are similar to those reported earlier in Caucasians [7].

Our aim in this study was to explore the disease characteristics and risk factors for gout in the Hmong patients compared to Caucasians. We hypothesized that in our sample: (a) Hmong have more severe gout (more tophi, more flares, higher serum uric acid) than Caucasians; (b) this increased risk of tophaceous gout persists even after adjusting for known risk factors for gout including age, sex, kidney disease, hypertension and diuretic use; and (c) that Hmong get less aggressive treatment despite earlier and more severe presentation, compared to their Caucasian counterparts.

MATERIALS AND METHODS

Using the HealthPartners clinical databases in St. Paul, Minnesota, we performed a retrospective chart review of Hmong and Caucasians with at least one International Classification of Diseases, ninth revision (ICD-9) code for gout, during the period 1/1/2001–3/31/2008. Electronic records were available from 2001 for patients in the health system's 36 clinics. Since the primary aim of this study was descriptive we searched from 1/1/2001–3/31/2008 in order to ensure a maximum number of patients studied.

Since documented codes can be inaccurate, we used the presence of physician-documented gout diagnoses on two or more occasions at least four-weeks apart in the chart to confirm the diagnosis of gout. These patients constituted the analytic dataset. Earlier studies have used this criterion for other rheumatologic diseases with good accuracy [13,14]. We also examined which patients in the analytic set had the preliminary ACR classification criteria for gout [14].

Individuals of Hmong ethnicity have one of 18 unique clan names. We searched for Hmong patients using these clan names and for documentation of the Hmong language being the main language to identify all Hmong patients from the database. This approach has been validated for descriptive studies of Hmong populations [8,12]. We selected the Caucasian controls by using the most common Caucasian last names in Minnesota of 'Anderson' and 'Johnson'. Caucasian ethnicity was confirmed by documentation in the electronic medical records. Patient-reported ethnicity was used to define the Caucasian group.

We used a standardized data extraction form to obtain the following data recorded in provider notes, pharmacy prescriptions, laboratory databases, and administrative/billing databases. Data were extracted by a trained rheumatology fellow (SW) for the entire

duration of follow-up at HealthPartners, starting from the initial visit for gout until the last note or 3/31/2008, whichever occurred later.

The following data were extracted: age at onset of symptoms, number and location of joints and tophi, presence of > 3 attacks/year, all SUA levels, glomerular filtration rates (GFRs), allopurinol dose and duration, and use of other drugs to treat gout including colchicine, uricosuric drugs, NSAIDs and corticosteroids. We also recorded age, height and weight to calculate body mass index (BMI), family history of gout, and alcohol use (heavy alcohol usage was defined as greater than seven drinks per week). When baseline alcohol use data was missing, we used the data from the earliest point of documentation as a surrogate for baseline. For missing height data on patients with weight documentation, mean heights for each ethnicity were used to calculate BMI (8 Hmong and 2 Caucasians). Co-morbidities such as hypertension, renal stones, diabetes, hyperlipidemia, and coronary artery disease were recorded. We recorded whether these were present at the time of onset of gout symptoms, at the time of physician diagnosis, and anytime during the follow-up. We recorded number and name of joints involved as right, left, or bilateral involvement at presentation and cumulative number of joints involved at initial attack and for the duration of follow up.

A patient was defined as having tophaceous gout if the examining physician documented the presence of tophi. Gouty attack was defined as documentation of acute onset of joint pain with some clinical features of inflammation, or treatment in keeping with that used for an acute flare, including NSAIDs, colchicine, or prednisone.

Clinical characteristics were compared between the Hmong and Caucasians using chi-square and t-tests, as appropriate. Univariate and multivariable-adjusted logistic regressions compared the association of Hmong ethnicity as the main predictor of interest and presence of tophaceous gout as the outcome of interest. Hierarchical regression models controlled for the following variables: (1) Model 1 included age, sex and ethnicity; (2) Model 2 included age, sex, ethnicity, hypertension and diuretic use (Model 1 + hypertension + diuretic use). Sensitivity analyses were conducted by restricting these analyses to the subset that met the ACR criteria (35%, 18%, 40% and 39% respectively) which would likely lead to further selection bias and potential for reporting/recall bias for symptom duration and alcohol use.

RESULTS

We identified 136 Hmong and 146 Caucasians with an ICD-9 code for gout from the database. Twenty five Hmong patients and 32 Caucasians were excluded because of incorrect or missing ethnicity in the chart. This provided us with 111 Hmong and 114 Caucasians with confirmed ethnicity. Eighty nine Hmong (80%) and 84 Caucasians (74%) met the two physician diagnoses criteria for gout and constituted the analytic cohort for most analyses. Of these, 61 Hmong and 47 Caucasians met the preliminary ACR criteria for gout used for sensitivity analyses (Figure 1).

Clinical Characteristics

The Hmong group was younger (58.3 vs. 66.3 years, $p = 0.04$), and consisted of more males (84.6% vs. 77.4%, $p = 0.25$). Hmong had lower prevalence of diabetes mellitus (DM), coronary vascular disease (CVD) and hyperlipidemia than Caucasians at initial presentation (Table 1). The Hmong were leaner (BMI, 28.3 vs. 32.9 $p < 0.001$), although the BMI data was only available for 53 Hmong and 51 Caucasian. Twenty percent Caucasians and 18 % Hmong had monosodium urate crystal (MSU) identification. Only 18 % of Caucasians had seen a rheumatologist as opposed to 30 % Hmong ($p = 0.06$).

Gout Severity in Hmong versus Caucasians

Hmong had a much earlier onset of symptoms (37.4 vs. 55 years, $p < 0.001$) and higher mean SUA levels during follow-up compared to Caucasians (9.1 vs. 7.6 mg/dl, $p = 0.001$) (Table 2). These results were unchanged when analyses were restricted to the ACR criteria subset. Surprisingly, 38.5% of Hmong presented with gout symptoms before the age of 30 as opposed to only 8.2 % of Caucasians. Hmong had more joint involvement (3 vs. 2.5, $p = 0.014$), renal calculi (11.2% vs. 2.4% $p = 0.023$) and increased frequency of acute attacks in the (45.9% vs. 28.9%, $p = 0.027$). There was no significant difference in the duration of gout symptoms between the Hmong and Caucasians (10.5 y vs. 11.4 y, $p = 0.6$). This result did not change when restricting the analysis to the ACR criteria subset (Table 2).

Treatment of Gout in the Groups

Allopurinol was prescribed more often in the Hmong (74.2% vs 59.5%, $p = 0.04$), however, the Hmong used allopurinol for fewer months than the Caucasians (30 vs 48, $p = 0.05$). These differences were not detectable in the ACR subgroup (table 3). There was a difference in allopurinol mean dose with the Hmong using 241 mg/day and the Caucasian group using 341 mg/day ($p = 0.1$). The Hmong had fewer gout-related outpatient visits with a diagnostic code of gout as compared to the Caucasians (7.1 vs. 5.8, $p = 0.012$).

Univariate and Multivariable Analyses of Ethnicity as Risk Factor for Tophaceous Gout

Hmong were more likely to have tophaceous gout than Caucasians (38.9% vs. 15.1%, $p = 0.004$). (Table 2) The mean number of tophi was 1.0 in Hmong, as opposed to only 0.25 in Caucasians ($p < 0.001$). In the ACR subgroup, Hmong also had more tophi (1.21 vs. 0.32, $p = 0.001$). In addition, Hmong had more hand tophi (21.3% vs. 3.6%, $p < 0.001$). This difference was also significant in the ACR subgroup (27.9% vs. 6.4%, $p = 0.004$). Multivariable analyses adjusted for age and gender found a significant association of Hmong ethnicity with risk of tophaceous gout (Table 4). The association was significant after adjusting additionally for hypertension and diuretic use.

DISCUSSION

Hmong are thought to originate from Southern Chinese aboriginal population [16,17]. In this study we examined if Hmong have more severe gout compared to Caucasians, using retrospective data. Our study made several interesting observations, which deserve further discussion.

The most striking observation was that the Hmong group had an earlier onset of gout symptoms. More than one-third of Hmong patients were less than 30 years old at presentation as compared to only 8 % of Caucasians. This earlier disease onset in Hmong is less likely to be due to detection bias due to physician visits for other conditions, as these patients had lower comorbidity load compared to Caucasians. Hmong also had increased number of tophi especially hand tophi and higher mean SUA at disease onset and during follow up. Increased tophi, renal calculi, higher number of acute gout attacks and more joints involved are all manifestations of related to prolonged duration of elevated SUA and/or lower rates of allopurinol use in the Hmong. Some well-known risk factors for gout were far less prevalent in Hmong compared to our Caucasian group including hypertension, chronic kidney disease, diuretic use, obesity, and heavy alcohol use. Thus, at least the traditional risk factors do not explain the risk of earlier onset or more severe gout in the Hmong. Generally the comorbidity load is expected to less in a younger group and this could explain the lack of traditional risk factors.

Hmong patients used allopurinol for fewer months, even though they were prescribed allopurinol more often and had fewer visits for gout, despite having more frequent attacks and more tophi. The cumulative allopurinol dose was also lower than Caucasians. Thus, medication compliance and follow-up in Hmong population are challenges to successful gout treatment and management. Many factors such as barriers of language, access, documentation and socioeconomic factors are likely to impact access and awareness of risks/benefits of treatment [3]. Cultural disease models and culture based alternative treatment can also impact effectiveness of care of chronic disease. [3] Treatment in the Caucasian group was also inadequate as their SUA was greater than the goal of 6 mg/dl. This is in accordance with the inadequate treatment of gout in general in the US [11].

Hmong ethnicity was associated with tophaceous gout after controlling for age, and gender, hypertension, and diuretic use. We could not control for duration of gout, BMI, GFR, and alcohol use because of missing data in these variable to control for further bias. Another reason for not controlling for gout symptom duration is that this is on the pathway to tophaceous gout and adjusting for it would lead to over-adjustment in models. Hmong may have genetic predisposition to hyperuricemia similar to Polynesian or South Chinese groups, a hypothesis that needs further study [3]. It is possible that decreased urinary urate excretion as noted in the Polynesian population may also be present in the Hmong population.. GLUT9 polymorphism affecting urate under-excretion was recently identified in New Zealand's Maori population [20]. Genetic susceptibility loci [18, 21], including polymorphisms in Hypoxanthine guanine phosphoribosyl transferase [19] have also been reported in other studies of Taiwanese aboriginal families. Since all Hmong have a limited set of clan names they are most likely related closely and thus there may be a genetic risk factor Dietary differences between US Hmong and Caucasians may also be responsible for the higher prevalence of tophaceous gout noted in the Hmong [10,22].

Our study has several limitations. Retrospective study design makes our findings susceptible to residual confounding bias. Documentation of BMI and alcohol use was missing in almost half of the patients. Documentation of BMI and alcohol use was missing in almost half of the patients. Duration of gout symptoms was not available in 40% of the Hmong. Quantitative alcohol consumption was rarely documented unless it was excessive, limiting the ability to see the effect of this important risk factor.

This study has several strengths. We had almost 70–80 patients in each group to perform most comparisons. The main results of our study were robust, since they were confirmed when we restricted the analyses to patients meeting the ACR classification criteria. Although it is challenging to find documentation of ACR criteria in database studies [23], this approach is considered valid by most researchers. Crystal identification which is considered the gold standard is not frequently available and clinical criteria have reasonable predictive value [24]. Previous studies also found that physician documentation of rheumatic diseases (rheumatoid arthritis and spondyloarthropathies) in medical records on two or more occasions agreed well with ACR criteria, another definition we used in our study [13, 14].

In summary, Hmong need aggressive treatment of their gout because of their tendency to have earlier presentation, tophaceous gout with more frequent acute attacks, and hand tophi which can be particularly disabling and predict poor functional capability [25]. Increased duration of untreated or undertreated gout will lead to more severe clinical gout. Control of gout with close follow up can prevent the chronic manifestations [26]. Further population-based studies may be necessary to determine if Hmong have similar prevalence and genetic risk factors for hyperuricemia as compared to other Aboriginal Chinese and Polynesian populations.

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Figure 1.
Source and Study Group

Demographic and comorbidities in Hmong vs. Caucasian patients with gout

	Patients with Physician-diagnosed Gout			Patients Meeting Gout ACR Classification Criteria		
	Hmong (n=89)	Caucasian (n=84)	p-value	Hmong (n=73)	Caucasian (n=53)	p-value
	Mean ± SD or % (n)	Mean ± SD or % (n)		Mean ± SD or % (n)	Mean ± SD or % (n)	
% Male	84.3% (75)	77.4% (65)	0.25	85.2% (52)	74.5% (35)	0.161
Age (yrs)	58.3 ± 18.7	66.3 ± 17.1	0.004	54.9 ± 17.7	68.5 ± 14.2	<0.001
Gout symptom duration (yrs)	11.4 ± 5.9	10.5 ± 10.8	0.58	11.5 ± 5.9	9.7 ± 5	0.14
Cardiovascular Disease	2.2% (2)	8.3% (7)	0.07	3.3% (2)	10.6% (5)	0.12
Diabetes Mellitus	2.2% (2)	15.5% (13)	0.002	3.3% (2)	19.1% (9)	0.007
Hyperlipidemia	13.5% (12)	51.2% (43)	< 0.001	16.4% (10)	55.3% (26)	< 0.001

SD = standard deviation, yrs = years.

Table 2

Clinical features of and risk factors for gout in Hmong versus Caucasian patients

	Patients with Physician-diagnosed Gout		p-value	Patients Meeting ACR Classification Criteria for Gout		p-value
	Hmong (n=89) Mean $\pm$ SD or % (n)	Caucasian (n=84) Mean $\pm$ SD or % (n)		Hmong (n=73) Mean $\pm$ SD or % (n)	Caucasian (n=53) Mean $\pm$ SD or % (n)	
Gout symptom onset age (yrs)	37.4 $\pm$ 15.9	55.0 $\pm$ 17.0	< 0.001	37.3 $\pm$ 14.7	58.2 $\pm$ 16.1	< 0.001
% with tophaceous gout	31.5% (28)	10.7% (9)	0.001	39.3% (24)	14.9% (7)	0.005
Mean # of tophi	1.0 $\pm$ 1.6	0.3 $\pm$ 0.8	< 0.001	1.3 $\pm$ 1.72	0.3 $\pm$ 1	0.001
% with Hand tophi	21.3% (19)	3.6% (3)	< 0.001	27.9% (17)	6.4% (3)	0.004
% with renal calculi	11.2% (10)	2.4% (2)	0.023	11.5% (7)	4.3% (2)	0.18
# joints ever involved	3.0 $\pm$ 1.2	2.5 $\pm$ 1.3	0.014	3 $\pm$ 1.26	2.63 $\pm$ 1.25	0.106
% with ≥ 3 attacks/yr	45.9% (39)	28.9% (22)	0.027	50% (29)	37.2% (16)	0.2
Initial SUA mg/dl	9.3 $\pm$ 1.8	8.2 $\pm$ 1.7	0.027	8.9 $\pm$ 1.46	8.18 $\pm$ 1.68	0.140
SUA during follow up (mg/dl)	9.1 $\pm$ 2.2	7.6 $\pm$ 1.5	< 0.001	9.1 $\pm$ 2.14	7.6 $\pm$ 1.31	< 0.001
Hypertension	12.4% (11)	52.4% (44)	< 0.001	11.5% (7)	57.4% (27)	< 0.001
% using diuretics	9% (8)	42.9% (36)	< 0.001	3.3% (2)	46.8% (22)	< 0.001
GFR at diagnosis (median)	59.2 $\pm$ 20.8	62.4 $\pm$ 19.1	0.341	64.3 $\pm$ 19.77	62 $\pm$ 17.5	0.58
BMI at diagnosis (or earliest)	28.4 $\pm$ 7.0	33.0 $\pm$ 7.7	0.002	27.8 $\pm$ 8.18	31.5 $\pm$ 7.47	0.05
Heavy alcohol use ^a	3.4% (3)	17.9% (15)	0.002	2.8% (2)	19.1% (9)	0.007

^a greater than seven drinks per week,

BMI = body mass index, SD = standard deviation, SUA = serum uric acid, yr = year, yrs = years.

Table 3

Treatment differences between Hmong and Caucasian patients with gout

	Patients with Physician-diagnosed Gout			Patients Meeting ACR Classification Criteria for Gout		
	Hmong (n=89) Mean ± SD or % (n)	Caucasian (n=84) Mean ± SD or % (n)	p-value	Hmong (n=73) Mean ± SD or % (n)	Caucasian (n=53) Mean ± SD or % (n)	p-value
Cumulative allopurinol dose (mg/day)	241 ± 247	341 ± 309	0.10	252 ± 285	301 ± 251	0.51
Duration of allopurinol use (months)	30.2 ± 40.4	48.0 ± 41.3	0.052	34.2 ± 50.6	46.9 ± 40.3	0.26
% currently using or ever used						
Allopurinol	74.2% (66)	59.5% (50)	0.04	63.9% (39)	61.7% (29)	0.81
Colchicine	51.7% (45)	36.9% (31)	0.08	54.2% (32)	44.7% (21)	0.37
Probenecid	2.2% (2)	6.0% (5)	0.22	3.3% (2)	4.3% (2)	0.79
Corticosteroids	34.5% (30)	22.6% (19)	0.13	42.4% (25)	31.9% (15)	0.27
NSAIDs	89.7% (70)	81.3% (61)	0.14	92.7% (51)	86% (37)	0.28

NSAIDs = nonsteroidal anti-inflammatory drugs, SD = standard deviation,

Table 4

Multivariable model examining Hmong ethnicity as predictor for odds of tophaceous gout

	Model 1		Model 2:	
	OR (95% CI)	p-value	OR (95% CI)	p-value
Age	1 (0.98, 1.02)	0.8	1 (0.98, 1.02)	0.7
Sex	0.7 (0.2, 1.9)	0.5	0.7 (0.22, 1.94)	0.5
Hmong	3.8 (1.7, 9.62)	0.002	3.9 (0.58, 9.55)	0.003
Hypertension			0.6 (0.25, 1.49)	0.3
Diuretic use			1.5 (0.46, 5.2)	0.5
GFR at presentation				

OR = odds ratio; CI = confidence interval, GFR = glomerular filtration rate

Hypertension and diuretic use (furosemide, hydrochlorthiazide or other diuretics) were defined as present at the time of presentation

Model 1: age, sex, ethnicity

Model 2: age, sex, ethnicity hypertension, diuretic use