ISSN 1021-366X

Department of Health
REPUBLIC OF CHINA

Vol. 12 No. 5/May 25, 1996

Epidemiology Bulletin

An Epidemiological Survey of Hyperuricemia among the Atayals of Nanau Township, Ilan County

Abstract

The serum uric acid levels of elderly people are highly related to many chronic diseases such as hypertension, heart disease, diabetes and kidney disease. Gout is a kind of hyperuricemia originating in disorders of either purine metabolism or purine excretion. Lai et al., in their 1991 survey, found that the aborigines of Tungyueh village, Ilan County, seemed to have a high serum uric acid level. The present survey studied the Atayals, aged 20 years and above, of Nanau Township in Ilan County, for likely risk factors possibly concerned with their high serum uric acid levels. The average uric acid value for the male Atayals was found to be 8.31± 1.90; that for the female, 6.58 ± 1.75. Using as a definition of hyperuricemia a uric acid level for males of > 7.7mg% and for females of > 6.6mg%, the prevalence rate of hyperuricemia was up to 49.4%, and the rate of gout, 14.9%. The prevalence rate of gout was 30.1% of all hyperuricemic cases. Hyperuricemia is considered to be associated with both ethnicity and dietary habits. To prevent hyperuricemia, one should be particularly concerned with good dietary habits. Further studies, using molecular biology, are needed thorough investigation of hyperuricemia in different ethnic groups.

Introduction

With rapid economic development, possibility of improved nutrition and promotion of successful health and medical care programs in the Taiwan Area, life expectancy has been prolonged and the elderly population has increased; thus prevention and control of chronic diseases have become more important. Among common disorders of the elderly, high serum uric acid levels are found to be directly correlated to hypertension, heart disease, diabetes, kidney disease, dietary habits and nutrition. Hyper-uricemia may induce several complications such as acute and chronic gout, distortion of joints, urethral stones and renal failure. These complications adversely affect the quality of life and may also increase medical care costs.

Epidemiological surveys in some communities in Puli Township of Nantou County and Kinghu Township of Kinmen County by Sun[21] found that the prevalence rates of hyperuricemia in a population 30 years old and above in Puli Township were 20.3% for males (N=542) and 14.6% for females (N=616); these rates in Kinhu Township (1991) were 12.3% for males (N=1,334) and 5.3% for females (N=1,534); the prevalence rate of gout was 11.8% of all hyperuricemia cases. Those findings reflected an increasing trend in the prevalence rates of hyperuricemia and gout among the population. A survey by Lai et al.[20] in Ilan County in July 1991 found a relatively high prevalence rate of hyperuricemia and gout among the aboriginals of Nanau Township. The literature also shows that some ethnic groups in the Pacific have higher prevalence rates of either hyperuricemia or gout: examples include Polynesians, such as the Maori of New Zealand and the Samoans[4] as well as the Micronesians on, for example, Nauru and the Marshalls[2,8]. Similar surveys have not yet been reported in Taiwan. The present survey was designed to investigate both the prevalence rates and the risk factors of hyperuricemia and gout in the aboriginals of Nanau Township of Ilan County.

Materials and Methods

Nanau Township, a mountain area whose residents include primarily the Atayal people, is located in the southern part of Ilan County, facing the Pacific Ocean to the East and neighboring Hualien County to the South. The total population of the Township is 5,560 of whom 3,370 are aged 20 years and above. There are seven villages in the Township: Nanau, Pihou, Tungyueh, Kinyueh, Wuta, Kinyang and Auhua. Of those places, Nanau village has the most non-aboriginals (of the population 20 years and above, 370 are aboriginal and 104, non-aboriginal). The other villages have a primarily aboriginal population, with fewer than 10% non-aborigines.

Both two-stage random sampling techniques were applied. From the seven aboriginal villages, four (Kinyang, Pihou, Nanau and Tungyueh) were selected at random. From each village, 60 aborigines were selected; the non-aboriginals in each village were used as controls. Interviews were conducted by public health nurses. After three failed attempts to make home visits, the selected sample was replaced by the person immediately previous on the list; if that person also proved impossible to meet for three times, the person immediately following the originally selected person was visited. Lacking that meeting, the case was dropped. An appointment was made with each case to collect a fasting blood to be used for biochemical tests. The case was further clinically assessed by a physician from the Immunology-Rheumatology Department of the Taipei Veterans' General Hospital. Any history of medication, of arthritis and any evidence of typical gout and gout stone was taken. The questionnaire used included items such a s personal background information, history of illness and medication, dietary habits, life style, findings of physiological testings and clinical assessment. A total of 252 aborigines and 95 non-aborigines were successfully interviewed. The bio-chemical testing of blood specimens was conducted by the Lotung Holy Mother Hospital, using the Hitachi-750.

Results

Of the 360 samples, 347 (or 252 aborigines who included 249 Atayals, 2 Amis and 1 Bunun and 95 non-aborigines) were successfully interviewed, with their blood specimens collected. Table 1 shows that of the 249 Atayals (95 males and 154 females), the average age was 47.79 ± 14.63 years; of the 95 non-aboriginals (55 males and 40 females), the average age was 58.88 + 17.12. The average uric acid value for the male aborigines was 8.31 ± 1.90 and for the females, 6.58 ± 1.75; the values for the male non-aborigines was 6.83 ± 1.68 and for the females 6.00 ± 2.19. The prevalence rate of hyperuricemia for the male aborigines was 61.1% and for the females, 42.2%; that for the male non-aborigines was 25.5% and for the females, 30.0%. Table 2 shows that the aborigines had higher average uric acid values and higher prevalence rates, especially the residents of Tungyueh village where there was a 61.4% prevalence rate of hyperuricemia and a 28.6% gout prevalence rate. Table 3 shows that hyperuricemia is related to creatinine, cholesterol, triglyceride, body weight, obesity index, systolic and diastolic pressures; it is not related to either age or blood sugar. Table 4, a single-variable analysis of risk factors, shows that hyperuricemia is related to the intake of alcohol and sea foods. Table 5 compares non-aborigines with the Atayals by drinking habit. The findings were that hyperuricemia-prevalence in the non-aborigines is not related to drinking, whereas in the Atayals it is so related (the confidence interval was 1.3 – 4.0).

Table 6 shows that the chances of developing hyperuricemia for the Atayals, who also drink, are higher than for the drinking non-aborigines. For the non-drinking groups, hyperuricemia potential is similar for both the Atayals and the controls.

Table 7 shows by logistic regression analysis that hyperuricemia among people in the Nanau area is related to drinking habits and to ethnic groups.

Discussion

Uric acid is the end product of purine metabolism. In a normal person, the production and excretion rates of purine are balanced, and therefore the serum uric acid level remains constant. Many studies in Europe and the United States have shown that the average serum uric acid value in the male is around 5.0 to 5.7 mg/dl, and slighter lower in the female at 3.7 to 5.0 mg/dl[3]. When this value reaches 7.0 mg/dl in the male and 6.0 mg/dl in the female, the person concerned is hyperuricemiac. Uric acid values differ with ethnicity and geographic area[1-4]. At 37° centigrade and when the blood concentration is higher than 7 mg/dl, purine reaches the saturation level and crystallizes. The above criteria of hyperuricemia therefore are accepted by many[2,4]. By this definition, hyperuricemia in European countries and the United States is estimated to occur in around 2 to 18% of the total population[5]. Some scholars, however, advocate a stricter definition of hyperuricemia, using > 7.7 mg/dl for the male and > 6.6 mg/dl for the female. No information is currently available about the average serum uric acid level of the Chinese population on Taiwan. In 1974, the Taipei Veterans' General Hospital, by a survey of serum uric acid levels of 11,000 hospitalized patients,

Table 1. Prevalences of Hyperuricemia and Gout by Ethnicity

Ethnicity	Sex	No	Average age in years mean (SD)	Average uric acid (mg/dl) mean (SD)	Prevalence of	
					Hyperuricemia %	Grout %
Atayals	M	95	46.38 (12.99)*	8.31 (1.90)	61.1	27.4
	F	154	48.66 (15.53)#	6.58 (1.75)	42.2	7.1
	Both	249	47.79 (14.63)!	7.24 (1.99)	49.4	14.9
Non-Aboriginals	M	55	63.36 (13.61)	6.83 (1.68)	25.5	0
	F	40	52.73 (19.56)	6.00 (2.19)	30.0	7.5
	Both	95	58.88 (17.12)	6.48 (1.95)	27.4	3.2

Definition of hyperuricemia: for male ≥ 7.7 mg/dl; for female ≥ 6.6 mg/dl
*p ≤ 0.001 the Atayal male vs. the non-aboriginal male
#p ≤ 0.001 the Atayal female vs. the non-aboriginal female
!p ≤ 0.001 the Atayal total vs. the non-aboriginal total
Vs = Versus
SD = standard deviation

Table 2. Prevalences of Hyperuricemia and Gout by Village, the Atayals

Village	Age (years) mean (SD)	Average uric acid (mg/dl) mean (SD)	Prevalence of hyperuricemia %	Prevalence of gout %
Nanau (n=65)	47.75 (14.23)	7.16 (2.05)	47.7	13.8
Tungyueh (n=70)	44.33 (11.38)	7.71 (2.00)	61.4	28.6
Pihou (n=60)	52.10 (15.76)	7.07 (1.82)	48.3	10.0
Kinyang (n = 54)	47.54 (16.49)	6.91 (2.02)	37.0	3.7
Non-Aboriginals (n=95)	58.99 (17.19)	6.48 (1.95)	27.4	3.2

SD = standard deviation

Table 3. Single Variable Analysis of Hyperuricemia and Measurements

Measurements	Hyperuricemia (n = 150)	Normal uric acid (n = 157)	p-value
Uric acid value (mg/dl) mean (SD)	8.82 (1.48)	5.66 (1.02)	< 0.01
Age (years) mean (SD)	51.10 (14.81)	50.62 (17.14)	NS
Creatinine (mg/dl) mean (SD)	1.19 (0.28)	1.06 (0.20)	< 0.001
Blood sugar before meal (mb/dl) mean (SD)	98.03 (33.62)	96.87 (34.63)	NS
Cholesterol (mg/dl) mean (SD)	192.25 (50.52)	178.65 (44.81)	< 0.01
Triglyceride (mg/dl) mean (SD)	233.22 (195.28)	158.10 (149.18)	< 0.001
Body height (cm) mean (SD)	158.23 (7.56)	158.10 (6.97)	NS
Body weight (kg) mean (SD)	64.15 (10.92)	60.45 (10.73)	< 0.01
Body mass index (kg/m^2) mean (SD)	25.53 (3.89)	24.07 (3.86)	< 0.01
Systolic (mmHg) mean (SD)	127.05 (15.23)	121.14 (16.01)	< 0.01
Diastolic (mmHg) mean (SD)	80.75 (9.39)	77.44 (9.07)	< 0.01

Body mass index (BMI): body weight (kg) / body height (m^2)

NS = no statistical significance

SD = standard deviation

Table 4. Single Variable Analysis of Hyperuricemia and Dietary Habit

	Uric acid in blood		X^2	p-value
	High	Normal		
Drinking.			11.90	0.00056
Yes	95	88		
No	55	109		
Smoking:			4.84	NS
Yes	57	53		
No	93	144		
Betel-nut chewing:			2.76	NS
Yes	27	23		
No	123	174		
Eating meat:			10.58	NS
Every day	67	64		
Often	49	57		
Occasionally	24	52		
Rarely or none	10	24		
Eating organs of animals:			3.64	NS
Every day	1	1		
Often	15	21		
Occasionally	75	79		
Rarely or none	59	96		
Eating sea food:			12.63	0.0055
Every day	36	26		
Often	54	57		
Occasionally	38	67		
Rarely or none	22	47		
Eating beans:			4.60	NS
Every day	6	18		
Often	75	99		
Occasionally	42	43		
Rarely or none	27	37		
Eating preserved food:			6.15	NS
Every day	3	4		
Often	10	16		
Occasionally	69	65		
Rarely or none	68	112		
Eating vegetables.			2.20	NS
Every day	125	152		
Often	20	34		
Occasionally	3	7		
Rarely or none	2	4		
Eating fruits			1 74	NS
Every day	65	83		
Often	47	68		
Occasionally	34	37		
Rarely or none	4	9		

NS = no statistical significance

Table 5. Drinking Alcohol and Hyperuricemia

Ethnicity	Drinking Alcohol	Uric acid in blood		Odd ratio (95% CI)
		High	Normal	
Non-aboriginal	Yes	7	20	0.9 (0.3 — 2.7)
	No	19	49	
Atayals	Yes	88	66	2.29* (1.3 — 4.0)
	No	35	60	

*x^2 test p-value < 0.01
CI = confidence interval

Table 6. Drinking Alcohol and Hyperuricemia by Ethnicity

Drinking Alcohol	Ethnicity	Uric acid in blood		Odd ratio (95% CI)
		High	Normal	
Yes	Atayals	88	66	3.81* (1.41 — 10.61)
	Non-aboriginals	7	20	
No	Atayals	35	60	1.50 (0.73 — 3.12)
	Non-aboriginals	19	49	

*x^2 test p-value < 0.01
CI = confidence interval

Table 7. Logistic Regression Analysis of Hyperuricemia Risk Factors

	B	S.E.	x^2	p-value
Drinking Alcohol (yes, no)	0.6299	0.2285	7.60	0.0058
Ethnicity (Atayal, non-aboriginal)	−0.4629	0.1723	7.22	0.0072

B : Estimated Logistic Regression Coefficient
S.B.: Standard Deviation of B

arrived at some average values of 5.30 ± 1.35 mg/dl for males, and 4.60 ± 1.24 mg/dl for females. A 1988 survey taken by National Yangming Medical College in Puli Township, Nantou County, resulted in some average uric acid values of 6.38 ± 1.84 mg/dl for males and 5.15 ± 1.47 mg/dl for females. These findings led to the decision to apply the more strict definition of hyperuricemia noted above for the present survey.

Major causes of hyperuricemia include primary metabolic disorders of unknown origin and other secondary causes such as deficiency of certain enzymes, blood diseases, inadequate kidney function and the use of diuretics and other drugs[9]. Epidemiological studies of the risk factors of hyperuricemia give body weight and an obesity index as the primary risk factors, though triglyceride and creatinine in blood are also significantly related[3,5,10,11,12]. Although some studies suggest that uric acid value is related to cholesterol, some studies maintain that they are not so significantly related[13]. As to blood sugar and uric acid, it is commonly accepted that hyperuricemia is negatively related to diabetes but is positively related to poor glucose tolerance[14,15]. The famous Framingham study in the United States indicated that serum uric acid value is significantly related to the blood pressure value, and that persons with higher uric acid are at higher risk of developing coronary artery heart disease[16].

The attack rate of gout increases with rising of serum uric acid level. In 1967, Hall et al. studied a group of people whose average age was 58 for prevalence rates of articular gout when there were different blood concentrations of uric acid. They found that in both males and females, the chances of developing gout increased with the increase in serum uric acid values. For instance, the prevalence of gout for a male with a serum uric acid value of 6.0 to 6.9 mg/dl was 1.9%, rising to 16.7% at 7.0 to 7.9 mg/dl[18]. Long-term hyperuricemia can lead to kidney diseases, urethral stones and complications such as hyperlipemia, diabetes and cardiovascular disorders. With adequate dietary habits and life styles, many diseases can be prevented. Hyperuricemia and gout are very good cases-in- point. Study of the risk factors of hyperuricemia can help to prevent other related diseases, as well as to reduce the emotional burden and medical costs brought about by those diseases. The study of hyperuricemia is thus very important.

The present survey showed that in the Atayals, drinking was found to be closely related to hyperuricemia and gout. Alcohol was useful for the excretion of uric acid, and that the excretion rate of uric acid by the kidney did not decline because of drinking. Purine nucleotide and adenosine triphosphate (ATP) expedited dissolution, to increase de novo purine synthesis[17]. This was thought to be the major reason that drinking promoted the increase of uric acid in blood and also explained why drinkers were more prone to gout.

The present survey also found that hyperuricemia was related to hypertension It is generally believed that sedimentation of uric acid in the kidney causes kidney inflammation and impairs the excretory and secretory functions of that

The proportion of Atayal women developing hyperuricemia after menopause was not statistically significant (Table 8) Literature, however, shows that menopause is positively related to uric acid Different findings in Atayal women could come from differences in ethnicity and diets.

Table 8. Menopause and Hyperuricemia

Ethnicity	Menopause	Hyperuricemia		OR	95% CI	p-value
		Yes	No			
Ayatlas	Yes No	31 32	33 52	1.53	0.75-3.11	0.207
Non-aboriginals	Yes No	10 3	13 15	3.85	0.73-22.61	0.067

OR : Estimated Odd Ratios

Many young people have moved out of the mountain areas. The average age of the samples was therefore higher, particularly among the non-aborigines who served as the controls whose average age was 58.99 years. This could be the reason why hyperuricemia prevalence in the non-aborigines was found to be higher than for other non-aborigines.

The present survey also showed that the great prevalence of hyperuricemia in the residents of Nanau township was highly related to ethnicity. Further studies should, in addition to dietary survey, apply a molecular biological approach of HLA and HGPRT to patients to identify specific pathogenic factors.

Prepared by: C. L. Huang, K. T. Chen (FETP, National Institute of Preventive Medicine, Department of Health)

References:

1. Healey LA. Epidemiology of hyperuricemia. Arthritis Rheum 1975; 18: 709-719.
2. Zimmet P, Whitehouse S, Jackson L, et al. High prevalence of hyperuricemia and gout in an urbanised Micronesian population. Br Med J 1978; 1: 1237-1239.
3. Kilsey JL. Epidemiology of musculoskeletal disorders. New York: Oxford University Press, 1982: pp 102-108.
4. Prior IAM, Ross BS, Harvey HPB, et al. Hyperuricemia, gout and diabetic abnormality in Polynesian people. Lancet 1966; 1: 333-338.
5. Killey WN, Palella TD. Gout and other disorders of purine metabolism. In: Thorn GW, Adams, RD, Braunwald E, Isselbacher K, Petersdorf RG, eds. Harrison's Principles of Internal Medicine. 12th ed. New York: McGraw-Hill, 1991: pp1834-1843.

6. Loenen HM, Eshuis H, Lowik MR, et al. Serum uric acid correlates in elderly men and women with special reference to body composition and dietary intake. J Clin Epidemiol 1990; 43: 1297-1303.
7. Jackson L, Taylor R, Faaiuso S, et al. Hyperuricemia and gout in Western Samoans. J Chronic Dis 1981; 34: 65-75.
8. Adams WH, Harper JA, Heotis PM, et al. Hyperuricemia in the inhabitants of the Marshall Islands. Arthritis Rheum 1984; 27: 713-716.
9. Palella TD, Kelley WN. An approach to hyperuricemia and gout. Geriatrics 1984; 39: 89-102.
10. Fessel WJ, Barr GD. Uric acid, lean body weight, and creatinine interactions: Results from regression analysis of 78 variables. Semin Arthritis Rheum 1977; 7: 115-121.
11. German DC, Holmes EW. Hyperuricemia and gout. Med Clin North Am 1986; 70: 419-436.
12. Yano K. Thoads GG, Kagan A. Epidemiology of serum uric acid among 8000 Japan-American men in Hawaii. J Chronic Dis 1977; 30: 171-184.
13. Yu TF, Dorph DJ, Smith H. Hyperuricemia in primary gout. Semin Arthritis Rheum 1978; 7: 233-244.
14. Herman JB, Goldbout U. Uric acid and diabetes: Observations in a population study. Lancet 1982; 2: 240-243.
15. Tuomilehto J, Zimmet P, Wolf E, et al. Plasma uric acid level and its association with diabetes mellitus and some biologic parameters in a biracial population of Fiji. Am J Epidemiol 1988; 127: 321-336.
16. Brand FN, McGee DL, Kannel WB, et al. Hyperuricemia as a risk factor of coronary heart disease: the Framingham study. Am J Epidemiol 1985; 121: 11-18.
17. Maclachlan MJ, Rodnan GP. Effects of food and alcohol on serum uric acid and acute attacks of gout. Am J Med 1967; 42: 38-57.
18. Hall AP, Barry PE, Dawber TR, et al. Epidemiology of gout and hyperuricemia — A long-term population study. Am J Med 1967; 42: 27-37.
19. Wynguarden JB, Kelley WN. Gout and hyperuricemia. New York: Grune & Stratton, 1976: 22-33.
20. Lai SC, Chu YJ, Kuo CF, et al. A survey of hyperuricemia in Tungau and Tungyueh of Ilan County. Epidemiology Bulletin 1991; 99-105.
21. Sun LN. Epidemiological study of hyperuricemia in community — a Puli study. Master's degree thesis; National Yangming Medical College, School of Public Health. 1989, pp 160.