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Gout in South African blacks

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SUMMARY A retrospective study was carried out to determine the frequency, age of onset, mode of presentation, pattern of joint involvement, and incidence of primary and secondary gout in black patients with gout who were admitted to the King Edward VIII Hospital in Durban, South Africa. Nineteen patients were admitted to hospital with gout over a 5-year period from 1977 to 1981. The admission rate was found to be 4·7/100 000 hospital admissions. Five patients (26%) presented with monoarthritis and 14 patients (74%) had polyarthritis on admission. The joints most frequently involved were the knees (74%), the first metatarsophalangeal (MTP) joint (58%), and ankles (42%). The serum uric acid (SUA) was increased in 94%, and tophi were noted clinically in 47%. Eight patients (42%) with hypertension were on treatment with diuretics and 7 of these patients had a raised blood urea. These 8 patients (42%) were considered to have secondary gout, while no secondary causes were noted in the remaining 11 patients (58%) who had primary gout.

One of the first descriptions of a black patient with gout was a South African Negro reported on by Andrew in 1807.[1] Talbott *et al.*[2] reported their observations in 65 black patients with gout seen by them over a period of less than one year in Miami, USA. On the basis of their experience they emphasised the relatively higher incidence of gout in blacks than was commonly held.

Gout has been long recognised as uncommon in Africa and there have not been any large series of blacks with gout. Thus Hall[3] reported on three patients with gout in a total of 26 370 admissions in Kenya, while Gelfand[4] in Zimbabwe saw only 6 patients with gout over a 6-year period at a large hospital. Seven patients were seen over a 6-year period in Uganda,[5] of whom 5 belonged to the upper social class. The largest series of gout in blacks in Africa was of 14 cases diagnosed over a 14-year period in Zaire.[6]

The relative rarity of gout blacks prompted an analysis of the records of black patients with gout seen at the King Edward VIII Hospital, Durban, South Africa, from 1977 to 1981. King Edward VIII Hospital is a teaching hospital with 2000 inpatient beds, and it is the major hospital attached to the University of Natal Medical School.

Patients and methods

The King Edward VIII Hospital admission records of 19 black patients with gout were studied. The age of onset, duration of disease, mode of presentation, pattern of joint involvement, criteria for diagnosis, and the presence or absence of any secondary causes of gout were noted.

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Results

Frequency of gout. The 19 patients with gout accounted for 22 admissions to the Hospital. The total number of hospital admissions over the 5-year period was 467 948. The admission rate for gout was 4·7/100 000. The numbers of patients seen annually were as follows: 1977, 3; 1978, 7; 1979, 2; 1980, 5; and 1981, 2.

Sex, age of onset, and duration of disease (Table 1). The male to female ratio was 3·8:1, and females accounted for 21% of the patients with gout. The

Table 1 *Sex, age of onset and duration of disease*

Sex	M : F	3·8:1
	UK[8]	9·3:1
	Talbott *et al.*[2]	2·0:1
Age of onset	Males	Females
30–39	5	–
40–49	4	2
50–59	3	2
60+	8	–
Mean	50	52
UK[8] peak	5th decade	6th decade
Duration of Disease		Number
1 year		6
1– 5 years		9
6–10 years		4
Mean duration of disease		3·1 years

Table 2 *Pattern of joint involvement*

Joint	*No.*	%	*UK %*[a]
Knees	15	79	32
1st MTP	11	58	76
Ankles	8	42	50
Hands	6	32	25
Wrists	5	26	10
Elbows	5	26	10
Other	3	16	4
Extra-articular (bursitis)	2	11	3

mean age of onset was 50 years in males and 52 years in females. The mean duration of disease was 3·1 years, and the disease was not present for longer than 10 years in any of the patients.

Clinical features. Five patients (26%) presented with monoarthritis, while 14 patients (74%) had polyarthritis (2 or more joints involved) on admission. Tophi were noted clinically in 9 patients (47%). The commonest sites were the elbows, ears, and feet. The incidence of past or present joint involvement is shown in Table 2. The joints most frequently involved were the knees (79%), first metatarsophalangeal (MTP) joint (58%), and ankles (42%).

Laboratory findings (Table 3). The serum uric acid (SUA) was normal in one patient who had a history of recurrent involvement of the first MTP joint and the knees, and the diagnosis was established by the finding of monosodium urate (MSU) crystals in the joint aspirate. The SUA result was not recorded in one patient who also had MSU crystals in the joint aspirate.

Another patient who presented with recurrent episodes of monoarthritis of the right knee had a raised SUA concentration and a synovial biopsy which showed deposits of crystalline material surrounded by a foreign-body giant cell reaction. This patient subsequently developed acute arthritis of the right first MTP joint while in hospital.

MSU crystals were noted in the aspirate from the knee in 5 patients, while microscopy of tophaceous material from the foot showed the presence of MSU crystals in 2 patients. The blood urea was elevated in 7 of 16 patients (44%). In 3 patients the blood urea was not recorded, none of whom had any evidence of renal disease. The latex test for rheumatoid factor was negative in 12 patients in whom the results were recorded.

Table 3 *Laboratory findings*

	No.	%
Elevated SUA (1 normal, 1 not known)	17/18	94
Elevated blood urea	7/16	44
MSU crystals	7	37
Synovial biopsy	1	5

Table 4 *Criteria for diagnosis*

	No.	%
1st MTP joint involvement	11	58
Tophus (clinically)	9	47
Elevated SUA	17	94
MSU crystals + biopsy	8	42
Radiological changes	10	59

Criteria for diagnosis (Table 4). The diagnosis of gout was established by the finding of MSU crystals in 7 patients, and 1 patient had synovial histology compatible with gout together with a raised SUA and involvement of the first MTP joint. Four patients had recurrent arthritis of the first MTP joint with elevated SUA, and 2 of these patients also had *x*-ray changes compatible with gout. Three patients had elevated SUA, first MTP joint involvement, and tophi, and one of these patients also had *x*-ray changes of gout. One patient with asymmetrical polyarthritis had a raised SUA with a tophus and *x*-ray changes of gout. The SUA was increased in the remaining 3 patients; 1 patient had acute monoarthritis of the ankle, another patient had asymmetrical polyarthritis and tophi, while the remaining patient had acute asymmetrical polyarthritis with *x*-ray changes compatible with gout.

Associated diseases. Two patients (11%) had urolithiasis, one of whom had a bladder calculus, while another patient passed a calculus in hospital which was found to be composed predominantly of urates. Eight patients (42%) had hypertension; 2 of these were diabetic, and 1 had chronic obstructive airways disease. These 8 patients were on diuretic therapy; 7 of them (44%) also had an elevated blood urea (range 7·6 to 18·1 mmol/l), and they were considered to have secondary gout. In the remaining 11 patients (58%) no secondary cause for hyperuricaemia was noted, and they had primary gout.

Other findings. A negative family history of gout was recorded for 10 patients, but this information was not recorded for the remainder. All 19 patients were resident in urban areas. All the patients belonged to the poorer social class.

Discussion

This study supports the findings of other workers that gout is an uncommon disease in blacks in Africa.[3-7] The admission rate for blacks with gout was 4·7/100 000. The male to female ratio in our study

was 3·8:1, while Talbott *et al.*[2] found a male to female ratio of 2:1 and Grahame and Scott[8] in the United Kingdom noted a male to female ratio of 9·3:1. The mean age of onset was 50 years in males and 52 years in females, while in the UK series[8] there was a peak incidence in the 5th decade in males and the 6th decade in females. The mean duration of disease was 3·1 years with all the patients having their disease for less than 10 years. Only half the patients in the UK study had disease of less than 10 years' duration.

Fourteen (74%) of our patients had polyarthritis on admission. The joints most frequently involved were the knees (79%), first MTP joint (58%), and ankles (42%), and tophi were present in 47% of the patients. In the UK study only 11% of the patients had more than one joint involved simultaneously. The joints most frequently involved were the first MTP joint (76%), ankles (50%), and the knees (32%), and tophi were present in 47% of the patients. 42% of our patients had hypertension, 44% had an elevated blood urea, and 11% had urolithiasis. Grahame and Scott[8] found hypertension in 52% of their patients, an elevated blood urea in 25%, and urolithiasis in 11%. All our patients were resident in an urban area and belonged to the poorer social class. Most of the patients in the Ugandan series belonged to the upper social class.[5]

Gout in the Maoris has been reviewed by Rose.[9] Population surveys[10-12] have shown that when hyperuricaemia is defined as 7 mg/dl (0·4 mmol/l) and above in males and 6 mg/dl (0·36 mmol/l) and above in females, approximately 50% of New Zealand Maoris, and Rarotongan, Puka Pukan, and Tokelauan Polynesians can be classed as hyperuricaemic. The prevalence of gout ranges from a maximum of 10·2% in New Zealand Maori males and 1·8% in Maori females aged 20 and over, through 5·3% in Puka Pukan and 2·4% in Rarotongan males to 2% in New Zealand European males.[7 10-12]

In South Africa epidemiological surveys[7 13] have been carried out in the urban and rural black population. Four hundred and twenty-four urban and 370 rural Africans were assessed, and there were a few patients with hyperuricaemia in each group, but no patients with gout were seen. The serum uric acid concentrations in the rural population were similar to the mean levels reported for European communities. The average SUA concentrations in the urbanised group were significantly higher than those of an ethnically similar rural community. Lowenthal and Dymond[14] reported on 11 African patients with gout seen over a 38-month period at the Baragwanath Hospital in Johannesburg.

In the population study conducted in Framingham, Massachusetts,[15] half of the new cases of gout detected over a 14-year period occurred in patients taking a thiazide diuretic or ethacrynic acid. Epidemiological surveys conducted in the urban black population in Durban[16] have shown an overall prevalence of hypertension of 25% based on the WHO criteria. Thiazide diuretics have also been recommended as the first-line therapy for hypertension in blacks, and their use is widespread in the treatment of hypertension and cardiac diseases. In view of the high prevalence of hypertension in blacks, the frequent use of diuretics, and the findings of the Framingham study one would expect to find many more blacks with gout. Since this expectation is not fulfilled it would seem that dietary, environmental, genetic and/or other known factors acting either singly or in combination are responsible. There is a need for prospective studies to try to determine the factors which are responsible to help in our understanding in gout in blacks.

Conclusion

The small number of patients in the present study, even though it is one of the largest in Africa, does not allow us to make any definite inferences regarding gout in blacks. However, on the basis of our findings we note that in comparison with whites (*a*) gout in blacks is still an uncommon disease; (*b*) it may frequently present as a polyarthritis, as was noted in 74% of our patients; (*c*) the knees, first MTP, and ankle joints are most commonly involved.

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Book review

The Heart and Rheumatic Disease. Vol. 2. Ed. by Barbara M. Ansell and Peter A. Simkin. Pp. 292. £24·00. Butterworths: London.

This book is the second in a series of reviews of topics in rheumatology, and follows *The Kidney and Rheumatic Disease.* It has an unusual plan: rather than dealing seriatim with the different rheumatic diseases and their effect on the heart, its chapters, by different authors, are devoted to different aspects of heart disease and the role of rheumatic diseases in each. This is particularly suited, for example, to pericarditis, which can arise in a number of rheumatic diseases and for which a comparative approach is useful. The same applies to the myocardium and to aortic and mitral valve disease.

Other chapters deal with the conducting system, pulmonary and systemic hypertension, coronary vasculitis, and rheumatic fever, and the final chapter is on diagnostic methods and techniques. Altogether the rheumatic diseases lend themselves well to this approach. There is little overlap between chapters and authors, and a good index facilitates cross-reference on individual diseases.

The book is well produced on high quality paper with excellent illustrations and many references to each chapter. It is a splendid reference book for rheumatology departments and medical libraries.

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