The Medical Journal of Malaya
Vol. XIX, No. 1, September 1964.

THIRTY-THREE CASES OF ACUTE ARTHRITIS IN SABAH

Dr. C. J. BURNS-COX, M.B., B.S. Lond., M.R.C.P. Lond.,
Medical officer, Queen Elizabeth Hospital, Jesselton,
The State of Sabah.

Introduction

Acute arthritis is a common world-wide diagnostic problem. The treatment depends to such an extent on the cause that it is vital to reach an accurate diagnosis.

Gout may be a fatal disease due to its involving the kidneys but there is reason to believe that early diagnosis and adequate treatment can prevent the development of chronic arthritis, tophi and renal damage. Unfortunately both early diagnosis and adequate management are extremely difficult here for social and geographical reasons.

Since acute arthritis is common in this area it was thought worthwhile to gather the cases together in order to find the more common causes.

Method

It was found to be impossible to obtain really accurate histories from most of these patients despite repeated attempts. This has made data on family histories valueless and the duration of joint symptoms is only accurate to the nearest year or two.

A complete physical examination was made and recorded at the time for the purpose of this survey. Only the few relevant facts are included in the tables.

Investigations on all patients included haemoglobin estimation, white blood cell total and differential counts, examination of the urine for protein, sugar, cells and casts, serum uric acid, blood urea and chest Xray. Most patients had Rose Waaler and Latex agglutination tests. Many patients also had Xrays of affected joints and Anti-Streptolysin O titres done.

Most of the Rose Waaler and Latex agglutination tests and many serum uric acid estimations were carried out by the Department of Pathology, Singapore. Many were done in the hospital laboratory here.

Serum uric acid was estimated by the method of BROWN (J.Biol.Chem.1945,158, 601.) using phosphotungstic acid as a reagent. The Singapore department of Pathology takes 5.0mg./100ml. to be the upper limit of normal in all people. For this series the upper limit of normal was taken to be 5.5mg./100ml. for males and postmenopausal females.

Materials

In the eleven months June 1963 to April 1964 inclusive, thirty-three adults have been admitted to the Queen Elizabeth Hospital, Jesselton suffering with acute arthritis either for the first time or giving a history of previous attacks. These all had subjective and objective evidence of arthritis. They were over sixteen years and none had a purely chronic arthritis or only the arthralgias which accompany many fevers. Expatriates are not included.

The Hospital serves a population of about 120,000 people scattered over an area of about 3,400 square miles. About 70% of the population is indigenous — Kadazans and Bajaus. 25% is Chinese and 5% is composed of Malays, Indians, Phillipinos, Indonesians, Timorese and others.

It is certain that many people with acute arthritis have been missed from this series for many reasons. The chief of these are that many will have found it difficult to reach hospital quickly, many will have accepted local remedies, some will have attended government dispensaries or private practitioners and some may have come with a mild attack and been given outpatient treatment.

Results

Twenty-two of the thirty-three cases were suffering from an acute attack of gouty arthritis. Five of these had tophi and in thirteen the first metatarsophalangeal joint was or had been previously affected.

TABLE

CASES OF GOUT

No.	Race	Age of Onset	Age of Presentation	Presenting Joints	Other Joints Involved	Serum Uric Acid	Rose Waaler	Latex	Other Details
1	Ch	65	80	Ankles	Knees, Big Toes	10.4	1/40	—	Proteinuria BP 190/100
2	Ch	50	56	Big Toe	Ankles Knees Metacarpo Phal. J.	7.5	—	—	Presented with execerbation and acute Polio.
3	Ch	36	40	Ankle	Knees, Wrists	8.3	1/10	+	Ureteric Colic
4	KAD	35	38	Knee	Wrists Knee, Ankles	8.5	—	—	—
5	KAD	40	43	Knee	Wrists, Big Toes	7.0	1/10	—	Tophi both ears
6	SINO KAD	30	34	Ankle	Big Toes, Knees	10.7	1/80	—	Ureteric Colic Large Renal Calculus
7	INDON	56	68	Ankles	Big Toes	8.4	—	—	Tophi both ears Proteinuria BP 160/105
8	KAD	24	27	Big Toe	Ankles, Elbows	7.0	—	—	—
9	Ch	51	60	Ankle	Big Toes, Wrists	9.0	1/20	—	Tophaceous Olecranon Bursae
10	Ch	60	65	Ankles Knees	Big Toes	7.4	1/10	—	Tophi in ears and toes Proteinuria
11	KAD	55	55	Big Toe	Knees Ankles	11.6	—	—	—
12	KAD	37	38	Big Toe	—	6.1	—	—	—
13	KAD	35	40	Knees	Ankles, Big Toes	6.8	1/20	—	Tophaceous Olecranon Bursae
14	KAD	49	55	Knee	Elbow Ankles Shoulders	12.5	1/160	—	—
15	BAJAU	55	56	Big Toe	Knees	8.6	—	—	Proteinuria
16	Ch	56	60	Big Toe	Ankle	11.3	1/40	—	—
17	BAJAU	46	52	Ankles	Nnee	6.6	1/80	—	—
18	KAD	43	43	Ankles	—	11.9	—	—	On Diuretics Proteinuria BP 200/110
19	KAD	43	46	Ankle	Wrists, Knees	7.4	1/40	—	
20	KAD	40	40	Wrist	—	6.0	—	—	Proteinuria

TABLE

CASES OF GOUT

No.	Race	Age of Onset	Age of Presentation	Presenting Joints	Other Joints Involved	Serum Uric Acid	Rose Waaler	Latex	Other Details
21	KAD	62	77	Ankles	Knees	10.2	—	—	Duodenal Ulcer Proteinuria with Pus cells + +
22	KAD	38	50	Wrist	Elbow, Knees	7.4	1/20	+	—
	RHEUMATOID ARTHRITIS								
23	KAD	46	53	Knee	—	3.6	1/320	+	Differential Agglutination Titre 1/160
	OSTEO ARTHRITIS								
24	Ch	48	56	Knees	Heberden's Nodes Ankles	3.7	—	—	—
25	KAD	44	51	Knees	—	4.0	—	—	Proteinuria Typical X-ray changes
26	Ch	53	58	Knees	Heberden's Nodes	2.7	—	—	Proteinuria
	ANAPHYLACTOID PURPURA OF SCHONLEIN								
27	Ch	20	20	Ankles Knees	—	2.7	—	—	Petechiae on Limbs and Buttocks
28	Ch	62	62	Shoulders Elbows Wrists	Knees	3.0	—	—	Histological Proof Petechiae Limbs.
	PETER'S SYNDROME								
29	Ch	28	28	Knees Elbows	—	3.2	—	—	Both had GCFT— No Rash VDRL—
30	PHILIP.	46	46	Ankles Knees	—	4.9	1/80	+	Red Eyes Urethral Discharge
	UNDIAGNOSED								
31	INDIAN	23	23	Knees Ankles	—	5.2	1/10	—	ASOT 180 TODD Unit Bronchiectasis with Pneumonia
32	KAD	37	38	Prox Inter Phal. Joint	Shoulder Knee	3.4	—	—	
33	TIMORESE	24	24	Ankle	—	4.8	1/20	+	

All cases were male except 15, 16 and 24.

The blood urea was not more than 35mg./100ml. in any patient.

Serum uric acid levels were repeated after three months in only four cases:—

Case 2 the level fell from 7.5 to 3.6mg./100ml.

case 3 the level rose from 8.3 to 9.8mg./100ml.

case 5 the level fell from 7.0 to 3.3mg./100ml.

case 6 the level fell from 10.7 to 2.2mg./100ml.

Most of the cases of gout were rubber tappers or padi farmers but some were from higher social grades.

There were no geographical areas with an unexpectedly high incidence of arthritis.

Treatment

Cases of gout were treated initially with colchicine or phenylbutazone orally until the acute attack had largely subsided. At the same time probenecid 0.5G. b.d. with Soda Bicarbonate 2G.q.d.s. was started. This was increased to probenecid 1.0.G. b.d. after four days and continued for at least three months. It was hoped to obtain treated serum uric acid levels after three months in all cases but most were not available at the time. A urinary output of at least 2,500ml. daily was insisted on while in hospital.

Case 1 was treated only with aspirin, in view of his age and the severe degree of chronic deformity, for its analgesic and anti-inflammatory effect rather than for its uricosuric power.

No blood dyscrazia or dyspepsia was encountered with probenecid but the soda bicarbonate had to be reduced at times because of nausea.

All other types of arthritis were treated with aspirin 600mg. four hourly in the first instance. Case 30 needed phenylbutazone and a ten day course of cortisone before obtaining relief.

Discussion

"Gout is almost unknown in the Orient and Tropics." Cecil and Loeb. A textbook of Medicine. 10th.Edition.p595.

This series is not sufficiently comprehensive to estimate the incidence of gout in this district but it does show that it is a common cause of severe acute arthritis.

Unfortunately owing to lack of facilities here a comparison between other similar series and this cannot be made.

The diet here consists chiefly of rice, dried fish, fruit and a little meat. It is now believed that the inherited predisposition to gout or hyperuricaemia is the cause of gout and that certain foodstuffs have the ability only to trigger off an attack. For this reason a detailed examination was not made of the diet of these patients. One single alcoholic drink cannot be blamed as several sufferers come from kampongs where beliefs prohibit alcohol.

There was no evidence of any other cause for the high serum uric acid levels such as hypothyroidism, renal failiure, starvation or severe infectious disease with much tissue breakdown. Case 18 had been treated with guanethidine 30mg. and hydroflumethiazide 50mg. daily for three months before his first attack. He also had generalised psoriasis of six months duration. The only affected joints were his two ankles. The distal interphalangeal joints of his fingers and toes were normal.

The only case with an obvious precipitating factor was 2. This attack started with the fever of the minor illness of acute paralytic polio in the opposite leg.

In patients with gout the commonest cause of death associated with the disease is renal involvement. Gouty nephritis may cause hypertension, renal failiure or predispose to chronic pyelonephritis with or without stone formation. In this series 6 out of 22 gouty subjects had proteinuria, two have recently suffered from ureteric colic and one also has a large renal calculus. 3 had a blood pressure over 95mm.hg. diastolic.

Only one of the patients in this series had been diagnosed as acute gout in a previous attack.

Rheumatoid arthritis is uncommon here when compared with the incidence in temperate climes.

Osteoarthritis is probably as common here as it is in the rest of the world but seldom has severe enough exacerbations to need hospital admission.

Only three cases did not fit a well recognised diagnosis. None of these had any residual deformity or pain after the acute attack had subsided.

Summary

In an eleven month period thirty-three adults were admitted to the Queen Elizabeth Hospital, Jesselton suffering with acute arthritis. Investigations showed that twenty two were suffering from acute gout, three had exacerbations of osteoarthritis, two had Reiter's syndrome, two had anaphylactoid purpura with arthritis of Schonlein and three remain undiagnosed.

The incidence of gout is surprisingly high in this series. This is of importance as the treatment of gout is specific and quite different from that of the other disease mentioned.

I am grateful to colleagues for referring cases to me. My thanks are due to Dr. J. A. B. Nicholson, Acting Director of Medical Services, Sabah for permission to publish.