GOUT

Some Clinical Observations

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SUMMARY. A series of twenty patients with gout managed at the British Military Hospital Rinteln is described, and some effects of treatment are discussed. Observations on the control of hyperuricaemia with uricosuric drugs and/or with allopurinol in tophaceous as opposed to non-tophaceous gout are offered. A biochemical "stress" test is suggested in the diagnosis of gout, where facilities for the demonstration and identification of crystals with polarised light do not exist.

Introduction

Twenty male cases of primary gout were seen in a two and a half year period. These were assessed clinically and investigated to exclude the presence of underlying disease producing secondary gout. Three cases of hyperuricaemia without gout are also briefly described as they illustrate some facets of the pathogenesis of secondary gout.

Social status

Seven cases were of officer status; nine were from among the senior non-commissioned officers, and only four were junior ranks—privates, lance-corporals and corporals.

Distribution by age

The ages recorded were those at each patient's first attack of gout, and not the age when they presented at the British Military Hospital Rinteln. The average age of the group was 37.9 years. While this is fairly young, it is biased by the selected population being screened in a military population. The range of ages varied from the early twenties to fifty-two years. The largest number of cases were in their late forties.

This age distribution must influence the rank distribution commented upon. The average of those with a family history of gout was 34.6 years, slightly younger than the average for the whole group.

The length of history

Thirteen of the twenty patients had histories of recurrent attacks of arthritis over two or more years. In the extreme case there was a patient with a 21 year history of numerous attacks of gouty arthritis. Table I details the length of history of these thirteen patients. While the length of history of gout in these patients is striking it must be recorded that only four of these patients had ever received uricosuric treatment, and none fully appreciated the importance and long term need for such management. This is surprising in view of the recent literature discussing the modern management of gout (Barnes and Mason, 1967), (Scott, Hall and Graham, 1966). Table II illustrates the variability of experience of the patients. Fifteen cases are detailed: those with similar lengths of history.

Associated Features

The associated features noted in the patients include some which can be considered

Table I
Length of history of thirteen patients with recurrent attacks of arthritis

Length of history	Number of patients	Length of history	Number of patients
2 years	1	6 years	1
3 years	1	7 years	1
4 years	3	10 years	2
5 years	3	21 years	1

Table II
Number of previous attacks of gouty arthritis in fifteen of the patients

Length of history	Number of Patients	Number of previous attacks for each patient
1 to 2 years	7	1. 1. 2. 3. 3. 4. 5.
4 years	3	4. 5. 6.
5 years	3	6. 6. 12.
10 years	2	4. 20.

as predisposing to the disease. A family history of gout, in a close relative, was obtained in six out of the twenty patients.

Thirteen of the group were considered to be obese on clinical examination; the average weight of the twenty patients was 192 pounds.

Twelve patients gave a history of an excessive intake of alcohol. An excessive intake was defined as any daily intake of six pints or more of beer or the equivalent in wines or spirits.

Three examples of active arthritis, having been precipitated by trauma were seen. One attack occurred in a patient, apparently well controlled with uricosuric treatment, after an acute streptococcal throat infection associated with a high fever.

One soldier was classified as having a racial pre-disposition; he was of mixed British, Malay, Italian and Japanese origin. It was considered that this parentage might predispose to hyperuricaemia, as other Pacific peoples (Maori, Filipino and Polynesian) have well documented hyperuricaemia (Kellgren, 1964).

When the relationship between all of these features was studied it was clear that there was a considerable overlap between obesity and excessive alcohol intake, ten patients being grouped under both headings. However, none of these features could be related consistently to the presence of tophaceous gout.

Joints involved in gouty arthritis

The site of involvement of first attacks of arthritis was the metatarso-phalangeal joint of a big toe in fifteen instances; the knee in three instances; the ankle on one occasion, and the finger also on one occasion. The case of finger involvement was determined by local trauma.

The site of involvement of all attacks of gouty arthritis was similar, in that the big toe was most commonly involved—in nineteen patients—with the knee and ankle each being affected in six patients. The wrist was affected in four patients and the shoulder and finger each in one patient.

Site of tophaceous deposits

Seven of the twenty cases had tophaceous deposits. In four patients they were present in auricular cartilage and in two instances the tophi were present in both ears. Four patients had deposits in bursae and one in a synovial sheath. Four patients had tophaceous deposits recognised as cystic spaces in bone in relation to affected joints.

General investigation

Simple haematology, urinalysis and blood sugar estimates were carried out in all patients. A high erythrocyte sedimentation rate was found in the presence of an active arthritis. Haemoglobin estimation and blood films were normal in all patients. Urinalysis revealed a mild proteinuria in three patients, two with tophaceous gout and one without. During follow up, after treatment had been initiated, the urines of these three cases returned to normal with clearing of the proteinuria. The results of blood sugar estimations both fasting and random were not remarkable although one case of diabetes mellitus, not previously diagnosed, was found.

Renal investigation

Blood urea estimations, water concentration and dilution tests and creatinine clearance estimation were carried out in all cases. Apart from a rather poor urine concentration as measured by the specific gravity the results were normal.

Radiological investigation

Intravenous pyelograms were carried out in fourteen patients and the appearances were normal in all. This included one patient who had previously passed a urate calculus. Radiographs of the affected joints showed cystic spaces in adjacent bone in four cases. In three patients non-specific osteo-arthritis was noted.

Uric acid levels

Five of the twenty cases had uric acid levels within the normal range when first seen. The remainder had high levels. The speed with which elevated levels settled when the patient was kept at bed rest and under treatment with an anti-inflammatory compound such as Butazolidin or Indomethacin is illustrated in Figure 1. It is important to recognise that uric acid levels may fall to within normal limits quickly and that this does not invalidate a diagnosis of gout. MacLachlan and Rodnan (1967) described this and state that many of their patients were intoxicated on admission and the rapid fall was related to the previous effect of alcohol in elevating the uric acid level. This did not apply to the patients in this series.

Where facilities exist for the demonstration and identification of synovial crystals clearly diagnosis can be made independently of uric acid levels. However, where crystal demonstration using polarised light, as described by Currey (1968), is not possible the diagnosis of gout, in a first attack, may be made difficult by failure to find elevation of the serum uric acid level. In two such doubtful cases a form of stress test has been applied. Use has been made of the observations by MacLachlan and Rodnan (1967) and Lieber, Jones, Losowsky and Davidson (1962) that fasting is associated with a rise in uric acid levels and that this rise is accentuated by alcohol taken during the period of fasting. It was considered that the "patients" would show a greater swing in uric acid levels

Fig. 1. The fall in serum uric acid with bed rest and treatment (three patients). The pecked line is the upper limit of normal.

than the control subjects. The patients and the controls were starved for twenty-four hours and given two ounces of whisky after starving for twenty hours. The results are given in Table III. The first doubtful case had injured a foot while playing football and

Table III
Investigations: stress test
Twenty-four hours starvation and 2 ounces alcohol at hour twenty

Serum uric acid levels	Initial per cent	After 8 hours per cent	After 16 hours per cent	After 24 hours per cent
Doubtful. Case 1	6.0	6.6	7.1	6.0
Control	4.4	4.0	3.8	4.1
Control	6.6	4.0	4.6	4.0
Control	5.1	4.6	4.8	4.4
Doubtful. Case 2	6.2	6.3	8.2	9.0

had been admitted to a surgical ward. The "reaction" on the foot did not settle and his uric acid levels were normal when they were measured. The second doubtful case was referred for opinion, having been treated with colchicine by his own doctor. He gave a history of a painful foot nine years previously. On admission uric acid levels were

normal. In the stress test the doubtful cases reached abnormal levels while the control cases stayed within the normal range. The controls showed no clear rise in uric acid levels over the test period. It is suggested that this test is worth trying in doubtful cases and, if further observations confirm that a rise to abnormal levels occurs in these circumstances, it would be suitable for use as a diagnostic test. In MacLachlan and Rodnan's study one non-gouty subject showed as great a rise in uric acid levels as the patients during a period of forty-eight hours starvation with alcohol given early in the period of fasting.

Cases of hyperuricaemia (without gout)

The three cases described illustrate some of the facets of the urate problem.

Case 1. A man (J.W.) aged 60 years who presented in 1960 with acute nocturnal dyspnoea. He was admitted to hospital in acute left ventricular failure with a hypertension of 240/150. He was successfully treated with guanethidine and chlorothiazide 250 mg twice daily. He has remained clinically well with a standing BP of 150/90 over nine years.

The blood urea was elevated at 80 mg/100 ml in 1962 and fluctuated between 62 and 86 mg/100 ml, until 1967 when it rose to 104 and 120 mg/100 ml. In 1967 the serum uric acid was twice measured at 13 and 12 mg/100 ml and this fell to 6.2 mg/100 ml three months after the chlorothiazide was stopped. The elevation of serum uric acid appeared to be related to the chlorothiazide therapy rather than to the underlying renal damage.

Breckenridge (1966) demonstrated that 58 per cent of treated hypertensive patients had an elevated serum uric acid and that this did not correlate with blood urea levels.

Case 2. A man (P.W.), aged 35 years, who was first seen in July 1966. He gave a history of pain of renal colic type in 1956 and in 1960. The latter episode was associated with overt haematuria and the passage per urethram of a small stone, which was not recovered for examination.

In 1960 attention was diverted from his calculi by the finding of a palpable spleen and an abnormal peripheral blood film. The bone marrow was considered compatible with chronic myeloid leukaemia. The neutrophil leucocytes were noted to have a lower quantity of alkaline phosphatase than normal. At that time the serum uric acid was stated to be normal. The total white blood count never rose above 20,000 cells per mm². The patient remained in good general health and by 1964 the spleen was no longer palpably enlarged.

In December 1965 he had a further episode of renal colic and a calculus lodged in the left ureteric orifice. This was delivered at cystoscopy and the calculus was found to contain fibrin, phosphate and urate. While studies of his calcium and phosphate metabolism had been normal his serum uric acid in January 1966 was recorded as 9.4 mg/100 ml.

In July 1966 his general health was good. The white blood count was 13,400 per mm². There were occasional metamyelocytes and smear "basket" cells were present. The serum uric acid was 10.3 and 9 mg/100 ml on two occasions. In September 1966 treatment with allopurinol 100 mg thrice daily was instituted and the serum uric acid was 7.4 mg/100 ml in November 1966; 5 mg/100 ml in January 1967; and 4.6 mg/100 ml in June 1967; 5.1 mg/100 ml in November 1967; 4.9 mg/100 ml in February 1968.

Case 3. A lady aged 65 years, who presented in March 1967 with osteoarthritis of the right hip. She gave a previous history of many attacks of pyelonephritis. Investigation revealed urea retention with a blood urea of 72 mg/100 ml and she could not concentrate urine beyond a specific gravity of 1010. Three estimations of serum uric acid were recorded as 12, 7.5 and 11 mg/100 ml. Although there was no specific therapeutic indication she was given allopurinol 100 mg thrice daily and her uric acid level in June 1967 was 4.7 mg/100 ml and in August 1967 it was 6 mg/100 ml.

Treatment

The general lines of treatment, as discussed by Barnes and Mason (1967) and Scott Hall and Graham (1966) have been followed. Acute attacks of gouty arthritis have been managed with a regime of bed rest, a high fluid intake, a normal diet without alcohol and the use of Indomethacin 25 mg six hourly; or phenylbutazone 100 mg six hourly.

In the cases in which long term management was indicated it consisted of an enhanced fluid intake, a normal diet, avoidance of alcohol if at all possible, or if not, a consistently modest intake. Salicylate was not used and patients were advised against casual self medication. The uricosuric agent used has been Probenecid. This has been given in an initial dose of 500 mg twice daily combined with colchicine 0.5 mg also twice daily for three months. After the first three months the dosage of Probenecid would generally be increased to one gramme twice daily.

Allopurinol has been used as the main alternative or supplementary therapy. It has been given to two cases with multiple tophi in a dosage of 100 mg thrice daily in addition to Probenecid. As alternative treatment it has been given to two patients.

Effects of treatment

The benefit from treatment, with which the patient is mainly concerned, is freedom from the attacks of painful arthritis. The physician is concerned not only with this but with evidence to show that the underlying biochemical abnormality has been corrected and that the predisposition to recurrent attacks has been reduced or removed. Treatment to achieve these objectives should not increase the risk of renal damage and should be free from other toxic effects.

The reduction in the predisposition to attacks of arthritis is illustrated in Table IV,

Table IV
The reduction in the predisposition to attacks of arthritis

Before treatment	After treatment	Before treatment	After treatment
2 per 12 months	1 in 36 months	1 per 6 months	1 in 7 months
1 per 10 months	Nil in 29 months	1 per 10 months	Nil in 7 months
3 per 12 months	1 in 24 months	1 per 12 months	Nil in 6 months
1 per 12 months	Nil in 18 months	4 per 12 months	Nil in 5 months
4 in 14 months	Nil in 18 months	4 per 12 months	3 in 5 months
3 in 2 months	Nil in 12 months	5 per 12 months	1 in 5 months
1 attack	Nil in 9 months	1 attack	Nil in 3 months
1 per 8 months	Nil in 8 months	3 per 12 months	Nil in 3 months
3 in 3 months	1 in 8 months	2 in 12 months	1 in 1 month
1 per 10 months	4 in first 3 months	2 in 2 months	
	Nil in next 8 months		

where the incidence of attacks before uricosuric treatment is compared with the incidence of attacks after treatment was started, and while the patient was under supervision. There is a bias against this type of comparison being favourable as attacks of gout are frequently precipitated by starting treatment. All the patients who have received uricosuric therapy for twelve months or more show a reduction in the incidence of attacks. The benefit in terms of a reduced incidence of attacks, is less definite for shorter periods.

To the clinician the general improvement in confidence in walking, driving and increased freedom of movement is quite striking. Also impressive is the influence of treatment upon tophi. Four tophi in ears and one in a bursa disappeared; other tophi were seen to decrease in size.

The measurement of biochemical control used has been that of studying the hyperuricaemia and its response to treatment. This is only an indirect reflection of the quantity of miscible urates held in the body, and is subject to the influence of many environmental factors. Many of these factors have been recognised as capable of precipitating an acute attack of gout and include diet, fasting or starvation, alcohol, trauma and acute infections.

Measurement of the serum uric acid is an even more oblique assessment of the state of the underlying abnormality in cases of tophaceous gout, where a large proportion of the urate is not available in solution but is in crystals of varying size. With this difference in the physical state in which the chemical is held in the body it is not surprising that the response of the serum uric acid during the early months of treatment is different in tophaceous, compared with non-tophaceous, cases of gout. This is illustrated in Figure 2 which details the response obtained in the two groups of patients. The wide fluctuations observed during treatment of the tophaceous cases implies a coarse homeostatic mechanism in the handling of the urate held in tophi. It would appear that the tophi are not absorbed by a simple pressure gradient created by lower blood levels as increased excretion or decreased formation of urate with allopurinol therapy occurred.

It is also of interest to note that fluctuation of uric acid levels in the tophaceous cases occurred over fairly long periods but were less obvious with intensive therapy (i.e. Probenecid plus allopurinol). These swings of uric acid while the patient was under treatment did not correlate with clinical attacks of gout. In Table IV it is not possible to distinguish the tophaceous from the non-tophaceous cases, when the incidence of attacks is calculated for a period of treatment of more than twelve months. The tophaceous cases did have a greater tendency to attacks of gout in the first one to two months of treatment.

Side effects of Treatment

The regimes described in the management of the acute attack of gout and long term treatment have been remarkably free from side effects.

In the acute attack Indomethacin or phenylbutazone is much preferred to colchicine which, in addition to nausea, vomiting and diarrhoea, produces marked malaise. Even in the small dosage used in the prophylaxis of acute attacks it was associated with impotence in one patient.

In the long term management no side effects from Probenecid and allopurinol have

been encountered. It was confirmed that a modest elevation of transaminase levels occurred during Indomethacin treatment. No upset of liver function tests was recorded on long term management, and no rise of alkaline phosphates levels was found in patients on Probenecid or Allopurinol or on a combination of both drugs. This had been described by Scott, Hall and Graham (1966).

Discussion

The general features of this small series do not differ remarkably from other series. The age distribution and the grouping of patients within social groups reflect the special features of the population being screened. The association with obesity and alcohol indulgence is confirmed.

The variability of the patients' experience in terms of numbers of attacks of arthritis is quite striking and the length of some of the patients' histories before they have been started on persistent and adequate therapy is noteworthy. This suggests that there are still a considerable number of cases of gout who are not receiving adequate treatment by present standards.

The site of attack of gouty arthritis conform to the classical descriptions and the distribution of tophi is again not unusual. It was seen mainly on the cartilage of the ear and in the olecranon bursa, and as deposits in bone seen on radiographs of joints.

General investigation excluded causes of secondary gout. Radiological investigations especially of joints were useful in showing urate deposits in bones and in assessing the degree of development of secondary osteoarthritis. This was noted in three cases. Intravenous pyelograms were not helpful in this group. No example of chondrocalcinosis articularis was found.

The results of renal investigations included three cases of slight proteinuria; in two instances the patients had tophaceous gout. The occurrence of proteinuria probably represents a degree of renal damage caused by the deposition of urate crystals in the renal tissue. Inflammatory reactions to the presence of such crystals must be responsible for the damaged kidneys seen in late cases of gout. One patient in this series, and one of the cases of hyperuricaemia without gout, had suffered from renal colic caused by urate calculi. It has to be considered whether the treatment of gout with uricosuric drugs, with the passage through the tubules of increased quantities of urate, might increase the liability to crystal formation in the kidney tissue and of calculus formation in the renal pelvis or ureters. The formation of urate renal stones is one of the indications for treatment with allopurinol listed by Barnes and Mason (1967). In the present series the fact that the proteinuria detected in these three cases cleared during treatment might suggest that the regime of management used was not associated with an increased risk of progressive renal damage.

The inability of several of these patients to produce high urine concentration specific gravities is more difficult to interpret. It may be related to the type of renal damage discussed above, or it is possible that it is related to the abnormal handling of urate by the tubules which may be part of the pathogenesis of gout. Nugent, MacDiarmid and Tyler (1964) have shown that gouty subjects excreted in the urine less of a urate load, producing similar plasma urate levels, than a control group of normal subjects. That is, in some gouty patients the kidney is under-excreting uric acid.

Some observations have been offered on the urate levels found in the patients in this series. These fall into three groups. The first emphasises the observation, previously made by others, that the uric acid level can fall to normal levels quickly after a patient with acute gouty arthritis is put to bed rest and started on treatment. It is clear that this is true even when the patient is not intoxicated on admission. In early cases this rapid fall to normal levels may cause diagnostic doubt, particularly as some cases may not have uric acid levels taken on admission. This occurred in one case in this series who was initially admitted to a surgical ward.

The second group of observations offered relate to the adaptation of the knowledge of the influence of alcohol and starvation on uric acid metabolism. Lieber, Jones, Losowsky and Davidson (1962) showed that intoxication, with blood alcohol levels in the 200 to 300 mg/100 ml range was associated in normal subjects with elevated serum lactate levels and a rise of serum uric acid, which reached abnormal levels in half the subjects studied. MacLachlan and Rodnan (1967) demonstrated that the combined effects of starvation and alcohol produced marked rises in the level of uric acid in gouty subjects and in normal controls. Their subjects were starved for 24 or 48-hour periods and the alcohol was administered early in the morning of the day or days of fast. Many of their subjects suffered repeated episodes of gouty arthritis in relation to the studies and one patient took his own discharge from hospital.

It is suggested that a modified regime of fasting and alcohol might provide a diagnostic stress test to assist in substantiating the early diagnosis of gout. Such a test was used in two cases in this series. One, originally admitted to a surgical ward with trauma to the foot, was seen on the sixth day of admission when the foot showed sub-acute inflammation. The second was admitted for opinion having partially settled with colchicine given empirically by his general practitioner.

Clearly when acute inflammation of a joint is seen and facilities for examining joint fluid with polarised light exist as described by Currey (1968) then the diagnosis can be based upon the recognition and identification of the crystals, and the distinction made from pseudo-gout due to crystals of calcium pyrophosphate dihydrate, as described by McCarty, Kohn and Faires (1962), even in the absence of radiological signs of chondrocalcinosis.

The two doubtful cases and three normal controls were exposed to twenty-four hours starvation, from mid-day to mid-day, and were given two ounces of whisky after 20 hours starvation. Uric acid levels were studied during the period and in the three control cases the levels remained fairly steady and within the normal range. Both doubtful cases showed a rise to abnormal levels. Neither experienced evidence of gouty arthritis. It is suggested that this test or a modification of it might be of use where facilities for crystal identification do not exist.

The third set of observations on urate levels relate to the levels obtained during the follow up of the patients while under uricosuric treatment, allopurinol or a combination of both. In the non-tophaceous cases the urate levels fell to within normal range fairly quickly and remained within the normal range for the duration of follow up. The tophaceous cases did not conform to this pattern. They showed wide fluctuations in uric acid levels over prolonged periods amounting to months in one case. The fluctuations (Figure 2) were not associated with clinical attacks of gout and in Table IV the tophaceous and the non-tophaceous cases can not be separated. They both showed a reduced

Fig. 2. The two groups of cases. Left—tophaceous. Right—non-tophaceous. The pecked line is the upper limit of normal. Treatment: white—Probenecid 0.5 g b.d.; black—Probenecid 1 g b.d.; stippled—allopurinol 100 mg t.d.

incidence of attack of gout when this is calculated over a period of treatment greater than one year. The tophaceous cases have a greater risk of gouty attacks being precipitated early in treatment.

The tophaceous cases all showed a reduction in the size of tophi while under treatment. The causes of the fluctuations was not apparent during follow up. The group was free of alcoholism or grossly aberrant behaviour. They admitted to occasional lapses in tablet taking but insisted that treatment was generally taken regularly. The decrease in size of the tophi and decreased incidence of attacks of arthritis would also indicate that treatment was being taken.

The reason for the fluctuations of uric acid levels in the blood is not clear. While the urinary urate excretion was not measured the fact that treatment was being effective must indicate that uricosuric treatment was increasing the excretion of uric acid. Uricolysis by other routes was not measured but no reason is seen for this changing

during this period of time. It is considered that the explanation probably relates to the means by which the urate, held in tophi, is mobilised to become incorporated in the miscible body pool of urates. This process must be regarded as having a course homoeostatic mechanism.

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SIXTY YEARS AGO

THE evergreen subject of the 'soldiers load' was dealt with by Lieutenant-Colonel Melville in a most excellent paper which appeared in the Journal in March 1911. The paper is too long to reprint completely and in consequence we print two extracts. The first contains Melville's comment on the greatcoat and the second deals with the views of von Moltke which are quoted in the paper. Both relate to topics on which division of opinion still exists today.

THE LOAD OF THE INFANTRY SOLDIER

by

Lieutenant-Colonel C. H. MELVILLE, R.A.M.C.

"In addition to the tent there is the greatcoat. This, again, is extremely heavy, our army heading the list with a weight of 6 lb. 13 oz. I am very doubtful if this weight is well expended. A greatcoat is excellent as far as the waist; below that it hampers a man in walking, and rucks up, leaving his legs exposed when lying down. What is needed for the protection of the lower limbs is either a full cape or kilt hanging round the waist (Burberry makes something of this nature, and I believe the 5th Gurkhas thought at one time of adopting this pattern), or a pair of stout overalls, such as men use for motor bicycling, or policemen wear on point duty. These last would not hamper the man to any extent in his movements, and would certainly keep in position during the night. These could be supplemented by a rather full-skirted pea jacket, such as the "coat, warm, British," which all who have served in India know well. It is to be noted that the Norwegians do not issue a greatcoat, but provide in lieu of this a thick "sweater" weighing 1,027 grammes, plus a sleeping bag weighing 1,500 grammes."

The following quotation, which appeared in the paper, is from a memorandum of Von Moltke's. It was originally written in 1860, in connection with a full report furnished by Oberstleutnant Ollech, of the Prussian General Staff who accompanied the French Army in the Italian Campaign of 1859 as Military Attache.