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GOUT AND THE HAEMOGLOBIN LEVEL IN PATIENTS WITH CARDIAC AND RESPIRATORY DISEASE

BY

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Secondary polycythaemia as a cause of gout appears to be rare, for it receives no consideration in textbooks by such authors as Cohen (1955) and Talbott (1957), and has not been observed by such rheumatologists as A. G. Mason (1960, personal communication) or A. T. Richardson (1960, personal communication). That this is so may in part be due to selection, for the usual causes of secondary polycythaemia are long-standing cardiac and respiratory disease. The presence of gout in these conditions may be overshadowed by the major disease, and hence this association is more likely to be seen by cardiologists and chest physicians.

The relatively high frequency of gout in primary polycythaemia contrasts with the apparently low one in secondary polycythaemia; this is surprising, since the production of red blood cells, as shown by radio-active-iron studies, is several times the normal in both, and the blood volume similarly increased (Lawrence *et al.*, 1953). Although the relationship is not mentioned in standard textbooks of cardiology, there have been occasional reports of gout in those with congenital heart disease. An example was reported by Yü *et al.* (1953) in a man aged 50 with haemoglobin level of 23 g./100 ml. and a serum uric acid of 9.8 to 11.2 mg./100 ml. who had had cyanotic heart disease from birth. At the age of 35 he had his first attack of gout, and subsequently developed tophi and heart failure.

There are probably a number of unpublished cases, for the combination of gout with congenital heart disease has been observed once by A. St. J. Dixon (1960, personal communication), and several times by J. Somerville (1960, personal communication), who regards gout as a not infrequent complication of long-standing congenital cyanotic heart disease. Talbott (1959) has described an example of gout in a man with acquired valvular heart disease and secondary polycythaemia. This form of gout has been shown by Yü *et al.* (1953) to be secondary to an increased red-cell turnover with an overproduction of uric acid from nucleo-proteins. Yü *et al.* (1956) demonstrated, using ¹⁵N-labelled glycine, that there was a different metabolic pathway involved from primary gout.

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Another condition characterized by levels of haemoglobin as high as those in congenital heart disease is mountain sickness. It is of interest that Monge (1943) noted that excruciating limb pain was common in this condition, but he did not state whether this was gout, or if hyperuricaemia occurred, though certain mountaineers, the Creoles, are subject to gout (Loewy and Wittkower, 1937).

There appear to be few, if any, reported cases of gout secondary to the polycythaemia of respiratory disease. This may be because polycythaemia is an unusual response to the anoxia of respiratory failure (Wilson *et al.*, 1951). Moreover, whereas patients with congenital cyanotic heart disease may have gross polycythaemia for years, this is unusual with anoxic pulmonary disease. However, with the use of antibiotics the prognosis of those with respiratory disease has improved so that it is feasible that they may now live long enough to develop hyperuricaemia, and eventually gout. There are, it should be noted, other causes of an elevated serum uric acid in patients with cardiac or respiratory disease, such as the effects of anoxia, congestive heart failure, and diuretics on the kidneys, as well as familial gout and polycythaemia vera.

The studies presented here were undertaken to determine the frequency of gout in patients with cardiac and respiratory disease who might be expected to have secondary polycythaemia. As a guide the readily available haemoglobin estimation was arbitrarily considered, when it was 120% or more, to indicate secondary polycythaemia. A previous study by Lewis and Gardner (1960) on patients with long-standing chest disease showed that hyperuricaemia occurred most frequently at these levels of haemoglobin.

Materials and Method

The investigation was carried out at the Brompton Hospital for Diseases of the Chest. In 90 subjects (77 men and 13 women) who were known to have had a haemoglobin level of 120% (17.5 g./100 ml.) or more during the period January, 1958, to March, 1960, the case-notes, and where possible the patients, were studied for a suggestive history of acute joint pain or a previous diagnosis of gout. In addition, the records over the past few years of all the patients with gout known to the hospital were examined with reference to the associated haemoglobin level. Serum uric acid levels were measured by Folin and Trimble's (1924) modification of the method of Folin (1922). The range of normal accepted was 1.5 to 6 mg./100 ml. The haemoglobin level was estimated by the oxyhaemoglobin method, using a photoelectric colorimeter with a neutral grey solution as a standard. A haemoglobin of 100% was taken to equal 14.6 g./100 ml.

Gout in Patients with a Haemoglobin of 120% or More

The number of patients attending the Brompton Hospital with cardiac and respiratory disease, who also have a very high haemoglobin level, is relatively small. During the 27-month period of survey there were approximately 7,500 estimations of the haemoglobin concentration, and only 90 patients had a value of 120% or over. The cardiac and respiratory diseases in these patients are shown in Table I. The serum uric acid was known in 21 of these patients (Table II); the values

ranged from 4.1 to 9.5 mg./100 ml., and in seven patients there was hyperuricaemia.

On reviewing the case histories of the 90 patients it was found that in three a diagnosis of gout had already been considered; a further patient gave a history of recurrent gout on direct questioning. In none was there a family history of gout. The case histories are as follows:

Case A.—A man aged 37 in 1959 was known to have had congenital heart disease since infancy. In 1953 he complained of shortness of breath and coughing up blood. Cardiac catheterization was performed, and a diagnosis of

TABLE I.—*Type of Disease in 90 Patients with Haemoglobin Levels of 120% or Above*

Disease	No.
Cardiac { Congenital	26
{ Acquired	16
Respiratory { With cor pulmonale	17
{ Without	29
Others { Polycythaemia vera	1
{ Undiagnosed	1

TABLE II.—*Serum Uric Acid Levels in 21 Patients From the Group of 90 With a Haemoglobin Level of 120% or Above*

Disease	Case No.	Age and Sex	Serum Uric Acid (mg./100 ml.)	Hb (%)	Disease
Congenital cardiac	1	18 M	8.5	140	Pulmonary stenosis with shunt
	2	23 F	5.9	140	Fallot's tetralogy
	3	18 F	7.4	164	"Cyanotic heart"
	4	38 F	7.5	150	Pulmonary stenosis with shunt
	5	5 F	4.7	144	Transposition of major vessels
	6	22 M	5.1	120	"Cyanotic heart"
	7	16 F	6.4	128	Fallot's tetralogy
	8	37 M	6.6	141	Patent ductus reversed shunt
Acquired cardiac	9	43 M	9.5	149	Fallot's tetralogy
	10	68 M	4.9	120	Ischaemic heart
	11	53 M	5.5	122	"Cyanotic heart"
	12	60 M	5.1	127	Cardiomyopathy
	13	51 M	6.0	122	Rheumatic heart
	14	38 M	4.6	128	Cor pulmonale
Respiratory	15	57 M	6.0	140	" "
	16	58 M	6.9	128	" "
	17	53 M	5.3	122	Emphysema
	18	61 M	4.3	120	Carcinoma
	19	53 M	5.0	120	Cor pulmonale
	20	57 M	5.8	124	Emphysema
	21	60 M	4.1	120	" "

pulmonary hypertension with a patent ductus arteriosus was made. In 1959 he was found to have tuberculosis; accordingly, treatment with streptomycin, *p*-aminosalicylic acid, and isoniazid was begun. Later in the year he complained of pain in the right big toe, which was swollen and hot, with red bands of inflammation over the two terminal joints. The condition resembled acute gout; the serum uric acid was 6.6 mg./100 ml. and the haemoglobin level 150%. There was no immediate response of the pain to colchicine, but the toe slowly became less painful; after the attack the serum uric acid was 4.9 mg./100 ml. The same year he was readmitted to hospital with heart failure, haemoptysis, and pulmonary arterial thrombosis, and was treated with diuretics.

Case B.—A man aged 34 in 1942 was rejected from military service because of valvular heart disease. From this time he had effort dyspnoea, which became increasingly severe during 1956–7. Early in 1957 he dropped a plank on his big toe. The pain quickly subsided and he was able to work and walk, but a week later his great toe suddenly became red and painful, and he was unable to walk home. He was subsequently seen by an orthopaedic surgeon, who diagnosed gout and treated him accordingly. The toe was radiographed, and no bony injury was seen. The pain disappeared in two weeks. At a later date he was referred to the Brompton Hospital because of his dyspnoea, and a diagnosis of mitral and aortic stenosis was made. His haemoglobin level was 122%. An operation was performed, since when he has been given diuretic therapy. When seen in 1960 he said he had had no further attacks of joint pain,

his haemoglobin concentration was 112%, and his serum uric acid 6 mg./100 ml.

Case C.—A man aged 30 in 1947, when he first complained of fatigue and coughing up blood, was diagnosed as suffering from Fallot's tetralogy. In 1952 his haemoglobin level was 152%. When seen at the Brompton Hospital in 1957 he gave a history of pain in his left big toe, which during an attack was sore, red, and extremely tender. He was later admitted to hospital, where he was noted to be plethoric, cyanosed, and clubbed, but had no gouty tophi. His haemoglobin was 149%. During his stay in hospital he had another attack, this time in his right big toe, and was treated with colchicine. In April, 1960, he was readmitted to hospital. His haemoglobin level was 134% and the serum uric acid 9.6 mg./100 ml.

Case D.—A man aged 58 in 1959 gave a history of winter bronchitis for 15 to 20 years, and progressive and incapacitating shortness of breath for four years. For three to four years he had recurrent but infrequent attacks of gout in his big toe. On examination he was plethoric, cyanosed, overweight, and dyspnoeic. There were no tophi, and a radiograph of the left big toe showed no abnormality. He had clinical and electrocardiographic evidence of cor pulmonale and radiological changes of emphysema. His haemoglobin was 128% and the serum uric acid 6.9 mg./100 ml.

None of these patients had gouty tophi. However, these do occur, as is shown by the following case which was under the care of Dr. J. F. Goodwin at the Hammersmith Hospital.

A man aged 49 had been short of breath since childhood. For 24 years he suffered from chronic bronchitis and for 16 years he had four or five attacks of gout each year. There was no family history of gout. In 1957 he was admitted to hospital for investigation, and was noted to be plethoric and cyanosed. He had gouty tophi in the left ear, and an effusion in the right knee. Cardiac catheterization revealed a patent ductus arteriosus, with pulmonary hypertension. His haemoglobin level was 163%, R.B.C. 9,500,000, P.C.V. 85%, W.B.C. 7,000/c.mm., and platelet count 72,000/c.mm. The red-cell mass was 4,000 ml., the plasma volume 2,060 ml., and the bone-marrow showed normoblastic hyperplasia. The serum uric acid was 16.2 mg./100 ml., and the blood urea 36 mg./100 ml. He was treated with probenecid, calcium aspirin, and colchicine, but the latter gave only limited relief.

Gout in Patients with Haemoglobin Level Below 120%

In the records of several thousand patients there were 13 in whom the diagnosis of gout was made. In all but one the haemoglobin levels were below 110%. Details of this exception are as follows:

A man aged 64 had had for 30 years respiratory symptoms, initially of cough and latterly of increasing dyspnoea. Over the same period he had had recurrent gout. On examination he was red-faced and cyanosed, and had small gouty tophi in his ears. His pulmonary disease was mainly emphysema. He had noticed no connexion between the two disorders, but when he was admitted to hospital severely ill with respiratory insufficiency he had an associated flare-up of his gout in both knee-joints. His haemoglobin level was 118% and the serum uric acid 7.8 mg./100 ml.

Discussion

In the four patients with gout and a haemoglobin level above 120% it can fairly be assumed that the combination was closely related to the polycythaemia from cardiac or respiratory disease. These patients are also likely to be treated with diuretics, since secondary polycythaemia is often associated with heart failure (Cournand, 1950; Wilson *et al.*, 1951). The role of such diuretics as the thiazide derivatives, which

may precipitate familial gout in these circumstances, may be difficult to assess, particularly in a first attack of gout. It can be argued that the combination of secondary polycythaemia, renal impairment, and diuretic therapy is a favourable one to cause gout, whether the patient is predisposed to familial gout or not.

It is reasonable to suppose that gout occurring secondarily to polycythaemia should be seen especially in the severe forms of disorder treated by cardiac and respiratory departments, into which, moreover, patients are not admitted for gout alone. The total incidence of gout in patients attending the Brompton Hospital is not known, for only the in-patients are coded for disease. It is known that the frequency of gout in the general population is low even though the disease is often unrecognized. In a general-practitioner survey based on consultations the figures were 1.2 per 1,000 for men and 0.4 per 1,000 for women (Logan and Cushion, 1958). Higher incidences may be expected in patients attending a hospital. In a general-hospital series of 200 patients, not admitted for the treatment of gout, there were two with gout (Beckett and Lewis, 1960), and in 200 consecutive in-patients with a haemoglobin level below 120% attending the Brompton Hospital there were also two with gout. Even so, the incidence of four diagnosed cases of gout in the 88 patients with cardiac or respiratory disease (Table I) and a raised haemoglobin concentration appears to be higher than expected by chance.

For a diagnosis of gout secondary to polycythaemia certain criteria can be postulated. The cardiac or respiratory disease which causes the polycythaemia should be long-standing and should antedate the gout. In such cases a family history of gout is not usually obtained. There should be clinical evidence of plethora, with an elevated haematocrit and haemoglobin level, but no abnormality of the white cell or platelet count or splenomegaly to suggest polycythaemia vera. In these patients the arthritis clinically resembles primary gout, but tophi should not always be expected, as they are often an index of long-standing gout. The response of the joint pain to colchicine is not a reliable diagnostic test, since in an appreciable minority the expected benefit is not seen (Dixon, 1960, personal communication). If these criteria are satisfied the diagnosis is likely, especially in congenital cyanotic heart disease where the polycythaemia is most extreme (Whitby and Britton, 1950) but cannot be proved without special studies.

Recognition of the significance of the joint pain in those with heart disease is of importance in distinguishing it from other causes of arthritis such as rheumatic fever, as well as from vascular thromboses and the embolic manifestations of bacterial endocarditis.

Summary

The incidence of gout was investigated in 90 patients who had a haemoglobin level of 120% or more, all but two of whom had cardiac or respiratory disease. There were four patients in whom a diagnosis of gout was made. It was considered that a haemoglobin level of 120% indicated the presence of secondary polycythaemia.

In a group of 200 patients with a cardiac or respiratory disease and a haemoglobin level below 120% there were two patients with gout.

The serum uric acid levels were estimated in 21 of the 90 patients with a haemoglobin level above 120%. The highest values of serum uric acid were seen in patients with congenital cyanotic heart disease.

It is considered that the importance of recognizing the association of gout with cyanotic heart disease is to distinguish it from other forms of arthritis, vascular thromboses, and bacterial endocarditis.

The association of gout in patients with a high haemoglobin level was seen more often with cardiac than respiratory disease.

I thank the consultant staff of the Brompton Hospital for allowing me to study their patients, and Dr. J. F. Goodwin, of the Hammersmith Hospital, for permission to include one of his patients. I wish to acknowledge the technical assistance of Miss J. Gardner in estimating the serum uric acid.

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SUCCESSFUL CARDIAC MASSAGE AFTER MYOCARDIAL INFARCTION IN A CASUALTY DEPARTMENT

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Cardiac massage after cardiac arrest under anaesthesia or during minor operative procedures has been commonly practised for many years. The first successful attempt was by Igelsrud in 1901 (Keen, 1904), and in 1953 Stephenson *et al.* were able to collect 1,200 cases. However, cardiac massage has been much less often attempted for cardiac arrest after myocardial infarction. The first successful attempt was made in 1953 by Reagan *et al.* (1956), and since then fewer than a dozen successes have been reported (Reagan *et al.*, 1956; Beck *et al.*, 1956; Celio, 1956; Mozen *et al.*, 1956; Hannon *et al.*, 1957; Bloomfield and Mannick, 1958; Nickel and Gale, 1959; Walton, 1960). The only one recorded in this country (Walton, 1960) occurred in a ward patient with a known myocardial infarct. In