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A CASE OF SPLENIC LEUKAEMIA COMPLICATED WITH GOUT AND ALBUMINURIA.

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INSTANCES of leukaemia with fulminating attacks of uratosis are sufficiently rare to justify an account of a well-marked case.

The patient, aged, 35, a fitter by trade, has been under treatment for eighteen months for splenic leukaemia with frequently-recurring joint troubles. In May, 1901, he was attacked by malaria while at a port in the United States. A year or two afterwards he was laid up suddenly with pains in the knee which came on after a heavy turn of night work. In January, 1905, he was admitted as an in-patient at the General Hospital, Bristol, with pyrexia, joint pains, marked anaemia and breathlessness. The big toe, ankle, and right knee were red, painful, and swollen, while the wrist, elbow, and fingers were also slightly affected. The urine was 1010, acid, and showed a trace of albumen, but no sugar. The retinal discs were normal. The spleen reached below the umbilicus, and a blood count showed red cells 2,610,000, white 223,200, and numerous myelocytes. There was a systolic murmur at the apex of the heart. The lungs were normal except for slight dullness at the bases. There was no tenderness over the sternum.

Under treatment by sodium cacodylate the red cells rose by March 21st to 3,260,000, the white fell to 71,000. The haemoglobin reached 48 per cent., but the myelocytes remained as high as 26 per cent. Treatment by the Roentgen rays was kindly undertaken by Dr. W. Kenneth Wills, and steady improvement followed. The spleen decreased in size, the blood became practically normal except that 3.2 per cent. of myelocytes were still present, and in June he resumed work, but came up for the x-ray treatment periodically till November. In February, 1906, he relapsed, and again came under treatment; the leucocytes now reached 134,000 and the myelocytes 27 per cent. By April the white cells had fallen to 33,000 and the myelocytes to 12 per cent. The spleen later on diminished and could no longer be felt below the ribs.

Frequent slight attacks of arthritis have occurred during all this time in various joints. There is no marked deformity, but slight creaking in the right knee and a deposit of urates along the extensor tendons of both index fingers near the proximal interphalangeal joints. One of these discharged for a time. In March a tophus found on the right ear was punctured. The contents proved to be sodium urate. After an inflammatory attack the joints show a fine desquamation. Colchicum, thymine acid, and other remedies have been employed, and appear to hasten the termination of an attack.

For some time there was reasonable doubt as to the nature of the arthritis. Garrod's serum test was tried unsuccessfully. The moderate pyrexia at first and the transference of attacks from joint to joint was in favour of true rheumatism. However, when uratic deposits were found the question was settled. He can give no history of gout in his family, and never suffered from serious illness before the malaria. As to the causation of the kidney trouble, he has no recollection of scarlatina as a child. As a fitter he has constantly used red and white lead in his work, but remembers no attack of colic, and his gums show no blue line. The heart is slightly enlarged, the apex $\frac{1}{2}$ in. external to the nipple line, the aortic second sound is accentuated, and there is a systolic murmur at the apex. The systolic blood pressure is 110 mm., and the vessels seem healthy. The liver dullness extends from the sixth rib to about a couple of inches below the costal margin.

The urine is clear, acid, has rarely any deposit, and has always shown a definite cloud of albumen. The quantity varies between 30 and 50 oz., and the specific gravity between

1010 and 1020; numerous granular and a few hyaline and fatty casts have been noted. No prolonged examination of the other constituents has been made, but I may give one report made during a period when the gout was quiescent:

"Quantity of urine 48 oz., urea 1.3 per cent., so that the total excreted was 272.8 grains of urea. The nitrogen represented by the purin bodies was found to be 0.0286 per cent., which gives 5.99 grains of purin nitrogen in twenty-four hours." During an attack the urea was 205 grains and the purin nitrogen 3.8 grains.

A severe inflammatory attack of gout occurred during May, 1906, when he had gone home to resume work; many joints were affected, and fresh uratic deposits appeared.

Blood Counts.

	Feb. 3, 1905.	Feb. 25, 1905.	March 21, 1905.	April 17, 1905.
Red cells ...	2,610,000	—	3,260,000	3,460,000
White cells ...	223,000	82,000	71,000	61,200
Polymorphonuclears ...	34.2	10	49	65
Myelocytes ...	25.2	79	30.4	15.8
Basophiles ...	16.6	—	10.2	4.4
Nucleated red cells ...	3.8	4.0	0.2	1.6
Haemoglobin ...	—	—	48	—

	June 7, 1905.	Sept. 12, 1905.	Sept. 19, 1905.
Red cells ...	4,850,000	3,540,000	3,750,000
White cells ...	20,000	67,000	44,800
Polymorphonuclears ...	77.8	—	—
Myelocytes ...	3.2	—	—
Basophiles ...	6.2	—	—
Nucleated red cells ...	—	—	—
Haemoglobin ...	60	—	52 per cent.

	Feb. 14, 1906.	Feb. 26, 1906.	March 3, 1906.	March 27, 1906.	April 10, 1906.
Red cells ...	3,220,000	2,310,000	3,040,000	3,400,000	3,310,000
White cells ...	134,000	116,000	108,000	140,000	63,000
Polymorphonuclears ...	51	44	532	48	66
Myelocytes ...	27	29	25	32	12
Basophiles ...	0.8	0.6	1.6	1.2	3
Nucleated red cells ...	—	—	—	—	—
Haemoglobin ...	50	40	55	55	60

The interest of the case depends largely on the importance we attach to the retention theory of gout. Garrod laid down that during acute gout uric acid exists in the blood in excess, and that very little appears then in the urine. As the attack passes off, it reappears in the urine, and sometimes in large amounts, but during the quiet intervals the excretion is deficient. This retention of uric acid by the kidneys forms an essential feature of gout. In leukaemia there is an excess of urates in the blood unequalled in any other condition except gout and temporary states such as pneumonia. Why then, it was asked, do we not get a deposit of urates and other symptoms of gout in leukaemia? Simply because the kidneys there are active, and the uric acid excretion is vigorous. The so-called quadriurates have not time to change into the gelatinous biurate, and this into the crystalline form. It is certainly curious that cases of gout combined with leukaemia are decidedly rare. I have only found three recorded—one mentioned by Roberts and two by Duckworth, but in two of the three gout had existed long before. Roberts says: "I am not aware that any case has been recorded in which gout supervened as a clear consequence and result of splenic leukaemia. In the present instance the evidence as to priority is not clear. The patient had first an attack of malaria, which is well known to be a precursor of leukaemia. Gowers, for instance, found a history of malaria in 20 per cent. A year later he was attacked by

gout. He cannot, of course, tell us whether the leukaemia began before or after that. The breathlessness of the advanced stage certainly came on later.

If we look on the kidney trouble, the gout, and the leukaemia as all following upon the malaria, there would on Garrod's theory be reason to consider the gout as the result of the failure of the kidneys to excrete the enormous amount of uric acid produced by the leukaemia. It is certain that xanthin bodies are found in great quantity in the blood of leukaemics, and their urine, which is generally free from albumen, shows a great excretion of the purin bodies, and a notable excess of uric acid, even $1\frac{1}{2}$ gram daily and upwards, while the urea is little altered. This patient's excretion is very scanty for a leukaemic.

It is open to us to say that the essence of gout does not consist in the retention of uric acid and its deposition in the joints when it is in excess. In short, we may call this process a uratosis, and think of gout as a toxæmia together with an inflammatory reaction which causes the precipitation of uric acid, or shows in some cases no arthritic symptoms. This is after all akin to the old idea of "suppressed gout," but in the present case we have an excess of urates in the blood together with damaged kidneys. Why, then, were they not deposited quietly without inflammatory symptoms, or what was the cause of the inflammation which, according to the toxin theory, preceded and led to their deposition?

We know that in some forms of kidney disease uric acid is found in excess in the blood. Von Jaksch found this was the case in nine patients out of twelve, and the effect of lead in southern England in producing both kidney disease and gout is well known. I say in southern England⁴ for, as Ewart and others point out, lead does not produce gout in Scotland and the north, so that the gout of lead workers must include some other factor. It may be said, too, that retention of uric acid cannot be the sole cause, otherwise gout would be far more frequent in ordinary Bright's disease; and, again, in typical gout the kidney trouble is not always that of advanced or even of definite structural disease. Still, the fact remains that in gouty persons the excretion of the endogenous purin bodies is distinctly low, and when a diet rich in purins is given the excretion does not rise to the extent it does in the healthy, while in an attack of acute gout it fails still more. We know that renal impermeability is not always the same as structural disease, though they coincide in some cases.

If a case of leukaemia, with their huge excess of endogenous purins, can be shown to produce the symptoms of gout when some forms of kidney disease are present, and only then, there is much reason to think that gout depends in some degree on renal failure. On the other hand, there may be in gout some peculiarity of the purin bodies which renders them difficult of excretion. In that case we may yet meet with gout and leukaemia in the presence of healthy kidneys, and the present patient's condition will then be held to be merely accidental. Without attaching too much weight to an isolated case, it is still significant that no one appears to have met with gout or uratosis combined with leukaemia when the kidneys were healthy.

For the careful blood enumerations I am indebted to Messrs. Fred. Leighton and Short.

LOCAL ANAESTHESIA BY NOVOCAIN.

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EVERY year the sphere of local anaesthesia is widening. Its use is no longer confined to minor surgery, and general anaesthesia has ceased to be an essential for major operations. Recently great advances have been made in the technique of injection of local anaesthetics; much has been done to ensure their purity, and we have learned the value of combining them with drugs which have a vaso-constrictor influence. Chemists are constantly endeavouring to produce a substance which is anaesthetic but yet non-irritant and non-toxic. Cocaine is an excellent anaesthetic, but it is not safe. There is always an element of uncertainty in its action; with some patients it produces a condition of painful or pleasurable excitement, with others a dangerous degree of respiratory and cardiac

depression. Moreover, it often causes destructive changes in the tissues at the seat of injection. Eucaine is more constant in its action, but it is much less potent. Stovaine was an advance on the older anaesthetics, but it, also, is not without general toxic and local irritant action.

In dentistry the use of drugs which are liable to exert a general toxic action is greatly to be deprecated. It is also most undesirable to introduce irritating substances into the gums for the purpose of ensuring painless extraction, for the mouth is a very septic cavity and any lowering of the tissue vitality may determine sloughing. Twenty years' experience did not give me either certainty or confidence in the older local anaesthetics, for failures sometimes occurred in a most unexpected and disconcerting fashion. Since December 5th, 1906, I have made extensive use of a new synthetic compound called novocain, a combination of an ethyl ester of para-amido-benzoyl with the active principle of suprarenal extract.

I first administered it to a clergyman who preferred not to take nitrous oxide gas, and who wished me to use cocaine. As I thought he was not a suitable patient for that drug I explained to him that I had a new substance which I had been told was safer and more efficacious. The tooth was a left superior wisdom, very much broken down, and quite firm; as the position was not an easy one, I had to use the mouth mirror to enable me accurately to insert the needle. The patient was delighted with the result, and declared the pain of extraction was very slight. Considering the difficulty of the injection, I was encouraged to proceed to an exhaustive trial of the drug. I did not specially select my cases. They were all either unsuited or unwilling for general anaesthesia. The following are a few of my most interesting experiences:

CASE XIV.

A medical man with a fractured left superior first molar declined "gas," and I am very glad he did, for I could not have extracted his tooth within the time one gets with a "gas" anaesthesia. I believe this was the most difficult extraction I have ever done. The crown broke off level with the gum in such a way that the palatine blade had to impinge on an inclined plane, with the result that I could not move the tooth outwards owing to slipping of the inner blade. I tried several times; between each attempt I expressed my regret, while he smilingly reassured me that he felt nothing. Eventually the roots separated, but even then I had difficulty owing to their curves. Afterwards he said he had felt nothing till the last root was being extracted. One injection of $\frac{1}{4}$ grain had ensured complete anaesthesia for almost twenty minutes.

CASE XII.

This patient was very neurotic and her teeth were very firmly fixed. In 1894, when she first came under my care, the futility of "gas" anaesthesia in such cases was distressingly borne in upon me. I subsequently removed the remaining teeth then requiring extraction under ether, but the patient was considerably collapsed for some hours after the anaesthetic. In February, 1907, I painlessly extracted at three sittings all her superior premolars, canines, and incisors with the aid of novocain. There were no untoward after-effects whatsoever.

CASE XXI.

This patient was a surgeon who, in the hard palate, had, arising from a first left superior molar, an abscess which was extremely sensitive to the slightest touch. A 10 per cent. solution of novocain was kept for six minutes in apposition to the surface of the abscess, which was then incised. There was absolutely no pain; the patient said the sensation of the knife cutting the abscess wall was distinctly pleasant. This case indicates the value of novocain for other than purely dental operations.

CASE XLII.

This patient was a medical man, whose left superior wisdom tooth presented the following condition: In the buccal roots the nerve was alive and hypersensitive; in the palatal root the nerve was dead and septic. I used novocain pressure anaesthesia, and painlessly drilled out the live nerve. Thus I was able painlessly to perform one of the most agonizing operations in dentistry. Sometimes I have applied novocain by pressure to live nerves and then immediately removed them without pain, but, owing to the position of the cavity, the application may be very difficult, and the procedure is not always possible.

As I gained experience my initial diffidence in the use of the drug gave way to complete confidence. Standing before a mirror, I have injected novocain into my own gum and painlessly removed a firm and offending right inferior incisor.