A CASE OF SPLENOMEDULLARY LEUKÆMIA.

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THE extraordinarily interesting paper on the surgery of the spleen by Sir Berkeley Moynihan in the last number of the *BRITISH JOURNAL OF SURGERY* prompts me to place on record the following case. I offer it as a further example of the changes seen in the blood and bone-marrow in association with splenic disease. In addition, the case seems to present some curious features: (1) A marked amelioration of symptoms after a course of *x*-ray treatment to the spleen; (2) A spontaneous fracture of the jaw; (3) Associated 'myelopathic albumosuria'.

The patient, Mrs. X., was sent to me by Dr. E. W. Lewis, of Southport. She is 52 years of age, and, until the present illness, has been fairly healthy but never robust. There is nothing apparently pertinent in the family history. She has two children, 21 and 10 years old respectively.

Eleven years ago, in February, 1910, she began to suffer from anæmia, and was diagnosed as a case of splenomedullary leukæmia. She was ordered a complete rest, and at the end of a year she says she "was in fair condition except for the anæmia and the enlarged spleen". One doctor stated that in February, 1911, "there were many myelocytes in the blood", and another doctor, in May, 1911, that "the hæmoglobin was over 70 per cent, the red cells 3,800,000, with obvious and considerable leucoeytosis".

In November, 1912, she was sent to Dr. Thurstan Holland for *x*-ray treatment for the condition; and I am indebted to him for the following notes.

"Duration of the enlarged spleen about two years. Spleen well over the mid-line. Splenic notch above and to the right of the umbilicus. Lowest border 7 cm. below the level of the umbilicus.

1912, November and December,	11 exposures to spleen.		
1913, January to December,	27	"	"
1914 " "	10	"	"
1915 " "	9	"	"
1916, January to May 26,	2	"	"

Each dose was about 1 S. with a tube of medium hardness, but filtered through three layers of thick felt.

<i>Blood-counts:</i>	Nov. 29, 1912	Reds 3,400,000	Whites 72,000.
	Dec. 29, 1912	" 2,160,000	" 80,000.
	Jan. 15, 1913	" 4,900,000	" 7,000.
	Mar. 11, 1913	" 3,904,000	" 7,000.
	Nov. 11, 1913	" 4,600,000	" 5,900.

Spleen: November, 1912, after three treatments, spleen a good deal smaller.

December 13, 1912: Spleen well over to the left side. Then follow many records of 'rapid diminution of the spleen'.

December, 1913, spleen not to be felt.

1916.—Patient perfectly well. Spleen not felt.

The same note on May 26, 1916, when all *x*-ray treatments were stopped."

Dr. Holland also saw the patient in June, 1919. His note runs: "Quite well. Good colour. Spleen normal in size".

To return to the patient's own narration. At the end of 1916 she felt perfectly well, her blood-counts were normal, and she could walk ten miles with comfort.

For over three years she remained apparently perfectly well.

In July, 1920, she noticed that she was more easily tired. A little albumin was found in the urine at this date. Some weeks later she complained of pain in the right iliac fossa. It was noted that the spleen was a little enlarged. She ran a temperature

for six weeks. Although there was no examination of blood for the Widal reaction, this attack was diagnosed as typhoid fever.

On December 11, 1920, she noticed for the first time a slight swelling in the lower jaw. Eating and talking were painful. The swelling gradually increased in size until on Christmas day, whilst eating a piece of bread and butter, she felt her jaw crack. Since then she has felt no further cracking. A loose tooth in the region of the swelling has been removed, and on two occasions the swelling was incised under the impression that it was a dental abscess. Blood only exuded from the incisions.

The foregoing is as complete an account as I have been able to gather of her past history. The interesting points are the diagnosis; the disappearance of the splenic enlargement; the disappearance of the leucocytosis; the attack of fever; the presence of albumin (or was it really albumose?); and the development of the jaw swelling.

FIG. 150.—Skiagram showing the tumour at the site of fracture.

When I saw her on January 21, 1921, she had an obvious fracture of the jaw just behind the last molar tooth on the right side, with the usual displacement of the posterior fragment inwards and the distal fragment downwards (*Fig. 150*). The fracture was not at all painful. She could talk comfortably and eat remarkably well.

An *x-ray* photograph showed that there was a tumour at the site of the fracture. This tumour was about the size of a walnut. It had absorbed and thinned out the compact bone, which was slightly expanded at the fracture. The mucous membrane was normal over the growth. There was no attempt at union. Otherwise the patient looked fairly well. She was anæmic, and the spleen could easily be felt about one inch below the costal margin. The spleen felt somewhat harder than normal.

No sign of any primary malignant growth was detected in breast, abdomen, uterus, etc.

A blood-count kindly taken by Dr. J. C. Matthews showed red blood-cells 4,500,000, normal morphologically; white cells 12,000 per cmm.

Differential Count.			Per cent.	No. per c.mm.	Normal.
Polymorphs	..	..	54.5	.. 6540	.. 6500
Lymphocytes (large and small)	..	..	33	.. 3960	.. 2500
Monocytes	..	..	12	.. 1440	.. 800
Basophils	..	..	5	.. 60	.. 100

In other words, distinct relative and marked absolute lymphocytosis. Slight monocytosis. No myelocytes or metamyelocytes found. Polymorphs show free division of nuclei. Arneeth count = right-hand deviation.

Examination of the urine shows the so-called myelopathic albumosuria. Equal parts of salicyl-sulphonic acid and urine give a flocculent protein precipitate which clears on boiling and reappears on cooling. If the urine is acidulated with a drop of weak acetic acid and heated in a water-bath, the protein is thrown down as a white precipitate

at about 60° C. This precipitate almost clears on boiling, and reappears on cooling (Bence-Jones' proteid).

Similarly, nitric acid and hydrochloric acid both precipitate the proteid in the cold, which precipitate is cleared on boiling. In this particular urine, however, the protein is not precipitated by half saturation with ammonium sulphate. The urine also shows the presence of urobilin; an indication of hæmolytic or some tissue deficiency. There is no evidence of sugar, bile pigments or salts, or acetone. (I am indebted to Dr. Coope for the urinary examination.)

This curious protein body in the urine (myelopathic albumosuria of Bradshaw) was first noted in the urine by Bence-Jones in 1847. It is a proteose, and is said to be one of the products of proteolysis on the way to the peptones and polypeptides. It occurs in the urine in cases where there is any absorption of partially digested pus in cases of empyema, pneumonia, etc., and in cases where there is tissue breakdown. It is also said to be diagnostic in myelomata and sarcomata of bone. Dr. T. R. Bradshaw¹ states that "the presence of albumose in the urine is one of the few signs of disease which can strictly be called pathognomonic, admitting, as far as our present knowledge goes, of only one interpretation; and it is usually for many months the only indication of the true nature of the disease from which the patient is suffering, and may for a long time be the sole evidence that he is seriously ill at all. Multiple myelomata, the morbid condition of which it is the sign, goes on to a fatal termination with the certainty of malignant disease, and no measure hitherto attempted appears to have the slightest influence in retarding its progress".

An x-ray of the chest revealed no definite shadows of other myelomata in the ribs, the usual situation of these growths.

The interest of this case lies in the diagnosis, and the question whether the two diseases leukaemia and myelopathic albumosuria are related, or independent of each other.

Further points of interest are the extraordinary effect on a leukaemic spleen of a course of x rays; and, lastly, the consideration of the prognosis. I think it grave.

ADDITIONAL NOTE—MAY 6, 1921.

This patient died on May 4, 1921.

Unfortunately, no post-mortem was obtainable. The tumour of the jaw increased to about twice its size, but there was no ulceration of the mucous membrane. There were no clinical signs of other tumours. During the last week of her life she suffered from persistent vomiting and diarrhoea. Myelopathic albumosuria was still present a month before she died.

REFERENCE.

¹ T. R. BRADSHAW, *Brit. Med. Jour.*, 1906, Nov. 26.

A CASE OF TUMOUR OF THE CAROTID BODY.

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Tumours of the carotid body are uncommon, and relatively few cases have been recorded in surgical literature. Keen and Funke¹ collected twenty-nine cases in 1906, and Callison and Mackenty² added a further thirty-one to the series in 1914. A perusal of the published accounts of these cases produces a feeling of doubt as to whether they have all been correctly diagnosed, and it is probable that the actual number of cases recorded in which the diagnosis of a tumour arising from the carotid body is clearly correct is considerably less than sixty. It is therefore worth while recording the history and treatment of another case in which the origin of the tumour was beyond doubt.

The patient, a married woman of 31, had noticed a lump in the left side of her neck