normal pressure, with one cell per c.mm. and a protein content of 32 mg. per 100 ml. The blood Wassermann reaction was negative. The electroencephalogram showed rather excessive low amplitude, slow activity.

At operation (Mr. N. O. K. Gibbon) on February 28 a single plum-coloured growth the size of a pea was found in the head of the pancreas and removed without undue difficulty. Convalescence was uneventful. Post-operative hyperglycaemia was not observed, the fasting blood-sugar values falling between 95 and 114 mg. per 100 ml. A glucose-tolerance test done ten days after operation gave the following results: fasting, 95 mg.; half an hour after 50 g. glucose, 130 mg.; 1 hour after, 120 mg.; 1½ hours after, 100 mg.; 2 hours after, 100 mg. per 100 ml.

Section of the tumour revealed an insulinoma, which was thought to be benign. The patient was discharged, symptomless, on March 20. When seen in the out-patient department six weeks later he felt very fit. He was working regularly and was symptom-free. The nystagmus had disappeared, but the muscle-wasting was unchanged.

C. J. WILLIAMS, M.D., M.R.C.P.,
Assistant Physician, Walton Hospital, Liverpool. 9.

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Foster Kennedy Syndrome Produced by Myelomatous Deposits in the Skull

The complete Foster Kennedy syndrome comprises an association of anosmia, primary optic atrophy and central scotoma, and contralateral papilloedema. The essential features of the syndrome are the atrophy and papilloedema, either or both of the other two components often being absent. The syndrome was first described in detail by Foster Kennedy (1911). It is usually produced by olfactory groove or sphenoidal meningiomas or by frontal-lobe tumours (Walsh, 1947). Its occurrence in any other condition is a rarity, and it is accordingly commonly seen in the 40–60 age group. Clearly, the syndrome may be due to any space-occupying lesion near to the olfactory tract.

The case described below is exceptional in that the compression was due to myelomatous deposits. Multiple myeloma also occurs most frequently after the age of 40 (Snapper, 1949). In this case the patient was unusually young, and the manner in which the case presented was more suggestive of a cerebral tumour than of myelomatosis.

CASE REPORT

A male steel worker aged 34 was first seen on July 30, 1953, complaining of generalized aches and pains which he had had for six months. These had at first affected his back and pelvic region. Later his shoulders and neck were involved. After these pains had been present for four to five weeks he developed severe frontal headaches. All these symptoms, with the exception of the headache, disappeared spontaneously five to six weeks prior to admission to hospital.

For five to six weeks before admission he had noticed that his left eye was beginning to protrude. There was no associated deterioration of vision. At the same time he developed intractable vomiting, which occurred 10 to 15 times a day. He also complained of general weakness, loss of weight, and recent nocturnal frequency of micturition. On inquiry he admitted that his food had recently become tasteless.

On examination he was clinically anaemic, but otherwise appeared in fair general health. There was marked proptosis and epiphora of his left eye. The patient had no sense of smell. There was neither diplopia nor ophthalmoplegia. The left fundus showed primary optic atrophy with

an occasional blotchy haemorrhage near the disk. In the right fundus the whole disk was ill-defined, the veins were prominent, and the complete lack of contour and blurring of the edge of the disk indicated a moderate degree of papilloedema. In addition several blotchy haemorrhages were present, mostly near the disk. The visual fields were normal, but the visual acuity of the left eye was diminished to 6/9, while that of the right eye was 6/6. He was tender over the fifth and sixth ribs on the left side. No further abnormalities were found.

Several laboratory investigations were carried out. The urine contained Bence Jones protein, but showed no other abnormality. The blood urea was 155 mg. per 100 ml. and his urea clearance was 15% of average normal standard clearance. The findings in the serum were: alkaline phosphatase, 13 K.A. units; acid phosphatase, 3 Gutman units; total protein 8.8 g. per 100 ml. (albumin 5.7 g., globulin 3.1 g.); sodium, 135 mEq/l.; potassium, 6.5 mEq/l.; alkali reserve, 18 mEq/l.; chloride, 88 mEq/l.; calcium, 12 mg. per 100 ml. The peripheral blood examination showed: haemoglobin, 51% (7.5 g. per 100 ml.); red cells, 2,500,000 per c.mm.; C.I., 1; leucocytes, 6,000 per c.mm. (neutrophils 3,000, eosinophils 60, lymphocytes 2,340, monocytes 600); no plasmacytes were seen; platelets, 250,000 per c.mm. (Lempert); E.S.R. (Westergren), 124 mm. in one hour.

Marrow smears from the right and left iliac crests and from a radiologically evident myelomatous deposit in the left clavicle showed gross infiltration with cells of the plasmacytic series, consistent with a diagnosis of myelomatosis.

Pentamidine isethionate was given by intramuscular injection on alternate days to a total of 4 g. over a period of six weeks. The patient also received deep x-ray therapy, 2,500 r depth dose to the anterior fossa of the skull, between August 12 and 28.

After these measures the proptosis and papilloedema disappeared, whilst the vomiting gradually ceased. It was at first thought that the vomiting may have been due to chronic renal failure, but as the blood urea level did not fall concurrently with the decline in vomiting it was concluded that the latter was caused by the raised intracranial tension.

COMMENT

This case presents several unusual and interesting features. Multiple myelomatosis in a man of 34 is rare. The youngest in Bayrd and Heck's (1947) series of 83 cases was 37. Though myeloma deposits are often found in the skull, producing the characteristic radiological appearance, it is relatively rare to find the tumour extending beyond the confines of these bones. This was observed in only two cases in the series quoted above. Accordingly, only exceptionally is intracranial tension raised in myelomatosis. Snapper (1949) states: "Although myeloma so often involves the skull, no convulsive seizures have been observed and the optic disks never show evidence of increased intracranial pressure." In the present case there were definite signs of raised intracranial tension—namely, papilloedema, retinal haemorrhages, and vomiting. Lumbar puncture was thought to be inadvisable in these circumstances, and so no direct measurement of the C.S.F. pressure was made. The only component of the Foster Kennedy syndrome that was lacking was the central scotoma.

I wish to thank Professor E. J. Wayne for permission to publish this case; Dr. E. K. Blackburn for the interpretation of the marrow smears and other haematological data; and Dr. G. M. Wilson for his help and encouragement.

W. K. C. MORGAN, M.B., Ch.B.,
Formerly House-physician, Sheffield Royal Infirmary.

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