hemicolectomy, and the continuity of the intestine was restored by means of an isoperistaltic, side-to-side ileo-transverse anastomosis.

The patient made a good recovery from the operation and was eventually discharged to his home when the fractures had united. He later returned to work.

COMMENT

This case is of interest, for it was practically a week before any symptoms or signs of an intra-abdominal lesion appeared. Had it not been for the multiple fractures it is quite possible that this patient would not have been admitted to hospital, or, if he had been, he might have been discharged before the intestinal perforation became manifest.

It is of interest also to speculate on the possible mechanism of the perforation of the caecum. We believe that minor damage to the caecum resulting in a haematoma in its wall occurred after the injury. It is well known that intra-abdominal traumatic lesions are common at the sites of junction of fixed and mobile intestine. The retroperitoneal haematoma probably caused some degree of ileus and caecal distension. The latter eventually became severe enough to cause the bowel wall to give way at the site of a haematoma.

We are indebted to Mr. J. Grant Bonnin, orthopaedic surgeon to the Central Middlesex Hospital, for permission to publish this case.

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Neurological Presentation of Glandular Fever

Neurological complications of glandular fever are not common. The following case is recorded because of its rarity in that cerebellar dysfunction provided the presenting symptoms.

CASE REPORT

The patient, a male shop-assistant aged 18, had been fit and well until about three weeks before admission to hospital, when he had an influenzal illness, with frontal headache, sore throat, and general malaise, which was treated with aspirin. Four days later he developed mild jaundice which lasted a day or two. He was confined to bed for two days, and subsequently felt well. Two weeks before admission he noticed that his legs were becoming weaker, and that there was a gradually increasing loss of balance and incoordination of his arms and legs. His handwriting became irregular, and while standing or walking he had to hold on to something to prevent his falling. In himself he felt well. There was no disturbance of speech, hearing, vision, appetite, bowel action, or micturition, nor any other complaint.

His past history included only chicken-pox and whooping-cough as a child. There was no known contact with any similar cases, with or without jaundice. There was no history of food or other poisoning, or of exposure to leptospires. His family were all fit, apart from his mother, who had a lymphatic obstruction of the right leg. There was no family history of neurological or other disease.

On examination when admitted to hospital on January 14, 1952, he was a slender but well-built youth. He was afebrile. His pulse was 68. No abnormality was detected in the cardiovascular and respiratory systems. The throat was healthy. Abdominal examination did not reveal any abnormality. The pupils gave a sluggish reaction to light, but otherwise were normal; there was no nystagmus; the fundi and cranial nerves were normal. In the upper limbs there was slight weakness of both triceps, and slight weakness and wasting of the small hand muscles; the biceps-jerks were present, the triceps-jerks weak, and the supinator-jerks absent; there was mild ataxia, but no muscle tenderness or sensory disturbance. The lower limbs showed gross bilateral pes cavus, slight weakness of extensors of hips, moderate ataxia, absence of both knee- and ankle-jerks, plantar responses both flexor, and no muscle tenderness or sensory disturbance. There were no meningeal signs or trunk weakness. Abdominal reflexes were present and equal. There was a very ataxic gait, and Romberg's sign was present. Multiple, discrete, non-tender glands were palpable in both anterior and posterior triangles of the neck, from clavicles to mastoids, and also small glands in the axillae. The spleen was not felt. The epitrochlear glands were also palpable.

A provisional diagnosis was made of mild peripheral polyneuritis, with additional cerebellar signs, the result of a recent infection, probably glandular fever.

Investigations.—Cerebrospinal fluid: clear; pressure 170 mm.; no block; protein, 80 mg./100 ml.; chlorides, 690 mg./100 ml.,; the film showed a very occasional lymphocyte, no organisms; culture was sterile; Wassermann reaction, negative; Lange colloidal gold curve, normal. Blood: haemoglobin, 108%; red cells, 5,620,000; white cells, 6,600 (polymorphs 52%, small lymphs 19%, large lymphs 25%, monocytes 3%, and eosinophils 1%); sedimentation rate: 8 mm. in 1 hour (Wintrobe); Wassermann reaction, negative. Paul–Bunnell test (January 16): sheep cells agglutinated to a titre of 1 in 16,384; guinea-pig kidney absorption titre, 1 in 8,192. Liver-function tests (January 24): alkaline phosphatase, 19 K.A. units/100 ml.; van den Bergh direct reaction, negative; 1.4 mg. bilirubin/100 ml.; thymol turbidity, 2 units/100 ml.; cephalin cholesterol, +++. An *x*-ray film of the chest did not show any abnormality.

Progress was satisfactory. The patient was well in himself the whole time, and throughout was afebrile. He was discharged home one month after admission, no specific treatment being necessary. When last seen as an out-patient 14 weeks after the onset of the symptoms he was in good health; the ankle-jerks, however, were still absent. There were almost no palpable glands, and the Paul–Bunnell and liver-function tests, which had been performed frequently, were now within normal limits.

COMMENT

Glandular fever is not an uncommon disease, and the presence of possible neurological complications is becoming increasingly recognized. It is seldom that the neurological complications provide the main features of the illness, and it is probably extremely rare for cerebellar dysfunction to provide the presenting symptoms.

Crowther (1951), reporting a case of glandular fever with encephalitis, reviewed the literature and could find only 24 cases of glandular fever confirmed by the Paul–Bunnell reaction and involving the nervous system since 1931, although several clinically suggestive cases without a positive Paul–Bunnell reaction have been described. Similarly, abnormal cerebrospinal fluids have been found without clinical manifestations. Of the 24 cases he quoted, 4 had a pure encephalitis, the others showing encephalomyelitis, cranial nerve lesions, meningitis, and peripheral nerve lesions. Another case with cerebral complications has since been reported by Librach (1952), who discussed possible ways in which the nervous system may become involved, and suggested that there may be an allergic origin.

The present case is unusual in several respects: the neurological symptoms were the presenting features, not merely complications; at no time after admission to hospital was the patient febrile; the cerebrospinal fluid showed only an increase in the protein content. The suggestion is therefore made that in cases of atypical infections of the nervous system the diagnosis of glandular fever should be considered.

I am indebted to Dr. I. Gordon, under whose care the patient was admitted, for his constant encouragement.

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