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COMMENT

Although no specific signs of glandular fever were present on admission, in the light of previous experience a white-cell count was done; this showed a characteristic mononucleosis. The Paul-Bunnell test on the blood was at first borderline, but later showed a diagnostic rise in titre to over 1 in 128, remaining at that level for seven days, and then falling quickly to 1 in 16. The maximum titre corresponded with the occurrence of gland and splenic enlargement, but with recovery from the nervous symptoms.

In contradistinction to the previous case, the cerebrospinal fluid was abnormal and showed 89 lymphocytes per c.mm. three weeks after the first complaint of headache, and when the neurological signs had gone, minimal gland and splenic enlargement occurred—that is, the lymphadenopathy followed the nervous signs.

The neurological manifestations have been reviewed briefly by Tidy (1952). He stresses the importance of the timing of the Paul-Bunnell test, which may be occasionally positive in the cerebrospinal fluid. It will be recalled that the present patient was admitted as a possible non-paralytic poliomyelitis case. It would seem eminently worth while to do routine white-cell counts and Paul-Bunnell tests in these cases, for in this way more might be learnt about the behaviour of this strange malady, and its true incidence appreciated. The relation, too, of the heterophil antibody titre to the stage and severity of the illness would also prove of value, especially if tested for both in blood and cerebrospinal fluid simultaneously, for detailed information regarding both in relation to one another and to the illness is lacking.

Clinically the most striking feature, apart from the bizarre nature of the symptoms, is the rapidity with which a structure that appears to be seriously affected recovers, and this is especially so in the neurological complications. Recovery, too, is usually complete and permanent, and without residual defects or deformities. Death is an uncommon termination of glandular fever, but in the absence of an extensive literature on the neurological complications it is difficult, if not impossible, to dogmatize on the prognosis. Generally speaking, it can be said to be excellent.

There have been few reports in the literature of the pathological findings in fatal cases with C.N.S. involvement. Dolgopol and Husson (1949) describe in detail such a case which occurred in a young white woman, aged 19, whose main symptoms were diplopia and dysphagia. Macroscopically, the brain was moderately congested, the meninges equally so, but the grey matter of the cord was diffusely haemorrhagic. Microscopically, the most important features in the brain were severe degenerative changes in the nuclei of the third and fourth cranial nerves and in the ventral portion of the inferior reticular nuclei just above the inferior olives. The spinal cord showed extensive recent perivascular haemorrhages limited to the posterior horns in the upper cord but also present in the anterior horns and white matter in the lumbar segment, where some demyelination was also present.

Thomsen and Vimtrup (1939) also described six fatal cases complicated by central respiratory paralysis, one of which showed similar degenerative changes in the medulla.

In view of the current interest in bulbar poliomyelitis it might be as well to consider in differential diagnosis the bulbar involvement which occurs in glandular fever, and to perform routine Paul-Bunnell tests in such cases.

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Medical Memoranda

Acute Meningo-Encephalitis as a Complication of Glandular Fever

I should like to add the following case to the one I recorded previously (Librach, 1952).

CASE REPORT

An unmarried female fitter aged 19 was admitted as a case of poliomyelitis on July 12, 1952. For five days she had complained of the gradual onset of headache associated with nausea and vomiting. Previous illnesses included measles, pertussis, and chicken-pox. Tonsillectomy had been performed in infancy. She had had no recent or remote inoculations. There was no one else ill at home.

On examination her temperature was 101.6° F. (38.7° C.), pulse rate 84, and respirations 16. She was not acutely ill. Her ears, nose, and throat were normal. A few small neck glands were present. The spleen was not felt. Apart from slight neck stiffness, there were no neurological signs. Treatment included sulphonamides and penicillin for the first 48 hours.

On July 14 the temperature was normal and no meningeal signs were elicited. On July 18 she was pale, and recrudescence of headache and vomiting occurred. There was marked oral foetor and furring of the tongue. Fine lateral nystagmus to the right was seen. On July 20 a normal menstrual period began, associated with continued vomiting and an irregular blotchy erythema of the trunk. Two days later she was quite well. On July 20 minimal gland enlargement affecting the cervical, axillary, and inguinal groups was noted, together with a palpable spleen. On August 2 she was discharged home. When seen again as an out-patient on August 16 she looked well, but felt tired.

Investigations.—July 15: White-cell count, 7,200 (polymorphs 40%, lymphocytes 44%, monocytes 16%). Glandular fever cells present. Cerebrospinal fluid: 10 ml. slightly opalescent fluid; cells, 89 per c.mm., all small lymphocytes; protein, 30 mg. per 100 ml.; chlorides, 760 mg. per 100 ml.; sugar normal; globulin normal; culture sterile; no organisms seen on direct examination. Paul-Bunnell tests (blood); July 17, 1 in 64; July 26, 1 in 128+; July 31, 1 in 128+; August 18, 1 in 16.