Discussion

During or immediately preceding cerebrospinal meningitis, meningococci have often been found in the conjunctiva. In some instances the eyes were apparently healthy (Randolph, 1893; McKee, 1908, 1909), but others showed a purulent conjunctivitis (Koplik, 1904). This type of conjunctivitis has become progressively rarer with treatment by specific antiserum and later by sulphonamides, so that it is now practically never observed (Lewis, 1931; Tillet and Brown, 1935). McKee (1908) discussed the probabilities of whether the conjunctiva acted as a portal of entry for the neisseriae or became invaded secondarily.

The occurrence of the conjunctivitis immediately before the onset of meningitis (Koplik, 1904) and its appearance in nurses attending patients with meningitis (Smith, 1905; Reese, 1936) support the first contention. There is no inherent reason why the conjunctiva should not, like the nasopharynx, harbour and even be infected by air-borne meningococci, but obviously the chance is less, inasmuch as a current of air containing the organisms is not drawn by each respiration over the conjunctiva. Meningitis resulting from meningococci which have gained access to the body via the conjunctiva, if it occurs at all, must be very rare compared with the usual sequence of events described by Wilson and Miles (1946), beginning with entry of the meningococci into the nasopharynx and their spread to the meninges either direct or by the blood stream.

Conjunctivitis due to *N. meningitidis*, unassociated as in this case with meningitis in either the host or any of his known immediate contacts, has often been recorded, and Stuart and McWalter (1948) give more than a dozen references. They themselves described six cases, from which they isolated five strains. Four of these were investigated serologically and, in contrast to the case here recorded, all belonged to Group I. It is a matter for speculation whether in the absence of treatment these patients would subsequently have developed meningitis. At present meningitis is usually due to Group I, and most of the strains found in the nasopharynges of persons who have not been in contact with cases of meningitis belong to Group II (Mackie and McCartney, 1948).

In the interests of public health as well as of the patient himself, rapid identification of the organism in neisserial conjunctivitis is of obvious importance. Difficulties due to absence of facilities for immediate culture may be overcome by the use of the thioglycollate method of transport (Stuart, 1946). As Stuart and McWalter suggest, meningococcal conjunctivitis is more frequent than commonly imagined, is often erroneously diagnosed as gonococcal, and may be as severe clinically as gonococcal ophthalmia. Although both conditions respond to sulphonamides, the sociological "follow-up" required is entirely different.

Summary

A case of purulent conjunctivitis due to *N. meningitidis* Group II is recorded and discussed. Contacts were examined for neisseriae without success.

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REFERENCES

- Koplik, H. (1904). *Med. News*, **84**, 1065.
 Lewis, P. M. (1931). *Sth. med. J.*, **24**, 101.
 McKee, H. (1908). *Ophthalm. Rec.*, **17**, 438.
 — (1909). *Ibid.*, **18**, 304.
 Mackie, T. J., and McCartney, J. E. (1948). *Handbook of Practical Bacteriology*, 8th ed. Edinburgh.
 Randolph, R. L. (1893). *Bull. Johns Hopk. Hosp.*, **4**, 59.
 Reese, F. M. (1936). *Amer. J. Ophthalm.*, **19**, 780.
 Smith, D. (1905). *Arch. Ophthalm.*, **34**, 481.
 Stuart, R. D. (1946). *Glas. med. J.*, **27**, 131.
 — and McWalter, D. (1948). *Lancet*, **1**, 246.
 Tillet, W. S., and Brown, T. McP. (1935). *Bull. Johns Hopk. Hosp.*, **57**, 297.
 Wilson, G. S., and Miles, A. A. (1946). *Topley and Wilson's Principles of Bacteriology and Immunity*, 3rd ed. London.

Medical Memoranda

Pulmonary Tuberculosis Complicated by Amoebic Hepatitis

The following case history may be of general interest because of its differential diagnostic problem, which is somewhat unusual, and its moral, which is becoming increasingly common.

CASE HISTORY

The patient, a married woman aged 36, has been suffering from pulmonary tuberculosis for at least ten years. A left-sided artificial pneumothorax was induced some two years ago, and she recently came for treatment of a fresh spread in the right lung. She had had appendicectomy twenty years ago.

For the last eight years she has been complaining of sharp, dragging pains in the right iliac fossa and lately also in the right epigastric region; the pains were independent of food, were worse when she stood up, and had grown in intensity during the last few years. There was moderate flatulence, but the bowels were regular and the stools macroscopically normal. She gave a vague history of three or four sharp attacks of diarrhoea in the course of the last nine years. The first attack, lasting four or five days, was in 1939; the others, lasting two or three days, occurred between 1939 and 1944. She thinks that on two occasions she passed some blood and mucus.

The patient has never been in tropical countries, and, apart from occasional pre-war trips to Paris and the South of France, her only foreign journey was a summer cruise in Dalmatian waters in 1937. The abdominal symptoms had been variously diagnosed as those of duodenal ulcer and abdominal tuberculosis. They have not responded to treatment.

On examination the caecum was ballooned, doughy and tender, and could be elicited by pressure on the colon as far as the splenic flexure. No mass or rolled-up omentum could be palpated. A small area of localized tenderness, which had been unknown to the patient, could be detected in the right lobe of the liver, about 1 in. (2.5 cm.) to the right of the xiphoid process. This tenderness was more pronounced in the intercostal space above than on the palpable liver-edge. On screening, the right diaphragm was observed to be slightly elevated and its movement was somewhat diminished. Sigmoidoscopy disclosed a congested oedematous rectal mucosa, several submucous haemorrhages on the haustra, and a few small round healed ulcers at 4 in. (10 cm.).

Investigation revealed slight hypochromic anaemia; W.B.C. 3,600, with normal distribution; van den Bergh quantitative test, 0.8 mg. per 100 ml.; nicotinic acid tolerance (Erdei, 1947, 1948), 450 mg.; galactose tolerance and thymol turbidity normal; cephalin-cholesterol test, weakly positive. On examination of the faeces two typical *Entamoeba histolytica* cysts could be demonstrated in the sixth specimen.

A course of ten injections of emetine, with concurrent penicillin and diodoquin and consecutive emetine bismuth iodide and carbarson, was given. After the second emetine injection the liver became very tender, but all tenderness disappeared after the sixth injection, thus justifying the diagnosis of chronic amoebiasis with hepatitis.

Further attacks of diarrhoea occurred some time after the improvement from the anti-amoebic treatment. First *Giardia lamblia* was demonstrated; afterwards, when the diarrhoea became worse and sigmoidoscopy revealed superficial ulceration of the rectal mucosa, *Shigella flexneri* (Newcastle-Manchester strain) was grown from a sigmoidoscopy swab. Several stool cultures proved to be negative. Sulphasuxidine treatment was given, and the patient is now free of the complaint, and her motions do not exceed two daily.

COMMENT

In the above case abdominal tuberculosis figured much too prominently in the differential diagnosis, and the correct diagnosis was arrived at only because it was realized that chronic amoebiasis is an increasingly common condition which must be considered when dealing with a very wide range of abdominal and general conditions. This case also demonstrates the value of a rapid liver-function test such as that of nicotinic acid tolerance.

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REFERENCES

- Erdei, A. (1947). *British Medical Journal*, **1**, 822.
 — (1948). *Lancet*, **1**, 368.