

## Clinical Notes :

### MEDICAL, SURGICAL, OBSTETRICAL, AND THERAPEUTICAL.

#### A CASE OF ACUTE SEPTIC MENINGITIS OF OTITIC ORIGIN; COMPLETE RECOVERY.

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THIS case is chiefly remarkable because of recovery.

The patient was admitted into hospital under my care on March 26th, 1918. A mastoid operation had been performed at a casualty clearing station 14 days previously. The notes stated that he had had sudden pain in the right ear with discharge; was delirious for a few hours; vomited. At the operation "offensive material" was found in the antrum and cells.

On admission on March 26th patient complained of headache and pain in the ear. Pus was freely draining through a small tube issuing from the mastoid incision and also from external meatus. Temperature, 99° F. No history of old-standing ear disease. On a daily antiseptic toilet the antro-tympanic cavity cleared up, and progress was good in every way. On May 14th he had an attack of benign tertian malaria which responded readily to quinine. From the 19th to 24th, diarrhoea; stools negative to bacilli and protozoa. On last date diarrhoea had ceased; complaint of headache and some aural pain; slight mastoid tenderness. Temperature rose to 100°. On the 25th temperature normal, but other symptoms had increased. From examination with aural speculum it was difficult to make out the anatomy of the operation cavity, so I reopened it under general anaesthesia. The antrum and tympanum had been thrown into one cavity and their roofs were absent, exposing a large area of dura covered with granulations. The antro-tympanic cavity was thoroughly cleansed and enlarged. No pus was found.

On May 26th patient had a much better night; the intense headache had gone. He now progressed well until June 5th, when he had a rigor; temperature 103°. Calomel gr. 3, aspirin gr. 15, followed by quinine hydrochloride gr. 20, given. On June 6th temperature 102°, pulse 94. Complained of headache; vomited in afternoon; no pain in ear; no mastoid tenderness; slight pain and rigidity back of neck; very slight Kernig's sign present. Reflexes normal; no definite signs of intracranial complications.

On June 7th temperature 101.2°, pulse 90; headache intense; slight retraction of head; Kernig's sign definitely present; no aural pain. Lumbar puncture; a test-tube full of cerebro-spinal fluid under pressure and cloudy in appearance evacuated. Microscopical examination on the spot showed numerous pus cells. Subsequent report from pathologist showed Gram + staphylococci. Under general anaesthesia the antro-tympanic cavity was again reopened and thoroughly explored; no pus found. The brain substance of the middle fossa was explored with a needle; negative result. On June 8th and subsequent days patient was distinctly more comfortable. Headache and pain were absent, but he was drowsy; Kernig's sign persisted. The temperature gradually came down.

On the 12th there was a turn for the worse. Temperature rose to 103.6°; headache, pain in back and legs, was increasingly drowsy, and became slightly delirious. Another lumbar puncture; coconut-milk-like fluid withdrawn under pressure, containing far more pus cells than before. Subsequent report from pathologist showed Gram + diplococcus present. Immediate improvement in all symptoms followed. The temperature remained in the region of 102° for three days and then came down. On July 1st he began to get up from his bed, and when evacuated to hospital ship on the 14th, could walk several hundred yards without fatigue. Kernig's sign was much less marked.

Cases of definite septic meningitis with pus in the cerebro-spinal fluid which recover must be very rare. I have never met one in my own practice. There are two suggestive points. The organisms found in the cerebro-spinal fluid were of low virulence, and the resisting power of the patient was much weakened by malaria. Had his powers of resistance not been weakened by malaria, it is more than likely that no suppurative lesion of the meninges would have occurred. From over three years'

experience in Macedonia I have no hesitation in saying that complications of middle-ear suppuration are more common<sup>1</sup> than they would be in England under the same circumstances, and the reason for this is the deleterious effect of malaria on the resisting powers of the individual. Organisms of low virulence are thus able to bring about infections, but this very fact gives the patient a chance of ultimately overcoming them. Also noteworthy is the marked relief afforded by each lumbar puncture.

#### A CASE OF PERITONITIS FOLLOWING A NON-PENETRATING WOUND OF THE ABDOMINAL WALL.

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THAT peritonitis may result from a non-penetrating wound of the abdominal wall, with the abdominal viscera showing no gross lesion, seems proved by the following case.

##### Account of Case.

Gunner J. H., recently wounded, was admitted into hospital on Oct. 2nd, 1917; very collapsed and appeared to have lost much blood. Entrance wound, about  $\frac{1}{2}$  in. in diameter, in right buttock just external to posterior superior spine of ilium. Exit wound, about 1 in. across, in lateral sector of right iliac region. The patient was treated in the resuscitation ward for some time; no signs of improvement; it was decided to operate lest internal hæmorrhage was going on.

I opened up the anterior wound and found that the right lateral peritoneum was exposed in track of missile. Careful examination by Lieutenant Camps, who assisted, and myself, revealed no penetration of abdominal cavity. The right iliacus muscle was severely lacerated. Also fracture of the right iliac bone, involving separation of greater part of crest with part of body; comminution not very great. The damaged tissue was cut away, but not in region of exposed peritoneum, as it would have necessitated opening the abdomen. Bipp was rubbed gently, in a thin layer, into the raw surfaces. The muscles and skin were partly closed in layers, but greater part of wound was left open and very lightly packed with gauze. The entrance wound was then dealt with, the edges being excised and subjacent injured tissue clipped away; bipp was applied. Prior to operation a catheter was passed; small quantity of clear, rather concentrated urine was drawn off; urine not examined.

On the following day the patient seemed better; rather persistent vomiting. It is unnecessary to detail general treatment; various remedies, including alkaline treatment, were employed. On the morning of Oct. 4th patient was much worse. 1 c.cm. of pituitrin was given hypodermically and sodium bicarbonate and glucose solution intravenously. Death took place about midday. During all this time very little urine passed. There did not seem any very definite indication to open the abdomen, especially as one felt certain that the parietal peritoneum had not been punctured. The man would almost certainly have been unable to stand such a procedure.

*Post mortem.*—The following conditions were found. Peritonitis, apparently rather subacute, involving chiefly lower half of small intestine, pelvis, and region of cæcum. There were a good many plastic adhesions and patches of adherent lymph; small quantity of dark reddish semi-purulent fluid in pelvis. No *B. coli* infection smell. The stomach was not distended; tendency for intestines to be somewhat collapsed, except jejunum, but this was not actually distended. The bladder contained only a little fluid. The appendix was bound down over brim of pelvis by a recent soft plastic adhesion; otherwise appeared healthy. The parietal peritoneum in relation to the wound was very carefully examined; no perforation found. The small intestine was carefully examined from the duodeno-jejunal flexure downwards; no perforation; cæcum and ascending colon also appeared intact. The rest of the large intestine, not in relation to the wound, appeared whole. On opening the abdomen no free gas was detectable. The kidneys appeared rather cloudy. A swab from fluid in the pelvis was taken, but there had been already contamination, being taken on a second examination.

##### Inferences.

It would seem that the intraperitoneal infection must have taken place in one of three ways: 1. By direct spread of infection from the wound through damaged parietal peritoneum. No swab was taken from the wound, but on the day after operation it looked fairly clean. 2. By

<sup>1</sup> See also THE LANCET, 1918, i., 704.