MASTOIDITIS LIMITED TO THE DEEP SUBANTRAL CELLS.

BY

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THE case here reported illustrates the potential difficulties and vagaries of mastoid surgery and the importance of Mouret's¹ teaching as to the necessity of exploring not only the superficial but the deep subantral cells for the possible extension of disease in every case of acute mastoiditis.

A professional man, aged 42, was referred to me on June 29th, 1926, suffering from severe pain in the left ear and left temporal region. It had been present for five days; a discharge from the left ear which had been in evidence for three days had ceased. I encountered some difficulty in viewing the left tympanic membrane owing to exostoses. It was hyperaemic, but the anatomical landmarks were still apparent and the hearing was but slightly impaired. The pulse and temperature were normal, but as there was marked tenderness over the mastoid and temporal regions I at once performed a linear paracentesis of the posterior segment of the membrane and confined the patient to bed. There was no discharge from the ear then or later.

During the succeeding days the patient complained of intermittent radiating pains in the left mastoid and temporal regions, especially at night; these were sufficiently severe to necessitate the administration of sedatives to induce sleep, but they tended to become less, as also did the mastoid and temporal tenderness. A four-hourly temperature and pulse record failed to reveal the smallest abnormality.

On July 9th I noticed very slight paresis of the left side of the face, only evident during emotional activity. As this became much more marked on the following day, I decided to explore the mastoid bone. This proved to be a matter of extreme difficulty owing to its density, the superficial and anterior position of the sigmoid sinus (which lay only a few millimetres below the cortex and immediately behind the posterior meatal wall), and to the downward and lateral inclination of the middle cerebral fossa.

The mastoid antrum, which lay at more than one inch from the surface, could only be reached by the use of a 2 mm. chisel in the very small triangle of dense bone which was circumscribed by the middle fossa, the sigmoid sinus, and the posterior wall of the bony meatus. It was small and contained a somewhat slightly turbid fluid. By applying pressure to the antral floor with a small sharp spoon I succeeded in opening into a small cellular area which lay in close proximity to the descending limb of the facial canal and which was found to contain purulent fluid. Cultural examination of both the antral and cellular contents yielded a pure culture of the *Streptococcus mucosus*. A careful examination failed to reveal any extension of the disease to the temporo-zygomatic cells. A small rubber drainage tube was inserted and retained for four or five days. The facial paralysis disappeared very quickly and the bone cavity was rapidly obliterated.

The deep subantral cells, which may often be prolonged into the interior of the pyramidal portion of the bone, in the space subtended by the labyrinth, the jugular bulb, the cerebellar fossa, and the facial canal, communicate with the inner half of the floor of the mastoid antrum. There may or may not be a connexion between them and the more commonly affected superficial subantral cells, which, as in this case, may be entirely absent. When both sets of cells are simultaneously affected the more apparent symptoms resulting from lesions of the external cells may mask those pertaining to a lesion of the deep, which latter, more especially in the absence of a direct communication with the superficial cells, may continue to advance. These deep lesions, which evolve slowly, may elude the attention of the less observant clinician and end by causing extensive havoc in the depths of the bone, implicating, on the one hand, the aqueduct of Fallopius or the cerebellar fossa (meninges or terminal part of the lateral sinus), or, on the other, the deeper planes of the neck.

REFERENCE.

¹ Mouret, J.: *Strasbourg Medical*, eighty-fourth year, No. 1.

Memoranda:

MEDICAL, SURGICAL, OBSTETRICAL.

HENOCH'S PURPURA IN AN ADULT.

THE following case appears of sufficient interest to be placed on record, since all the symptoms seemed to point to an "acute abdomen" until the purpura appeared.

A man, aged 44, came to my surgery in July, 1926, complaining of pain in the stomach, which was worse after meals; there was no vomiting and no pain on palpation. I gave him a rhubarb and soda mixture; one week later the pain had gone and he stated he was now quite well again.

On November 26th, in the afternoon, while the patient was walking he was suddenly seized with an acute abdominal pain "doubling him up"; this became gradually worse and he passed what he stated to be a good motion. The pain remained severe, and at 9 p.m. he looked collapsed, but the pulse was 74 and the temperature 97.4°. On examination his stomach was hard, and moving little if at all on respiration. By palpation it was difficult to make anything out as the muscles were rigid, but pain was found in the right iliac fossa. On percussion there was a resonant note all over; the liver dullness was lessened and the tongue was furred.

The patient had a bad night, and on the next morning the pulse was 76 and the temperature 97.2°. The pain, severe all over the abdomen, which was still rigid; it was worst in the left iliac fossa. The urine was smoky; it contained blood and slight albumin. On the next morning the pain continued severe, the pulse 82, and the temperature 100.1°. The abdomen was still hard and painful to palpation all over; he had been given small doses of calomel and salts but had vomited them, the vomit being bile-stained. After two enemata with no good result, he passed in the evening a motion which contained obvious blood, some bright red and some dark. In the early morning of November 29th he had severe pain over the left base, resembling pleurisy, but at 11 a.m. he was much better, the pulse and temperature were normal and there was less rigidity of the abdomen. At 11 p.m. he was reported to be unconscious after a bout of wild excitement in which he tried to get out of bed. I found the patient asleep, bathed in perspiration, and covered with a typical purpuric rash. Since then he never looked back; the pain entirely disappeared, and the urine and faeces contained no blood.

I think this is clearly a case of Henoch's purpura in an adult, but until the rash appeared I at first suspected, from the previous history, a perforation; when the temperature rose on November 26th acute appendicitis seemed possible. I notice that in the 1925 edition of Taylor's *Medicine* it

is stated that this purpura may be confused with intestinal obstruction, but this was put out of court in my case by the motion on the 26th; had not the pulse remained quiet I would certainly have sent him into a nursing home for surgical treatment. The pain and rigidity were obviously due to haemorrhage into the bowel wall.

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DIVERTICULITIS OF THE CAECUM.

THE following case appears to be worthy of record owing to its unusual features.

A woman, aged 54, had vomited and then was attacked with severe pain in the right lower abdomen at about 9.30 a.m. on October 31st, 1926. She had felt slight abdominal discomfort on the previous day, but put it down to "indigestion."

On examination there was rigidity and tenderness in the right iliac fossa and a small lump was felt in the appendix region. The pulse was 120 and temperature 99°. With these signs and symptoms the case was considered to be a small appendicular abscess with dense adhesions.

Operation.—The abdomen was opened in the afternoon by Mr. L. E. Barrington Ward, F.R.C.S., by a right paramedial incision, and the rectus muscle was retracted inwards. The appendix was apparently normal, but there was a large quantity of free fluid in the abdomen. On palpating the caecum, which was covered with plastic lymph, showing that definite peritonitis was present, a mass was felt which appeared to be incorporated in its wall. An alternative diagnosis was made of diverticulitis or new growth. The caecum and part of the ileum was excised and the terminal portion of the small intestine was anastomosed to the ascending colon. The patient stood the operation well and made a good recovery.

The pathological report was that the peritoneum covering the caecum was acutely inflamed and great engorgement was present. Opening into the caecum about one inch from the appendicular orifice was a diverticulum about half an inch long. The mucosa of the diverticulum was inflamed and contained a faecalith. Cross-section of the caecum showed the mucous membrane of the diverticulum partly ulcerated and extending through the muscular coat of the caecum as far as the subperitoneal.

This case is of interest on account of the uncommon situation for diverticulitis to attack the large bowel, and illustrates how difficult an apparent appendix operation may turn out to be in general practice.

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