AN UNUSUAL CASE OF MENINGITIS ASSOCIATED WITH HAEMATURIA

BY

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(With Special Plate)

Cases of acute haemorrhagic nephritis associated with meningococcal meningitis are uncommon. In the majority of cases they are associated with purpura, and are, as a rule, severe and often fatal. Arvid Wallgren (*Acta Med. Scand.*, 1920) has recorded three cases in which oedema and haematuria preceded the meningitic symptoms, and in which the diagnosis of uraemia was first made and the meningococcal infection proved by examination of the cerebro-spinal fluid. These cases were fatal.

In the following case the meningeal symptoms were atypical, and were at first masked by those of nephritis. The causal organism was not found, but in my opinion it was probably a subacute meningococcal infection.

CASE RECORD

Harold A., aged 3 years, was admitted to Selly Oak Hospital on October 24th, 1931. He had been taken ill three weeks previously with acute tonsillitis followed by cervical adenitis. On October 23rd he vomited, and was feverish and delirious. He had been healthy, except for measles in February, 1930. There was no history of otitis media.

On admission he was a normally developed fairly well-nourished child. He was pale, there was oedema of the face and eyelids, and herpes on the left side of his mouth. The tonsils were inflamed, and the upper cervical glands enlarged. Nothing abnormal was found in the heart, lungs, or abdomen. The central nervous system appeared normal. The temperature was 100°, pulse 120, respirations 30. On examination of the urine it was found to contain a large quantity of blood and albumin. The specific gravity was 1020, and there were many granular casts, but no organisms were found or grown in the urine. Streptococci and staphylococci were grown from a throat swab. The child seemed to be suffering from acute nephritis following tonsillitis.

During the following week he ran a high temperature, varying from 103° to 100°. He passed a fair quantity of urine, which, as he was often incontinent, could not be measured, and this, for the first four days, contained a large quantity of blood. Later, red blood corpuscles and casts were seen microscopically, and there was some albumin for ten to fourteen days. The oedema of face subsided in three or four days.

On November 2nd the child appeared worse. He lay curled up in bed, was very irritable, and resented any interference or handling. The limbs were held flexed and appeared to be stiff, but reflexes were normal; the neck muscles were slightly rigid, though there was no head retraction. The temperature was 99°, pulse 130. The specific gravity of the urine was 1018, and it contained a faint trace of albumin. The ear drums were examined and found normal. The blood urea was 20 mg. per cent. A lumbar puncture revealed cerebro-spinal fluid under slightly increased pressure, and contaminated with blood. It was sterile, but there seemed to be an increase of white cells. During the next few days the temperature was 98° to 100°, and symptoms of cerebral irritation subsided, though he occasionally vomited, and appeared to be losing weight, his limbs becoming wasted. The temperature remained normal. There seemed to be slight improvement until November 21st, when he had a rise to 102°, and vomited.

On December 1st he again had a rise of temperature, and complained of pain in the neck and head. The urine was clear, only occasionally showing a trace of albumin. The tonsils were a little injected. There was slight neck rigidity. Kernig's sign was absent, knee-jerks and ankle-jerks were normal, but there was an extensor plantar reflex on both sides. Ophthalmoscopic examination showed

some swelling of both disks. A lumbar puncture was done; the cerebro-spinal fluid was under increased pressure, and was turbid. It contained: 2,000 cells per c.mm., mainly polymorphs; 0.30 per cent. protein; and 678.2 mg. per cent. chlorides. No organisms were seen and the culture was sterile. The Wassermann reaction, both in the cerebro-spinal fluid and in the blood, was negative.

For five days the child was irritable, vomited, and complained of headache, these symptoms being relieved by lumbar puncture. On December 6th the temperature was normal and his general condition improved. For the next ten days he appeared happy and contented, and played with his toys, and on December 17th he again had a rise of temperature, with headache, vomiting, and irritability. Frequent lumbar punctures were done during the attacks and between attacks, and the cerebro-spinal fluid was always turbid, the cells varying from 2,000 to 3,000 per c.mm. On every occasion it was sterile. These recurrent attacks continued at intervals of ten to fourteen days, there being usually a rise of temperature to 100° or 101°. The child complained of frontal headaches, vomited, and was extremely irritable. Symptoms were relieved by withdrawing 10 to 15 c.cm. of cerebro-spinal fluid. The recurrent attacks of headache and vomiting were thought to be due to intermittent blocking of the foramina of Luschka and Magendie, giving rise to internal hydrocephalus, and it was decided to do an encephalography in order to ascertain the extent of the hydrocephalus and also because similar cases were said to have benefited by the introduction of air into the meningeal spaces.

On February 8th, 1932, a lumbar puncture was done; 10 c.cm. of cerebro-spinal fluid were withdrawn and 9 c.cm. of air injected. This was continued until 60 c.cm. of air had been injected. The child then complained of some headache, so the injection of air was discontinued and an x-ray photograph was taken. In this the air was seen to have filled the lateral ventricles, the aqueduct of Sylvius, and the third and the fourth ventricles. There was definite internal hydrocephalus. The cerebro-spinal fluid was again examined, and contained over 2,000 cells per c.mm., mainly polymorphs; 0.4 per cent. protein; 17 mg. per cent. glucose; and 636.9 mg. per cent. chlorides. No organisms were seen, and cultures were negative. The child vomited twice after the radiograph had been taken, and was lethargic that day. Next day he was cheerful and bright, and took his food well. The general condition improved during the next fortnight, and he put on weight for the first time since admission.

After this he had no recurrence of the cerebral attacks. The cerebro-spinal fluid examined early in March was normal: cells, 2.6 per c.mm.; protein, 0.01 per cent. On March 15th he had gained over 4 lb. in weight, was walking well, and showed a normal mentality. He was discharged home on March 26th. At this time his cranial nerves and optic disks were normal, abdominal reflexes brisk, knee-jerks and ankle-jerks normal, and there was an extensor plantar reflex of both feet. I have seen the child twice since, and he has maintained his satisfactory progress. On the right side there is still an extensor plantar response.

COMMENTARY

The points of interest in this case are:

1. The onset. The child was acutely ill, and the case resembled that of an acute haemorrhagic nephritis—probably a manifestation of meningococcal septicaemia.

2. The prolonged course and the late manifestations of meningeal symptoms. Nine days after admission the child appeared to have cerebral irritation lasting twenty-four to thirty-six hours; thirty days after admission he had neck rigidity and an extensor plantar response.

3. The rapid improvement after encephalography. This was probably due to breaking down of adhesions, or releasing of an encysted area, after which the chronic inflammatory process, shown by increased cells and protein in the cerebro-spinal fluid, ceased.

The points in favour of this being a meningococcal infection are:

1. The herpes at the onset, which occurs in a considerable number of cases (Rolleston: *Acute Infectious Fevers*).

2. The large numbers of polymorphs in the cerebro-spinal fluid, and the fact that in subacute cases meningococci may be very scarce.

3. Cases of meningococcal infection with an onset resembling nephritis have been described.

4. A prolonged course with relapses is one recognized form of meningococcal septicaemia.

CONGENITAL ABSENCE OF HEAD OF FEMUR

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The following cases of this anomaly seem of sufficient interest to warrant record. Briefly, they are examples of congenital absence of the head of the femur on both sides in two members of the same family, with a condition resembling double Perthes's disease in a third member. The particulars are as follows:

CASE RECORD

M. M. W., a female child of 5 years, was sent to me on March 12th, 1931. She complained of slight pain in the left hip, and the mother stated that she was limping. On clinical examination nothing definite could be made out. There was certainly no sign of limping when the child was made to walk, and her general condition was good. There was no muscular wasting, and movements of the joint were practically normal. There was no tenderness anywhere, and no pain on percussion of the heel or of the great trochanter. The patient was *x*-rayed, and an extraordinary condition was seen. The head of the femur on both sides was completely absent, and a space of about 1 inch existed between the neck and the acetabulum (Fig. 1). A report was sent to the tuberculosis officer concerned, giving particulars of what was found, and stating that the condition was not tuberculous, and that she should be recommended to an orthopaedic hospital for any treatment required.

At a later date Dr. Folliott (the tuberculosis officer who had sent the case) reported that he had reason to believe that two other members of the same family were affected by some hip condition, and they were requested to attend for examination. On January 8th, 1932, the first patient's mother and brother attended for examination. Neither of them complained of any symptoms whatsoever. The mother displayed rather a "waddling" gait, and her appearance was somewhat suggestive of a congenital specific condition. On *x*-ray examination of the mother, aged 44, it was found that the head of the femur on both sides was absent, and both the necks were considerably thickened (Fig. 2).

The boy, aged 7, showed nothing abnormal on clinical examination, but the *x* rays revealed a condition resembling double Perthes's disease (Fig. 3).

COMMENTARY AND REVIEW OF LITERATURE

In the *Annals of Surgery*, 1930, this condition is reported on by Dr. Franklin E. Walton of St. Louis. He describes congenital absence of the neck of the femur as a comparatively rare anomaly and gives the history as one of a painless, sinking limp, first noticeable when the child begins to walk, suggestive of the likelihood of congenital dislocation of the hip. He further states that the usual confirmatory clinical features are—shortening of the limb, displacement of the upper trochanter, absence of the head from its normal position (below the centre of the inguinal ligament), and mild limitation of abduction, with the other movements comparatively free. None of these symptoms or signs were present in any of the cases recorded above. Walton states that the cases on record are few, but he

refers to eleven. Of these six were bilateral cases (two of which later developed coxa vara), two unilateral (one of which showed absence of the upper epiphysis and part of the shaft), one showing absence of the lower femoral epiphysis, and one extensive arrest of development of the femur.

Treves, in his *Surgical Anatomy*, states that there are three epiphyses in the upper end of the femur, one for the head, which unites at between 18 and 19 years; one for the lesser trochanter, which unites at about 17; and one for the great trochanter, which unites at about 18. The neck is formed by extension of ossification from the shaft and grows as a part of the shaft. Slovitich, in a thesis for the doctorate of medicine of the University of Paris, wrote on these conditions. He stated that these malformations were unknown until forty years ago, the first observation of them being published in 1883 by John H. Morgan. In this case there was congenital absence of the right femur. Some authors, however, doubt the occurrence of total congenital absence. Slovitich refers to the work of Reiner, who has divided these malformations into five groups. In the first are placed all the cases in which the femur is shortened and thinned—in these there is an extreme degree of congenital coxa vara. In the second the femur does not exist as a complete unit, but is divided into several fragments—below, the inferior extremity of the femoral shaft with its epiphyses; above, the head and the trochanter. In the third group the same disposition exists as in the second, but the lower extremity of the femoral shaft is fixed like a cork on to the tibia, without the formation of a knee-joint. In the fourth group certain parts of the femur form a complete unit, but the upper part of the femoral shaft is arrested in its development and retains its embryo form. The head is situated in the prolongation of the shaft of the femur; the neck does not exist. Above, the axis of the femur is bent towards the pelvis. In the last group the malformation consists of extreme shortening of the shaft. The epiphysis, hip-joints, and knee-joints are normally developed and present their normal dimensions. It is pointed out by this author that there is one characteristic found in common—namely, that the proximal part of the femur is that which shows the greatest malformation.

Blencke, who has adopted Reiner's classification, has examined sixty-three cases, adding three new ones of his own—total sixty-six. Of these, thirty were males and nineteen females; in the remaining seventeen the sex was not stated. From this he assumed that the malformation was commoner in males than in females, but this point is not yet certain. Eleven times the deformity affected the femur alone; twelve times the femur and the fibula; eleven times the femur, the fibula, and the patella; and three times the femur, the foot, and the fibula. In twelve cases the deformity affected all the bones of the lower extremity without exception, and thirty-one times the femur, fibula, patella, and the foot. Detailed observations are wanting on nine of the cases. In the great majority the patella was absent. Naturally, the extent of the deformity differed in these cases. In some, absence of the femur was complete, these being very rare, and denied by some authors. In others the fragments, more or less large, were present as vestiges, and sometimes the malformation consisted in a true shortening of the hypoplasia, the shape being preserved. These statistics show that the deformity can be either unilateral or bilateral, and can exist alone or in association with other deformities of the lower limbs, having a predilection for certain of them—in particular for certain parts of the same bone—but not absolutely sparing any part of the lower extremity.

In discussing the pathology of this condition various authors have suggested alcoholism of the parents, emotion,

H. R. FRIEDLANDER AND J. E. BOWEN: CHRONIC APPENDICITIS IN SHANGHAI

FIG. 5.—Case 4. Forty-eight-hour picture showing slight residue in the caecum and ascending colon through which the appendix could be seen retrocaecally.

FIG. 6.—Case 5. The forty-eight-hour film showing caecal and appendix residue with some small concretions in the appendix.

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FIG. 1.—Antero-posterior encephalograph.

FIG. 2.—Lateral encephalograph.