on crutches. Two months later the swelling reappeared and was this time incised under a general anæsthetic and two sequestra removed. In the 20th month a further course of deep *x*-ray therapy was given as his leucocyte had risen to 200,000 per c.mm. and his spleen was again well across the mid-line. The count came down as before to within normal limits and he actually got well enough to leave hospital and go to a convalescent home for three months.

He then had to return to hospital with fever, caused this time by a *Bacterium coli communis* infection of his urinary tract. This cleared up quickly with hexamine and an autogenous vaccine course and he went back again to the convalescent home. Finally he was readmitted to hospital with high fever two and a half years after the date of his first arrival.

His spleen extended three inches across the mid-line once more and his leucocyte count was up to 480,000 per c.mm. with 54 per cent myelocytes. Broncho-pneumonia set in and he passed away after two days. Post mortem the spleen was found to weigh 18 lbs.—which in this country where so few post-mortem examinations are performed is probably a record. The case is also interesting for the tremendous resistance shown by this child to such severe diseases with so many serious complications.

A CASE OF ENCEPHALITIS PERIAXIALIS

(SCHILDER'S SYNDROME)

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ENCEPHALITIS PERIAXIALIS (Schilder's disease) is rare. So far as we are aware no case has yet been reported in India.

History of the case

On the 6th October, 1934, J. A., a Mohammedan boy of 8 years, a resident of Allahabad, was admitted to King George's Medical College Hospital, Lucknow, for :—

(1) Mental dullness—6 weeks.
(2) Loss of hearing in both ears—6 weeks.
(3) Loss of sight in both eyes—6 weeks.
(4) Loss of speech—2 weeks.
(5) Involuntary movements of the hands and feet—2 weeks.
(6) Spasticity of both lower limbs—2 weeks.
(7) Dribbling of urine and constipation—2 weeks.
(8) Difficulty in swallowing—15 days.

His father stated that he himself had suffered from gonorrhœa sixteen years previously and from syphilis twelve years previously but that his son had not been ill until six weeks ago when he complained of severe headache and became unconscious. During the first two hours of the coma some involuntary movements of the face, hands and feet were noticed. His abdomen became distended and he passed a motion in bed with much flatus, which relieved the distension. He vomited once and remained unconscious for three days, without taking food and without passing any motion. After these three days he asked for food and spoke frequently. But he remained completely deaf. This condition continued for a month when fifteen days before admission he became unable to speak. Involuntary movements in the limbs now appeared. Urine was passed in small quantities every hour or so and the bowels were never moved without a purgative. Seven days before admission the father noticed that the patient had increasing difficulty in swallowing.

Condition on admission

The young patient lies quietly in bed on one side, for the most part drowsy or asleep. He is not anæmic, nor emaciated. He does not speak, weep, cry or laugh. He cannot hear in either ear when spoken to. Vision in both eyes is lost. His eyes do not blink when a hand is waved suddenly before them. Apparently he cannot comprehend his surroundings and appears to exist without consciousness and without intelligence.

Nervous system

The pupil reacted to light. There was no squint. The eyes appeared to move freely in all directions. Both optic disc margins were blurred; this was probably due to œdema from the cerebral swelling and increased intracranial pressure. The blurring was more marked in the left disc. The lower limbs were spastic with brisk knee and ankle jerks. Babinski's reflex was present on both sides. The upper limbs were normal. Sensory functions could not be tested satisfactorily because of the patient's mental condition. Difficulty in swallowing food was noticed. Urine was passed in the bed in small quantities every hour or so. The patient will frequently abruptly throw his legs and hands in any direction. These movements appear more marked on his right side. The right hand is often placed over the right temporal region as though some trouble existed there.

Reports on blood and cerebro-spinal fluid

On the 8th October the polymorphonuclears were 60 per cent, the lymphocytes 33 per cent, the large mononuclears 5 per cent and eosinophils 2 per cent. No malaria parasites were detected. The Wassermann reaction was completely negative.

The cerebro-spinal fluid was under distinct pressure, and two test tubes, or about 40 c.cm., were drawn off before the fluid flowed at normal rate. The removal of this fluid did not result in any clinical improvement. The total cell count numbered 5 per c.mm. and these were mainly lymphocytes. The albumin was 0.025 per cent and the chlorides 0.74 per cent.

Diagnosis

A sequence of the development of the following symptoms in children or in young adults is pathognomonic—

1. Progressive bilateral cerebral blindness.
2. Progressive bilateral deafness.
3. Progressive bilateral ataxy.
4. Progressive spastic paralysis.
5. Progressive mental deterioration.

Differential diagnosis

(1) *Disseminated sclerosis.*—Schilder's disease has been described as 'disseminated sclerosis in childhood' because of the scattered lesions and of the obvious demyelination. But the lesions in Schilder's disease are massive as shown by the symptomatology and by the naked-eye post-mortem appearance whilst the

spread of these lesions clinically from one part of the brain to another is rapid. Moreover the brain is selected and the spinal cord is usually spared. Encephalitis periaxialis affects children mainly, and its symptomatology is pathognomonic and distinctive from that of disseminated sclerosis.

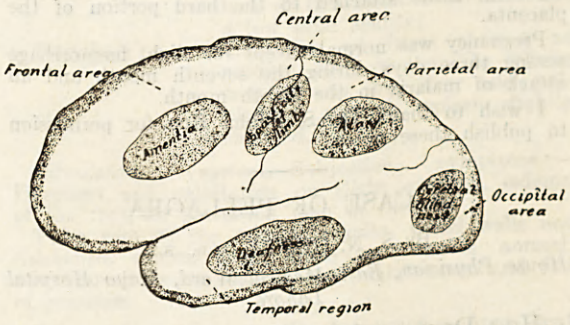

Diagram showing the areas of massive macroscopic gelatinization and microscopic demyelination in the white centres of the cerebral hemispheres, which in the case quoted started (1) symmetrically in both frontal centres (with the production of amentia) and (2) symmetrically in both temporal centres (with the production of bilateral deafness), and progressing to (3) both occipital centres (with the production of bilateral cerebral blindness), (4) whilst it spread after a month to both parietal centres (with the production of bilateral ataxy) and (5) to central area (with the production of bilateral spasticity of lower limbs). A more frequent manner of spread through the white centres is from the occipital lobes forward.
The surface and section of the cortical grey matter is normal.

(2) *Hyperkinetic type of encephalitis lethargica* is also characterized by spontaneous involuntary movements, restlessness, and mental symptoms. The absence of ophthalmoplegias in Schilder's disease and the presence of the pathognomonic signs is important in differentiating the two conditions.

(3) *Intracranial tumour* may be simulated when headache, vomiting, papillœdema, and perhaps fits, are present, but the absence of optic neuritis with the presence of severe loss of vision and with the signs of an extensive rapidly-spreading disease of the cerebral hemispheres point to Schilder's disease.

AN UNUSUAL CASE OF JAUNDICE ASSOCIATED WITH HODGKIN'S DISEASE

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K. A., Mohammedan male, aged 30, admitted to hospital 19th November, 1934, with fever, general pains, cough and jaundice.

There was no past history of any significance.

Present illness.—Some twenty-five days previously he experienced a chill, followed by fever and cough with expectoration and general body pain. About fourteen days ago he noticed jaundice and dark coloration of his urine, succeeded by epigastric pain and constipation.

Condition on admission.—Well made and nutrition good. Appetite good. Deep icteric colour; temperature 101°F., pulse normal, no respiratory symptoms.

Abdomen: upper part full; epigastric tenderness; liver enlarged, tender, smooth, and firm, extending from the fourth intercostal space to three finger-breadths below the costal margin.

Spleen: enlarged, smooth, and firm like an old malarial spleen, and extending four finger-breadths below the costal margin though patient said he had only noticed it recently. No ascites and no palpable abdominal glands.

Chest: no cardiovascular abnormality. Lungs normal except for signs of congestion at the right base. Sputum scanty and mucopurulent, with, on the second day, a small piece of rusty brown material, which showed many pneumococci.

Urine: dark, containing bile salts and pigment.

Stools: clay-coloured at first, but after saline purgative showing some bile; no parasitic infection.

Blood: Van den Bergh's reaction, immediate, direct, strongly positive, total white count, 1,600 only, with 80 per cent polymorphonuclears and 20 per cent lymphocytes. This was repeated the next day with result, 1,760, 76 per cent polymorphonuclears and 24 per cent lymphocytes.

22nd November. Wassermann reaction of blood, strongly positive.

23rd November. Tests for kala-azar, negative.

24th November. Total erythrocytes, 3,500,000; hæmoglobin 60 per cent, white count 2,400, with 70 per cent polymorphonuclears, 28 per cent lymphocytes, and 2 per cent eosinophils.

Progress.—Fever remained at 100°F. to 101°F. daily, but patient's general condition was good; four daily injections of one grain of emetine hydrochloride were given with no effect on the temperature or on the size or tenderness of the liver. His main complaint was that he was not given enough food. Jaundice remained unchanged, with light bile-coloration of the stools.

In the night of 25th November, 1934, he complained of vague discomfort, but did not look worse. About daybreak on the 26th he suddenly got out of bed and, before the nurse could reach him, fell in a fit, and expired almost before the house physician could arrive.

No post-mortem examination could be obtained, except that the liver was taken out, and an interesting condition was found.

Liver enlarged; weight 4 pounds. Greenish colour; on cutting, bile stained, with dilated portal veins: microscopically nutmeg condition with scattered bile pigment: liver cells around portal canal almost disappeared. Some proliferation of bile ducts and some pericholangitis. An infiltration around the portal canal and between the adjacent cells, consisting of lymphocytes and a few enlarged endothelial cells, some of the latter being multi-nucleated. At places there were isolated collections of such cells in the liver substance. The appearance was that of early Hodgkin's disease of the liver.

At the neck of the gall-bladder was an enlarged lymph gland which, on cutting, showed no necrosis nor degeneration, and microscopically was found to be a typical Hodgkin's gland without much fibrosis.

Comment.—The patient undoubtedly suffered from Hodgkin's disease, though there were no demonstrable enlarged glands during life. The diagnosis during life remained obscure, with syphilis as the most probable cause of the jaundice. The extreme leucopenia was not explained. In view of such an incomplete