by suturing its cut margin to that of the transverse mesocolon and the posterior peritoneal wall repaired in the usual way.

The patient stood the operation well, and recovery was uneventful, except that she had diarrhœa on the 4th and 5th post-operative day, probably due to errors of diet.

About 3 weeks after the operation she developed an abscess in the abdominal parietes which healed after incision.

Points of interest

1. No significant symptom except occasional vague abdominal pain, dyspepsia and anorexia.
2. Free mobility of the lump.
3. Large number of nodules present on the surface.
4. Easy separation of the mass.

Pathological report

(S. Bhattacharya)

The mass : 1. *Macroscopic examination.*—The specimen consists of about 6 inches of ileum, cæcum, appendix, ascending colon and about 4 inches of transverse colon. The area of the cæcum and the lower half of the ascending colon is occupied by a large indurated mass, which shows the presence of numerous fair-sized tubercles on the surface (size of mustard seeds). The indurated area extends on to about 3 inches of ileum.

2. *Microscopic examination.*—(1) Giant cells of tubercular type, fair number; (2) much lymphocytic infiltration; (3) large amount of fibrosis; (4) caseation not much in evidence.

The lymph glands : 1. *Macroscopic examination.*—(1) Enlarged; (2) cut surface homogeneous, caseation not seen.

2. *Microscopic examination.*—(1) Giant cells, fair number; (2) marked fibrosis replacing the lymphoid tissue which is not much in evidence; (3) caseation—very little.

Diagnosis

Tubercular intestine, hyperplastic type.

A CASE OF HYPERTENSIVE ENCEPHALOPATHY WITH SUBARACHNOID HÆMORRHAGE IN ACUTE DIFFUSE GLOMERULO-NEPHRITIS

By M. BASAVARAJ URS, B.SC., M.B., B.S.
Assistant Professor of Medicine and Assistant Surgeon, Krishnarajendra Hospital, Mysore

A SIXTEEN year old married girl entered Krishnarajendra Hospital, Mysore, on 8th March, 1947, with complaints of œdema of the face and lower extremities and scabies for the past fifteen days. One year before admission she had an attack of scabies which was treated and cured.

On admission, the girl was conscious, the feet were swollen, and lesions of scabies with secondary infection were found all over the body specially over the hands and feet. Blood pressure was 130/90 mm. of Hg. Urine revealed albumin +++, R.B.C. ++, and casts ++, and was high coloured with a specific gravity of 1025. There were no signs of upper respiratory infection. Pupils were normal. The heart was not enlarged. The sounds were of good quality. Lungs were normal. Liver and spleen were not palpable. W.B.C. count was 11,500 c.mm., R.B.C. count 3.5 million per c.mm., Hb. 75 per cent, sedimentation rate 40/10, blood urea 35 mg. per cent.

A diagnosis of acute nephritis with hypertension was made, the ætiological factor probably being pyodermia. After five days' stay in the hospital, she started complaining of headache and slight dimness of vision. The headache became intense towards the evening and the vision was almost lost. She vomited about six times and it was projectile in nature. At 6 p.m. on the same day she developed an epileptiform seizure which lasted for 2 minutes and became unconscious after the attack. Soon after the attack, the blood pressure was 150/110 mm. of Hg. She spent the night unconscious with six more attacks of convulsions, incontinence of urine and fæces, and frequent vomitings. The next morning, lumbar puncture revealed hæmorrhagic fluid which was under high tension. The fluid was intimately mixed with blood and never became clear even after taking a test-tubeful of fluid with frequent change in the position of the needle. The fluid did not clot on standing. On centrifuging, there was xanthochromia. There was no increase in the number of leucocytes and no micro-organisms could be detected under the microscope. The blood pressure was 140/110 mm. of Hg. On physical examination, there was paresis on the left side of the body with rigidity of the neck and a positive Kernig. She was found to curl herself to the left. Pupils were normal and fundii were normal. She was put on intravenous glucose 50 c.c. 25 per cent twice a day with injections of luminal and oral bromides. She was also put on penicillin 20,000 units every three hours. Within twenty-four hours the convulsions ceased. She regained consciousness, and complained of severe headache and dimness of vision. The lumbar puncture was repeated which had the same characteristics as before. Blood pressure had come down to 130/100 mm. of Hg. Blood urea was 45 mg. per cent. Paresis of the left side of the body, nuchal rigidity and Kernig's sign were still present. The treatment was continued with absolute rest in bed and orange juice. Four days later the patient was found to be quite conscious, speech was rational, external stimuli were appreciated. The quantity of urine passed by this time was 40 oz. a day. Blood pressure was 130/90 mm. of Hg. There was improvement in the paresis. Nuchal rigidity and Kernig's sign were negative. Lumbar puncture

revealed no pressure, the fluid coming out in drops in contrast to the continuous stream in the first two punctures. The fluid was coloured yellow and on microscopy there were no red cells nor leucocytes.

After a stay of twenty-eight days in the hospital she was discharged at her request with instructions to come and report after a month. At the time of discharge there was no œdema, blood pressure was 110/85 mm. of Hg., blood urea 35 mg. per cent. Few granular casts and few red cells were found in the urine and albumin was in traces. Scabies had been cured.

She was prompt in reporting her condition exactly a month later. Except for eczematous ulcerations around the waist, nothing abnormal was detected on physical examination. Urine was free from albumin, red cells and casts. Urine output was between 40 to 50 oz. a day. Blood pressure was 110/80 mm. of Hg. After a stay of one week, the ulcerations around the waist disappeared with local application of calamine lotion, oral cibazol and intravenous soluseptasine.

Comment.—The first thing that strikes in the differential diagnosis is an accidental puncture of the spinal veins. The fact that the fluid did not become clear with the change in position of the needle, the intimate mixture of the blood with the cerebrospinal fluid, the absence of clotting and the presence of xanthochromia speak strongly against the accidental puncture. Acute meningitis is out of question in the absence of an increased number of leucocytes and micro-organisms in the fluid. Rupture of a berry aneurysm either spontaneous or as a result of an increased arterial tension is the only other alternative diagnosis. But the cerebral episode in association with increased cerebrospinal pressure and increasing arterial tension is more in favour of hypertensive encephalopathy.

In the acute stages of glomerulo-nephritis, it is the danger to the heart and to the brain which is of first and highest importance rather than to the kidney itself. 'Almost every case that dies in the acute stage of this disease dies of cardiac insufficiency'.—Volhard. The danger to the brain though less frequent is more startling and dramatic because of the cerebral œdema causing the convulsions of the so-called eclamptic uræmia, which has nothing to do with the uræmia caused by renal insufficiency. This cerebral-symptom complex is termed hypertensive encephalopathy by Oppenheimer and Fishberg. It is to be noted that the higher the blood pressure the greater the danger to the brain. Headache becomes intense accompanied by vertigo. Vomiting is projectile and without effort. Amaurosis affects both eyes, the vision returning to normal within a few hours or days if the patient survives. The blood pressure is high before and during the attack. The cerebrospinal fluid is under increased pressure, the fluid gushing from the needle in a steady stream. Though unusual, it is possible to find blood in the cerebrospinal fluid. One case has been reported by Dewar and Walmsley (1945) where subarachnoid hæmorrhage was detected at post mortem in a case of relapsing fever with acute glomerulo-nephritis. The possibility of hæmorrhage into the subarachnoid space should be in mind whenever the epileptiform seizures are preceded by nuchal rigidity, a positive Kernig and an increasing blood pressure. 'It seems likely that . . . . the cerebral arterioles are unable to constrict with the force necessary to keep pace with the great rise in arterial pressure; the result is that blood enters the cerebral capillaries under preternaturally great tension and transudation is augmented'.—Fishberg (1939). The already injured capillaries as a result of generalized capillaritis may have to yield to this preternatural great tension.

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A CASE OF BLACKWATER FEVER

By R. N. CHAUDHURI, M.B., M.R.C.P. (Edin.), T.D.D. (Wales)

and

H. CHAKRAVARTI, M.B. (Cal.)

From the Department of Tropical Medicine, School of Tropical Medicine, Calcutta

Gupta *et al.* (1942) reported on the anti-hæmolytic property of *Vitex peduncularis in vitro,* while clinically Chaudhuri and Rai Chaudhuri (1945) observed its apparent efficacy in a case of quinine hæmoglobinuria. Singh and Singh (1944) claimed uniformly good results with antivenene in blackwater fever cases. No report is yet available to us on paludrine being tried in this disease. The following case in which all the three drugs were tried is therefore worth reporting although none of them seemed to influence the hæmolytic process.

Case report

A Bengali male, aged 24 years, was admitted to the Carmichael Hospital for Tropical Diseases on the 13th January, 1947, for blackwater fever of six days' duration. He lived in a hyperendemic rural area about 30 miles from Calcutta and gave a past history of