

blood pressure was then recorded as 210/110. On regaining consciousness he complained of pain in the head and the back, from where it radiated to the legs as well.

Next morning I was asked to see the case and found him less drowsy and more restless. He was then complaining of pain in the head, neck and back going down to the legs. Rigidity of the neck was present. Pain in the nape of the neck on deep pressure was also elicited. Kernig's sign was present. There was no paresis. Knee, ankle, biceps and triceps jerks were present; abdominal reflexes were also present. Plantar reflex was normal. Pupils reacted sluggishly and were equal. A hæmic bruit was present in the heart. Temperature 101°F., pulse 78 per minute, blood pressure 170/90. A diagnosis of spontaneous subarachnoid hæmorrhage was suggested. Lumbar puncture was done and blood-stained fluid came out under pressure. On keeping the fluid in a test tube for some time, the red blood cells settled down in a very small number and the supernatant fluid became clear but showed deep xanthochromia. There was no spontaneous coagulation of the fluid. Albumin in the fluid 0.25 per cent, chlorides 0.85 per cent and urea 0.19 per cent; culture, sterile; Wassermann reaction, completely negative; van den Bergh reaction, indirect positive.

The patient was put on bromides and calcium lactate by mouth and he passed a better night. Next morning, drowsiness and restlessness continued and he complained of less headache and less pain in the back. The reflexes with the exception of the abdominal had disappeared. Rigidity in the neck persisted. The blood pressure was as high as before and pulse 72 per minute. Three days later he became more restless; temperature went up to 103.4°F.; blood pressure, 200; pulse, 80 per minute; reflexes, absent. Lumbar puncture at this stage was refused. During the next three days he became more and more restless and delirious; the blood pressure had now a tendency to fall; the pulse and temperature were rising. Another lumbar puncture was done and it showed blood-stained cerebro-spinal fluid under pressure, but the colour of the fluid was not so high as on the previous occasion. Next day he became more deeply unconscious, and his pulse and respiration failed, resulting in his death. No post-mortem examination was allowed.

The typical history of severe headache followed by pain in the back accompanied later on with rigidity of the neck and tenderness of the nape of the neck led me to think that he had some intracranial hæmorrhage, probably subarachnoid. Meningitis could be ruled out on account of the peculiar onset of the symptoms. Cerebral apoplexy is also not considered possible because of the insidious onset and long course of the illness. The typical cerebro-spinal fluid with slight increase in the amount of protein and with van den Bergh indirect positive confirmed the diagnosis that hæmorrhage had taken place somewhere under the arachnoid.

The above case conforms with the 'lumbago and sciatica syndrome' described by Professor Arthur Hall in connection with the different manifestations of spontaneous subarachnoid hæmorrhage and impresses on us the importance which should now be attached to cases of lumbago, especially when they are either accompanied with or preceded by severe headache or migraine. A lumbar puncture in such cases

may help in warding off a fatal issue in a certain number of patients.

As regards the pathogenesis of this condition, it is supposed to be due to leakage from and subsequent rupture of a 'berry aneurysm' situated on a vessel of the brain especially the basilar artery and the circle of Willis. Leaving out trauma, neoplastic growths and the infective processes, such aneurysmal dilatations may be due to three different pathologico-ætiological groups:—

1. Arterio-sclerotic—due to degenerative condition of the vessels.
2. Syphilitic (rare).
3. Congenital.

Out of the above three groups, the first is usually met with in the young adult life, the second one in the middle life, while the third is found among the old. There may be some exceptions to the above rules. The case quoted above was an old man and hence the chances were that he had a degenerative condition of the vessels at the base of the brain. The hæmorrhage was due to an arterio-sclerotic aneurysm probably on the basilar artery. A condition of general arterio-sclerosis was further suggested by the increased amounts of chloride and urea in the cerebro-spinal fluid. I consider the basilar artery the most probable site because aneurysm situated above that area would almost certainly show definite localizing signs, which were absent in this case.

A CASE OF SEPTICÆMIC PLAGUE SIMULATING PERNICIOUS MALARIA

By H. A. YENIKOMSHIAN, M.D., D.T.M. & H., M.R.C.P.
Associate Professor of Medicine, American University of Beirut

PLAGUE is sporadic in Beirut. During autumn and winter we see more cases, and occasionally we get a scare of an epidemic. But during the spring and summer the number of cases diminish. I have no satisfactory knowledge about the rodents and fleas of the country to explain this phenomenon. The following case is quite unusual in Beirut and of sufficient interest to warrant publication:

A school girl, twelve years of age, was admitted to the American University of Beirut Hospitals in a state of coma on the 4th of March 1933. She was in perfect health on the 2nd of March. On that day she woke up with a sore throat and vomited bilious fluid twice. At noon her mother was frightened to find that she could not wake up the child. A doctor was called who found that she was in a state of light coma, responding only to painful stimuli, and had high fever. She lives in a malarial district, has a large, hard, spleen and has had malaria, lasting for two months, several years previously. The doctor gave her an injection of quinoplasmine. She had an attack of convulsions during the night, and the next day she was brought to this hospital.

The state of coma, that she was in on admission, simulated a cerebral form of pernicious malaria.

Physical examination.—Well developed, fairly well nourished, temperature 40.5°C. (105°F.), pulse 120, respiration 28 per minute.

Face congested, no rigidity in the neck. Only slight redness in the throat.

Pupils equal and react to light.

No apparent disease in the chest.

Liver palpable, spleen hard and reaches three fingers below the costal arch.

No glandular enlargement.

LABORATORY EXAMINATIONS

Blood count.—Leucocytes 13,700, polymorphonuclears 82 per cent, lymphocytes 6 per cent, large mononuclears 11 per cent and eosinophils 1 per cent.

No malarial parasites found. Several smears were examined.

Urine.—Albumin ++, acetone ++, microscopic examination—many granular casts.

Lumbar puncture obtained clear fluid, which did not come under high tension and showed no pathological changes.

Blood sugar 80 mgm./100 c.cm.

Urea N 50 mgm./100 c.cm.

Chlorides 370 mgm./100 c.cm.

Diagnosis was not clear. We had no cases, at that time, of cerebro-spinal fever or epidemic encephalitis in Beirut. The patient was not diabetic and had no history of chronic kidney disease. Comparatively high blood urea nitrogen in the presence of low blood chlorides could be explained by dehydration due to vomiting and starvation. Apparently this was an acute infection with cerebral involvement. A blood culture was taken, also a throat swab. Her acidosis and dehydration was treated by intravenous glucose and saline, and despite the absence of malarial parasites in the peripheral blood, diagnosis of pernicious malaria was entertained and she was treated by injections of quinine and plasmochin.

Next morning the patient seemed to be much better. The temperature came down to 37.8°C. (99°F.), she could retain some food in the stomach, and could speak intelligently. In the afternoon of the same day she had another rise of temperature and vomited, the vomitus containing dark blood, went into coma again, the temperature rose, the coma got deeper and deeper, and she died at midnight of that day with a temperature of 42°C. (107°F.).

Another blood count made on that day was:

Leucocytes 18,000, polymorphonuclears 72 per cent, mononuclears 24 per cent, lymphocytes 3 per cent, and eosinophils 1 per cent.

As no autopsy was permitted we made a splenic puncture after death and found bi-polar staining oval bacilli, morphologically similar to the plague bacillus. On the same day the laboratory reported that her blood culture showed a growth of a short non-motile Gram-negative bacilli, suggestive of *Bacillus pestis*. This was confirmed by inoculating the culture into a white mouse and rubbing the same on the shaved skin of a guinea-pig. The mouse died three days after inoculation, and the guinea-pig after eight days. On autopsy of these animals, typical *Bacillus pestis* were obtained.

Dr. G. C. Low (Price, 1930) describes a cerebral form of plague which clinically resembles very closely cerebral pernicious malaria. In my case there was a high mononucleosis—11 per cent and 24 per cent—and moderate leucocytosis. Though we have examined many smears, Leishman stain, for plasmodium malaria, we have seen no organisms suggesting *Bacillus pestis*.

REFERENCE

Price, F. W. (1930). *A Textbook of the Practice of Medicine*, Humphry Milford, London.

AN INTRACTABLE ULCER ON THE SCALP

By P. A. MAPLESTONE, D.S.O., M.B., CH.B., D.T.M.
and

L. M. GHOSH, M.P. (Cal.), D.T.M.

(From the Calcutta School of Tropical Medicine, Medical Mycology Inquiry under the Indian Research Fund Association)

A boy aged 14 years came to the outpatient department with an ulcer about one and a half inches in diameter on the top of his head.

Previous history.—About three years ago the boy had a continued fever lasting for about six weeks. This was very severe and during its course he had convulsions and was delirious. Towards the end of this pyrexial attack the lobes of the ears, the alae nasi and the scalp on the vertex became 'black' (probably gangrenous). One day he scratched his scalp and the whole of it came away over an area about six and a half by four and a half inches, leaving the skull exposed. The ears and nose gradually recovered and appear to have healed without any scarring. The large exposed area on the scalp also gradually lessened and in about six months there was a granulating ulcer about one and a half inches in diameter which was surrounded by a ring of dense scar tissue of the already-healed portion.

Various treatments were tried for a period of about two years without any permanent benefit, though at times the ulcer diminished in size, but it always broke down again and reached its former dimensions. On one occasion a skin graft was done in another hospital and after about six months he was discharged with the ulcer reduced to about one-third of an inch in diameter, but it very soon broke down again.



Before treatment.

Condition on coming to the School.—There was a superficial ulcer on the top of the head covered with unhealthy indolent granulations. The margins showed no signs of healing and the blood supply was poor on account of the ulcer being completely surrounded by a dense ring of scar tissue about two inches in width.

Cultures from the pus only produced ordinary pyogenic organisms, the Wassermann reaction was negative, and the boy appeared otherwise healthy; the only other abnormal condition present was a hookworm infection of moderate severity.