Medical Memoranda

Tuberculous Meningitis with Paraplegia as the Dominant Feature

The following case is put on record as it presented certain unusual features which caused difficulty in arriving at a correct diagnosis.

CASE REPORT

A Gurkha recruit aged 16 was admitted to hospital on May 14, 1948, with a small septic laceration of the left knee. This healed without obvious complication, but on May 18 he developed a sudden rise of temperature. This was provisionally diagnosed as a malarial attack, which was treated by routine methods. However, as no malaria parasites were seen in blood-slides and his pyrexia did not subside with antimalarial measures, it was considered that he might have developed a secondary generalized infection from the primary focus on the left knee. Penicillin and sulphathiazole were therefore given, but without clinical improvement.

On May 23 he had to be catheterized, and patches of anaesthesia were found distributed over the lower chest and flanks. During the next 24 hours he developed a rapidly progressive paraplegia and became drowsy. Lumbar puncture performed at this time revealed diminished C.S.F. pressure, a raised protein content, and 9 cells per c.mm. A blood count showed 27,000 white cells (90% polymorphs).

I was invited at this stage to see the case in company with Lieutenant G. Watts, R.A.M.C., and found the patient practically comatose. There was some stiffness of the neck, the pupils were dilated and fixed, and he had a complete flaccid paraplegia with loss of all reflexes, including the abdominal reflexes. The level of loss of sensation was difficult to determine accurately owing to his inability to co-operate, but he reacted to pin-prick above approximately the fourth dorsal level. There was no clear line of demarcation of sensory loss. The spine showed no obvious abnormality.

A cisternal puncture revealed a pressure of 200 mm. of water. The C.S.F. was clear and there were only 4 cells per c.mm. Tubercle bacilli were not found. A tentative diagnosis of spinal compression following an epidural abscess was made. It was surmised that the septic laceration of the knee had been the portal of entry for organisms causing a generalized infection.

The patient died on May 25. Owing to religious objection a post-mortem examination limited to the spine and skull only was permitted.

Pathology.—On exposing the theca no epidural abscess was found. The dura was much thickened, and on being incised the subdural space was seen to be filled with thick gelatinous and fibrinous pus. The dura was covered with shaggy exudate and studded with tubercles. A similar appearance was observed on the surface of the cord. These changes were most pronounced between the levels of D 10 and D 4, and had apparently produced a complete spinal block. The base of the brain presented similar but very much less marked appearances, and the lining of the ventricles was studded with small tubercles.

Histological examination showed that the cord itself was largely unaffected, but the pia and arachnoid were grossly thickened and contained large numbers of lymphocytes, histiocytes, polymorphs, and giant cells. Bacteriological culture gave a growth of human tubercle bacilli.

COMMENT

A case of tuberculous meningitis with pronounced early spinal symptoms is described. This form of the disease seems to be comparatively rare, and in this instance it ran a very rapid course, there being only seven days between the onset of the pyrexial stage and death. The sudden fever

and the presence of a focus of sepsis on the knee suggested a pyogenic cause for the spinal signs, and the true diagnosis was revealed only at necropsy.

Four cases with a similar chain of features occurring in Singapore between 1938 and 1942 were described by Ransome and Montiero (1947). Though there are certain differences between their cases and the one described here, the march of events was the same—pyrexia, paraplegia, meningeal signs, and death. In their cases the onset and course of the condition were more gradual, the illness lasting about three weeks. In describing the nervous signs they noted the patchy sensory loss and the difficulty in demarcating the limit of sensation, but stated that the muscular paresis of the legs never became complete. However, in the main essentials the case here described corresponds with the clinical and pathological picture as observed by Ransome and Montiero.

I am indebted to Captain J. C. Gould, R.A.M.C., for his report on the pathological findings.

EDWARD KNOWLES, M.Ch.Orth., F.R.C.S.,
Late Lieutenant-Colonel, R.A.M.C., Orthopaedic
Specialist, Far.E.L.F.

REFERENCE

Ransome, G. A., and Montiero, E. S. (1947). *British Medical Journal*, 1, 413.

Case of Oesophageal Carcinoma

This is, I believe, the first recorded case of carcinoma of the oesophagus in a patient suffering from anorexia nervosa.

CASE REPORT

The patient was an unmarried woman aged 38. During a pleasure cruise in the Thames Estuary 21 years ago she became much alarmed when a large wave broke over her. She began to "tremble and go stiff all over," she lost her voice; all inclination for food left her, and she had a "breakdown." She had to give up her job. Anorexia persisted thereafter, and she said that she had lived on 2 pints (1.1 litres) of milk and two cups of sweet tea each day until her admission. Her weight, which was 5 st. 1 lb. (31.75 kg.), tended to confirm the truth of this statement. Eighteen years ago amenorrhoea and daily vomiting began and persisted for a year; there was no further change until three months before admission, when retrosternal oppression occurred during meals, and rapid loss of weight began. A month before admission vomiting started, and for the last few days had been almost continuous.

On examination she was a tall, emaciated virgo intacta, with no stigmata of hypovitaminosis. Slight proteinuria and a few small, easily felt hard lymph nodes in the left posterior triangle were the only positive physical signs. A provisional diagnosis of anorexia nervosa was made, but the recent alteration in symptoms suggested oesophageal obstruction, and a barium swallow was carried out; this revealed a tight stricture in the lower third of the oesophagus, which was subsequently shown by oesophagoscopy and biopsy to be due to the presence of a squamous-celled carcinoma.

Radiotherapy was begun, and gastrostomy was performed. The liver was rapidly invaded, however, and became enormously enlarged and irregular. After the appearance of ascites and oedema of the legs, downhill progress was rapid, and four months after admission the patient died.

At necropsy an ulcerating growth of the oesophagus involving the whole wall from the bifurcation of the trachea to the cardia was found. The liver weighed 4,150 g. and was packed with carcinomatous masses. There were two enlarged lymph nodes containing secondary deposits at the bifurcation of the aorta, and one in the left side of the neck. Microscopically, many branches of the portal vein in the liver were shown to contain plugs of growth.

COMMENT

The feature of interest in this case is the relation of the long-standing psychological disturbance to the development of oesophageal cancer. Lawrence Abel (1929) states that two-thirds of his cases of carcinoma of the oesophagus were preceded by an incident of emotional stress, and Guisez (1925) has said that half of the cases in his series had a similar history. Abel suggests that the neoplasm arises at the site of pre-existing spasm of the muscle wall, and refers to his own observations and those of Guisez that mental stress is often a cause of oesophageal spasm.

If the patient's symptoms before the development of carcinoma had been due to oesophageal spasm, it is reasonable to suppose that during the twenty-years history some of the oesophageal dilatation and hypertrophy characteristically the result of spasm ought to have developed. This was not so, and one is forced to the conclusion that in this patient, at any rate, whatever aetiological relation there may have been between mental stress and oesophageal cancer, spasm played no part.

Dr. E. R. Cullinan, under whose care this patient was admitted, has kindly given permission for the publication of this report.

IAIN MACDOUGALL, M.D., M.R.C.P.,
St. Bartholomew's Hospital.

REFERENCES

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Guisez, J. (1925). *J. Laryng.*, **40**, 213.

Localized Fat Atrophy After Jelly-fish Sting

The following case is worth recording because of its unique nature.

CASE REPORT

A V.A.D. nursing member aged 27 was bathing in the sea at Trincomalee, Ceylon, at about 10 p.m. on February 26, 1948. As she was climbing up the steps to come out of the water the anterior part of the left thigh and the left leg just above the ankle came in contact with the tentacles of a jelly-fish, which for the moment became wrapped round the limb. A severe stinging sensation was at once felt, followed almost immediately by large urticarial weals on the sites of contact. She returned home and went to bed, though the pain continued throughout the night and was severe enough to interfere with sleep. There was never any collapse, dyspnoea, respiratory embarrassment, or generalized urticaria, and she reported sick at the hospital the next morning.

When seen about 12 hours after the accident she was still complaining of an exceptional amount of pain, and urticarial weals with vesiculation were present across the anterior aspect of the middle third of the thigh and the leg for about 4 in. (10 cm.) above the ankle. There were no general symptoms beyond slight malaise. The patient was rested in bed and calamine lotion applied; the urticaria subsided in a week, leaving a purplish red discoloration of the skin, which persisted, though gradually fading, for about six months. Some oedema

of the left foot and ankle appeared on the second and third days and lasted for some three weeks.

The discoloured skin of the affected areas remained remarkably sensitive, and even three months later a stinging sensation could be appreciated on contact with water.

On May 9 the patient again reported, stating that for the past three weeks she had noticed her left leg becoming thinner. The cutaneous discoloration was still just apparent; there appeared to be a complete disappearance of the subcutaneous fat in these areas, the left thigh being 1½ in. (4.4 cm.) thinner at 6½ in. (16.5 cm.) above the patella, and the left leg ¾ in. (1.9 cm.) thinner at 2 in. (5 cm.) above the ankle. There was no loss of power, no muscular wasting, and no reflex or sensory changes. A radiograph of the lower limb showed no abnormality. No treatment was given and the patient was again seen on November 21. A certain amount of redeposition of fat had taken place in the intervening six months—the left thigh now showing 1¼ in. (3.16 cm.) of wasting and the left leg about ½ in. (1.25 cm.)—and the tendency seemed to be towards a slow return to normal. The accompanying photograph, which was taken on November 22, shows the degree of fat atrophy then present.

COMMENT

Jelly-fish of various types are very prevalent round the coast of Ceylon during the months of February and March, and a number were known to be about at the time; it is regretted, however, that the exact species responsible was not identified.

Jelly-fish stings are capable of causing a variety of local and general symptoms, mostly suggestive of excess histamine liberation. Urticaria in varying degrees of severity is the most usual local reaction, and may be followed by a profuse weeping eczema (Allen, 1920: quoted by Joslin *et al.*, 1940). General symptoms include shock, cardiac distress, and dyspnoea, but these were entirely lacking in this case. The degree of skin discoloration and the length of its persistence were both exceptional, though it is not very uncommon for some reddening of the skin to be present at the end of three months.

Lipodystrophy, or atrophy of the subcutaneous fat, is well recognized in diabetic patients treated with insulin, and is especially frequent in children and to a less extent in women, but is scarcely ever seen in adult males (Joslin *et al.*, 1940). This observation may have some bearing on the present case: it has been suggested that the androgen may protect the adult male and lack of it may account for the frequency in women and children, for hypogonadal adult males may occasionally suffer from insulin lipodystrophy (Walderstrom: quoted by Joslin *et al.*, 1940). I have been unable to trace any similar case in the literature.

My thanks are due to the Medical Director-General of the Navy for permission to publish this case.

M. A. RUGG GUNN, M.D., M.R.C.P.,
Surgeon Commander, R.N.

REFERENCES

- Joslin, E. P., *et al.* (1940). *The Treatment of Diabetes Mellitus*, p. 597. London.
Strong, R. P. (1944). *Stitt's Diagnosis, Prevention, and Treatment of Tropical Diseases*, p. 1549. London.

A new type of rubber surgical glove has been developed in the U.S.A. to relieve cases of dermatitis caused by the continual wearing of rubber gloves, which is a problem chiefly faced by surgeons, who may have to cease working for considerable periods to obtain relief. A rubber company of Akron, Ohio, has announced that it is now producing this special glove, and a number of doctors have reported that they are effective.—B.U.P.