bone marrow, neutropenia, and hyperchlorhydria. He stated that his tongue had been sore, but examination showed no abnormality.

The anaemia was undoubtedly due to stagnation of bowel contents interfering with the normal fermentative processes in the ileum, affecting folic acid and/or vitamin-B_{12} production. The recent work of Dyke *et al.* (1950) would seem to support this contention, more particularly if it can be assumed that the effects of the stagnation would extend to include the colon with consequent folic acid and vitamin-B_{12} deficiency.

Summary

A case is presented of a man aged 35, with loss of weight, gastro-intestinal symptoms, and a macrocytic anaemia, which seems to give clinical support to experimental work on the aetiology of macrocytic anaemia. It is noteworthy that the condition developed after surgical procedures which led to small-bowel stasis, and was cured by surgical removal of the lesion, without recourse to liver, folic acid, or vitamin B_{12}, which were not necessary in order to effect a readjustment of the normal intestinal metabolic processes and of the normal relationship between host and intestinal bacteria.

This case and a study of the literature emphasize again the necessity for a complete intestinal investigation in all cases of macrocytic anaemia which do not fall into well-recognized clinical groups, such as Addisonian anaemia, the macrocytic anaemia of pregnancy, or those associated with diagnosed constitutional disease.

It seems that short-circuit operations below the stomach are unsound except as a temporary measure. This is particularly the case when an antiperistaltic anastomosis is fashioned. There is a danger of the development of a macrocytic anaemia in such procedures as ileo-colostomy, in which a blind length of bowel is left.

The literature is reviewed.

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In the annual report for 1949 of the Medical Department of the Uganda Protectorate there is a description of the methods by which information is obtained about the state of public health in the Protectorate. Some of the notifications of deaths and of infectious diseases are made by chiefs of tribes, and, though the total deaths notified in this way are reasonably accurate, the allocation of deaths to the primary cause leaves much to be desired. The compiler of the report writes: "While the infectious diseases are generally diagnosed accurately, many deaths from 'old age,' 'chest,' or 'njoka' (literally, a snake in the stomach) afford little information about the real cause of death. Less than one-twentieth of all deaths occur in Government hospitals, and it is rare for deaths outside to be attended by medical practitioners."

Medical Memoranda

Pericardial Effusion as a Sequel to Subphrenic Abscess

The rarity of pericardial effusion as a sequel to subphrenic abscess has prompted the recording of this case. Of 182 cases of subphrenic abscess reviewed by Harley (1949), none were complicated by pericarditis. Ochsner and De Bakey (1938), reviewing the world literature, recorded pericarditis as a complication in 5% of 1,380 cases. This incidence, which largely results from one series of cases, is probably above the average.

Case Report

On June 4, 1948, a boy aged 11 was admitted to hospital with a 36-hours history of abdominal pain. Operation revealed a gangrenous appendix with pus in the peritoneal cavity. Appendicectomy was performed and the peritoneum drained. During the next three weeks two further abdominal abscesses were evacuated. As his fever persisted he was screened, and this showed the right dome of the diaphragm to be raised and immobile. An anterior subcostal incision was made and two pints (1.14 litres) of pus were obtained from the lateral and superior aspects of the liver. On culture this grew coliform organisms, non-haemolytic streptococci, and *Staphylococcus albus*.

On August 17, as these measures were not entirely successful, 6 in. (15 cm.) of the tenth rib was resected posteriorly. The diaphragm was incised through a transpleural approach. Adhesions were broken down and a cavity was found lying well towards the midline. Thick pus was removed and the site drained. His condition steadily improved, and on September 27 he was discharged. While in hospital he had received 18,200,000 units of penicillin and 64 g. of sulphadimidine.

Three weeks after discharge he vomited and complained of headache, which was later associated with severe upper substernal pain. After one week in bed he felt better.

Three weeks later he was readmitted to hospital following a recurrence of the pain. Clinical findings suggested the presence of a pericardial effusion. In particular, the apex beat could not be seen or felt and the area of cardiac dullness was greatly increased. The heart sounds were notably faint. An electrocardiogram showed no abnormality. Screening revealed a grossly enlarged heart shadow with little movement. The lung fields were clear. A needle was inserted in the fifth left intercostal space, 1 in. (2.5 cm.) medial to the anterior axillary line, and 300 ml. of greenish-yellow opalescent fluid removed. This contained lymphocytes and polymorphonuclear leucocytes in equal numbers and was sterile on culture. The effusion recurred during the next three days, but by the end of a week had receded to normal.

Three weeks later he complained of substernal pain and the effusion was found to have recurred. A small right-sided pleural effusion was also noted—a new feature. The pericardial fluid had the same characteristics as before. The symptoms and signs gradually receded over the next five days.

He remained well until the beginning of February, 1949—an interval of two months. On this occasion the effusion appeared on the day after he had been allowed up for the first time. Bilateral small pleural effusions were also present. He improved with rest. At this point he was given 1 g. of streptomycin daily for eight days. He has had no episodes of pericarditis since.

He was last seen on December 19, 1949, when there was no clinical or radiological evidence of pericardial or subphrenic disease.

Comment

The four attacks of pericarditis with effusion were considered to be secondary to the subphrenic abscess. Although two loculi of pus were drained, a small focus must have persisted. It is unlikely that there was a direct invasion of the pericardium, as the fluid was repeatedly sterile on culture. The effusion is probably analogous to the oedema surrounding any acute inflammation. The immunity of the pericardium, as compared with the pleura, to involvement by subphrenic inflammation may be due to its relation to the avascular central tendon of the diaphragm.

In conclusion, the importance of early diagnosis and treatment of subphrenic infection is again stressed.

I am indebted to Professor J. Craig and Mr. G. Gordon Bruce for help and criticism.

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An Adreno-renal Syndrome in Malignant Tertian Malaria

The patient whose case is described below developed algid malaria in Co. Down after living with impunity in West Africa for 20 years. It could be postulated that during his journey home he was infected with a different strain of *Plasmodium falciparum*, to which he was not immune. Certainly his peripheral blood showed evidence of a heavy infection with the parasite.

Case Report

The patient, a man aged 41, had lived in West Africa since 1930. He was flown home at the end of August, 1950. When seen on September 16 he was in a grave condition, which, by clinical and pathological interpretation, was found to be a state of uraemia arising as the result of a heavy infection with malignant tertian malaria. Before examination he had had preliminary symptoms of pains in the limbs and general fatigue. His illness can be described in two phases—that of serious peripheral vascular failure, followed by the phase of uraemia with deepening coma.

During the first phase the patient was mentally alert and complained bitterly of severe pains in the arms. On examination his whole body, and especially the limbs, was cold and sweaty, and the joints of his right arm were swollen and painful. It was almost impossible to feel his pulse and to record the blood pressure. The systolic blood pressure was below 60 mm. Hg. His temperature was 95° F. (35° C.) and his spleen was greatly enlarged. A sample of urine was found to be free from albumin. At this stage an electrocardiogram showed normal complexes in the standard limb leads and in unipolar chest leads, and an emergency radiograph of the chest revealed a normal heart and lungs. It was therefore evident that he was in a state of grave circulatory failure of peripheral origin, with hypotension as an outstanding feature, and, at the beginning, a normal urine. Appropriate stimulants were given for his state of collapse.

The next day the second stage of the illness had begun. He gradually passed into a state of coma, from which he could scarcely be aroused. An extensive haemorrhagic skin rash developed on the limbs and in the folds of the groins. Marked oliguria occurred, and his urine was loaded with albumin. The following tests were carried out to determine the cause of his coma.

Investigations.—Blood urea; 120 mg. per 100 ml. Blood slides (thick and thin): accolé forms of *Plasmodium falciparum* and male and female gametocytes in abundance; neither trypanosomes nor *Treponema recurrentis* were seen. A hanging drop of blood contained no trypanosomes. Blood counts: red cells, 3,740,000; Hb, 70% (Haldane); white cells, 7,800 (neutrophils 50%, eosinophils 24%, lymphocytes 22%, basophils 2%, monocytes 2%). Urine: albumin +, no casts, no red cells, no haemoglobin, no ova. Weil-Felix OX2, OX19, OXK were negative in all dilutions. The Widal reaction was negative for the salmonella group. Agglutination tests for *Leptospira icterohaemorrhagiae*, *Leptospira canicola*, and other West African species of leptospira were negative.

The second phase was therefore characterized by disturbance of renal function with albuminuria and urea retention. As the patient ultimately made a complete recovery and the condition responded dramatically to proguanil, it is reasonable to assume that the heavy malarial infection was responsible for the renal damage, especially as other likely causes had been excluded. Proguanil, 200 mg. daily, was given, and in a few days he made a dramatic recovery. Within seven days his temperature, which had risen at the beginning of the second phase, had settled down, the urinary output was normal, and the urine was free from albumin. All parasites had disappeared from the blood. Later, renal-function tests showed normal function and intravenous pyelography revealed normal kidneys with good concentration power.

Comment

The clinical description of this case of malignant tertian malaria is divided into two distinct phases. The first phase, with its marked hypotension and cold extremities, resembled an acute hypoadrenalism. Significantly there were no urinary changes, and the pain and swelling of the joints are interesting in view of the relation of arthritis to the adrenal glands. Unfortunately, details of electrolytic balance are not available. At any rate, the cardiovascular phenomena were peripheral in origin.

The second stage of the illness has two possible explanations. The albuminuria and features of uraemia may have been due to actual involvement of the kidney by malarial infection—that is, a true glomerulonephritis or nephrosis due to tubular damage—or they may have been due to a dynamic disturbance of renal function consequent on the very low blood pressure of the first phase. It is significant that, although the urine was loaded with albumin, repeated microscopical examination failed to show the presence of red blood cells or casts. Thus the urine did not indicate the presence of glomerular inflammation or rupture of capillaries as would have been expected had a true glomerulonephritis been present. The absence of casts in the urine and the raised blood urea do not suggest a primary nephrosis.

In the first phase the marked and persistent hypotension would be sufficient to cause the second phase of the illness with impairment of renal function due to interference with glomerular filtering pressure, the albuminuria being due to the state of anoxia consequent on the reduced renal blood flow. The urine showed a sharp onset of albuminuria, which cleared up abruptly with treatment. This also supports the dynamic theory, as a true nephritis would most likely have shown a persistent albuminuria for some time even after the acute inflammation had cleared up. Moreover, a few weeks later there was no evidence of renal disease.

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