100 ml. Of the 12 cases above this figure, one was complicated by amyloid disease, while another was a diabetic with a glucose level in the pleural effusion of 220 mg./100 ml.

A glucose level of under 60 mg./100 ml. would therefore appear to be strongly suggestive of the tuberculous origin of a serous pleural effusion. Where the glucose level is over 100 mg./100 ml. the effusion is probably of non-tuberculous origin.

The six "undiagnosed" cases had glucose levels ranging from 62 to 96 mg./100 ml. Four of these six cases were considered clinically and radiologically to be of tuberculous post-primary type, but bacteriological confirmation was lacking. The four patients were adolescents or young adults, with straw-coloured lymphocytic effusions of recent onset, and gave positive Mantoux reactions.

There were eight tuberculous patients with glucose levels between 60 and 100 mg./100 ml. and seven non-tuberculous. The glucose values in the six "undiagnosed" cases would therefore be of no value in deciding the aetiology of the effusion.

Summary

The pleural glucose level has been studied in 61 cases of serous effusion.

In these cases: (a) a glucose level of less than 60 mg./100 ml. was found only in tuberculous effusions; (b) the majority of tuberculous effusions had glucose levels of less than 100 mg./100 ml., while most non-tuberculous effusions had levels above 100 mg./100 ml.; and (c) no significance could be attached to glucose levels of between 60 and 100 mg./100 ml.

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ACUTE PORPHYRIA WITH UNUSUAL FEATURES

BY

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In view of the recent interest in this disease and its comparative rarity in this country it is thought worth while to record the following case, which presents some unusual features.

Case Report

The patient, a woman aged 23, was admitted in December, 1949, to a surgical ward of the Southern General Hospital with a history of attacks of abdominal pain and vomiting of one week's duration. The pain usually started in the right hypochondrium and travelled forward across the epigastrium and through to the back in the upper lumbar region. It was spasmodic in nature, and severe, and an attack lasted for 15 to 30 minutes. Shortly after the onset of the pain she vomited. These attacks occurred three or four times a day. Her bowels had been regular until two days before admission, since when they had been constipated. Three days before admission her urine was red in colour on one occasion. Her menstrual history was normal, and she had just finished a period when the pain began.

There was nothing relevant in her past history except that she had had a similar attack of pain and vomiting with the passage of red-coloured urine one year previously, when

she was treated for "kidney trouble" with rest in bed for six weeks at home. No further information is available about this illness.

On examination the patient was seen to be a well-grown and well-nourished woman who looked ill. She had definite guarding and tenderness in the right loin and hypochondrium but none elsewhere. Her temperature was normal, pulse 72, and respirations 16. No abnormality was detected clinically in any of the other systems. The urine was dark in colour but gave a negative reaction for both blood and bile. The test with Ehrlich's aldehyde reagent, however, was strongly positive. The importance of this was not appreciated at the time. On the fourth day a trace of bile was detected in the urine (iodine test) and a faint icteric tinge was noticed at the corners of both sclerae. The blood bilirubin then was 0.6 mg. per 100 ml., the thymol turbidity test for liver function was ++, prothrombin time 22 seconds, icteric index 4. The blood picture was normal.

In the meantime the patient's condition had not improved. The attacks of pain had persisted with almost unabated severity and were still accompanied by guarding and tenderness in the right hypochondrium. In view of this fact and the laboratory findings it was decided to perform a laparotomy with the tentative diagnosis of gall-stones. At operation, however, the biliary tract appeared perfectly normal and no other intra-abdominal abnormality was found. It was then that the diagnosis of acute porphyria was proposed. Chemical tests and spectroscopy of the urine speedily established it.

The patient had a post-operative cough and later developed pneumonia. It was noticed at that time that she had some difficulty in coughing and that her sclerae assumed a plum-coloured tinge. Temporarily she made a partial clinical recovery, but her cough persisted. On the seventh post-operative day she felt so well that she asked to be allowed to get up, and enjoyed it. She then complained of attacks of numbness in both thighs, occurring only at night and lasting for about one hour; but there was no objective evidence of peripheral neuritis, and the cerebrospinal fluid was normal. On the 14th day she first complained of weakness and could hardly raise herself in bed. The trunk muscles, thighs, and upper arms showed great loss of power, while the reflexes appeared normal. There was some improvement in the right arm and the thighs after two days, but the other affected muscles remained weak. At this time she developed tachycardia, 120 a minute, and her blood pressure rose from 108/75 to 135/95 mm. Hg. The attacks of abdominal pain had returned on the ninth post-operative day and were severe. On the 18th post-operative day she lost her voice and did not regain it. On the 24th day she had great difficulty in swallowing, and next day the diaphragm became involved and she died with a generalized flaccid paralysis.

During the whole course of her illness the urine was pigmented, the intensity varying from a faint pink tinge to a deep port-wine colour.

Further Investigations.—Once the diagnosis of acute porphyria was established a number of other investigations were carried out in an attempt to find out to what extent the different systems were involved in the disease. The liver function tests carried out on the 19th post-operative day were as follows. Plasma proteins: total, 8.12 g.% (albumin 3.88 g., globulin 4.24 g.). Laevulose-tolerance test: fasting 2.4 mg., half-hour 8 mg., one hour 13.1 mg. per 100 ml. Thymol turbidity test ++; cephalin-flocculation test negative; van den Bergh test negative, direct; blood bilirubin, 1.35 mg. per 100 ml.; icteric index, 6.

Bile could not be detected in the urine post-operatively. On the 12th post-operative day the serum chloride and sodium levels were 325 mg.—336 mg. per 100 ml. respectively. Renal-function tests were carried out from the 12th to the 18th post-operative day and showed no abnormality: urea-clearance test 82%; urea-concentration test, concentrated to 4.6 g.% in the second hour; blood urea, 26 mg. per 100 ml. The blood picture on the ninth post-operative

day was: red cells, 5,840,000 per c.mm.; Hb, 14 g.; P.C.V., 52%; M.C.V., 89 μ^3 ; M.C.H.C., 27%; white cells, 10,000 per c.mm. The water-excretion test (Kepler-Power) was carried out on the 24th post-operative day and gave the following results: largest volume of day urine, 21 ml.; volume of night urine, 72 ml.; urine urea, 3.4 g.; urine chlorides, 320 mg./100 ml.; plasma sodium chloride, 565 mg./100 ml.; plasma urea, 45 mg./100 ml.; value of "A," 33. The urine contained 285 mg. of uroporphyrin per litre at the time of death.

Post-mortem Examination.—The lungs showed mucopus in the larger bronchi and patches of unresolved pneumonia at both bases. No abnormality was detected macroscopically in any of the other systems. Sections of the lungs showed bronchopneumonia. Sections of the brain and spinal cord showed none of the characteristic features of the disease.

Family History.—The mother died, aged 49 years, of a "stroke," the father in his seventies with "dropsy." Three brothers and two sisters are well; their urines were examined, and none contained porphyrins. Two cousins on the mother's side died young—one at 23 years, diagnosed as from acute ascending myelitis, the other at 21 years, diagnosed as from anxiety neurosis and pneumonia.

Discussion

Porphyria is a disease believed to be due to an inborn error of metabolism which results in the circulation of an excess of porphyrins in the blood. It has been suggested that porphyrins exert a toxic action, particularly on nervous tissue (Mason, Courville, and Ziskind, 1933; Vannotti, 1937; Thorner, 1937). The condition was first described as a clinical entity by Günther (1911). Since then a number of cases have been reported, particularly in Scandinavian and American literature, and the condition has been excellently reviewed by Nesbitt (1944). In this country attention has been drawn to the disease only recently (Chandler, Harrison, and Rimington, 1939; Discombe and D'Silva, 1945; Jörgensen and With, 1947; Abrahams, Gavey, and MacLagan, 1947; Discombe, 1948; Petrie, 1948; Davies, 1949; Gibson, Harrison, and Montgomery, 1950; Hart and Collard, 1950).

This case presents a number of the usual features of the disease. The patient had abdominal pain, neurological manifestations, tachycardia, and arterial hypertension, and she passed pigmented urine. But the most interesting and confusing feature was the marked guarding and tenderness in the right hypochondrium; this is a feature which has not been reported before, and was very largely responsible for the delay in arriving at the correct diagnosis. Her family history is also interesting, as the stories of the fatal illnesses of her two cousins suggest that they, too, may have suffered from acute porphyria.

The serum chloride and sodium levels were estimated and the Kepler-Power test was carried out because it has been suggested that there is some adrenal insufficiency in this disease (Abrahams, Gavey, and MacLagan, 1947). This contention cannot be supported by the present case. The lowering of the serum chloride and serum sodium has also been attributed to impaired renal function rather than adrenal disturbances (Davies, 1949), but again in this case we could find no evidence of renal disorder. Since this patient's serum sodium and chloride values showed no significant deviation from normal we cannot expect to throw light on this particular problem, but it is evident from this case that lowering of sodium and chloride concentration in the serum is not an invariable accompaniment of the disease.

Various drugs such as sulphonal, methylsulphonal, barbiturates, lead, etc., have been incriminated in the production of this disease. This patient stated that she did not take any drugs before her admission.

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Medical Memoranda

Failure of Aureomycin in Dermatitis Herpetiformis

Conflicting reports of the value of "aureomycin" have been published recently. In America Robinson *et al.* (1949) treated five cases of dermatitis herpetiformis with from 1 to 2 g. of the antibiotic daily. They reported invariable relief from itching and involution of vesicles within one to two weeks, and that the response was more rapid and satisfactory than could be expected with any previously known treatment. Clarence Shaw (1950) used aureomycin in two patients suffering from dermatitis herpetiformis and reported complete failure to influence the course of the disease in either patient. The dosage he used was 1 g. daily for two weeks.

Recently I have had the opportunity of treating a severe case of dermatitis herpetiformis with this drug.

Case Report

A married woman aged 68 developed erythema of both forearms in May, 1950. By July the lesions had become vesicular, involving the forearms and backs of hands, and spreading rapidly to the legs, thighs, buttocks, face, and neck, with groups of vesicles and bullae scattered over the chest and back, and accompanied by considerable itching. At no time were the mucous membranes affected.

Before admission she had received a course of penicillin injections (2,500,000 units in a week), then a course of sulphadimidine (32 g. in four days), and, finally, sulphapyridine 1 g. four times daily for a week (total, 28 g.). No improvement was noted with these measures, vesicles and bullae continued to form, and she was admitted to hospital.

Investigations.—Bulla fluid contained "a few leucocytes—no eosinophils." The patch test (potassium iodide, 20% in soft paraffin) was strongly positive. Nikolsky's sign could not be elicited. Blood eosinophilia varied between 13% and 34%. Biopsy report: "A subepidermal bulla, in the floor and lumen of which are numerous eosinophils. There is no evidence of pemphigus. Diagnosis—dermatitis herpetiformis."