Investigation.—Blood picture: Hb, 90% (13.2 g.); leucocytes, 8,000 per c.mm.; platelets, 140,000 per c.mm.; bleeding-time 2 minutes (Ivy); clotting-time, 5½ minutes (Lee and White); film appeared normal. The E.C.G. showed changes typical of an old anterior cardiac infarction. X-ray film of chest showed emphysematous and atheromatous changes. The blood urea was 48 mg. per 100 ml.; serum protein, 7 g. per 100 ml.; albumin, 3.9 g. per 100 ml. The urine contained occasional pus cells with a moderate growth of *Bact. coli*. No acid-fast bacilli were seen in the sputum. Sigmoidoscopy revealed no organic lesion in the distal 10 in. (25 cm.) of bowel.

Tentative diagnoses of anterior cardiac infarction, haemorrhagic diathesis (probably scurvy), and epilepsy were made. Accordingly he was given a high protein diet and was saturated with ascorbic acid, 1 g. daily for two weeks, followed by 150 mg. daily for a month. During the course of treatment the haemorrhages rapidly decreased, and no new ones appeared. By June 27 occult blood had disappeared from the faeces. The epilepsy was satisfactorily controlled with ½ gr. (32 mg.) of phenobarbitone three times a day. But in view of the presenting symptoms of anorexia, epigastric discomfort, loss of weight, and haematemesis, it was decided to examine the gastro-intestinal tract further, in order to exclude the possibility of a neoplasm.

On June 6 a barium-meal examination demonstrated an ulcer crater in the pre-pyloric area. The site of the ulcer led the radiologist to suggest a gastric neoplasm as the likely diagnosis. A surgical specialist confirmed the radiologist's opinion; and the patient was placed on the waiting-list for a laparotomy.

Following ascorbic acid treatment the patient continued to improve. He cast off the air of lassitude, gained weight, and began to relish his meals. On July 26 his blood chemistry was as follows: blood urea, 28 mg. per 100 ml.; serum protein, 6.3 g. per 100 ml.; albumin, 3.9 g. per 100 ml.; thymol turbidity, negative; colloid gold, negative.

Such satisfactory progress seemed to warrant a further radiological examination, and on July 11 an x-ray film revealed a normal pylorus, which was confirmed by gastroscopy the following day. Another barium-meal examination on August 12 was also normal. On September 12 gastric biopsy of a specimen taken as near as possible to the original site of the tumour revealed a natural glandular pattern, with haemorrhage into the superficial layer of the gastric mucosa.

The patient was discharged on August 29, and when subsequently seen at follow-up clinics his condition had remained satisfactory. The intrinsic pre-pyloric tumour first demonstrated was therefore thought to be a submucosal haematoma which had resolved following a course of ascorbic acid.

COMMENT

Examples of the protean haemorrhagic manifestations of scurvy are numerous. In the central nervous system the disease has simulated infantile paralysis (Lellis, 1927). Cases have been recorded with haemorrhage into the cord (Prakken, 1925), and into the brain and retina (Shapiro and Hurwitz, 1938); and the less bizarre complication of scorbutic neuritis was reviewed by Stewart (1925). Death from suprarenal haemorrhage whilst under treatment was reported by Worms *et al.* (1937); and pericardial haemorrhage complicating scurvy was discussed by Barton and Freeman (1934). There was no anaemia in this case, but, as pointed out by Crandon *et al.* (1940), anaemia and scurvy need not coexist.

Gastro-intestinal complications of scurvy are not common; a dyspeptic type of diarrhoea with a sanguineous discharge may on occasions be present (Price, 1933), and Whitby and Britton (1950) mention haematemesis as one of the more rare complications. Foote (1926) reported a case of scurvy simulating acute intussusception, but I can find no record of a gastric submucosal haematoma. Nowadays, when gastrectomy is one of the commoner operations, and

intensive radiological investigations are part of a hospital routine, this case may be of interest; though it illustrates a rare complication of scurvy, it is one which could be too easily mistaken for a gastric carcinoma.

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Scurvy in Old Age

There are so many sources of vitamin C in a natural diet that the gross deficiency which produces scurvy occurs only in exceptional circumstances. Such circumstances are illustrated in the following case.

CASE REPORT

A man aged 80 was admitted to Stapleton Hospital on June 4, 1953. He and his wife, a childless couple both old-age pensioners, lived together until December, 1952, when the wife was taken to hospital under a statutory order. She was found to be a senile dement and died a month after admission.

The patient continued to live alone, and for a time meals were supplied to him by the local authority mobile meals service. Being deaf and forgetful, he failed to answer the door so often when the van called that the service was discontinued. In June, 1953, a neighbour informed the police that he had not been seen for some days. He was therefore taken to a hostel and subsequently transferred to hospital.

The house in which he lived was stated to be in a filthy condition. There was no evidence of recent cooking, and money was found scattered about the rooms. Further investigations revealed that he had assets of over £1,500.

On examination he was found to be very deaf, mentally confused, and incapable of giving any account of himself. There was no evidence of wasting, but his skin and conjunctivae were icteric and he had large extravasations extending practically from the buttocks to the heels on both sides. The left leg was also oedematous. There were smaller areas of bruising on the posterior surfaces of the arms, and pin-point haemorrhages on the chest, which were not definitely related to the hair follicles. Splinter haemorrhages were seen under the nails and ecchymoses on the soft palate and conjunctivae. The patient was edentulous but the gums were healthy. No abnormalities of the hair follicles were seen. Blood pressure was 130/80 mm. Hg. There was evidence of a moderate degree of generalized arteriosclerosis in conformity with his age. No gross clinical abnormalities were revealed by further examination.

The blood count on admission showed 2,250,000 red cells per c.mm., of which 5% were reticulocytes; haemoglobin, 6.5 g. per 100 ml.; white cells, 3,590 per c.mm. (49% polymorphs, 47% lymphocytes, 4% monocytes). Mean corpuscular volume, 97 cubic μ ; plasma bilirubin, 2.6 mg. per 100 ml. The reticulocytes had dropped to 2% by the sixth day after admission.

A preliminary examination of the urine showed him to be excreting 7.3 mg. of ascorbic acid per 100 ml. After ingesting 700 mg. of ascorbic acid daily for 12 days the concentration rose slowly and only reached 19.5 mg. per 100 ml. on the twelfth day. His petechial haemorrhages had disappeared by the third day after the saturation regime was begun and the large areas of bruising on the legs had faded at the end of a week.

Six days after admission his blood count showed 3,890,000 red cells per c.mm.; haemoglobin, 9.5 g. per 100 ml.; white cells 3,100 per c.mm. (46% polymorphs, 50% lymphocytes, 1% basophils, 1% eosinophils); M.C.V., 68 cubic μ ; the film showed hypochromia. The patient had received no iron or other therapy apart from his ascorbic acid.

At this stage he was put on iron and ammonium citrate mixture, 1 g. three times daily, and given totals of 350 mg. of thiamine, 350 mg. of nicotinic acid, and 10 ml. of crude liver extract by injection in the course of 10 days. The red cells increased to 4,600,000 and the haemoglobin to 14.5 g.% on July 3.

His physical condition improved rapidly, and within a fortnight he was up and about and doing odd jobs in the ward. His mental condition unfortunately showed no parallel improvement. He was discharged to the care of a niece, as he was no longer able to look after himself.

COMMENTS

Although mild degrees of nutritional deficiency are frequent enough in old people who live alone, gross deficiency syndromes amounting to clinical scurvy or pellagra are exceedingly rare. Senile purpura is often mistaken for the former, and glossitis originating from dentures or the use of antibiotics combined with peripheral vascular disease can produce a clinical picture which is sometimes mistaken for pellagra in aged persons.

During the past three years we have seen only one other case of scurvy among the aged sick, this patient being a feeble-minded man who lived alone. During the course of two years in Belmont Road Hospital, Liverpool—a hospital which catered for the aged sick of that city—one of us saw four cases of scurvy.

The history in all these cases is much the same. The old person lives alone and never gets a "cooked meal." Among this population, making tea to go with bread-and-butter or frying meat, egg, or fish is not reckoned as cooking a meal. A "cooked meal" includes potatoes and a vegetable.

Sheldon (1948), in his survey of Wolverhampton old people, showed that widows tended to preserve the home and the home routine when the partner died, whereas widowers did not. Elderly women when living alone will cook a meal in the sense described above, whereas elderly men will not. It is not surprising, therefore, that all the scorbutics we have met with in this age group were old men.

There are two institutions in our cities which cater for the needs of lonely old people and help to keep the incidence of scurvy low. For those who can walk there is the local fish-and-chip shop. For those confined to the house there is the mobile meals service. The cooked meal delivered by the local authority three times a week supplies many potential deficiencies, and the potatoes which it always contains are an excellent source of vitamin C. It is frequently reported that these people, rather than cook a meal for themselves, will consume half the meal on the day it is delivered and save half for heating up on the next day. The nutritionist might object to the wastage of vitamin C by this procedure, but in practice it seems that the meal retains sufficient vitamins to supply the needs of the old people.

The case recorded illustrates destitution in the midst of plenty which is occasionally a tragic feature of old age. There was enough money to purchase food, but neither partner was mentally capable of looking after the home. Although some improvement occurred in the patient's mental condition after iron and vitamin C and B complex supplements, there was no cure such as is seen in cases of pellagra. The mental deterioration in both partners obviously antedated the scurvy—it was a cause rather than a consequence of the deficiency.

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Kwashiorkor Syndrome in a 3-months-old African Infant

Kwashiorkor is believed to be exceedingly rare under the age of 6 months (Trowell and Davies, 1952), and in a study of 1,000 cases recorded in the literature no case was reported under the age of 3 months. Cases in the 4–6 months group were those in which breast-feeding had failed and the infant was fed on starchy foods from birth.

The following case is of interest in that the infant was breast-fed from birth and presented at the age of 3 months with all the signs of advanced kwashiorkor.

CASE REPORT

A female Jalu baby aged 3 months was admitted to hospital on March 5, 1953, weighing 6 lb. (2.7 kg.). The mother gave a history that the child had been born at home one month prematurely (birth weight not known), and although small was apparently healthy. Breast-feeding was established without difficulty and the child thrived. At the age of 1 month she developed oedema of the face and feet and started to have loose stools—an average of four bulky stools being passed in twenty-four hours. The baby continued to feed well, with very occasional vomiting after feeds. There was no history of an infection or other illness during the first three months of life.

On examination she appeared contented and quite mentally alert. There was marked oedema of the face, hands, and feet, but no apparent loss of subcutaneous fat. The skin was pale and depigmented and the hair quite white. Examination of the abdomen showed the liver to be enlarged to three fingerbreadths below the costal margin. No abnormalities were detected clinically in any of the other systems.

Laboratory Investigations.—Blood slide, negative for malaria parasites. Mantoux 1:1,000, negative. Hb, 85%; red blood cells, 3,210,000 per c.mm.; white blood cells, 4,900 per c.mm. (polymorphs 42%, lymphocytes 57%, monocytes 1%). A blood film showed no abnormalities. Urine contained no albumin. Blood urea, 25 mg. per 100 ml. Plasma proteins: total serum protein, 4.02%; albumin 1.73%; α globulin 0.27%; β globulin 1.39%; γ globulin 0.63%. Ide test, negative. Mother's Kahn test, negative. Mother's breast milk: protein, 1.42%; lactose, 7.1%; fat, 2.4%.

Breast-feeding was maintained while the infant was in hospital, as the mother had an adequate supply, an average gain of 3 oz. (85 g.) being recorded after test weighing. An additional supplement of 8 g. of protein in the twenty-four hours was given in the form of dried skimmed milk, which the child took readily.

No clinical improvement occurred, and on March 19 it was noticed that the child had become more oedematous and was disinclined to feed. Tube feeding was instituted for twenty-four hours, with expressed breast milk and dried skimmed milk, after which she seemed better and oral feeding was recommenced. On March 24 the condition deteriorated suddenly. The child became dyspnoeic and was found to have a left-lower-lobe pneumonia. Penicillin was given immediately, but the child died two days later.

At post-mortem examination the clinical finding of a left-lower-lobe pneumonia was confirmed. Examination of the abdomen revealed an enlarged pale liver. Kidneys, spleen, and pancreas were all macroscopically normal. No abnormality was discovered in any other system. The pathological report on sections of liver and pancreas was: "The liver shows generalized fatty change. There is no evidence of cirrhosis. There is some atrophy and fibrosis of the pancreas."

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