Haemosiderosis (also known as siderosis or Bantu siderosis) is known to be a common condition in the Africans of South Africa (Higginson et al. 1953, Wainwright 1957), Rhodesia (Buchanan 1966), Tanzania (Haddock 1965), and Ghana (Edington 1959). This is also true of patients admitted to the Ndola General Hospital and in traumatic cases on whom forensic autopsies are performed at the Ndola Hospital mortuary (Lowenthal, unpublished data). The subject has been reviewed by Bothwell et al. (1965).

The clinical associations of haemosiderosis are portal fibrosis and cirrhosis often leading to liver failure, diabetes mellitus, porphyria cutanea tarda, and scurvy (Bothwell et al. 1965). Pathologically the condition is characterized by the excessive deposition of iron in the form of haemosiderin granules in the reticulo-
endothelial system. (Bothwell and Bradlow 1960).

Sefet et al. (1966) have reported a series of 32 cases in Johannesburg Africans in whom siderosis was associated, probably causally, with osteoporosis, vertebral collapse and scurvy. This paper describes a similar case which we believe to be the first one reported from Zambia and possibly the first from outside South Africa.

CASE REPORT

The patient is an elderly African male who was admitted to the Kitwe Central Hospital in December 1966. The complaints were of chronic severe dorso-lumbar pain for many years which 5 years previously been unsuccessfully treated by immobilization in a plaster jacket. During the weeks preceding admission the patient's health had been very poor with anorexia, malaise and probably some weight loss. Since his retirement six years previously he had had no paid employment and had subsisted on a grossly inadequate diet consisting of maize meal or rice, with meat or fish only once or twice weekly. The only vegetable eaten was pumpkin, never more than once or twice a week. He never ate fruit. The patient readily admitted to consuming large quantities of local beer for many years and in the period preceding admission he had taken 2 gallons daily.

Physical examination revealed a frail, sick, anaemic Zambian African man aged about 60. Several small bruises were scattered over the trunk and limbs, but there was no history of trauma. Folliculitis with perifollicular haemorrhages were present on the buttocks and thighs. Typical scorbutic changes were present with swollen bleeding gums. A marked lower dorsal kyphus was present. The liver was just palpable below the costal margin, but the spleen could not be felt. Rectal examination revealed no abnormality. Abnormal findings were absent from the cardiovascular, respiratory and nervous systems. Blood pressure was 140/80 mm. Hg., the pulse 96/min. and the temperature 98 deg. F.

Investigations: Stool and urine were normal. Bence-Jones protein was absent from the urine. Blood smear was negative. Haemoglobin was 5.6 G/100 m.L., MCHC 25%, reticulocytes 2% serum iron 112 microgrammes %, platelets 100,000/cub. mm., white blood count 5,200/cub. mm. with polymorphs 40% and lymphocytes 60%. Sedimentation rate was 65 mm. in the first hour (Wintrobe). Fasting blood sugar was 80mg.%, and blood urea 36 mg.% Serum calcium was 9.3 mg.%, and phosphorus 2.7 mg.%, alkaline phosphatase 12 and acid phosphatase 3.6 King Armstrong units.

Liver function tests: urinary bilirubin was negative and a trace of urobiligenen was present. Serum bilirubin was 1.3 mg.% all unconjugated, serum protein 7.2 G % (albumin 2.3, globulin 4.9) and prothrombin index 83%.

Chest X-ray showed clear lung fields, a normal cardiac shadow and unfolding of the aorta.

X-ray of the spine showed gross vertebral osteoporosis and "codfish deformity" or collapse of thoracic vertebrae 9, 10, 11, and 12 and of the all lumbar vertebrae. (Figs. 1 & 2).

Liver biopsy showed masses of haemosiderin granules in the parenchymal cells, Kupffer cells and portal tracts Fig. 3) grade ++++, for each entity based on the code of Bothwell and Bradlow (1960).

The quantitative estimate of liver iron was 30,581
The patient was treated with blood transfusions, high-protein diet and vitamin supplements and symptomatically he has made a good recovery. However, the dorsal kyphus persists and in the 12 weeks since admission the vertebral appearance on X-ray are unchanged.

DISCUSSION

The features of the patient described here are identical with those of the patients described by Seftel et al. (1966) although our patient is older than the majority of those in the Johannesburg series. The fact that this man had scurvy can be explained by his poor diet.

Many Zambians take similar diets yet scurvy in the wards of the Kitwe and Ndola hospitals is rarely seen. Seftel et al. (1964) suggest that in siderotic individuals subsisting on a borderline diet the available ascorbic acid is irreversibly oxidized by the deposits of ferric iron. Ascorbic acid is known to be essential for the formation of bone matrix, thus deficiency of this vitamin over a long period could lead to osteoporosis and vertebral collapse. We believe this to be a likely mechanism in our patient.

Haemosiderosis in South Africa has been explained as being due to the excessive absorption of iron from a diet rich in iron and more particularly from the ingestion of alcoholic drinks with a high iron content as a result of home brewing in iron containers (Bothwell et al. 1965). This too is probably the cause in the patient reported here who had been a heavy drinker for many years. The iron content of the beer consumed in the Ndola area is at present being investigated. One sample of a home-brewed alcoholic beverage ("munkoyo") has been found to contain 2.02 mg. of iron per 100 ml.

We believe that the investigations carried out in our patient exclude other conditions likely to cause vertebral collapse, such as osteomalacia, myelomatos is or bony metastasis.

It is likely that with vigilance more such cases will be found in Zambia.

SUMMARY

A case of haemosiderosis, osteoporosis, vertebral collapse and scurvy occurring in a Zambian African male is described in detail.

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