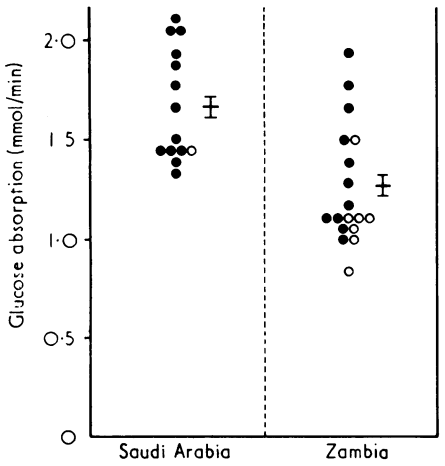
Results

The figure summarises results for the Arabs and Africans.[1] For all results ($t=4{\cdot}07$; DF=30; $P<0{\cdot}001$), and also when the perfusion segment was proximal ($t=2{\cdot}72$; DF=22; $P<0{\cdot}02$), the difference was significant (Student's t test). Mean net water absorption rate in the Arabs was 4·49 (2·30-7·44) ml/30 cm of jejunum/min and in the Africans 1·88 (−1·15-4·72) ml/30 cm of jejunum/min; this difference was significant ($t=4{\cdot}99$; DF=30; $P<0{\cdot}001$).



Glucose absorption rates (mmol/30 cm jejunum/min). Means (±SE of mean) are indicated. ● = Infusion ≤20 cm past duodenojejunal flexure. ○ = Infusion >20 cm past duodenojejunal flexure.

In the Arabs the correlation between glucose absorption rate and age, body weight, and serum albumin and globulin fractions was not significant, and absorption rates were distributed fairly evenly between the various nationalities. The correlation between glucose and water absorption rates was significant ($r=0{\cdot}77$; DF=12; $P<0{\cdot}01$).

Mean serum γ-globulin concentration was significantly lower in the Arabs than in the Africans[1] ($t=3{\cdot}05$; DF=30; $P<0{\cdot}005$), but mean serum albumin and α_1-, α_2-, and β-globulins were not significantly different.

In the additional two men glucose absorption rates were 0·22 mmol/min [0·04 g/min] in the first patient, and 1·22 mmol/min [0·22 g/min] in the second patient.

Discussion

Rapid glucose absorption in the Arabs seems most likely to have been related to the lower incidence of subclinical systemic infection[1 2 5]; mean serum γ-globulin was the only index that was significantly different from the findings in the Africans. The difference was not due to subclinical malnutrition in the Africans,[6] and, although not studied in the Arabs, jejunal morphology seems unlikely to be relevant.[7] Genetic factors have not been excluded.

Maturity onset diabetes mellitus and impaired glucose tolerance are common in Arabs in Riyadh; they are not usually associated with obesity (G C Cook, unpublished). Much refined sucrose is taken throughout the day, usually in tea; mucosal hydrolysis of sucrose is not rate-limiting in its absorption.[8] Impaired glucose tolerance might therefore be due to pancreatic β-cell stress associated with repeated portal hyperglycaemia.[9] Africans in Lusaka now consume refined sucrose in addition to their staple carbohydrate diet, but impaired glucose tolerance and maturity onset diabetes are relatively uncommon (G C Cook, unpublished); their slower glucose absorption presumably prevents excessive insulin secretion. Systemic infections may partly explain, therefore, the apparent rarity of diabetes and impaired glucose tolerance in Africans in Africa.

I thank Dr M T Al-Torki for explaining the procedure to the patients.

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SHORT REPORTS

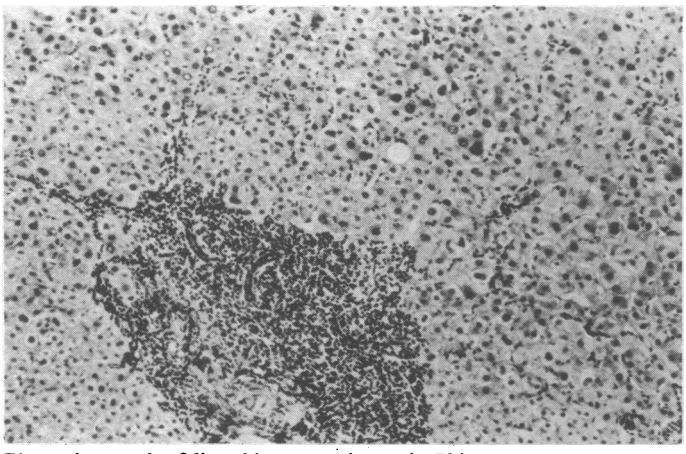
Portal hypertension in Waldenström's macroglobulinaemia

Intrahepatic presinusoidal portal hypertension and bleeding oesophageal varices due to Waldenström's macroglobulinaemia occurred in one of our patients.

Case report

A 60-year-old man, admitted as an emergency with acute retention of urine and benign prostatic hypertrophy, was seen with an additional complaint of upper gastrointestinal bleeding. He had vomited 570/ml of altered blood and passed two malaena stools on the day before admission. Bleeding continued for four days. He admitted to ill health in the previous two months, with 5 kg weight loss, increasing breathlessness on exertion, and ankle swelling. For 40 years he had suffered from psoriasis, at one time treated with methotrexate, but more recently controlled by local steroid applications. During this illness he had iritis and an arthropathy of the knees and ankles, for which he was receiving indomethacin. He was wasted, anaemic, and had extensive psoriasis. Enlarged, fleshy lymph nodes were felt in the axillae and groins, and there was moderate firm hepatosplenomegaly. Initial haematological investigation showed haemoglobin 3·6 g/dl, white blood count 5×10^9/l (5000/mm³), platelets 150×10^9/l (150 000/mm³), and the blood film showed definite rouleaux formation. The erythrocyte sedimentation rate (Westergren) was greater than 140 mm in 1 h. Total serum protein concentration was 87 g/l, with albumin 36 g/l, and gammaglobulin raised at 37 g/l. This last fraction contained a wide fast-moving paraprotein band. The bone marrow aspirate was hypercellular owing to a heavy infiltration with lymphocytoid and plasma cells, both erythropoiesis and leucopoiesis being depressed. Immunofluorescent staining showed an IgM kappa monoclone of plasma cells predominating. Waldenström's macroglobulinaemia was diagnosed. Uncorrected whole blood viscosity at two rates of shear was normal. The only coagulation abnormality was a minor increase in prothrombin time to 14·5 s against a control of 12 s. A 24-hour urine specimen contained 1·5 g protein, but Bence Jones protein was not detected. At gastroscopy oesophageal varices with an overlying erosion were seen. No peptic ulcer was found either at this examination or on barium studies.

Initial liver function tests showed a mildly raised bilirubin (which did not persist), but normal transaminases and alkaline phosphatase. The remarkable feature on liver biopsy (fig) was the heavy infiltration of the portal tracts with lymphocytoid cells of the type present in the bone marrow; the parenchymal cells and liver architecture being normal. Trans-splenic portal venography showed the portal vein to be patent and demonstrated the oesophageal varices. At puncture the intrasplenic pressure was 20 mm Hg. A hepatic



Photomicrograph of liver biopsy specimen. (×70.)

blood flow study[1] was carried out using a constant infusion method with ^{131}I-labelled rose bengal. The estimated hepatic blood flow was 1230 ml/min (normal). The hepatic vein was patent; the wedged (left) hepatic vein pressure was 15/10 mm Hg, with a mean of 8 mm Hg, and inferior vena cava pressure was 5/2 mm Hg.

Comment

Investigation showed this patient had portal hypertension of the intrahepatic, presinusoidal type.[2] This has been described in various myeloproliferative disorders and lymphoma,[3 4] but not in reports on Waldenström's macroglobulinaemia, which included a series of 40 cases,[5] although several of these patients had heavily infiltrated portal tracts. Our patient's estimated hepatic blood flow was normal, indicating that increased resistance to blood flow through the infiltrated portal tracts was causing portal hypertension rather than increased splanchnic blood flow in association with the splenomegaly. Treatment has been started with cyclophosphamide, which, if it reduces the portal tract infiltration, may also reduce the degree of portal hypertension.

I wish to thank Dr C D Holdsworth, Dr F E Preston, and Mr W Morris-Jones for permission to report this case, and Mrs N Hobson for technical help in performing the hepatic blood flow study.

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Factitious hypercalcaemia

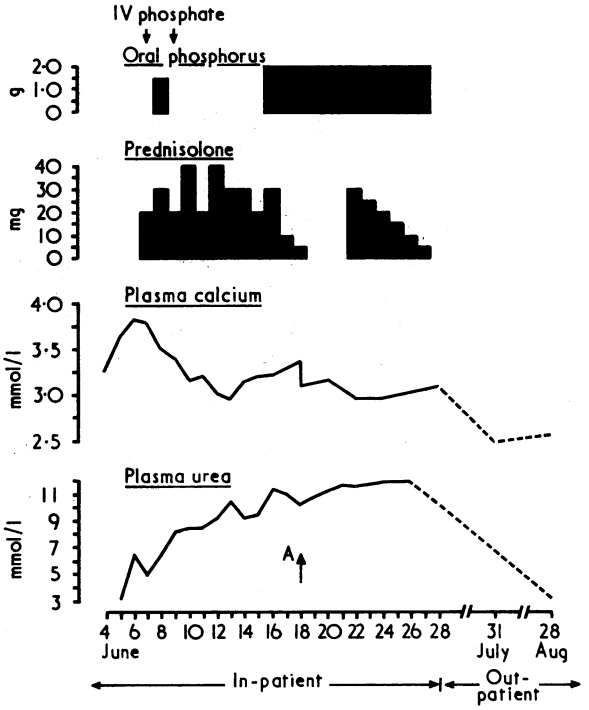
Deliberate self-intoxication with vitamin D does not appear to have been reported. In the case described here it occurred in a patient with the Munchausen syndrome.[1] The case also illustrates the value of parathyroid hormone assay in refuting a tentative diagnosis of hyperparathyroidism. The diagnosis of vitamin D intoxication was established by finding an extremely high level of serum 25-hydroxycholecalciferol.

Case report

A 34-year-old woman with a long history of deep venous thrombosis and recurrent pulmonary embolism had the first of several episodes of right loin pain in July 1972. It was discovered that her plasma calcium concentration was 2·9 mmol/l (11·4 mg/100 ml) and she was admitted for further investigation. The results of physical examination were unremarkable. Hypercalcaemia was confirmed, and the phosphorus excretion index (+ 0·09) was at the upper limit of normal, but otherwise full biochemical and radiological investigations gave normal results. Within two days she began vomiting. Concomitantly her plasma calcium rose rapidly to more than 3·5 mmol/l (14 mg/100 ml), requiring urgent rehydration and treatment with prednisolone (see figure) and two infusions of a 2·9 % sodium phosphate and 0·26 % potassium acid phosphate mixture.

Preparations were made for emergency exploration of her neck as hypercalcaemia was uncontrolled, and her blood urea was rising. At this point it was reported that parathyroid hormone was undetectable in her serum by immunoradiometric assay.[2] Aspects of her behaviour had already aroused suspicion and she was found to be in possession of about 500 strong calciferol tablets (stated strength 1·25 mg) and ampoules of calcium gluconate for injection. When confronted with this discovery she initially denied all knowledge, but then claimed that she added the vitamin D to a tumbler of water in which she kept her false teeth. During psychiatric interview there was definite communication block and a diagnosis was made of behaviour consistent with a hysterical personality disorder. It was subsequently discovered that since the age of 14 she had attended no fewer than thirteen hospitals, five being London teaching hospitals, with a wide variety of complaints including pyrexia of undetermined origin, cystitis, venous thrombosis, and abdominal pain (negative laparotomy).

The original sample of serum was found to contain 1298 nmol/l (520 ng/ml) 25-hydroxycholecalciferol (normal range 10·0-80·0 nmol/l (4-32 ng/ml)),[3] confirming vitamin D intoxication. After discharge from hospital her plasma calcium and urea concentrations fell to normal (see figure), but she has subsequently defaulted from follow-up.



Biochemical and therapeutic data. The arrow marked A indicates the point at which the patient was found to be in possession of calciferol tablets and ampoules of calcium gluconate for injection.

Conversion: SI to traditional units—Calcium: 1 mmol/l ≈ 4 mg/100 ml. Urea: 1 mmol/l ≈ 6 mg/100 ml.

Comment

Clearly this patient's illness was a manifestation of the Munchausen syndrome. Hyperparathyroidism was tentatively diagnosed before the report of serum parathyroid hormone assay became available. Although some patients with hyperparathyroidism have serum parathyroid hormone concentrations within the normal range, they usually also have a minimal rise in plasma calcium concentration.[4] Additional features which cast some doubt on such a diagnosis were the persistently normal plasma bicarbonate and chloride concentrations; unusually low plasma alkaline phosphatase, levels ranging from 3·8 to 6·3 King Armstrong units; and the unstable concentrations of the plasma calcium. There was no obvious reason for the initial rapid rise in plasma calcium, and, in our experience, such instability without clear cause is unusual in hyperparathyroidism. Preece *et al*[5]