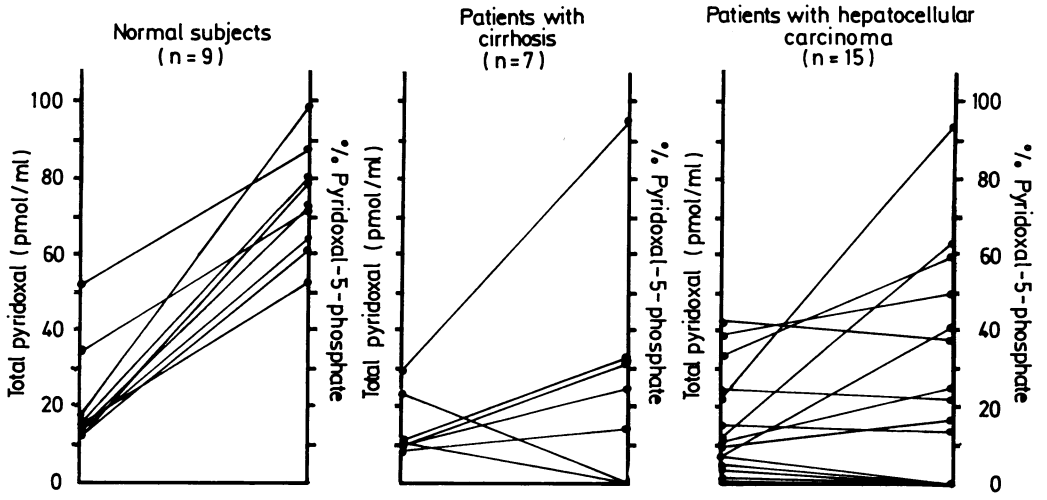
SHORT REPORTS

Vitamin B6 concentrations in patients with chronic liver disease and hepatocellular carcinoma

Vitamin B6 is the generic term for three closely related and interconvertible compounds. Most of the vitamin B6 obtained from the diet is rapidly converted by the liver to its active coenzyme, pyridoxal-5-phosphate, which has a central role in the metabolism of amino acids. Patients with chronic liver disease are commonly deficient in pyridoxal-5-phosphate, and this may be due to decreased conversion of vitamin B6 to pyridoxal-5-phosphate, although increased breakdown of pyridoxal-5-phosphate[1,2] or decreased intake or poor absorption of dietary vitamin B6 may be responsible.



Total pyridoxal concentrations and proportion of existing pyridoxal-5-phosphate in patients and controls.

Recently a method permitting estimation of concentrations of pyridoxal-5-phosphate from direct measurement of plasma total pyridoxal and free (that is, non-phosphorylated) pyridoxal concentrations was described,[3] and we applied this to patients with chronic liver diseases. Because of the high risk of hepatocellular carcinoma in patients with chronic liver disease we also studied patients with hepatocellular carcinoma with or without associated cirrhosis.

Patients, methods, and results

We studied 17 patients with cirrhosis (cryptogenic, two; chronic active, five; alcoholic, five; primary biliary, two; haemachromatosis, two; Wilson's, one), 10 of whom had histologically confirmed hepatocellular carcinoma; five patients with hepatocellular carcinoma but without cirrhosis; and nine healthy control subjects. None gave a history of dietary supplementation with vitamin B6 during the previous six months. The age range was 30-55 in the normal subjects, 18-69 in the patients with uncomplicated cirrhosis, and 20-68 in the patients with hepatocellular carcinoma. Total and free pyridoxal concentrations were measured by the method of Smith *et al*[3] and serum α fetoprotein by radioimmunoassay (AFP-RIA, Amersham, United Kingdom).

Total pyridoxal concentrations ranged from 10 to 51 pmol/ml in the nine normal subjects, a range similar to that reported by Smith *et al*.[3] Although seven of the patients with hepatocellular carcinoma had concentrations below the lowest values recorded in the control group, there was no significant difference overall between either patient group and the control subjects (figure). The concentrations of pyridoxal-5-phosphate, however, were much lower in the patients with uncomplicated liver disease, being unrecordable in seven ($p < 0{\cdot}01$, Wilcoxon's rank sum test). The proportion of total pyridoxal existing as active pyridoxal-5-phosphate was more than 50% in all control subjects but less than 50% in all patients with liver disease apart from one with cirrhosis and three with hepatocellular carcinoma. None of the patients with uncomplicated cirrhosis had increased serum and α fetoprotein concentrations, and in the patients with hepatocellular carcinoma there was no correlation between α fetoprotein and pyridoxal-5-phosphate concentrations.

Comment

The normal concentrations of total pyridoxal in most of the patients with cirrhosis suggest that deficient intake and poor absorption of vitamin B6 are not the major cause of the deficiency of pyridoxal-5-phosphate in such patients found in this and other studies. This deficiency may be due either to failure of hepatic conversion of vitamin B6 or to enhanced degradation of pyridoxal-5-phosphate. Although in this study there was no relation between α fetoprotein concentrations and pyridoxal-5-phosphate deficiency, experimental studies have shown that dietary deprivation of vitamin B6 may lead to increased α fetoprotein concentrations and preneoplastic nodules in baboons.[4] The possibility that vitamin B6 deficiency is a risk factor for the development of hepatocellular carcinoma in cirrhosis cannot be excluded, particularly as seven of the patients had lower concentrations of pyridoxal-5-phosphate than any normal subject.

1 Labadarios D, Rossouw JE, McConnell JB, Davis M, Williams R. Vitamin B6 deficiency in chronic liver disease—evidence for increased degradation of pyridoxal-5-phosphate. *Gut* 1977;**18**:23-7.
2 Lumeng L, Ting-Kai-Li. Vitamin B6 metabolism in chronic alcohol abuse. *J Clin Invest* 1974;53:693-704.
3 Smith GP, Samson D, Peters TJ. A fluorimetric method for the measurement of pyridoxal and pyridoxal phosphate in human plasma and leucocytes, and its application to patients with sideroblastic marrows. *J Clin Pathol* 1983;**36**:701-6.
4 Foy H, Kondi A, Davies JNP, *et al*. Histologic changes in livers of pyridoxine-deprived baboons—relation to alpha-fetoprotein and liver cancer in Africa. *Journal of the National Cancer Institute* 1974;53:1295-311.

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Prevalence of antibody to HTLV-III in haemophiliacs in the United Kingdom

Haemophiliacs receiving treatment with blood coagulation factor are at risk of infection with human T cell lymphotropic virus type III (HTLV-III) (or lymphadenopathy associated virus),[1] the agent thought to cause the acquired immune deficiency syndrome (AIDS).[2,3] Studies carried out in different parts of the United Kingdom have shown that many haemophiliacs now have