Serum pyridoxal in patients with gastric pathology

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SUMMARY Fasting serum pyridoxal was assayed by an automated microbiological system in 60 patients investigated for dyspepsia, patients with active peptic ulceration being excluded from the study. Gastritis was present in 30 patients, gastric carcinoma in 16, six patients had benign polyps, and, in eight patients, radiology, endoscopy, and biopsy failed to show any abnormality. Of the 52 patients with gastric pathology, 44 had a low serum pyridoxal.

We have previously shown that active gastric ulceration is associated with a low serum pyridoxal (Sanderson and Davis, 1975), whereas patients with active duodenal ulceration had normal values and that this was unrelated to diet, alcohol intake, drug therapy, age or sex differences between the two groups. This present study was performed to investigate whether this abnormality was unique to peptic ulceration or if it occurred in other types of gastric pathology.

Methods

Blood was taken immediately before upper gastrointestinal endoscopy and the serum separated and stored at -20°C until required for assay. Patients who in the month preceding endoscopy had either overt gastrointestinal bleeding or had received a blood transfusion were excluded from the study. Endoscopy was performed with the Olympus GIF Type D, and biopsies were taken in all cases *via* the endoscope. Serum folate was assayed by the method of Davis *et al.* (1970) using *Lactobacillus casei* as the test organism (reference range 2.5-18.3 $\mu\text{g/l}$). Serum B_{12} by the method of Nicholas and Pitney (1958) using *Euglena gracilis* as the test organism (reference range 160-850 ng/l), and pyridoxal by the method of Davis *et al.* (1973) using *L. casei* as the test organism. The laboratory was unaware of the endoscopic or histological diagnosis until the end of the study.

Sixteen patients had gastric carcinomas, six had benign polyps, nine had superficial gastritis, 19 had atrophic gastritis, and gastric atrophy was

present in two patients. A further eight control patients were investigated for dyspepsia, and both radiology and endoscopy plus biopsy failed to show any abnormality.

Results

A total of 60 patients (25 males and 35 females) were investigated. Their ages ranged between 41 and 89 years with a mean of 64 years. Serum folate was below normal in 13 and serum pyridoxal below normal in 44 patients, and vitamin levels in individual patients are shown in Fig. 1.

Serum pyridoxal normally decreases with age and is slightly lower in females than males (Hamfelt, 1964; Anderson *et al.*, 1970) and individual results are shown in Fig. 2 (males) and Fig. 3 (females).

Serum B_{12} was raised in two cases: 1250 ng/l and 1400 ng/l , and below normal in none. Two patients with pernicious anaemia were receiving vitamin B_{12} at the time of this study.

Of the eight patients in whom no abnormality was found, serum folate was normal in all eight and one patient had a low serum pyridoxal. Individual pyridoxal values found in the various types of gastric pathology are shown in Fig. 4, and the results for those patients in whom no abnormality was found are shown in the normal column.

Discussion

Dietary insufficiency, malabsorption, increased utilization or excretion, or any combination of these factors, could be responsible for the low serum pyridoxal found in the majority of patients with gastric pathology.

Anorexia and a consequent poor diet frequently

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Fig. 1 Serum pyridoxal/serum folate in individual patients. ● Gastritis. ▲ Carcinoma. ▼ Polyp.

Fig. 2 Serum pyridoxal/age in males. The lower limit of the normal range is shown as a continuous line.

accompany gastric pathology and dietary insufficiency may be the cause of a low serum pyridoxal found in our patients. An associated deficiency in folate might be expected to accompany such an abnormality and, of 13 patients with a folate below 2.5 ug/l, 12 had a serum pyridoxal of 12 or less.

However, in the 26 patients with a serum pyridoxal of this order, serum folate varied between 0.9 ug/l and 15.5 ug/l. No significant difference ($P > 0.05$) was found between the serum folate values of the gastritis and carcinoma groups, although weight loss of greater than 3 kg in the preceding three

Fig. 3 Serum pyridoxal/age in females. The lower limit of the normal range is shown as a continuous line.

Fig. 4 Individual serum pyridoxals in pathological groups. The mean values for each group are shown as an interrupted line. (Because we are unable accurately to assay values below 12 nmol/l, values below this figure are assumed to be 11 nmol/l for the purpose of this comparison.)

months was common in the latter group and infrequent in the former, as far as could be ascertained by reference to previous hospital attendances or historically when no record was available. This

group of patients is obviously highly selected as all were referred for endoscopy because of clinical suspicion of gastric pathology, whether or not this was confirmed by radiology, but, in those eight patients who were radiologically, endoscopically, and histologically normal, only one had a low serum pyridoxal, and we were unable to ascertain that the dietary habits of this group varied markedly from those patients in whom gastric pathology was found.

Animal experiments (Booth and Brain, 1962) have shown that no significant absorption of pyridoxine occurs from the stomach and that the jejunum is the site of absorption of this form of vitamin B6. There is also strong circumstantial evidence available (Morris *et al.*, 1970) to show that the jejunum is the main site of vitamin B6 absorption in man. None of our patients is known to suffer from small bowel disease, although no studies of small bowel function were performed.

Surveys of alcoholic patients (Hines and Grasso, 1970; Davis and Smith, 1974; Lumeng and Li, 1974) have shown that about half have low serum pyridoxals, but only two of our patients admitted to a regular large intake of alcohol. No patient was receiving anticonvulsant therapy, L-dopa, isoniazid, or penicillamine, drugs previously reported (Biehl and Vilter, 1954; Hagberg *et al.*, 1966; Hollister *et al.*, 1966; Evered, 1971) to be associated with abnormalities of vitamin B6 metabolism, and we have previously shown that our premedication of pethidine and atropine does not effect fasting serum pyridoxal levels (Sanderson and Davis, 1975).

We have recently investigated a patient who, in 1974, was one of the controls used in the establish-

ment of a normal range of serum pyridoxal in this hospital and the serum B₁₂, folate, and pyridoxal were within the normal range (serum B₁₂ 335 ng/l; serum folate 10.2 ug/l; serum pyridoxal 49.8 nmol/l). Six months after these results were obtained, she complained of episodes of epigastric discomfort and a barium meal was strongly suggestive of an antral ulcer. This was confirmed at endoscopy and biopsy and cytology showed the lesion to be benign. Vitamin assays were repeated at endoscopy and serum B₁₂ and folate were still within the normal range (B₁₂ 250 ng/l; folate 8.1 ug/l), but the serum pyridoxal had now fallen to below normal (16 nmol/l). This patient is unique in our experience and it is improbable that we will have further cases in our control population; however, it does suggest that the development of gastric ulceration is accompanied by a fall in serum pyridoxal to below normal values.

This present report shows that a low serum pyridoxal is not restricted to patients with active gastric ulceration but occurs with other common forms of gastric pathology. Further study of absorption and excretion of vitamin B₆ in patients with gastric pathology appears to be indicated before relevant conclusions can be drawn regarding this particular association.

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