

Hepatic microsomal enzyme induction: treatment of vitamin D poisoning in a 7 month old baby

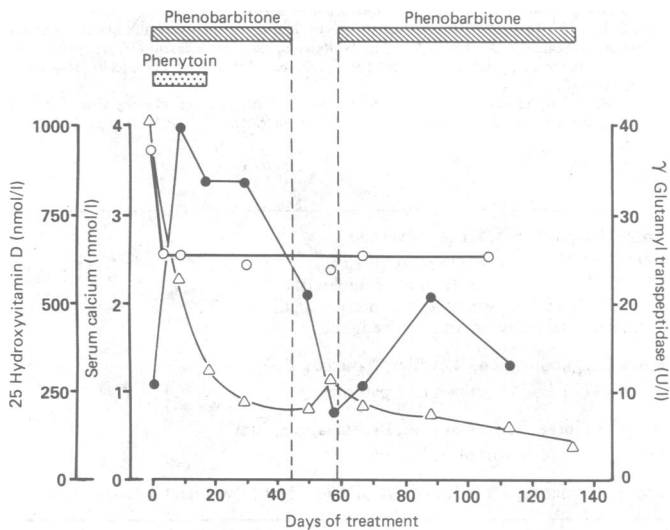
Although it is well known that vitamin D should be used with care, cases of serious and sometimes lethal intoxication have been reported.^{1,2} Vitamin D can be stored in adipose tissue in large quantities, and its removal takes a long time. Iqbal and Taylor used induction of hepatic enzymes to treat a 77 year old woman intoxicated with vitamin D.^{3,4} We recently adopted this method to treat a baby who had received an overdose of vitamin D.

Case report

The patient, a boy, was born at term (birth weight 3800 g, Apgar score 10); he was breast fed during the first two and a half months of life and received 400 U cholecalciferol daily from the neonatal period. He remained in good general and nutritional health, but when he was 5 months old an "adequate" dose of cholecalciferol was prescribed. By mistake he was given 60 000 U daily for 50 days and then 120 000 U (3.0 mg) daily for 39 days. The treatment was stopped after he developed severe symptoms of intoxication, and he was admitted to hospital.

Examination and a history obtained from the mother showed him to have severe anorexia, constipation, and pallor; he was irritable or sleepy with conjunctivitis and moderate hepatomegaly. Urea, creatinine, and bilirubin concentrations and glutamic oxaloacetic transaminase and glutamic pyruvic transaminase activities were normal. Urinary and serum calcium concentrations were both 3.75 mmol/l, serum phosphate concentration was 1.29 mmol/l, and alkaline phosphatase activity was 94 U/l. Vitamin D concentration, measured by high performance liquid chromatography, was 419 nmol/l and 25-hydroxyvitamin D concentration, measured by a radiocompetitive test,⁵ 1025 nmol/l.

All dairy products were immediately removed from the diet, and frusemide 1 mg/kg body weight/day was given for one week. Simultaneously phenytoin 5 mg/kg/day was given for 17 days and phenobarbitone 5 mg/kg/day for 133 days. Phenobarbitone was stopped for 14 days on the 44th day of treatment. After the first nine days of treatment there was a great improvement with serum and urine calcium concentrations becoming normal. When the phenobarbitone treatment was stopped the symptoms returned, but when it was restarted the child again improved. Treatment was stopped after 133 days.



Serum calcium (○—○) and 25-hydroxyvitamin D (△—△) concentrations and γ glutamyl transpeptidase activity (●—●) during treatment. Vertical broken lines indicate 14 days when treatment was stopped.

The figure shows serum calcium and 25-hydroxyvitamin D concentrations and γ glutamyl transpeptidase activity (a measure of microsomal enzyme induction) during treatment. At the end of treatment the child's general condition was good, vitamin D concentration was moderately raised at 67.1 nmol/l, but 25-hydroxyvitamin D concentration (100 nmol/l) was within the normal range; roughly three months later these concentrations were 53.6 nmol/l and 122 nmol/l, respectively.

Comment

To speed up the removal of vitamin D from this child we induced the hepatic microsomal enzymes by giving phenobarbitone and phenytoin.

These drugs are routinely used as anticonvulsants and are known to decrease vitamin D concentration and often lead to faulty bone mineralisation. Iqbal and Taylor used glutethimide for the same purpose.³ Our case shows that excess vitamin D in a baby can be dealt with by hepatic enzyme induction. Treatment should be continued for at least three months and should be ended only when the 25-hydroxyvitamin D concentration has dropped to within the range 100-150% of normal.

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Jazz ballet bottom

Abscesses in the natal cleft region are usually associated with infection of a pre-existing sacrococcygeal pilonidal sinus. The patients affected are commonly young men, and typically there is appreciable local growth of hair. The abscesses in such cases usually are associated with a proved sinus or pit and usually contain hairs.

We report three cases of natal cleft abscesses in young women in which there was no evidence of a pilonidal aetiology. All the cases were strongly associated with local trauma caused by jazz ballet exercises.

Patients, methods, and results

During the six months from November 1986 to April 1987 three young women were referred to this hospital as emergencies with presumed pilonidal abscesses of the natal cleft. In all three cases the onset of symptoms was clearly related to a period of jazz ballet exercises.

In all three cases the patients had no relevant medical history, and the result of a random blood glucose test was normal. The abscesses were all drained under general anaesthesia, and none was found to contain hairs or have other evidence of a pilonidal origin. In all three cases *Staphylococcus aureus* was cultured from the pus. The wounds were dressed regularly with eusol and gauze, and two healed uneventfully. The third patient, however, promptly returned to her jazz ballet classes after her wound had healed and equally promptly returned three weeks after her first admission with a recurrence of her abscess. This was treated in a similar fashion, and again *S aureus* was cultured from the pus. The wound healed uneventfully, and after advice to avoid those jazz ballet exercises that entail trauma to the sacrococcygeal region there was no further recurrence.

Comment

Jazz ballet is a fairly new form of recreation that includes various exercises to music, some of which require the participant to sit on the floor with most of the body weight supported by the sacrococcygeal region. Considerable frictional trauma to this part of the body may occur, and it is apparently common to experience transient soreness after a class (personal communication). In our patients this progressed to the formation of a subcutaneous abscess, presumably due to infection of a local haematoma.

The role of local trauma in the aetiology of true pilonidal disease of the natal cleft has been discussed extensively in medical reports^{1,2}; indeed, this condition has been termed jeep disease from the association of trauma with the seat of a jeep.⁴ Several reports of series of pilonidal abscesses describe atypical cases in which hairs could not be found associated with the lesions,^{1,2} and we believe that our cases show that local trauma can produce abscesses in

this region without a pre-existing pilonidal sinus. In other sites infection or inflammation related to trauma is associated with the presence of a bursa—for example, olecranon bursitis and the parapatellar bursitis—but there was no evidence in our cases of an adventitious bursa at operation. It would be interesting, however, to survey a sample of jazz ballet enthusiasts to see whether any have an uninfected bursa in this site.

Cases like those reported here are worth recognising, because in the absence of an underlying sinus the prognosis for full recovery without recurrence must be much better. Patients who are predisposed to infection should perhaps be advised against those exercises that result in trauma to the sacrococcygeal region.

We thank Mr W G Everett, consultant surgeon, for permission to report on his patients.

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Campylobacter pylori, gastritis, and peptic ulcer disease in central Africa

Peptic ulcer disease and gastritis are common medical problems in Rwandese patients. Among 3040 patients referred to the endoscopy clinic of the Centre Hospitalier de Kigali between April 1981 and April 1986, almost 900 (29%) had active duodenal ulcer and over 2550 (84%) various degrees of gastritis (unpublished). Recently a correlation has been reported between the presence of *Campylobacter pylori* in gastric mucosa and peptic ulcer disease.¹ The aim of this prospective study was to discover the prevalence of *C pylori* among Rwandese patients.

Patients, methods, and results

During May and June 1986, 184 upper digestive fibroscopies were performed in 173 patients with dyspepsia (94 women (mean age 34.3), 79 men (mean age 35.0)) and the following appearances recorded: duodenal ulcer (43 cases), healed duodenal ulcer (6), gastric ulcer (2), gastritis (90), neoplastic disease (7), normal (25). Eleven patients with duodenal ulcer had repeat endoscopy with antral biopsy for culture of *C pylori* after six weeks' treatment with ranitidine. Two antral samples and one fundal biopsy sample were taken from 166 patients. One antral and the fundal sample were submitted in 10% formaldehyde solution for histopathology. All sections were stained with haematoxylin and eosin and samples from 17 patients by the Warthin-Starry technique. Gastritis was graded as superficial if the infiltrate was limited to the upper layer of the lamina propria and global if the lamina propria was entirely invaded. The pattern of inflammation was graded 1 to 6 according to its activity (linked to the presence of

polymorph infiltration) and severity. Lymphocytic gastritis, recently described as a histological entity,² is characterised by massive lymphocytic infiltration of the epithelium. Antral biopsy samples for microbiology were crushed between two sterile slides. One slide was examined after staining with 1% gentian violet solution. The tissue fragment adhering to the other slide was plated on to Skirrow's medium³ and incubated for seven days at 37°C in an anaerobic jar containing a Gas-Pak without catalyst. All 20 isolates examined in Belgium were confirmed as *C pylori*. Among the seven patients with neoplasm only one biopsy sample was sent for microbiology, the others being used for diagnosis of the tumorous process.

A total of 131 patients were infected with *C pylori* with no difference in prevalence between men and women. They included all patients with duodenal ulcer, 67 of the 90 (74%) with an endoscopic diagnosis of gastritis (52 of the 72 (72%) with mucosal oedema, 15 of the 18 (83%) with atrophy), three of the seven with neoplasm, and 12 of the 25 (48%) with endoscopically normal gastric mucosa (table). Various degrees of gastritis were present in all patients with adequate biopsy samples. *C pylori* was isolated from 18 of the 39 (46%) with quiescent gastritis and from 95 of the 99 (96%) with active gastritis. Active gastritis was present mainly in groups with the highest level of infection: in 29 out of 39 (74%) patients with duodenal ulcer, 43 out of 64 (67%) with mucosal oedema, and 13 out of 15 (87%) with atrophy, a lower level being found in the "normal" group (10 out of 19; 53%). Repeat endoscopy in the 11 patients with duodenal ulcer disclosed complete healing of ulcer, though nine remained infected. There was a strong correlation between the results of culture and direct visualisation after staining with gentian violet or by the Warthin-Starry technique.

Comment

This study disclosed a high level (75.5%) of *C pylori* gastric infection in Rwandese patients and a close relation with duodenal ulcer and active gastritis. The clinical significance of the organism remains to be elucidated. This study on *C pylori*, the first from central Africa, suggests a widespread prevalence of the organism, similar results having been reported from other continents.^{4,5}

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Histopathology in diagnostic groups and numbers of patients from whom *C pylori* was cultured (figures in parentheses)

	Grade of inflammation	Duodenal ulcer	Healed duodenal ulcer	Gastric ulcer	Gastritis			Total
					Oedema	Atrophy	Normal	
Quiescent gastritis	S 1		2 (1)		1 (0)	2 (1)	1 (0)	6 (2;33%)
Active gastritis	S 3						1 (1)	1 (1)
Quiescent gastritis	G 1	8 (8)	3 (2)	1 (1)	14 (4)		7 (1)	33 (16;48%)
	3	20 (20)	1 (1)	1 (1)	35 (33)	7 (6)	6 (5)	70 (66;94%)
Active gastritis	4	9 (9)			10 (10)	4 (4)	3 (3)	26 (26;100%)
	5					1 (1)		1 (1)
	6					1 (1)		1 (1)
Lymphocytic gastritis		2 (2)			4 (3)		1 (0)	7 (5;71%)
Inadequate		4 (4)			8 (2)	3 (2)	6 (2)	21 (10;48%)
Total		43 (43)	6 (4)	2 (2)	72 (52)	18 (15)	25 (12)	166 (128;77%)

*S=Superficial. G=Global.