Age and sex distribution of 116 children with paracetamol poisoning

Age (years)	No of patients (male:female)	Total No of patients
1-4	44:42	86
5-10	2:1	3
11-13	6:21	27

with a maximum plasma SGOT of 3129 IU/l; after haemodialysis on two occasions she made a full recovery.

Liver function tests were performed in 19 patients aged 1 to 4 years: 16 patients had normal results and three patients had maximum plasma SGOT concentrations not exceeding 250 IU/l. Ten of the 19 patients aged 11 to 13 years had normal liver function values; one patient had a maximum plasma SGOT concentration of over 250 IU/l but less than 1000 IU/l; and two patients, one of whom has already been discussed, developed plasma SGOT concentrations greater than 1000 IU/l.

Twenty-seven patients received gastric lavage, 60 ipecacuanha syrup, 19 methionine, and 20 supportive therapy only. All patients made a satisfactory recovery.

Comment

It is remarkable that with one exception there were no serious complications in the 116 children reviewed and that all made full recoveries. The child who developed hepatic encephalopathy was given methionine at a later stage than recommended,³ which may have contributed to the encephalopathy. The patients seemed to fall into two groups: children aged 1 to 4 years, who had an equal male:female ratio and ingested small quantities of paracetamol with minimum liver damage; and children aged 11 to 13 years with a female: male ratio of 3:1 who ingested larger quantities of paracetamol with a greater likelihood of liver damage. The peak incidence of all forms of poisoning in young children is known to occur at 1 to 4 years, and this probably represents true accidental poisoning; in contrast, the high incidence of paracetamol poisoning at 11 to 13 years is likely to be due to self-poisoning. From these findings it is clear that small children rarely require treatment with a specific antidote, though older children of about 11-13 years of age may require active measures. It would seem prudent to administer to this latter group the adult dose of oral methionine (2.5 g every four hours to a total of 10 g), so long as no more than 10 hours have elapsed from the time the drug was ingested.

¹ Volans, G N, *Journal of International Medical Research*, 1976, 4, suppl No 4, p 7.

² Office of Population Censuses and Surveys, *Mortality Statistics: Accidents and Violence*. London, HMSO, 1975.

³ Crome, P, *et al*, *Lancet*, 1976, 2, 829.

(Accepted 7 June 1978)

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Pseudomembranous colitis in a colostomy

Pseudomembranous colitis is now a well-recognised complication of antibiotic treatment,¹ but has also been reported after surgery and in patients with chronic debilitating diseases.² The aetiology is not yet proved but circumstantial evidence suggests that it results from an alteration in colonic bacterial flora allowing overgrowth of a pathogenic bacterium.³ A case of pseudomembranous colitis causing colostomy dysfunction without any preceding antibiotic treatment is described.

Case report

A 69-year-old woman was admitted to hospital with a three-month history of gradually worsening intractable diarrhoea and leakage from her colostomy. She had a history of Crohn's disease necessitating a left hemicolectomy four years before this admission. It was expected that the colostomy would require to be refashioned. On admission the mucosa of the colostomy was noted to have white-yellow plaques and endoscopy showed similar lesions within the ascending colon. A biopsy specimen of the plaques on the colostomy showed the typical features of pseudomembranous colitis. There were focal areas of glandular disruption and necrosis with associated acute inflammation and an overlying membrane of mucin, polymorphs, and fibrin typical of a type 2 lesion (see figure). A biopsy specimen of the surrounding mucosa appeared normal and there was no evidence of

Glandular disruption associated with mucin and polymorph pseudomembrane. (H and E. x 100.)

Crohn's disease. Antibiotics had not been prescribed by her hospital doctors or her general practitioner in the preceding two years. Culture of faeces failed to grow *Clostridium sordellii* or *Cl difficile*. Treatment with metronidazole (1.5 g a day) resulted in disappearance of the plaques and improvement in the patient's symptoms within seven days. The patient was discharged without requiring surgery.

Comment

The diagnosis of pseudomembranous colitis rests on the awareness of the condition and the typical histological features of an appropriate mucosal biopsy specimen,⁴ and is supported by a rapid improvement in the patient's condition after treatment with vancomycin or metronidazole.⁵ This case shows that pseudomembranous colitis may occur in the absence of a preceding history of antibiotic treatment and may be an unreported cause of colostomy dysfunction.

I thank Mr H I Tankel for permission to publish.

¹ Ecker, J A, *et al*, *American Journal of Gastroenterology*, 1970, 54, 214.

² *British Medical Journal*, 1978, 1, 669.

³ George, R H, *et al*, *British Medical Journal*, 1978, 1, 695.

⁴ Price, A B, and Davies, D R, *Journal of Clinical Pathology*, 1977, 30, 1.

⁵ Dink, H T, Kernbaum, S, and Frottier, J, *Lancet*, 1978, 1, 338.

(Accepted 21 June 1978)

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Correction

Prevalence of latent perhexiline neuropathy

In the paper by Dr Alain Sebillé (20 May, p 1321) the name of L Rozensztajn, MD, associate professor of experimental medicine, Institute of Cardiology of the Hôpital Saint-Antoine in Paris, was omitted as a coauthor.