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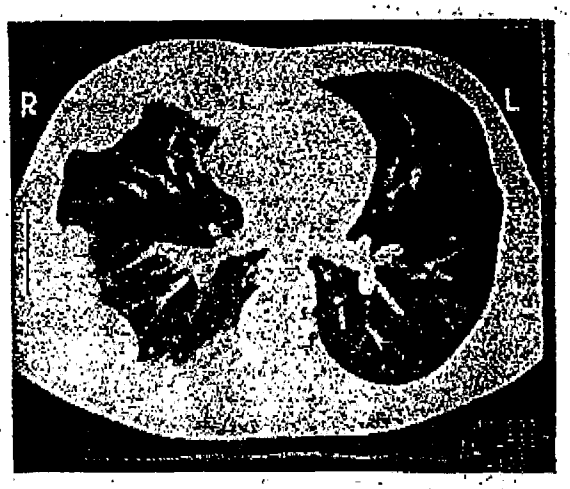
Letters to the Editor

MALIGNANT PLEURAL MESOTHELIOMA FIVE YEARS AFTER DOMESTIC EXPOSURE TO BLUE ASBESTOS

SIR,—An association between malignant pleural mesothelioma and asbestos exposure has been confirmed,[1] and the latent period between exposure and emergence of the tumour is usually said to be 20–60 years (average 35–40 years).[2] We report a case in which a 28-year-old man presented with advanced pleural mesothelioma, 5 years after brief domestic exposure to blue asbestos. We believe this to be the youngest reported case. Although it is recognised that a few cases of malignant pleural mesothelioma have been recorded without documented evidence of industrial asbestos exposure,[3] we believe that there is sufficient evidence in this case to suggest that the tumour was initiated by a short period of domestic blue asbestos exposure.

This 28-year-old man presented with a 1-year history of weight loss with 6 months of night sweats and general malaise. His haemoglobin was 9·2 g/dl, erythrocyte sedimentation 128 mm/h (Westergren), and a chest X-ray revealed multiple pleural nodules, as did computerised tomography (see figure).

He had knocked down a wall whilst renovating his house 5 years previously. The wall partition was packed with blue asbestos, and over a 72 h period he had been heavily exposed to the dust. He had not worn a face mask.



Computerised tomographic scan of lung showing nodules.

A pleuropneumonectomy was done, and the tumour was found to have sufficient histological characteristics to merit a diagnosis of malignant mesothelioma—namely, focal cellular positivity for cytokeratin, negative immunoperoxidase stain for carcinoembryonic antigen, presence of cytoplasmic tono filaments, and poorly formed microvilli. There are several histological subtypes of mesothelioma;[4] this tumour was the mixed type, since it also contained both spindle and pleomorphic cells.

The patient died before external beam radiotherapy could be started, and necropsy was not done.

This case is remarkable for the short history between exposure and tumour presentation and for the brief period of exposure, which was domestic. It highlights the need for extreme care during exposure to dust environments.

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SUCRALFATE AMELIORATES PERIRECTAL PAIN DUE TO BILE ACID MALABSORPTION

SIR,—One experimental approach to treating hypercholesterolaemia is partial ileal bypass,[1] a surgical procedure that reduces bile acid reabsorption in the distal ileum. One adverse effect of this operation is persistent rectal pain induced by the bile acid malabsorption.

A man found to be hypercholesterolaemic at age 23 had a myocardial infarction at the age of 34. Coronary angiography demonstrated 90% occlusion of his circumflex artery and 50% occlusion of his right coronary artery. His persistently raised total and low-density-lipoprotein (LDL) cholesterol levels were not effectively lowered by several dietary and pharmaceutical interventions, and a partial ileal bypass was performed. In terms of plasma total and LDL cholesterol concentrations the procedure was effective:

	Plasma cholesterol (mg/dl)				*Plasma triglyceride (mg/dl)*
	Total	*VLDL*	*LDL*	*HDL*	
Before (n=4)	376±27	39+9	306±23	31+2	187±18
After (n=3)	211±18*	34+6	158±13*	20+2*	178±48

Values as mean±SEM. *Significantly lower (p<0·05).

However, after the ileal bypass the patient had intermittent diarrhoea (5 or 6 loose stools daily) with severe and persistent anal pain. Despite 8 g cholestyramine at bedtime to bind bile acids and palliative treatments such as sitz baths, topical lignocaine, suppositories, aluminium hydroxide tablets, and paracetamol, the patient continued to complain of severe pain. During the first 8 postoperative months he was admitted to hospital three times for treatment of an anal fissure and abscess. Although increasing cholestyramine to 16 g per day and adding loperamide decreased the stool frequency, the constant perirectal burning did not subside. This pain and/or the use of these medications meant that the patient was unable to carry on normal daily activities, resulting in discord within the family.

Before reversing the bypass we decided to try sucralfate. After only 4 days of sucralfate therapy (2 g every 6 h), the patient's perirectal burning was totally relieved for the first time in nearly 9 months. He reported pain relief irrespective of the amount of daily intermittent diarrhoea. Discontinuing and restarting sucralfate confirmed this agent was relieving the patient's perirectal pain. Sitz baths and the other medications were no longer needed. During the past 10 months the patient has been able to taper his dose of sucralfate to 1 or 2 g per day with symptomatic relief.

Sucralfate is comparable with cholestyramine in its bile-binding capacity[2] and has a high binding affinity for defective mucosa, forming a protective barrier over ulcer sites.[3] More than 90% of an administered dose is recovered unchanged in the faeces.[4] The most common adverse effect of sucralfate is constipation (Marion Laboratories product information on 'Carafate'). These combined properties led to the successful use of sucralfate in this patient. While a previous case-report describes the successful treatment of cholerectic diarrhoea with sucralfate,[5] we believe that this is the first evidence that sucralfate is effective in the management of perirectal pain associated with bile acid malabsorption.

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