Results of testing of specimens produced by transtracheal saline injection for 42 patients with community acquired pneumonia

No of patients	Result of testing lower respiratory tract secretion*
22	Pneumococcal antigen positive: no pathogens on culture
13	Pneumococcal antigen negative: no pathogens on culture
4	Pneumococcal antigen positive: *Str pneumoniae* on culture†
2	Pneumococcal antigen not tested: no pathogens on culture
1	No specimen obtained

* Pneumococcal antigen detected using countercurrent immunoelectrophoresis.[5]
† *H Influenzae* also cultured from two specimens.

respiratory tract secretions. Of these, five were subsequently diagnosed as having legionnaires' disease, four pneumococcal pneumonia, two psittacosis pneumonia, one Q fever pneumonia, and one pulmonary tuberculosis. Troublesome complications of transtracheal injection did not occur. Occasionally blood streaking of the specimen was noted. Three of the 42 patients subsequently had transtracheal aspirations performed and legionnaires' disease was diagnosed in two and *Haemophilus influenzae* infection in one.

Comment

Transtracheal injection of saline proved to be a safe and effective method of obtaining lower respiratory tract secretions from patients with pneumonia who were unable to produce sputum spontaneously. Examination of such specimens by culture and by countercurrent immunoelectrophoresis for pneumococcal antigen was particularly useful for diagnosing pneumococcal infection in our series. Other supporting evidence of pneumococcal infection was found for the majority of patients diagnosed in this way. The low success rate for culturing *Str pneumoniae* was probably related to frequent use of antibiotics before hospital admission.

We have used a similar technique for several years for giving intratracheal lignocaine anaesthesia before fibreoptic bronchoscopy. No troublesome complications have been noted after over 2400 such injections, confirming the safety of this technique. A detailed post-bronchoscopy questionnaire survey of 166 consecutive patients disclosed that 140 found the transtracheal injection aspect of the bronchoscopy "all right" or "a little unpleasant," 15 "unpleasant," and 11 "extremely unpleasant." Both of us have experienced a transtracheal injection of saline and neither found it painful or particularly distressing.

We also find the technique useful for the investigation of suspected pulmonary tuberculosis. When sputum is unobtainable the injection may obviate the need for gastric washings or bronchoscopy.

Although invasive techniques such as transtracheal aspiration and percutaneous lung aspiration provide specimens of lower respiratory tract secretions uncontaminated by oropharyngeal commensals, reports of serious complications and occasional deaths suggest that they should not be used routinely.[4]

When investigating a patient with pneumonia or a lower respiratory tract infection we suggest that a transtracheal injection of saline should be performed when a sputum specimen is required but is not available. Only if examination of the resulting specimen is unhelpful should more invasive techniques such as transtracheal aspiration be performed.

We thank our clinical and laboratory colleagues for their help and co-operation.

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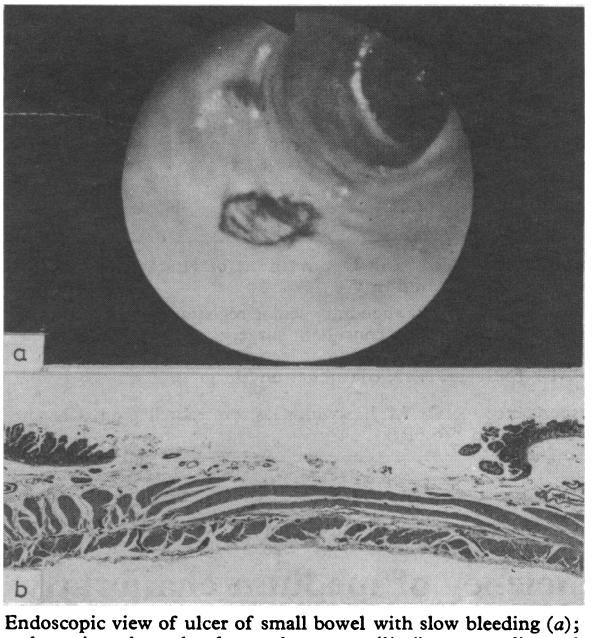
Recurrent bleeding from idiopathic ulceration of small bowel

Ulceration of the small bowel other than that seen in peptic ulcer disease and Crohn's disease is rare. Its causes include the Zollinger-Ellison syndrome, tuberculosis, syphilis, typhoid, fungal infections, enteric coated drugs such as potassium, steroid treatment, intestinal lymphoma, polyarteritis, and non-specific ulceration associated with malabsorption.[1 2] We report two cases of idiopathic small-bowel ulceration not associated with villous atrophy. Both cases were diagnosed during investigations for recurrent gastrointestinal bleeding.

Case reports

Case 1[3]—A woman aged 57 presented with heart failure and mitral incompetence and was treated with digoxin and diuretics but no potassium supplements. She was found to have anaemia with occult blood in her stools. Full endoscopy, radiology, angiography, and scintigraphy showed no cause. At diagnostic laparotomy two years later inspection and transillumination of the bowel showed no abnormality: her small bowel was inspected with a fibreoptic colonoscope (Olympus CFLB3R) introduced by mouth. Multiple superficial ulcers, all less than 1 cm in diameter and bleeding slowly, were found in the ileum (figure). Full thickness biopsy specimens showed



Endoscopic view of ulcer of small bowel with slow bleeding (*a*); and section through ulcerated mucosa (*b*) (haematoxylin and eosin) × 16.

congestion and superficial ulceration with no underlying inflammation or vasculitis and no evidence of villous atrophy or Crohn's disease (figure). After various empirical and unsuccessful medical treatments she underwent a second laparotomy in 1982 with a view to ileal resection to control the bleeding. Enteroscopy showed similar ulcers confined mainly to the jejunum. Resection of the most severely affected segment failed to control the blood loss.

Case 2—A 71 year old man was treated for cardiomyopathy with digoxin, diuretics, and potassium supplements for six years until 1980 (no potassium was given after early 1980). In 1980 worsening heart failure coincided with the finding of anaemia and occult blood in his stools. Full endoscopy, radiology, angiography, and scintigraphy failed to show a cause. In 1983 worsening anaemia and heart failure warranted diagnostic laparotomy. Inspection and transillumination of the bowel showed no abnormalities, but enteroscopy showed jejunal ulcers similar to those seen in case 1. Biopsy specimens were also of similar appearance. He had a stormy postoperative course and died of heart failure 36 days later. The bowel was unsuitable for detailed examination at necropsy.

Comment

Both patients were extensively investigated for blood loss, and no cause was found other than the ulcers described. Idiopathic ulceration associated with malabsorption is invariably visible radiologically and on inspection at laparotomy.[2] The ulcers that we found were seen

only on endoscopy of the small bowel; this operative technique was first described in 1975.[4]

Neither patient showed histological evidence of any known cause of intestinal ulceration, and neither had received potassium tablets for at least three years. Potassium tablets usually cause solitary and appreciably scarred ulcers.[5] We do not think that the ulcers were caused traumatically during enteroscopy as we did not find any in two other patients investigated by this technique. Both our patients had underlying cardiac disease, and intestinal haemoperfusion may perhaps be so compromised in such patients that minimal changes in blood flow—for example, as a result of arterial spasm or micro-emboli—can produce transient mucosal ischaemia leading to focal haemorrhage and sloughing.

This type of ulceration may be more common than published reports indicate as it would not have been diagnosed in our two patients if they had not bled appreciably and undergone enteroscopy. We believe that these two patients showed a previously undescribed form of small-bowel ulceration that may be diagnosed only by enteroscopy, which should be available as a final test in the routine and emergency investigation of gastrointestinal bleeding.

We thank Dr I M Murray-Lyon for allowing us to report on a patient under his care.

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Deficiency of medium chain fatty acylcoenzyme A dehydrogenase presenting as the sudden infant death syndrome

Disorders of fatty acid metabolism due to deficiencies of several acylcoenzyme A dehydrogenases (glutaric aciduria, type II)[1] or to a more specific deficiency of a single acylcoenzyme A dehydrogenase[2] have recently aroused great interest. These enzymes play a part in β oxidation of fatty acids in mitochondria, and disorders resulting from their deficiency may present clinically as an illness resembling Reye's syndrome, manifesting the pathological changes seen in that syndrome.[3] Deficiencies of acylcoenzyme A enzymes are thought to be inherited as an autosomal recessive trait.

We report on a boy, initially considered to be a victim of the sudden infant death syndrome, in whom pathological examination suggested Reye's syndrome. Subsequent evaluation, however, showed the absence of medium chain fatty acylcoenzyme A dehydrogenase as a single defect of mitochondrial enzymes.

Case report

An 18 month old boy started vomiting; this became more frequent with worsening, non-specific malaise and mild infection of the upper respiratory tract. Sixty hours later he suffered a grand mal convulsion and died. There was no relevant family history, and he was his parents' firstborn child.

Macroscopically, the brain was oedematous; the liver was pale but normal in size; and the nasopharynx was inflamed. Microscopic examination showed diffuse microvesicular fatty change without necrosis in cardiac and skeletal muscle, renal tubules, and the liver. No glycogen was detected in the liver.

Histochemical examination of frozen liver showed normal activities of cytochrome oxidase and succinic dehydrogenase. Homogenised frozen liver showed normal glutamate dehydrogenase activity. Activity of medium chain fatty acylcoenzyme A dehydrogenase was absent and activities of short and long chain dehydrogenases were moderately reduced when octanoyl, palmitoyl, and butyrylcoenzyme A respectively were used as substrates (table). Serum obtained from blood post mortem was analysed for organic acids by gas-liquid chromatography after discontinuous solvent extraction, and trimethyl silyl derivation showed the presence of octanoic acid. Two dimensional thin layer chromatography for amino acids did not show the presence of sarcosine. Post mortem vitreous humour glucose concentration was 0·2 mmol/l (3·6 mg/100 ml).

Activities of liver acylcoenzyme A dehydrogenases to various substrates (expressed as μmol substrate/min/mg protein)

Substrate	Patient	Mean (SD) in controls (n = 5)
Butyrylcoenzyme A	$0{\cdot}98 \times 10^{-3}$	$2{\cdot}27 \times 10^{-3}$ ($0{\cdot}77 \times 10^{-3}$)
Octanoylcoenzyme A	None detected	$1{\cdot}24 \times 10^{-4}$ ($0{\cdot}87 \times 10^{-4}$)
Palmitoylcoenzyme A	$0{\cdot}19 \times 10^{-4}$	$0{\cdot}62 \times 10^{-4}$ ($0{\cdot}46 \times 10^{-4}$)

Comment

Diffuse fatty change in the liver of a severity similar to that found in Reye's syndrome was reported in 5% of cases of the sudden infant death syndrome in one study,[4] and the authors suggested that this might be associated with some unrecognised but specific disease process in a small proportion of these deaths. The case described here appears to belong to this group and would not have been diagnosed had the enzyme investigations not been carried out.

Reye's syndrome is probably a heterogeneous disorder, and certain metabolic disorders may mimic it.[3] In Reye's syndrome there is preservation of cytoplasmic enzymes and, unlike in the present case, a reduction in most mitochondrial enzymes.[5] The activities of short and long chain fatty acylcoenzyme A dehydrogenases were moderately low in our patient (table), being 31% and 43% of the respective mean normal values. This may be explained by overlapping specificities of the three enzymes to the various substrates used. The absence of sarcosine suggests that our patient did not have a defect in the electron transfer chain distal to the dehydrogenase step.

Deficiencies in fatty acid acylcoenzyme A dehydrogenase enzymes are thought to be inherited in an autosomal recessive manner, and these inborn errors of metabolism may help to explain the slightly increased incidence of the sudden infant death syndrome in siblings of those who have already died of that syndrome. Screening of siblings is therefore indicated in families with a history of the sudden infant death syndrome. Gas-liquid chromatography of urine for organic acids is a simple way of detecting deficiencies in acylcoenzyme A dehydrogenase. These disorders may also be diagnosed antenatally by analysis of amniotic fluid cell enzymes and of liquor metabolites.

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