diagnosis of inflammatory bowel disease. They may have an inherent relationship. More subtle psychological mechanisms must also be considered.

The women in our study probably represent the severe end of the range of the irritable bowel syndrome. Thus extrapolation of these results to the syndrome as a whole is not justified. Our findings suggest, however, that sexual dysfunction in the irritable bowel syndrome is a hitherto completely unrecognised aspect of this condition that needs to be addressed.

Comment

We were unable to define a range of normal scintigraphic appearances and found no relation between areas of increased radioactivity and the distribution of inflammatory bowel disease. In addition, there was no relation between the maximum of retained abdominal radioactivity and the extent or activity of disease, some normal subjects having a higher retention of radioactivity than those with extensive inflammatory bowel disease.

One explanation for the difference between our results and those of Dawson et al may be related to bowel preparation. Unlike us, Dawson et al used mannitol; this disaccharide undergoes bacterial fermentation in the colon, tending to produce an acidic environment favouring sucralfate binding to ulcerated mucosa. After ingestion of the undigested carbohydrates, lactitol and lactulose intraluminal pH of the ascending colon falls from 6.5 to 5.2-5.6, whereas the pH in the descending colon and rectum is unaltered; probably similar changes occur after mannitol. Binding of $^{99m}$Tc-sucralfate occurs only when the exudative mucosal proteins are positively charged and, as their isoelectric points range from 4.8 to 7.2, the relatively modest change in pH that occurs after mannitol is unlikely appreciably to enhance $^{99m}$Tc-sucralfate binding.

In vitro tests of adherence of the isotope to the parent molecule and the absence of gastric mucosal uptake show that our disappointing findings were not due to disruption of the $^{99m}$Tc-sucralfate complex. We therefore suggest that labelled sucralfate attaches to luminal contents rather than adhering to the mucosa. This is supported by the caecal "hot spots" found in those patients shown to have caecal pooling at colonoscopy and by the observation that the one poorly prepared patient who had pronounced faecal retention at the time of endoscopy also had increased retention of radioactivity throughout the colon in the absence of any mucosal abnormality.

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$^{99m}$Tc-Sucralfate scintigraphy and colonic disease

Sucralfate is an aluminum substituted disaccharide, which at low pH binds to sites of ulceration in the gastrointestinal tract. Dawson et al have recently reported that radionuclide abdominal scans taken 12-24 hours after ingestion of $^{99m}$Tc-sucralfate provide a range of normal appearances in healthy subjects and the pattern of ideal or colonic abnormalities in inflammatory bowel disease. This is surprising in view of the low binding affinity of sucralfate at ileocolonic pH. We have studied a series of consecutive patients undergoing routine colonoscopy immediately preceded by $^{99m}$Tc-sucralfate scintigraphy.

Patients, methods, and results

We studied 18 patients. Bowel preparation comprised clear fluids for five days preceding colonoscopy and Picolax taken over the last 36 hours of this period. Seventeen to 20 hours before colonoscopy, at least two hours after the last dose of Picolax, each subject drank 100 MBq of $^{99m}$Tc labelled sucralfate in 10 ml fluid.

Comparison of colonoscopic findings and interpretation of $^{99m}$Tc-sucralfate scintiscans (figures are numbers of cases)

<table>
<thead>
<tr>
<th>Colonoscopic diagnosis</th>
<th>Areas of uptake</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>Caeum (2), whole colon (3), transverse colon (1), hepatic flexure (1)</td>
</tr>
<tr>
<td>Left sided colonic neoplasms (6) (6 benign polyps, 1 carcinomas)</td>
<td>Caeum (4), whole colon (1), hepatic flexure, ileum, and rectum (1)</td>
</tr>
<tr>
<td>Inflammatory bowel disease (5)</td>
<td>Caeum and ascending colon (3)</td>
</tr>
<tr>
<td>Left sided (3) Pancolitis (2)</td>
<td>Ascending and transverse colon (1), caeum (1)</td>
</tr>
</tbody>
</table>

This was prepared with a 1 g tablet of sucralfate according to the method of Vázquez et al. Labelling efficiency was 99.5% (SD 0.6%, n=10). Patients underwent abdominal scintigraphy next morning, roughly one hour before colonoscopy. Scans were reported without knowledge of the colonoscopic findings. The study was approved by the local ethical committee.

The quality of bowel preparation assessed at colonoscopy was good in all but one patient, who had adult Hirschsprung’s disease. Panocolonoscopy was successful in all patients. The table shows the final diagnoses and scintigraphic findings.

Mechanisms responsible for thirst and polyuria associated with primary hyperaldosteronism

Renal resistance to the antidiuretic action of vasopressin is the recognised cause of the polyuria and thirst that are associated with primary aldosteronism and is believed to be due to chronic hypokalaemia, which leads to renal tubular dysfunction and nephrogenic diabetes insipidus. We describe a patient with this disorder whose thirst and polyuria were principally due to an unusual form of hypothalamic diabetes insipidus; nephrogenic diabetes insipidus, although present, was minimal.

Case report

A 43 year old woman with no medical history presented complaining of thirst and polyuria, lethargy, and weakness that had persisted for six months. She was not taking any drugs and did not eat liquorice. Blood pressure was persistently raised at 170/180/100 mm Hg. She was otherwise normal on physical examination. Urine volume ranged from 4.0 to 5.7 24 hours. Preliminary investigations showed mild hypokalaemia (plasma potassium concentration 2.8-3.0 mmol/l), plasma sodium, urea, creatinine, and calcium blood glucose concentrations were all normal. Renin supine plasma renin activity was low at 3.0 pmol/min (normal range 0.8-14.5), but plasma aldosterone concentration was raised at 879.2 pmol/l (normal range 60-350)
The observation that the thirst, polyuria, and posterior pituitary function improved with spironolactone and after surgery suggests that there may be a causal link between primary hyperaldosteronism, or its metabolic consequences, and this form of diabetes. Clearly the improvement was not just related to the drug treatment. Alternatively, a coincident selective hypo-physiophobia may have occurred that caused bilateral first hypothalamic diabetes insipidus, but we believe this to be unlikely. Previous studies have indicated minor reductions in the release of vasopressin but not frank hypothalamic diabetes insipidus in association with primary hyperaldosteronism.

We are grateful to Dr D Carr, consultant physician, North Tees General Hospital, Stockton, for allowing us to study this patient. We thank Miss Wendy Pearson for secretarial help. Observational studies were supported by grants from the Medical Research Council and the scientific and research committee, Newcastle Health Authority.


(Accepted 5 June 1987)

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Very low birthweight survivors: illness and readmission to hospital in the first 15 months of life

Though there has been a decline in perinatal mortality in all birthweight groups over the past five years, there is concern about the quality of survival of very low birthweight babies (<1500 g). Reports of follow up of these babies deal almost exclusively with neurosensory development, although these children experience a wide range of illnesses and other problems.

We present information on illnesses and readmission to hospital during the first 15 months of life among surviving very low birthweight babies of singleton births born to residents of Greater Glasgow Health Board during 1983.

Patients, methods, and results

A list of registered births of babies weighing <1500 g was obtained from birth notifications. Obstetric and paediatric information was abstracted from case notes for infants who survived for at least 30 days, and parental consent for follow up was obtained at this time. Developmental and physical assessment was carried out in the children’s homes at four and 15 months, and the parent was interviewed on both of these occasions about topics including illnesses and admission to hospital.

Severe morbidity was defined as serious, prolonged, or repeated illness that was treated, usually by hospital admission, and considered to be qualitatively different from normal childhood illness. Significance of differences in mean values was calculated by a one tailed t test and differences in proportions by the standard normal curve of sample differences centred at 0.

There were 101 singleton births with infants weighing <1500 g. Of these, 17 were stillborn, 25 died in the neonatal (21) or infant (4) period, and 59 were discharged home from the nursery. Two children were lost to follow up; results for the 57 survivors are therefore presented.

Twenty six children (46%) were classified as having severe morbidity, and the prevalence of morbidity did not differ with developmental state (table). Nineteen of the 26 children with morbidity had serious or repeated respiratory tract infections, of whom 13 were admitted to hospital at least once for this indication, while four of the 26 were admitted to hospital for other reasons. Five of the 31 children classified as having no severe morbidity were admitted to hospital for minor problems such as constipation. Thus the overall rate of readmission to hospital was 42% (24 of 57 children; 45 admissions). The rate for all children in Glasgow by the age of 18 months is 13-9%.

Babies weighing <1000 g at birth or who develop abnormally are known to have a higher incidence of neonatal problems such as respiratory difficulties and...