SHORT REPORTS

Perforation of ileal duplication in old age

Intestinal duplications have an individual muscle coat, submucosa, and an epithelium corresponding to the structure of the adjacent bowel, although heterotopic mucosa is common. Such anomalies generally lie within the mesentery or mediastinum and may communicate with the bowel or remain discrete. They are either tubular or cystic in nature. Most are found during childhood, often at operation. Out of 315 cases recorded up to 1950, Dohn and Poulsen¹ found that 30% were first diagnosed in patients aged 16 and only 5% in patients over 50. Anderson *et al*² reviewed 63 cases in adults recorded between 1949 and 1959; the oldest patient was 68, the condition being found incidentally at necropsy. We report a case of peritonitis occurring after perforation of an ileal duplication in old age.

Case report

An 81-year-old man was admitted to hospital as an emergency case with a three-day history of central abdominal and right hypochondrial pain that was colicky initially but had remained constant for 24 hours. He was nauseated but had not vomited and had developed diarrhoea two days before admission. His previous history included cardiac failure (treated with digoxin and frusemide), asthma, and iron-deficiency anaemia treated with iron for 15 years. He was also taking prednisolone 5 mg thrice daily for iritis. On examination he was found to be dehydrated and feverish (37.8°C), with slow atrial fibrillation. There were signs of peritonitis from the right hypochondrium to the right inguinal ligament.

Serum electrolytes, serum amylase, and liver function values were normal. Blood urea was 16 mmol/l (96.4 mg/100 ml) (normal 2.5-7.1 mmol/l; 15-43 mg/100 ml), and a hypochromic microcytic anaemia (haemoglobin 10.6 g/dl) and neutrophil leucocytosis (white cell count $14.5 \times 10^9/l$; $14\,500/mm^3$) were found. Erythrocyte sedimentation rate (Westergren) was 110 mm in the first hour. X-ray pictures of chest and abdomen were normal. Acute perforated appendicitis was diagnosed initially, but at laparotomy a mass of oedematous omentum, mesentery, and ileum was found. Dissection showed a 6-cm duplication of the mid-ileum. Segmental ileal resection with end-to-end anastomosis was performed. The postoperative course was uneventful.

The surgical specimen consisted of 32 cm of ileum with a duplication 6.5 cm long and 1.5 cm in diameter included in the mesentery. Purulent exudate coated the bowel, and the tip of the duplication was covered with slough. On section the mucosa was seen to be arranged in coarse spiral folds, creating apparently independent loculi filled with pus (see figure). Microscopically most of the duplication was lined with congested ileal mucosa with patches of heterotopic gastric mucosa. Near the blind tip the wall had perforated, destroyed by acute transmural inflammation.

Comment

Although the pathogenesis of duplications of the gut is not yet entirely understood, the structural characteristics are well documented and the clinical features recognised. Neonates commonly present with intestinal obstruction. In infancy and childhood rectal bleeding and peritonitis occur after perforation of peptic ulcers, as do abdominal masses.³⁻⁵ Common complaints in adults are vague abdominal pain and dyspepsia, and rarely obstruction or bleeding. Intrathoracic duplications may cause dyspnoea or dysphagia, but Anderson *et al*² found no instance of sealed or free perforation among their adult cases. These differences may arise because of the much lower prevalence of small-intestinal anomalies in adults.

In this case perforation was related to several factors. Longstanding hypochromic anaemia might have indicated peptic ulceration, but none was found on microscopy. The arrangement of mucosal folds could have led to inspissated mucus plugs, obstruction of the lumen, and acute suppurative inflammation. Perforation of the tip through an area of gangrene suggests that this was the sequence of events. Steroid treatment may also have contributed to the pathogenesis.

This case shows that congenital abnormalities may remain asymptomatic until old age and should be considered in the differential diagnosis of acute abdominal complaints at any age.

We thank Professor J C Goligher for allowing us to make this report on a patient under his care.

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⁴ Collins, C D, *British Journal of Surgery*, 1972, **59**, 159.

⁵ Forshal, I, *Postgraduate Medical Journal*, 1961, **37**, 570.

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Improved method of recovering toxocara species ova from soil

The presence of ova of *Toxocara* spp in the soil is of considerable importance for public health, and Borg and Woodruff have shown that such ova are often present. Until now it has been possible to determine whether ova are present in a given soil sample, but counting the number of ova per unit of soil has presented difficulties. Such a demonstration is important for assessing the severity of environmental contamination with the ova. As a result of continued work on this subject in our laboratory the following method has been devised and has been found to be satisfactory.

Method

From the uppermost inch of the area being examined soil is collected with a trowel. When an area is covered in grass it is best to sample grass and the underlying soil separately. There may be recently deposited ova on the grass, whereas the soil will probably contain ova that have accumulated over weeks, months, or years. The samples, each of which need weigh no more than 15-20 g, are stored in screw-capped plastic containers.

Resected specimen of ileum showing duplication.

In the laboratory 5 g of soil is weighed, transferred to a stout 50-ml round-bottomed centrifuge tube, and shaken with enough water to fill the tube to within about 2.5 cm from the brim. The tube is then centrifuged at 2000 rpm for one minute. The supernatant is poured away and the process repeated. Zinc sulphate solution of specific gravity 1.180 (33%) is then added until the tube requires about 1 ml to fill it completely. The tube and zinc sulphate solution are shaken and allowed to stand for 10 minutes. Using a plastic syringe and disposable needle the clear supernatant is then withdrawn from the tube and passed through a Millipore filter pad of pore size $14.0\ \mu\text{m}$ (Millipore Corporation, Catalogue number NC WP 02500). The filter pad is held in a Millipore Swinnex 25 syringe attachment, and after filtration the pad is removed, mounted on a microscopic slide, and any ova present are counted. The count represents those present in the 5 g sample of soil.

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Time course of changes in concentration of some plasma components after frusemide

Spontaneous diurnal variations in the concentration of plasma constituents are mostly small or difficult to assess in the supine position¹ and are without consistent pattern. Change in position may, however, greatly influence the plasma concentrations, particularly of non-diffusible ions² but also of some diffusible ones,² as may meals^{1,3} and exercise.^{1,3} Potent short-acting diuretics may disturb these normally small variations, influencing many important physiological processes governing homeostasis—such as diuresis, urinary excretion of electrolytes, plasma volume,⁴ and general haemodynamic^{4,5} and hormonal balance—especially in the renin-angiotensin-aldosterone system.⁴ We report changes in some plasma concentrations after frusemide administration.

Methods and results

Two healthy people and five ambulant hospital patients volunteered for the experiments. One patient had aortic incompetence, two aortic stenosis, one coronary heart disease, and one pulmonary emphysema. Three of the patients were on long-term diuretic treatment until two days before the experiment. The previous day and throughout the day of the experiment everyone received a low-salt diet and was instructed to avoid foods rich in potassium (according to a list). Fluid intake was not restricted. Physical activity was kept to a minimum.

Special care was taken with blood sampling.^{2,3} Samples were taken from an antecubital vein through an indwelling Teflon catheter, which was kept clear by intermittent flushing with small amounts of heparinised isotonic saline. The first portion of each sample was discarded. The tubes were centrifuged at 2000 rpm for 15 minutes and the plasma was separated and recentrifuged for five minutes and then stored in sealed containers before analysis in one batch next morning. The following were measured: packed cell volume (in duplicate), total protein, sodium, potassium, chloride, calcium, magnesium, inorganic phosphorus, urea, creatinine, and urate. Baseline samples were taken at 9 am, then everyone was given 80 mg frusemide by mouth. Samples were withdrawn after 1, 3, 5, 8, and 24 hours, the volunteers having been recumbent for half an hour before sampling to ensure plasma volume equilibrium. Differences between the mean baseline values and mean values after frusemide were assessed by Student's *t* test on paired observations.

No significant changes in the mean concentrations of sodium, calcium, or magnesium were observed. The figure gives values that did change sig-

Changes in packed cell volume and in plasma concentrations of protein, potassium, chloride, inorganic phosphorus, urate, creatinine, and urea after administration of frusemide 80 mg by mouth (at time 0). Values are means $\pm$ SE of mean.

Conversion: SI to traditional units—Potassium: 1 mmol/l = 1 mEq/l. Chloride: 1 mmol/l = 1 mEq/l. Inorganic phosphorus: 1 mmol/l $\approx$ 3.1 mg/100 ml. Urate: 1 mmol/l $\approx$ 16.8 mg/100 ml. Creatinine: 1 μmol/l $\approx$ 0.0113 mg/100 ml. Urea: 1 mmol/l $\approx$ 6.0 mg/100 ml.

nificantly, the mean concentration of each constituent $\pm$ SE of the mean being related to time.

Comment

These observations show that frusemide causes acute changes in the levels of several plasma components and should be considered in any assessment of electrolyte disturbances. Different diuretics probably all have their own characteristic plasma concentration/time relationship.

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³ Statland, B E, Winkel, P, and Bokelund, H, *Clinical Chemistry*, 1973, **19**, 1380.

⁴ Rosenthal, J, et al, *Canadian Journal of Physiology and Pharmacology*, 1968, **46**, 85.

⁵ Lal, S, et al, *British Heart Journal*, 1969, **31**, 711.

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