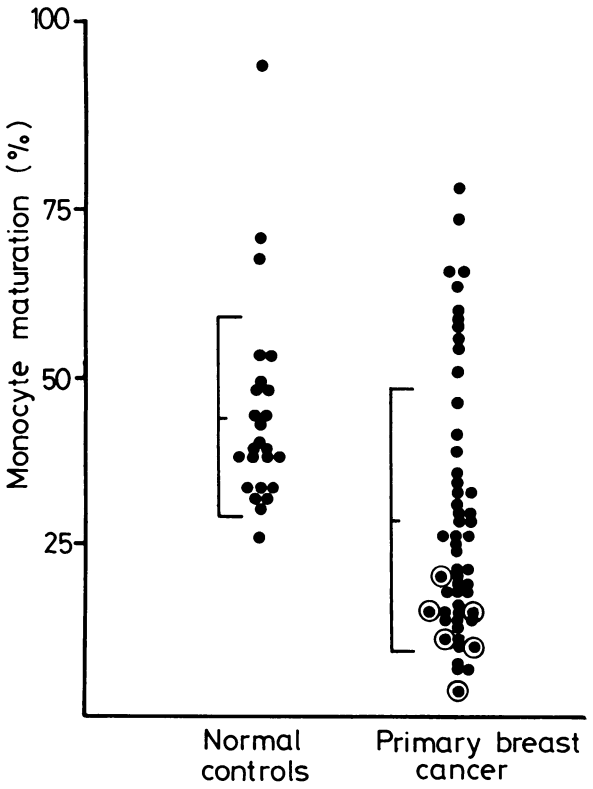
Patients, methods, and results

Blood samples for assays of monocyte function were taken before surgery from 125 women presenting to a breast clinic and the assays performed in ignorance of their clinical condition. Benign lesions were subsequently found in 36 patients and operable primary breast carcinoma in 54. None of these 54 women showed any evidence of distant metastases at the time of primary surgery. Preoperative assessment included full blood count; liver function tests; serum calcium, phosphate, and alkaline phosphatase estimations; chest radiography; skeletal survey; liver and bone scans; hepatic ultrasonography; and bone-marrow examination. A further 35 patients with overt disease, either local or distant, were examined. Age-matched normal healthy women served as controls.

In-vitro maturation of monocytes was assayed as described.[2] Briefly, defibrinated venous blood was layered on to Lymphoprep (Nyegaard) and centrifuged. Washed mononuclear cells were counted, made up to 2×10^9/l in 50% fresh autologous serum, and cultured as described. A drop of the cell suspension was also stained for non-specific esterase (NSE)[3] and the proportion of NSE-positive cells counted. Maturation was expressed as the yield of attached macrophages assayed after seven days of incubation as a percentage of the number of monocytes added (NSE positivity being used as a monocyte marker).

The figure gives the results. In normal donors the mean percentage



In-vitro maturation of monocytes in 54 women with operable primary breast cancer and age-matched normal control donors. Six patients relapsed; their values at presentation are encircled. Bars are means ±1SD.

maturation was 48·3±SD19·7 and in the patients with benign disease 45·31±9·0 (t±0·52; P>0·1). In the patients with primary breast cancer the mean maturation was 29·3±18·9%, whereas in the appropriate age-matched controls it was 44·3±14·8%; this difference was highly significant (t=3·42; P<0·001). At the time of writing, nine months after starting the study, six of the 54 patients with primary cancer had relapsed with overt recurrent disease. Their mean monocyte maturation (at presentation) was 12·6±5·0% —that is, all their values were extremely low. Furthermore, the 35 patients with overt dissemination also showed depressed maturation. Those with distant metastases (24) had a mean maturation 15·9±12·9%, whereas those with local spread (11) had a mean maturation of 28·8±16·3%. The overall group was significantly depressed (20·2±15·2%) when compared with age-matched normal women (t=6·07; P<0·001) and those with primary tumours (t=2·47; P<0·05), and the difference between women with distant and local spread was also significant (t=2·54; P<0·01).

Maturation of monocytes correlated with the diameter of the primary tumour, the larger the tumour the greater the suppression (r=0·290; t=2·184; P<0·05). Nuclear grade 3 tumours were associated with significantly lower maturation than grade 1 (P<0·02). The presence of sinus histiocytosis in the axillary nodes was also associated with significantly higher monocyte maturation (t=2·31; P<0·05). There was, however, no correlation with axillary node state (t=0·276; P>0·1).

Comment

The use of tumour-product markers[4] and immunological state[5] has failed to provide assays suitable for detecting patients whose poor prognosis dictates the need for some form of adjuvant treatment. The suppression of in-vitro differentiation of monocytes has been described in patients with malignant melanoma[2] and is related to tumour burden. In our study of 54 women with primary breast cancer (with no evidence of distant metastases) the maturation of monocytes was significantly depressed when compared with that in normal women or those with benign breast disease. Monocyte maturation in half of the patients was lower than in the lowest normal control. The most startling finding was the prognostic significance of the in-vitro results. Out of 54 women presenting over nine months, six relapsed with overt metastatic disease. These six when first seen showed severely subnormal monocyte maturation in vitro. No patient relapsed who presented with normal maturation. The number of relapses was still small. We report the data at this stage, however, to encourage the evaluation of this observation in other laboratories.

Studies in these laboratories have been supported by a programme grant from the Medical Research Council. GAC gratefully acknowledges support from the Cancer Research Campaign. We are grateful to our many clinical colleagues at the Royal Marsden Hospital who helped in the organisation of the study.

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Small-bowel volvulus in association with progressive systemic sclerosis

Progressive systemic sclerosis (PSS) is a generalised disorder characterised by deposition of increased amounts of collagen in skin, heart, lung, gastrointestinal tract, and kidney. The small intestine is affected in some 50% of cases, and in about half of these small-bowel lesions cause symptoms. We describe a patient who presented with the hitherto unrecognised complication of small-intestinal obstruction as a result of volvulus.

Case report

A 69-year-old Caucasian man was admitted to hospital as an emergency case shortly after eating a large lunch. He complained of the sudden onset of dysphagia associated with upper abdominal pain, nausea, and vomiting. His history included Raynaud's phenomenon for four years. He was a thin man with telangiectasia on the arms and neck. His facial skin was smooth and flattened with lip puckering. The fingers were cold and cyanosed. There was proximal muscle wasting of the arms. Bilateral basal crepitations were heard in the chest. The upper abdomen was distended, tympanitic, and tender. Plain abdominal radiography showed eventration of the left hemidiaphragm and multiple dilated loops of small bowel with fluid levels in the erect film suggesting obstruction. Conservative treatment with analgesics, nasogastric intubation, and intravenous fluid replacement failed to alleviate the symptoms. At laparotomy eventration of the left hemidiaphragm was seen; the stomach was rotated in a ventroaxial direction; the small bowel, which showed massive dilatation throughout its length, had volvulated around the base of a thickened, foreshortened mesentery; the sigmoid colon was also dilated. The condition could not be treated surgically, so the

abdomen was closed. A mesenteric biopsy specimen contained fibroblastic and collagenous connective tissue. Postoperatively the patient's symptoms slowly regressed, and after one week he was able to take solids.

The haemoglobin concentration was 12·6 g/dl, erythrocyte sedimentation rate 33 mm in the first hour (Westergren), and blood urea concentration 4·9 mmol/l (29·5 mg/100 ml). Tests were positive for antinuclear factor (1/1000) and rheumatoid factors (sheep cell agglutination test 1/1256; latex test 1/640). Chest radiography showed bilateral lower zone reticular shadowing consistent with pulmonary fibrosis. Results of barium studies several months after laparotomy were compatible with PSS.[1] Barium meal and follow-though examination showed a widened, atonic oesophagus 7 cm diameter at its lower end. The duodenal loop and proximal two-thirds of the small bowel were severely dilated, and transit was delayed. Barium enema disclosed a voluminous rectum and sigmoid colon, which were atonic and smooth in outline. Reduced xylose excretion (3·2 g 45 min after a 25 g load) and increased three-day faecal fat excretion (128 mmol; 36·4 g) were also compatible with gastrointestinal PSS.

The patient was treated with penicillamine and tetracycline and showed modest improvement.

Comment

Acute and chronic complications of gastrointestinal PSS are recognised. Chronic complications may present as malabsorption syndrome secondary to bacterial overgrowth of the dilated, atonic bowel[2] or as pseudo-obstruction of the small bowel[3] resulting from dilatation, atony, and delayed transit. Acute complications include fatal peritonitis and haemorrhage[4] and also acute pseudo-obstruction.

Although colonic volvulus associated with PSS has been reported,[5] small-bowel volvulus has not been documented before. Several mechanisms have been suggested for the production of various types of intestinal volvulus. The present case appears to have been due to a combination of factors—namely, suspensory ligament dysfunction resulting from the abnormal collagen of PSS; disordered peristalsis after a heavy meal; and the enlarged intra-abdominal cavity, resulting from diaphragmatic eventration, allowing abnormal movement of the small intestine.

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Genitofemoral neuropathy

Genitofemoral neuropathy appears to be a rare entrapment syndrome, and all cases reported have resulted from local injury, usually appendicectomy. I describe a young woman with an apparently idiopathic entrapment neuropathy of this nerve; her condition might have been aggravated by modelling tight jeans.

Case history

A 20-year-old model first noticed an area of numbness about 5 cm diameter just below the middle of the inguinal ligament in November 1977. Occasionally she had an unpleasant tingling in this region and it was sore to touch. It was aggravated by standing, and when severe her hip ached. Modelling jeans in January 1978 aggravated the symptoms. There had been no recent weight increase. She had been taking oral contraceptives for three years. When she was first seen, in March 1978, she had a constant ache in the left groin, hyperpathia, and numbness.

Examination showed impaired pinprick and cold appreciation with hyperpathia to light touch in the cutaneous distribution of the femoral branch of the left genitofemoral nerve. There was no triple response in this area. A depot preparation of methylprednisolone (1 ml) was injected just lateral to the femoral artery and just below the inguinal ligament. This relieved the hyperpathia within 24 hours, and one month later appreciation of pinprick was only slightly impaired. This residual numbness gradually faded, and by the end of May she was symptom-free. In July, however, the symptoms recurred, with tingling and sensitivity to light touch, and in October these symptoms faded a little to be replaced by numbness. As before, standing for any length of time made it worse. Once again the symptoms responded to local injection of methylprednisolone.

A special technique is used to put on jeans that are several sizes too small. It requires the help of three assistants. The model wears nylon pants which extend from the waist to the knees to overcome friction. Two assistants, one on each side, help to pull on the jeans while the model lies on her back. The third assistant kneels at the head of the model holding a wooden coat-hanger, whose hook is looped into the zip fastener ready to pull as soon as a special device to hold the front of the jeans together has been applied, and provided the material does not tear. Once encased it is impossible to stand up without help or to sit down.

Comment

The genitofemoral nerve arises from L1 and L2. It passes down through psoas and divides into two major branches. The genital branch enters the inguinal canal through the deep inguinal ring and continues to supply the cremaster and upper lateral part of the scrotum or mons pubis and labium majus. The femoral branch accompanies the external iliac artery, passes under the inguinal ligament, and enters the femoral sheath lateral to the femoral artery and supplies the upper part of the femoral triangle.

Only 10 cases of genitofemoral neuropathy have been reported.[1 2] The patients were equally divided between the sexes, and their average age was 27 years (11-65). In all cases identifiable trauma to the nerve was the cause, usually appendicectomy (eight cases). One patient also had a herniorrhaphy, one a psoas abscess, and one young girl fell off her bicycle and injured her groin on the handlebar. The onset of symptoms was immediate in two patients but was usually delayed by months or years. One of these patients had the nerve decompressed, and in seven others the nerve was cut. Operative treatment was successful in all eight.

Pressure from clothing, usually corsets, is a well-recognised cause of damage to the lateral cutaneous nerve of the thigh, producing meralgia paraesthetica, but this nerve is more superficial and more lateral; the genitofemoral nerve is relatively protected in the hollow of the groin. Wearing tight jeans might have been an aggravating factor in this patient, but the onset of symptoms predated the time she was modelling them. There was good response to local steroid injection on two occasions, but this treatment may not be sufficient when the nerve is damaged by operation, and it might be difficult to locate or reach the site of damage. Exploration and nerve section seem to be effective. In another patient seen recently, whose lesion occurred after three lower abdominal operations, including appendicectomy, the symptoms were relieved by an epidural phenol injection at L1.

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Corrections

Camplylobacter colitis

We regret that an error occurred in this article by Dr M E Lambert *et al* (31 March, p 857). On page 858 under "Results" and "Discussion" the word "irritable" should have read "inflammatory."

Serum ferritin concentration and oral iron treatment in patients on regular haemodialysis

We regret that a printing error occurred in this paper by Mr A M Cotterill and others (24 March, p 790). The eighth sentence in the patients, methods, and results section should have read "Only three patients had received intravenous iron at any time and none had received it within a year of starting the study."