CASE REPORTS

Gastric outlet obstruction as a presenting manifestation of systemic lupus erythematosus

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Abstract

A patient with nausea and vomiting who subsequently proved to have systemic lupus erythematosus is described. Although gastrointestinal involvement is common in systemic lupus erythematosus it is rare as an initial manifestation. Gastric outlet obstruction was shown on the air contrast examination while the mucosa at endoscopy was normal. The gastric symptoms regressed after treatment with high dose corticosteroids and a repeat air contrast examination of the stomach was normal. This stricturing process may have been caused by a local peritonitis.

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Systemic lupus erythematosus (SLE) is a multisystem disease of uncertain aetiology. For reporting purposes the American Rheumatism Association has defined criteria.¹ Gastrointestinal symptoms are common in the course of SLE.²⁻⁴ About 50% of patients with SLE suffer from anorexia, nausea, vomiting, diarrhoea or abdominal pain.⁵ Gastrointestinal symptoms are only rarely involved as the initial manifestation of this disease.⁵ We describe a patient with gastric outlet obstruction as

the first and rare manifestation of SLE. Aetiological mechanisms are discussed.

Case report

In September 1991 a 27 year old Dutch woman of Asiatic origin was admitted because of persistent vomiting. The history comprised a tonsillectomy in 1966 and an admission in July 1991 because of dehydration attributed to a gastroenteritis acquired during a vacation in Florida. In that same vacation she had been treated with cotrimoxazol because of dysuria and developed a facial rash, which was attributed to the cotrimoxazol treatment. On admission the patient complained of nausea, vomiting, diarrhoea, and abdominal cramp for three months. During that period her weight had dropped from 46 to 41 kg. A careful history showed some intolerance to sunlight. She had also suffered from pain in her hand joints. She used butylscopolamin for the abdominal cramp.

Physical examination showed a moderately ill woman with a height of 1.60 m and weight of 40 kg. The blood pressure was 110/80 mm Hg, pulse 64 beats/min, and temperature of 36.4°C. The face showed some exanthema. The proximal and distal interphalangeal joints of the second and third finger of the right hand were swollen and painful when flexed. The remainder of the physical examination was unremarkable; later an abdominal examination showed ascites.

Laboratory results were: erythrocyte sedimentation rate 8 mm in first hour; haemoglobin, leucocytes, and thrombocytes were within the normal range. Serum urea, creatinin, albumin, electrolytes, liver function tests, glucose, and amylase were within normal limits. The antinuclear factor was strongly positive, lupus erythematosus cells were positive, class 5 B. Anti-DNA antibodies were negative. The complement profile was: CH50 45 (256-580 u/ml), C₁q 4 (10-14 mg %), C₁q bindings assay 29 (0 mgeq/l), C3 47 (68-104 mg %) C4 26 (17-30 mg %). The urine analysis showed 0.4 g protein/24 h, red blood cells, and casts of leucocytes and erythrocytes. The endogenous creatinin clearance was 72 ml/min. An air contrast examination showed a stricturing lesion in the antrum that did not dilate after glucagon was

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Figure 1: (A) Double contrast phase of the air contrast examination of the stomach: a nasogastric tube is seen in the body of the stomach. There is a short stricturing lesion in the gastric antrum. Slight shouldering is suggested in the proximal part of the lesion. The structure ends at the pylorus. After 0.5 mg glucagon was given intravenously we did not see any change in the stricturing lesion; (B) after three months of treatment a normal gastric antrum is seen. There is a good flow of barium into the small bowel.

Figure 2: (A) Detail of the stricturing lesion in the gastric antrum; (B) detail of the antrum in the double contrast phase of the follow up examination shows a normal distending antrum and an unremarkable mucosa.

given (Fig 1A, 2A). This finding was suggestive of a malignant process.

At gastroscopy there was slight narrowing of the antrum with thickened folds of the mucosa. The mucosa itself had a normal aspect. Multiple gastric biopsies were normal and repeat biopsies did not show evidence of carcinoma or lymphoma. A scintigraphic study showed severely impaired gastric emptying for both solid food and fluids. Ultrasonography of the abdomen showed a thickened wall of the gastric antrum. In addition the small bowel wall was also thickened and a small amount of free fluid was seen in the peritoneal cavity. Hand radiographs showed soft tissue swelling of the second and third digit of the right hand whereas a thorax radiograph was normal. An electrocardiogram showed non-specific ST segment deviations. The diagnosis SLE was made on the presence of at least four criteria: non-erosive arthritis of the fingers, a butterfly like exanthema in the face, light hypersensitivity, proteinuria and casts in the urine, positive antinuclear function and lupus erythematosus cell tests. During the next three months SLE developed fully with pancytopenia and diffuse proliferative glomerulonephritis. The erythrocyte sedimentation rate rose to 117 mm in the first hour; proteinuria increased to 8.9 g/24 h. The patient lost another 5 kg of weight because of persistent vomiting, for which she had to be fed parentally. An insulin hypoglycaemia test

showed a normal pancreatic polypeptide response thereby excluding an impaired vagal cholinergic stimulation of the stomach. Examination of ascites fluid did not show malignant cells but showed 0.5×10^9 leucocytes/l, mainly macrophages but no lupus erythematosus cells. Because there was no support for the diagnosis of malignancy the gastric outlet obstruction was assumed to result from focal SLE related peritonitis, and the patient was eventually treated with 60 mg of prednisone. During this treatment the clinical condition of the patient improved considerably. The parental feeding was stopped and the patient could eat normally again. Because of the good clinical response it was not thought ethical to perform laparotomy or laparoscopy to obtain biopsy specimens of the serosa to confirm the diagnosis histologically. A follow up air contrast radiographic examination of the stomach showed no abnormalities (Fig 1B, 2B).

Discussion

The patient described was admitted because of severe vomiting caused by a gastric outlet obstruction. As there was no evidence of malignancy, the gastric abnormality was attributed to SLE. This diagnosis was supported by the disappearance of symptoms and normalisation of the air contrast examination.

Gastrointestinal symptoms are common in SLE but as a presenting manifestation they are rare.²⁻⁶ Severe gastric outlet obstruction seems to be extremely rare. In patients with SLE who have gastrointestinal symptoms, drugs, uraemia, and other concurrent diseases may be involved. Possible mechanisms related to SLE are serositis of the peritoneum and lupus enteritis.² In necropsy studies signs of peritonitis have been reported in up to 70%.⁷ Gastrointestinal symptoms have also been attributed to lupus enteritis where a small vessel vasculitis has been found in the submucosa.^{2,8} In this last study, however, the diagnosis was based on clinical grounds without pathological proof.

In systemic diseases other than SLE, especially vasculitic syndromes like polyarteritis nodosa, Wegener's granulomatosis, dermatomyositis, and also in Behçet's disease peritonitis can be a feature too. In these diseases peritonitis usually occurs after the gut has been affected by vasculitis of intramural vessels. Isolated peritonitis, however, caused by arteritis of subserosal arteries most commonly occurs in SLE.^{9,10}

Motility dysfunction of the oesophagus, stomach, and small bowel can lead to dysphagia, outlet obstruction, and pseudo-obstruction.^{4,11}

Our patient had a gastric outlet obstruction with diminished gastric emptying. Possible mechanisms involved include vagal neuropathy or localised serositis of the peritoneum. Despite multiple gastric biopsies no evidence of vasculitis was found. The normal insulin hypoglycaemia pancreas polypeptic test excludes vagal neuropathy.¹² It is possible that

she suffered from a focal serositis of the gastric peritoneum restricting gastric dilatation. To our knowledge only one case of localised serositis of the stomach in a patient with SLE has been described before.¹³ The diagnosis was made at laparotomy. In this patient, however, there were no signs of gastric outlet obstruction. Other arguments in support of focal SLE related serositis as the cause of the gastric outlet obstruction, were the thickened wall of the stomach, the presence of ascites, and the clinical and roentgenological response to steroid treatment.

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