for a patient with renal failure. Lowered blood viscosity, which usually accompanies the anaemia of chronic renal failure, may prevent sluggish mesenteric blood flow in other patients undergoing dialysis who are subjected to similar haemodynamic stresses.

Digitalis should be considered as a possible cause when evaluating obscure abdominal pain in patients undergoing haemodialysis, who may benefit—and be rendered free of pain—when digitalis is withheld.

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Arthritis preceding fulminant ulcerative colitis and responding to colectomy

Although it has long been recognised that ulcerative colitis may be complicated by bouts of arthritis,^{1,2} the clinical course of "colitic arthritis" has only recently been characterised.³ Large joints are usually affected in a flitting and asymmetrical way, most commonly in chronic ulcerative colitis, and in a series of 269 patients never preceded the onset of bowel symptoms. Colectomy invariably results in prolonged remission of arthritic symptoms.⁴ We describe a patient with colitic arthritis whose joint symptoms preceded bowel symptoms by five years and in whom the onset of colitis was acute and fulminant.

Case report

A 19-year-old woman presented in December 1978 with a four-year history of recurrent pain in her shoulders, elbows, wrists, and knees. Her general health was otherwise good with no bowel symptoms. There was tenderness over the ulnar heads and discomfort on moving the shoulders and knees but no other abnormality. Radiographs were normal. Results of routine haematological studies and tests for antinuclear factor were normal. The presumptive diagnosis was seronegative rheumatoid disease. She was given a six months' course of chloroquine phosphate and naproxen and remained fairly well over the next 15 months with only one exacerbation of soreness and stiffness of the right shoulder, which responded to a local hydrocortisone injection.

In January 1980 she became generally unwell with lethargy, weakness, and an exacerbation of pain and stiffness in her large joints. She developed bowel symptoms for the first time with lower abdominal pain and loose, watery bowel motions up to eight times daily, streaked with mucus and blood. Several stool cultures were sterile, and symptomatic treatment had little effect. On admission to hospital she was pale and dehydrated with a temperature of 38.5°C, though her pulse and blood pressure were stable. There was no obvious swelling or inflammation around her large joints, but they were painful and stiff. She had generalised abdominal tenderness, particularly in the left iliac fossa, and bowel sounds were pronounced. Abdominal radiography showed a grossly dilated colon. Findings of routine haematological and serological studies were again normal. Her HLA type was subsequently reported as A2, B17, BW44, BW4 but not B27. Radiographs of her sacroiliac joints were normal.

Conservative treatment with intravenous fluids and high-dose corticosteroids produced a temporary initial response. Five days after admission,

however, she developed severe abdominal pain with guarding and rebound tenderness, and bowel sounds disappeared. Abdominal radiography confirmed perforation of the bowel. At operation the whole colon was distended, inflamed, and oedematous and there were perforations in the caecum and sigmoid colon. She underwent total colectomy and terminal ileostomy. Since the rectum had looked relatively normal on sigmoidoscopy it was conserved. Histological examination showed extensive ulceration with focal crypt abscess formation, the appearances being typical of severe ulcerative colitis.

The patient made an excellent recovery, and five months later remained entirely free of joint symptoms without anti-inflammatory drugs.

Comment

The pattern of joint disease, exacerbation of symptoms with the onset of colitis, and dramatic response to colectomy confirm that this patient had colitic arthritis.³ The unique features, however, were the long history of joint symptoms preceding the onset of colitis and the acute fulminant mode of onset of colitis. Significant colitis was probably not present during the five years when no bowel symptoms existed, but perhaps the relatively simple investigation of sigmoidoscopy with rectal biopsy should be considered in recurrent undiagnosed seronegative arthropathy. It might then be possible to offer specific treatment for colitis with resultant control of joint symptoms and to hope to prevent severe colitis.

Requests for reprints should be sent to Dr David M Fraser.

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Successful pregnancy in severe chronic renal failure not requiring dialysis

Chronic renal failure in women is usually associated with impaired fertility¹ and a high incidence of miscarriage.^{2,3} In women with creatinine clearance below 10 ml/min successful pregnancy not requiring dialysis has not been recorded. We report such a case.

Case report

A 31-year-old Asian housewife had three full-term normal deliveries in 1968, 1969, and 1974, during each of which her albuminuria was + + +. During the third pregnancy creatinine clearance was 133 ml/min. She was normotensive during all three pregnancies. In June 1979, however, she presented to her GP with headache, and her blood pressure was 220/120 mm Hg. Oxprenolol 320 mg daily reduced the blood pressure to 160/85 mm Hg by October. In November 1979 (at 17 weeks of pregnancy) her blood pressure was 170/100 mm Hg. Her haemoglobin concentration was 7.6 g/dl, blood urea and creatinine concentrations 27 mmol/l (162.7 mg/100 ml) and 500 µmol/l (5.7 mg/100 ml) respectively, proteinuria 3 g/day, and creatinine clearance 7.6 ml/min. She was admitted to hospital and received a 40 g protein "no added salt" diet, transfused with 2 units of blood, and prescribed only methyldopa 250 mg twice daily. Blood urea and creatinine concentrations rose slowly, though blood pressure and fetal growth were initially satisfactory.

On 12 February 1980 she was transferred to the Manchester Royal

Infirmery and St Mary's Hospital, Manchester, for consideration for haemodialysis. Her haemoglobin concentration was 8.8 g/dl, blood urea concentration 30 mmol/l (180.7 mg/100 ml), blood creatinine concentration 740 μ mol/l (8.4 mg/100 ml), creatinine clearance 5 ml/min, serum albumin concentration 27 g/l; proteinuria 3 g/day, plasma calcium concentration 1.5 mmol/l (6 mg/100 ml), plasma phosphate concentration 1.8 mmol/l (5.6 mg/100 ml), serum alkaline phosphatase activity 65 IU/l, and plasma urate concentration 0.6 mmol/l (10.1 mg/100 ml). Her blood pressure was 150/100 mm Hg, rising to 170/110 mm Hg. She was treated with methyldopa 1.5 g/day and a diet containing 50 g protein, 50 mmol (mEq) sodium, and 10.5 MJ (2500 kcal). Results of ultrasound scanning and radiography confirmed fetal gestation of 28 weeks. Urinary oestriol excretions were 15 and 24 μ mol/day (4.3 and 6.9 mg/day; normal value over 40 μ mol/day (11.5 mg/day)).

One week later the blood urea concentration was 33 mmol/l (198.8 mg/100 ml), creatinine concentration 800 μ mol/l (9.0 mg/100 ml), and creatinine clearance 3 ml/min. She entered spontaneous labour and delivered a 1600 g boy. The placenta (350 g) was macroscopically normal. The infant suffered respiratory distress syndrome, complicated by a pneumothorax requiring ventilation. His blood urea concentration on delivery was 29.4 mmol/l (177.1 mg/100 ml), falling to normal in three days. One month later he was discharged well and weighing 2000 g.

After delivery the patient's blood pressure rose, requiring methyldopa 500 mg three times daily, hydralazine 25 mg three times daily, and atenolol 100 mg daily. When she was discharged three weeks after delivery her creatinine clearance was only 3 ml/min. Renal function continued to deteriorate, and she was awaiting dialysis. Intravenous pyelography seven weeks after delivery showed small smooth kidneys. Investigation detected no underlying systemic disorder. Renal biopsy was not performed.

Comment

The reasons for poor fetal survival during pregnancy complicated by chronic renal failure are not understood. When serum urea concentration exceeds 7.5 mmol/l (45.2 mg/100 ml) and serum

creatinine concentration 275 μ mol/l (3.1 mg/100 ml), normal gestation is rare and conception not recommended, but these concentrations are symptomless and not troublesome in non-pregnant women.¹ Reciprocal creatinine concentrations showed a linear decline before and after birth (figure), though we do not know the rate of decline before pregnancy. Strauch and Hayslett suggested that pregnancy does not alter the course of the underlying renal disease.⁴

The ease of control of blood pressure was probably important in determining our patient's successful outcome. Felding⁵ showed that in mild renal failure poorly controlled maternal blood pressure increased prenatal mortality from 8% to 31% and prematurity from 12% to 37%. The degree of parity was also important. Mackay concluded that adaptation to pregnancy was less well tolerated in the first than in subsequent pregnancies.² Our patient received transfusions three times during pregnancy and also immediately after delivery (figure). We decided to keep haemoglobin concentrations above 8 g/dl, which was a compromise to meet fetal requirements without reducing maternal renal function.

Pregnancy in patients receiving haemodialysis or with renal transplants is now acceptable. In chronic, severe renal failure not requiring dialysis, however, there is insufficient knowledge and experience. The fear of causing deterioration in maternal condition with no guarantee of a viable infant results in extreme caution when advising on conception and easy acceptance of therapeutic abortion.

This case indicates that with joint care of an obstetrician and nephrologist successful pregnancy is possible when the creatinine clearance is below 10 ml/min on booking, provided that the patient is a multigravida and does not have severe hypertension.

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Haemoglobin concentrations, creatinine clearances, serum urea concentrations, and reciprocal of serum creatinine concentrations in patient with severe chronic renal failure before and after delivery.

Conversion: SI to traditional units—Serum urea: 1 mmol/l $\approx$ 6 mg/100 ml. Serum creatinine: 1 μ mol/l $\approx$ 0.01 mg/100 ml.

Ionised calcium in rheumatoid arthritis: effect of non-steroidal anti-inflammatory drugs

Many patients with rheumatoid arthritis have disordered calcium homeostasis, and some workers have suggested that a hyperparathyroid-like state may exist in rheumatoid arthritis.^{1,2} After correction for low albumin concentrations, hypercalcaemia was present in up to 46% of patients,² although parathyroid hormone concentrations were not raised.¹ Hypocalcaemia may also occur in rheumatoid patients with advanced bone disease, even allowing for low serum albumin concentrations.³ Diseases associated with deranged calcium metabolism are best assessed by measuring the physiologically important ionised fraction of serum calcium because extrapolating from "corrected" calcium values is unreliable.⁴ We decided to re-examine calcium balance in rheumatoid arthritis by measuring serum ionised calcium concentrations. The effects of non-steroidal anti-inflammatory drugs were also studied.

Patients, methods, and results

Serum ionised calcium, total serum calcium, total protein, albumin and phosphate concentrations and alkaline phosphatase activity were estimated in