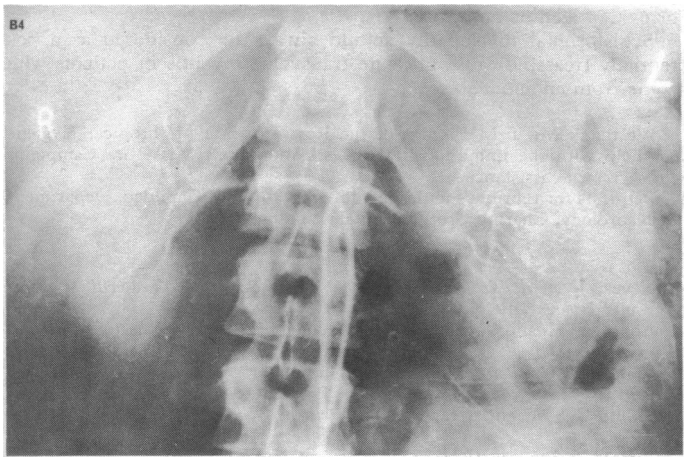
SHORT REPORTS

Postpartum acute renal failure: possible role for endarterial urokinase

Late postpartum acute renal failure is a rare complication of the puerperium, which, together with its insidious onset, probably explains why renal damage is generally irreversible by the time of diagnosis. It is important to note that the fully developed syndrome is usually preceded by definite, albeit non-specific, prodromal symptoms.

Case report

A 26-year-old gravida 5 (group A, rr; HLA 1, 2, and 8, W39) appeared to be well after an uncomplicated pregnancy. Two weeks post partum, however, she developed headaches and mild ankle oedema, and four weeks later she became anuric. Plasma urea was 48 mmol/l (289 mg/100 ml), haemoglobin was 7·1 g/dl, fragmented red cells were seen, platelets were 160×10^9/l (160 000/mm^3), and plasma fibrinogen was 7·5 g/l. Renal biopsy showed fibrin deposition in the afferent arterioles and interlobular arteries but no evidence of complement or immunoglobulin deposition. Bilateral renal arteriography showed symmetrical poor blood flow to the kidneys with no filling of the terminal branches. The catheters were left in position and 50 000 U urokinase was infused in bolus doses every eight hours to the non-biopsied kidney. A continuous infusion of heparin 50 U/hour was maintained to each side. Repeat angiography showed an increase in blood flow on the treated side only (figure). The catheters were removed on the third day. Only small volumes of urine were passed (300-400 ml/day), and peritoneal dialysis was begun. The plasma fibrinogen concentration remained raised (9·6 g/l), and fibrinolysis, as measured by the euglobulin lysis time, was reduced (patient 6 h; control 2 h). An induction dose of ancrod 1 U/kg body weight, was given by infusion over 12 hours, and the plasma fibrinogen concentration fell to 7·5 g/l. The same dose repeated as a bolus reduced the concentration to 0·85 g/l. Ancrod was used at intervals of three to seven days but the fibrinogen concentration recovered at a rate of $0{\cdot}1 \pm 0{\cdot}02$ g/l daily after each dose. After four weeks the plasma fibrinogen stabilised at 5·8 g/l. Fibrinogen turnover studies then showed a pool of 15·5 g (normal 4-7 g) and a half life of 2·3 days (normal > 3 days). When last seen the patient was managing well on intermittent haemodialysis.



Bilateral renal arteriogram after two days of urokinase treatment.

Comment

The pathogenesis of this syndrome is unknown, though the clinical picture and renal histology have been well described.[1] In this patient the very high plasma fibrinogen concentration was a persistent feature of the illness and was due at least in part to a rapid rate of synthesis. Fibrinolytic activity was reduced. Both the high rate of fibrinogen synthesis and decreased fibrinolysis would be reflected in enhanced fibrin deposition, but whether the raised plasma fibrinogen concentration was itself a contributing factor in the condition we cannot say. None the less, fibrin deposition in the renal microvasculature is the most important single aspect of the disease and the one to which attention should be directed initially. In our patient renal endarterial perfusion with urokinase to one kidney enabled comparison to be made with the effect of continuous low-dose heparin infusion to the other. Though this treatment had no effect in allowing recovery of renal function, the urokinase clearly produced an arteriographic improvement, while heparin did not. It has been suggested that anticoagulation aids recovery in some cases, but reports show that improvement is equally rare whether heparin is used or not.[1] Simple anticoagulation, though theoretically attractive, must therefore be regarded as inadequate in postpartum renal failure. Endarterial urokinase, however, might be of benefit provided the condition is recognised before renal damage becomes irreversible. Ancrod might be of value, but how low a concentration of fibrinogen would have to be achieved to alter fibrin deposition sufficiently is not known.

For treatment to be successful early diagnosis is necessary. Thus in the puerperal patient with apparently minor complaints of fluid retention, hypertension, and proteinuria appropriate biochemical and haematological investigations are indicated, and if the plasma urea concentration rises appreciably renal biopsy should be considered.

[1] Schoolwerth, A C, *et al*, *Archives of Internal Medicine*, 1976, **136**, 178.

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Royal Hospital, Sheffield S1 3SR
M COCHRAN, MD, MRCP, senior registrar, renal unit
J F MARTIN, MRCP, MRC fellow, department of medicine
B ROSS, MD, FFR, consultant radiologist

Gastric emptying in coeliac disease

Abnormally high concentrations of propranolol have been recorded in the plasma after oral administration of the drug to patients with treated coeliac disease.[1 2] Among explanations proposed for this apparently contradictory phenomenon has been the possibility of accelerated gastric emptying. A preliminary study carried out on a small group of patients with a tube dye-dilution technique suggested that liquids do, in fact, leave the stomach more rapidly in coeliac disease than normal.[3] We decided to investigate solid-phase emptying simultaneously with liquid-phase emptying in a group of patients with coeliac disease at various stages of treatment.

Patients, methods, and results

Ten patients with coeliac disease were compared with 10 normal controls. Four of the patients had been only recently diagnosed and were still on a normal diet, and six were on gluten-free diet. The fasting subject ingested a test meal of energy density 7·7 MJ/l (1·86 kcal/ml) at zero time and contain-

Mean gastric emptying times in minutes (ranges given in parentheses)

		Controls	Patients with coeliac disease	P*
Liquid phase (10 subjects in each group)	$t_{\frac{1}{4}}$	18·0 (7-31)	32·0 (11-85)	<0·05
	$t_{\frac{1}{2}}$	53·9 (20-77)	59·7 (27-143)	NS
Solid phase (5 subjects in each group)	$t_{\frac{1}{4}}$	64·5 (30-77)	67·6 (26-98)	<0·05
	$t_{\frac{1}{2}}$	74·0 (84-123)	73·6 (50-122)	NS

*Calculated by Wilcoxon's sum of ranks method.
NS = Not significant.