in liver transaminase or alkaline phosphatase activity. Haemoglobin concentration fell from 14·2 g/dl to 12·8 g/dl with a rise in reticulocytes to 3%. Intravascular haemolysis was confirmed by a positive methaemalbumin test, a raised plasma haemoglobin concentration at 0·26 g/l (normal range 0·01-0·04 g/l), and a reduced haptoglobin concentration at 0·028 g/l (normal range 0·3-2·0 g/l). The platelet count had fallen to 33×10^9/l, but a clotting screen was normal. Coombs test was negative.

A diagnosis of acute intravascular haemolysis with associated renal failure was made. Despite alkalinisation of his urine he remained anuric and the blood urea concentration rose to 48·3 mmol/l (290 mg/100 ml). He was transferred to the renal unit, where he required haemodialysis on six occasions over the next 10 days before entering a diuretic phase. Values of plasma haemoglobin, haptoglobins, reticulocytes, platelets, bilirubin, and urea returned to normal and remained so after termination of dialysis. Three months after discharge serum haptoglobin values, Schumm's test, Ham's test, haemoglobin electrophoresis, Coombs test, and red-cell enzyme activities were normal. There was evidence, however, of an increase in osmotic fragility (lysis beginning at 0·75% NaCl solution, control 0·55%).

Comment

Acute renal failure is a well-recognised complication of radiocontrast investigations but the mechanism is usually unknown.[2] Diabetes mellitus and pre-existing renal insufficiency appear to be predisposing factors but these were not present in our patient.

Haemolysis and haemoglobinuria after contrast angiography has been documented in six children with cyanotic congenital heart disease and secondary polycythaemia.[3 4] Martin[5] reported a case of acute renal failure with low serum haptoglobin values and methaemoglobinuria after myelography with sodium diatrizoate. It has been postulated[3] that rapid injection of hypertonic contrast medium causes initial loss of water and uptake of solute in the red blood cells. When isotonicity of the plasma is restored these erythrocytes may absorb excess water, swell, and be more easily haemolysed. That our patient had abnormal osmotic fragility three months after discharge suggests that he may have been susceptible to sudden increases in plasma tonicity. Intravascular haemolysis is thus a potentially serious, though fortunately rare, complication of contrast media investigations.

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(*Accepted 1 December 1980*)

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Possible transmission of malaria by renal transplantation

Malaria may present as a postoperative fever. This may be due either to infection by transfusion[1] or, in patients from endemic areas, to recrudescence of latent infection provoked by the surgery.[2] I report a case of malaria in a recipient of a renal transplant in which the infection may have been acquired from the donor kidney.

Case report

A 27-year-old Saudi man was admitted to this hospital on 17 December 1979 for living-donor renal transplantation. He had chronic renal failure of uncertain aetiology and had been on regular haemodialysis in Saudi Arabia for one year. Examination showed no other abnormality apart from a late systolic murmur. His brother, the donor, was 25 years old and in good health. Neither gave a definite history of malaria but both came from Jizan, a known malarial area of Saudi Arabia, and the donor was resident there. The donor was found to have mild splenomegaly for which no cause was found despite extensive investigations, including examination of blood films for malaria parasites.

On 29 December 1979 renal transplantation was performed and standard treatment with prednisolone and azathioprine begun. Good renal function was established immediately, with serum creatinine concentration falling to normal within three days. The postoperative course was uneventful apart from a dehiscence of the abdominal wound. Low-grade fever was noted, but no cause was found and the patient was discharged well on 21 January 1980.

On 6 February he was readmitted with aching in limbs and chest, occasional rigors, and a daily fever up to 38·5°C. Serum creatinine and urea concentrations rose and he was thought to be having an episode of rejection. Accordingly intravenous methylprednisolone was given, with subsequent return to normal of creatinine and urea values but without effect on his fever. On 22 February very occasional malaria parasites (*Plasmodium falciparum*) were seen in the blood film. Chloroquine was given and resulted in complete resolution of his illness.

Retrospective examination of a blood film from the donor taken the day before transplantation disclosed very scanty ring forms of *P falciparum*. No parasites were seen in the recipient's blood film on three occasions before the operation, though thick films were not available. The malaria indirect fluorescent antibody test was performed on available sera, and the table gives the results. This test is the most specific method of detecting malaria antibodies. Two antigens were used—*P falciparum* and *P fieldi*, which is a simian plasmodium found to be antigenically closely related to *P vivax*, though there is cross-reactivity between all human and some simian plasmodia.[3] High titres (⩾1/200) suggest recent infection in non-immune patients but may be associated with latent subclinical infection in those from hyperendemic areas.

Results of indirect fluorescent antibody test on sera from transplant recipient and donor

	Date	Titre to *P falciparum*	Titre to *P fieldi*
Recipient	14 June 1979	4096	256
	17 December 1979	4096	1024
	25 February 1980	4096	1024
Donor	29 December 1980	16304	4096

Comment

One previous case of malaria occurring in a renal transplant recipient has been reported,[4] though several cases have been described after cardiac surgery,[5] which were possibly due to recrudescence of a latent infection provoked by the stress of the operation. In this case the infection might also have been due to recrudescence provoked by surgery, and the high initial indirect fluorescent antibody test titres in both patients suggest that latent subclinical infection was present in both. The infection may, however, have been acquired from the donor in view of his very high titres before the operation, and, though a new infection usually causes a rise in titre against the homologous antigen, and possibly against the heterologous antigen also, this may have been prevented by immunosuppression.

Whatever the source of infection, the important conclusion is that malaria must be added to the long list of causes of fever in renal transplant recipients and donors from malarial areas. Moreover, malaria is endemic in most of the coastal regions of Saudi Arabia, a country usually thought of as a non-malarial desert area. Both *P vivax* and *P falciparum* are found, but infections due to *P vivax* occur much more commonly.

I thank Dr C C Draper, of the London School of Hygiene and Tropical Medicine, for performing the indirect fluorescent antibody test, and Drs N F Jones and P J Hilton for permission to report this case.

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(*Accepted 9 December 1980*)

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