by tests set up in adjacent wells, to give a reaction of partial identity with the reference antiserum *S. mansoni* and the reference *S. mansoni* antigen. Normal sera did not react when diffused against reference antiserum *S. mansoni* and reference *S. mansoni* antigen.

Discussion

As expected, many of the sera from the patients with schistosomiasis contained antibody to *S. mansoni* adult worm extracts. Not every serum contained antibody, a finding discussed by Kagan (1968). Antibody was more commonly detected in the *S. mansoni* patients, but this may have been because we used the homologous *S. mansoni* antigen for the tests. Though this antigen cross-reacts with *S. haematobium* antibody it may be less effective in this heterologous system.

The novel finding was the demonstration of circulating soluble schistosomal antigen in the patients' sera, particularly sera from the patients infected with *S. mansoni*. This is not too surprising when one considers that the adult worms are located in the blood stream. Interestingly, antibody to the circulating antigen was also demonstrable. Under the conditions which we employed, antigen bound up in immune complexes would not be demonstrable. Only when antigen was present (a) in the absence of its reciprocal antibody—for example, early in infection—or (b) in excess simultaneously with immune complexes would it be shown by our gel-diffusion tests. Of course, under conditions of antigen excess immune complexes are most likely to be formed and then deposited in the glomeruli, leading to the renal complications reviewed above. The formation of such immune complexes in schistosomiasis may be a common transient event followed by spontaneous resolution in most cases. Probably only a

minority go on to develop the chronic immune complex disease, which may then include an autoimmune element, possibly with antibodies to the original immune complex or altered host tissue. This would account for the difficulty in demonstrating schistosomal antigen in the kidneys of the patients with developed nephrotic syndrome.

In addition to their importance in immunopathology the presence of circulating antigens and immune complexes might be expected to interfere with serological tests designed to measure antibody to schistosomes. This may be the reason for the fairly common false serological negatives especially encountered in young patients with schistosomiasis.

We think that this work deserves to be extended to include longitudinal observations on patients with the disease before and after treatment. Longitudinal studies on induced infections in animals could also answer some of the questions about the origin, time course, and fate of the soluble circulating antigens of schistosomes.

We are grateful to Professor A. W. Woodruff for his constant encouragement and advice, and to Dr. D. Bidwell, who supervised the gel-diffusion tests. The reference antiserum to adult *S. mansoni* worms was kindly donated by Dr. John Kusel.

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MEDICAL MEMORANDA

Post-transfusion Purpura Associated with Unusual Platelet Antibody (Anti-PL^{BI})

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British Medical Journal, 1975, 1, 436-437

Post-transfusion purpura is a rare cause of thrombocytopenia occurring in women about a week after blood transfusion. Almost all patients with this disorder have had pregnancies or have had blood transfusions some years earlier (Shulman *et al.*, 1961; Morrison and Mollison, 1966; Cimo and Aster, 1972).

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The condition is usually self-limiting though one death from intracranial haemorrhage has been reported (Gockerman and Shulman, 1973). The aetiology is uncertain but all patients tested have had circulating antibodies to a transfused platelet antigen absent from their own platelets.

We report a case of post-transfusion purpura which differed from other cases in that the platelet antigen was PL^{BI} and there was a rapid improvement in the platelet count and clinical state after treatment with massive doses of corticosteroids.

Case Report

A woman aged 79 was admitted to hospital on 7 October 1973 for cholecystectomy. Preoperatively her haemoglobin was 14.1 g/100 ml, W.B.C. 7800/mm³ and M.C.V. 90 fl. Platelets were present in normal numbers on a blood film and liver function, urea, and electrolytes were normal.

On 10 October cholecystectomy with exploration of the common bile duct was carried out; a gall stone obstructing the duct was removed and a T-tube drain inserted. During the operation the patient was transfused with 2 units of group A, Rh-positive blood though the blood loss was not excessive and haemostasis was normal. On the first postoperative day the haemoglobin was 15.7 g/100 ml, the W.B.C. 14 800/mm³, and the platelets appeared normal on the blood film. She received two intramuscular injections of pentazocine 60 mg and one of metoclopramide 10 mg postoperatively.

On 17 October generalized purpura and bleeding from the operation wound and T-tube developed. The blood pressure became

unrecordable, the haemoglobin fell to 8.3 g/100 ml, and the platelet count fell to 3000/mm³. W.B.C. was 700/mm³, prothrombin time was 12 seconds (control 12), partial thromboplastin time with kaolin was 36 seconds (control 35), fibrinogen titre was 1/128, and fibrin degradation products were 40 µg/ml. A provisional diagnosis of Gram-negative septicaemia with disseminated intravascular coagulation was made and the patient was treated with 4 units of whole blood and 2 platelet concentrates (from 10 donors). Injections of 10 000 units of heparin and 200 mg of hydrocortisone were given intravenously together with ampicillin 1 g and gentamicin 80 mg intramuscularly. Cultures of blood and wounds swabs were subsequently sterile. Later that evening, when post-transfusion purpura was diagnosed, the heparin effect was reversed with protamine sulphate and transfusion was continued with platelet-poor, dextran-sedimented blood. The corticosteroid preparation was changed to intravenous methylprednisolone sodium succinate 500 mg six-hourly and after three days to oral prednisolone 60 mg daily, reducing to 2.5 mg daily over the next three weeks. Ampicillin was discontinued after nine days and gentamicin after 30 hours. In 24 hours clinical improvement was apparent, associated with an increase in the platelet count and haemoglobin (see fig.). Sternal marrow aspirate showed normal appearances with normal numbers of megakaryocytes, but most of these showed absent platelet budding. The patient was discharged on the 39th postoperative day. Haemoglobin and platelet values remained normal.

Platelet Studies.—Platelets harvested from whole-blood samples taken from the patient (in remission), her husband, and the donors of the two units of blood transfused during the operation were phenotyped by microcomplement fixation using 30 known antisera covering 15 specificities. The phenotype of the patient and her husband was also determined by a microlymphocytotoxic technique (see table). Four samples of the patient's serum (one pre-operative, 8 October 1973, and three postoperative, 17 and 18 October 1973 and 3 January 1974) were tested in parallel by micro-complement fixation at 37°C against a panel of 18 typed platelet samples; all contained antibody with anti-PIB¹ (HL-A 2) speci-

Results of Phenotyping Platelets from the Patient, Her Husband, and Two Blood Donors

Patient	HL-A Phenotype	Donor J	HL-A Phenotype
..	.. 1, 3, 8, W22	..	.. 2, 3, 5
Husband	.. 2, 12, W18, W25	Donor C	.. 1, 5, 12

city. In addition the patient's sera reacted strongly against her husband's platelets and those from donor J.

Comment

This case represents a further example of the rare syndrome of post-transfusion purpura and is the third to be recorded in Britain (Morrison and Mollison, 1966; Nicholls *et al.*, 1970). This is also the first report of the disorder associated with anti-PIB¹ (HL-A 2). Since there was no previous history of blood transfusion and platelet antibody was present in the pre-transfusion sample sensitization probably occurred during one or more of the patient's pregnancies. This is supported by the presence of the PIB¹ antigen on the husband's platelets.

The thrombocytopenia has been explained on the basis of destruction of the patient's platelets as "innocent bystanders" by complexes formed from the interaction of circulating antibody with transfused platelet antigen. The presence of immature megakaryocytes and failure of platelet budding seen in the bone marrow of our case and others, however, suggests that suppression of megakaryocyte maturation may also be of significance.

Corticosteroids in conventional doses have been used in a number of cases but there is no evidence that this treatment influenced the course of the disease since several weeks elapsed before the platelet counts returned to normal. Our patient, treated with massive doses (2 g daily) of intravenous methylprednisolone sodium succinate for the first three days, improved rapidly and the platelet count was normal by five days. Similar therapeutic results were obtained in two cases treated by exchange transfusion (Shulman *et al.*, 1961; Cimo and Aster, 1972). We therefore consider short courses of high-dose corticosteroids worthy of further trial in the treatment of post-transfusion purpura.

This rare syndrome should be borne in mind when any patient develops thrombocytopenia after blood or plasma transfusion and must be remembered as another potential hazard of these frequently used materials.

We thank Mr. J. A. Fox, consultant surgeon, for permission to report this case.

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