Platelet response.
out haemorrhagic complications and the patient was well when last seen in September 1989.

The figure shows his platelet response. The platelet count rose from $53 \times 10^9/l$ to $153 \times 10^9/l (289\%)$ by day 4, to a maximum of $416 \times 10^9/l (798\%)$ on day 14, and when last seen was $142 \times 10^9/l$ on day 320.

Fresh frozen plasma has been used before in the treatment of adult immune thrombocytopenia. On that occasion, however, it was used concurrently with other immunosuppressive agents such as vincristine, prednisolone, and azathioprine. Although a possible synergistic action between fresh frozen plasma and other agents can be postulated from that study, it is difficult to draw conclusions as to the likely role or mechanism of action of fresh frozen plasma in the treatment of immune thrombocytopenia. The use of compatible plasma ensures that the observed effect was not due to macrophage Fc receptor blockade by sensitised red cells. Studies in which technetium-labelled anti-D sensitised red cell removal by reticuloendothelial system macrophages was decreased after administration of intravenous immunoglobulin suggest that the immunoglobulin can induce widespread macrophage Fc receptor blockade. Other evidence suggests that this effect is dose dependent. The comparatively small amount of immunoglobulin present in fresh frozen plasma (10 mg/ml—total dose infused 14 g), however, suggests that if indeed the observed effect is immunoglobulin dependent, then factors more related to the nature of the Ig component infused are likely to be responsible for it.

Infusion of IgG in immune thrombocytopenia modulates the immune system as early as three days after infusion, and there is circumstantial evidence of a rapid fall in a platelet associated Ig after intravenous immunoglobulin. We feel that the most likely explanation is that there is a specific modulatory effect of the immune system from infused immunoglobulin, resulting in decreased anti-platelet antibody production.

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Leukaemic phase of mantle zone lymphoma
Mantle zone lymphoma, also called intermediate lymphocytic lymphoma, is a histologically distinctive form of non-Hodgkin's lymphoma with morphological features ranging from those of small lymphocytic lymphoma to small cleaved cell lymphoma. Leukaemic manifestation of this lymphoma seems fairly uncommon and its detailed morphological and phenotypic characteristics have only recently been described.

A 60 year old man was admitted into hospital in May 1987 because of a two month history of anorexia, weight loss, and fever. Physical examination showed enlargement of cervical, axillary, and inguinal lymph nodes. Both the liver and the spleen were also enlarged. Laboratory data were as follows: leucocyte count was $9.7 \times 10^9/l$; the differential was 0-29 segmented neutrophils, 0-67 lymphocytes, 0-02 monocytes, and 0-01 eosinophils. Lymphoid cells were heterogeneous, with respect to size and morphology. The lymphocytes were medium to large in size, with scanty to moderate cytoplasm, and a nucleus with an irregular outline some of which bore pronounced indentations. The chromatin was moderately coarse with nucleoli in some cells. Haemoglobin concentration was 115 g/l; platelet count was 156 x $10^9/l$. The erythrocyte sedimentation rate was 16 mm in the first hour. Gamma globulin was 5-48 g/l, and lactic dehydrogenase was 634 U/l. Liver function tests were normal. A chest x-ray film was reported as normal.

A left axillary lymph node biopsy was carried out. Histological examination showed diffuse infiltration by well differentiated lymphocytic cells, together with atypical lymphocytes resembling small cleaved cells. Germinal centres were not observed. Intermediate lymphoma was diagnosed. A bone marrow biopsy specimen disclosed nodular and interstitial infiltration by medium sized lymphocytes. An ultrasound scan showed multiple, enlarged, abnormal lymph nodes. Cyclophosphamide was started with poor results. Radiotherapy (20 Gy) to the spleen was carried out, resulting in pronounced improvement of abdominal symptoms. In the last follow up carried out in January 1990 enlarged lymph nodes were present and the spleen was palpable 10 cm below the costal margin. Laboratory data were as follows: leucocyte count $28 \times 10^9/l$; the white cell differential was 0-08, segmented neutrophils, 0-05 monocytes, 0-01 eosinophils, 0-86 atypical lymphocytes, haemoglobin concentration...
tration was 87 g/l, platelet count was 123 × 10^9/l and lactic dehydrogenase was 939 U/l. Surface membrane markers were identified by indirect immunofluorescence and examined with a FacScan Flow cytometer (Becton Dickinson, Mountain View, California, USA). The following antibodies were analysed: T4, T3, T8, J5, B4, B1 (Coulter Clone); LeuM5, Leu15, Leu14, IL-2, Leu17 and Leu8 (Becton Dickinson); anti-LFA 1 and anti-βLFA 1 B (Janssen); Cris 1 (Dr R Villa), Hospital Clinic Provincial, Barcelona; FMC7 (Sera-Lab); 1B08 (Immunotech); and surface immunoglobulins (Kallestad). Mouse rosettes were also sought. Detailed results are shown in the table.

B cell chronic lymphoid leukaemias comprise a broad spectrum of lymphoid proliferations classified according to the cytological and phenotypic features of the leukaemic cells. Our case was a mantle zone lymphoma in leukemic phase, which is a rare form of B cell chronic lymphoid leukaemia (B-CLL). Cytological and immunologic features of the lymphoma had pronounced heterogeneity of size and a fairly pleomorphic appearance. The surface marker analysis of the leukaemic cells (table) showed a monoclonal B cell proliferation that was primarily CD5 in the early chronic phase. Surface immunoglobulin was strong, FMC7 was positive, and there was no formation of mouse rosettes. All these features differ from typical B-CLL leukaemia but resemble the surface phenotype of prolymphocytic leukaemia and that of follicular lymphoma in leukemic phase. Overall, it seems that the characteristic phenotypic profile of mantle zone lymphoma in the leukemic phase reflects strong surface immunoglobulin and positivity for FMC7 and CD5. Reactivity with CD10 and mouse rosette formation is variable. Data on the antibodies Leu8, CD11, CD22, CD23, CD25 and CD38 are scarce. Further studies are needed to clarify precisely the phenotype of this particular lymphoid leukaemia.

Hypercalcaemia and osteolytic lesions associated with chronic lymphatic leukaemia (CLL)

Case 1
A 72 year old man had cervical and axillary lymphadenopathy and an enlarged spleen palpable 1 cm below the left costal margin. A blood count showed that his haemoglobin concentration was 11.5 g/dl (normal range: 12.5-16.0 g/dl), his white cell count was 1.14 × 10^9/l (normal range 4.0-10.0 × 10^9/l), his lymphocytes were 105 × 10^9/l and his platelet count 250 × 10^9/l (normal range 150-400 × 10^9/l). A biochemical screen, including that for serum calcium concentration, was normal. A bone marrow aspirate and trephine biopsy specimen showed diffuse infiltration with small mature lymphocytes, and chronic lymphatic leukaemia (CLL) was diagnosed. The disease was easily controllable by short, intermittent courses of chlorambucil.

Three years from diagnosis and while not receiving treatment, the patient was admitted with a two week history of thirst, malaise, and vomiting. Examination showed that he was dehydrated, had enlarged cervical lymph nodes, an enlarged liver palpable 3 cm below the right costal margin and an enlarged spleen palpable 4 cm below the left costal margin. The haemoglobin concentration was 9.1 g/dl, the white cell count 14.8 (small mature lymphocytes 9.1 × 10^9/l, pro-lymphocytes 3.9 × 10^9/l), and the platelet count 142 × 10^9/l. Serum calcium was 5.66 mmol/l (normal range 2.20-2.65 mmol/l), phosphorus 0.9 mmol/l (normal range 0.70-1.30 mmol/l), and alkaline phosphatase activity 101 IU/l (normal range 28-142 IU/l). Serum albumin was 34 g/l (normal range 35-45 g/l). The urea, creatinine, amylase and lipase concentrations were normal. The serum parathormone concentration was <0.1 µg/l (normal range <0.5 µg/l) and vitamin D concentration was 10 µmol/l (normal range 15-100 µmol/l). A chest radiograph showed increased osteoporosis and multiple lytic lesions throughout the skull. No serum or urinary paraprotein was detected.

Treatment with chlorambucil 6 mg/day, prednisonole 40 mg/day and intravenous frusemide 40 mg/day and intravenous fluids was begun, and after three days the calcium had fallen to 3.0 mmol/l. Intravenous mithramycin (25 µg/kg/day) for three days was given, after which the calcium concentration was 2.95 mmol/l. Two weeks later a further course of mithramycin was necessary as the calcium concentration had risen to 3.7 mmol/l. A further short-lived response was achieved but three weeks later, the patient fell, fractured his femur and pelvis, and died shortly afterwards from bronchopneumonia.

Thoracic aortitis due to salmonella

Case report
A 62 year old college lecturer was admitted with a six month history of night sweats, arthralgia, and lethargy. Two weeks before admission he developed haemoptysis, hoarseness, and continuous left shoulder pain. There was no history of recent foreign travel, nor diarrhoeal illness in the patient or his family, nor a notable medical history. On examination he had fluctuating fever up to 38.5°C. His blood pressure was 110/80 mm Hg in both arms with a systolic murmur at the left sternal edge and a paraduodenal rub. On chest X-ray picture, which had been normal four months earlier, showed a left hilar mass. His white cell count was raised at 18.6 × 10^9/l, with an erythrocyte sedimentation rate of 116 mm in one hour and a positive anti-nuclear antibodies test. Serum IgG and IgM anti-salmonella antibodies were 1:160 and 1:320; respectively.

Blood cultures and culture of urine were negative. At bronchoscopy the left vocal cord was seen to be paralysed, with extrinsic compression of the trachea and left main bronchus. Culture of bronchial washings was negative. A computed tomogram of the thorax (figure) showed aneurysmal dilatation of the thoracic aorta; this was confirmed at surgery.