surface immunoglobulin and were characterised by convoluted nuclei and scarce cytoplasm. The cells were 10-15 μ m in diameter and were negative for acid phosphatase, non-specific esterase, Sudan black, and periodic-acid Schiff stains. No abnormal lymphocytes were detected in the blood or bone marrow. Cranial radiation and intrathecal methotrexate were started on day 48, but no systemic cytotoxic therapy was given. Severe visual impairment persisted and his general condition remained unchanged. His spleen, lymph nodes, bone marrow, and peripheral blood remained normal. On day 71 occasional primitive lymphocytes and numerous smear cells were found in his blood; the white count was $2.7 \times 10^9/l$. The patient deteriorated until death on the 81st day after admission. There was no necropsy. Analysis of the bone marrow and peripheral blood at death showed infiltrating neoplastic cells with E and C3 receptors as detected previously in the CSF.

Discussion

This case may be regarded as acute leukaemia or lymphoma with early involvement of the meninges. It is unusual for acute leukaemia to present with CNS symptoms.³ The incidence of meningeal symptoms at diagnosis of non-Hodgkin's lymphoma varies—6/30 children reported from Toronto⁴ and 0/64 by Pinkel *et al.*⁵ This case appears to be the first documented of a T cell lymphoma presenting with CNS symptoms and no clinical evidence for other tissue lesions. The neoplastic cells had convoluted nuclei, but, unlike other cases of T cell ALL, expressed the C3 complement receptor. Tests with unsensitised and IgM-sensitised ox cells were negative.

This case is reported to emphasise that ALL enters into the differential diagnosis of CSF lymphocytosis. This point was not appreciated initially, since there was no indication that the lymphocytes in the first CSF were malignant, and the thymic shadow, although enlarged, was not considered abnormal.

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Acquired circulating anticoagulant in a 4-year-old child

Acquired inhibitors of coagulation have been reported in several conditions including haemophilia, various autoimmune disorders, elderly patients with no demonstrable underlying disease, and in women post partum. We wish to report a case occurring after infection.

Case report

A 4½-year-old girl presented with a six-week history of spontaneous bruising, anorexia, headaches, and lassitude. She had a recent history of an upper respiratory tract infection, which had been treated with a course of erythromycin. There was no family or previous history of a haemorrhagic diathesis. Clinical examination showed numerous small bruises over the trunk and limbs and one large subcutaneous haematoma in the left parasternal region. Discrete cervical lymphadenopathy was present. At the time of presentation a sibling had proved infectious mononucleosis.

The results of haematological studies were as follows: haemoglobin 12.0 g/dl; total white cell count $6.8 \times 10^9/l$; differential count, neutrophils

62%, lymphocytes 35%, monocytes 3%, no atypical cells seen. The ESR was 30 mm in one hour and the platelet count $171 \times 10^9/l$. A Paul-Bunnell screen (Monospot-Ortho Diagnostics) gave a negative result. Viral serological investigations showed significant titres for adenovirus (1/320), respiratory syncytial virus (1/80), *Mycoplasma pneumoniae* (1/80), and Epstein-Barr virus (IgG-1/128). Cytomegalovirus and rubella titres were 1/8 and 1/5 respectively. The results of immunological investigation were as follows: no antinuclear, gastric parietal cell, smooth muscle, or mitochondrial antibodies were detected; a screen for rheumatoid factor was negative. Serum complement levels were: C3 (65 mg/dl)—slightly reduced levels—and C4 (25 mg/dl) normal levels. The immunoglobulin levels were normal.

Anticoagulant effect of patient's plasma

Test	Patient	Normal range
Prothrombin time (seconds)	14.8	11.8–15.3
Kaolin-cephalin time (seconds)	62.6	33.4–52.4
Kaolin-cephalin time + equal amount normal plasma	61.6	
Thrombin time—5u (seconds)	14.0	12.0–16.0
Factor VIII assay 2-stage method (% normal)	130	50–200
Factor IX assay 1-stage method (% normal)	65	50–200
Factor II assay 1-stage method (% normal)	50	60–200

The anticoagulant effect of the patient's plasma (see table) was confirmed by a modification of the agarose gel inhibitor technique.¹ The degree of inhibition did not appear to be influenced by varying the concentration of phospholipid (Diagen-Bell and Alton platelet substitute). The same reagent was used in the kaolin-cephalin clotting time test. Antibody neutralisation experiments using sheep anti-human IgG and IgM (Burroughs Wellcome) indicated that the inhibitor was an IgG antibody. Initial clotting investigations were limited by the amount of plasma available. Three months after presentation the patient was clinically well and the inhibitor was undetectable. In view of the possible association of this inhibitor with infectious mononucleosis, samples from twenty individuals with serologically proved mononucleosis were tested using the agarose gel technique. Four samples showed a variable degree of inhibition of coagulation but further studies were not possible.

Discussion

The appearance of a circulating inhibitor of coagulation in a child is rarely reported. Lechner² in a review reported only one case of a coagulant inhibitor occurring after infection. The nature of the associated infection in our case is uncertain. The absence of diagnostic haematological and virological findings consistent with recent infectious mononucleosis³ could be due in part to the late presentation of the case. Bleeding problems occurring in this illness are usually associated with thrombocytopenia but abnormalities of coagulation have also been described.⁴

Circulating anticoagulants which do not appear to consume coagulation factors but otherwise interfere with the coagulation process have been described in systemic lupus.⁵ The mechanism of inhibition is poorly understood but may affect interaction of prothrombin with prothrombin activator. An associated variable decrease in prothrombin levels also occurs. Thus our inhibitor resembles the lupus anticoagulant. Inhibition of phospholipid, which has also been suggested for the lupus-inhibitor activity, was not found in our limited experiments.

When bleeding problems occur in association with infectious mononucleosis or other viral infections with a normal platelet count the possibility of an acquired circulating anticoagulant should be considered.

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