weeks' duration. She was taking diclofenac 50 mg twice a day for osteoarthritis of the hips.

On examination she was pale, ill, and had generalised lymphadenopathy and hepatosplenomegaly. Investigations showed a normocytic normochromic anaemia with an erythrocyte sedimentation rate of 117 mm/first hour. The white cell count was 17×10^9 with an absolute lymphocytosis. The direct Coomb's test was positive and the reticulocyte count was 4%. The bone marrow was hypercellular, but otherwise normal. Serum protein electrophoresis showed a large polyclonal increase in gamma globulins. Serum electrolytes and renal function tests were normal, but the corrected calcium was 3·1 mmol/l (normal 2·25–2·65 mmol/l) and phosphate was 1·22 mmol/l (normal 0·80–1·45 mmol/l). Hypercalcaemia persisted. Serum parathormone concentrations were inappropriately high and varied from 0·22 μg–0·26 μg/l (normal range less than 0·0·1–0·73 μ73 μg/l) over a period of six months.

During her illness, the serum thyroxine (T4) concentration fell from 57 nmol/l to 15 nmol/l (normal range 50–150 nmol/l) and thyroid stimulating hormone activity remained normal. A thyroid scan showed no abnormality and there were no thyroid antibodies in the serum. A cervical lymph node biopsy specimen showed deposits of papillary thyroid carcinoma and a striking lymphoproliferative reaction, thought possibly to be an immune response to the tumour, or angioimmunoblastic lymphadenopathy.

She was discharged home, but returned nine days later, having deteriorated rapidly and was sleepy and confused. On readmission she had widespread, large, firm, tender, generalised lymphadenopathy. A right axillary lymph node biopsy specimen showed features of angioimmunoblastic lymphadenopathy. She was treated with prednisolone 20 mg four times a day with a good response and was well for five months. She then became lethargic and there was a rapid deterioration of renal function. A renal biopsy specimen showed focal segmental proliferative glomerulonephritis. She died of renal failure and bronchopneumonia three weeks later.

Comments

The first patient is the fourth reported example of histologically confirmed Hashimoto's disease, associated with angioimmunoblastic lymphadenopathy,[1] and there have been reports of associated abnormal thyroid function tests in this disease. The patient was taking fenoprofen before the onset of the disease and it is well known that acute T cell leukaemia can be precipitated by a variety of drugs, and a patient who already has autoimmune disease would be particularly susceptible to developing abnormal lymphoproliferative reactions to foreign antigens such as drugs. It is also reasonable to speculate that an abnormal immune system might predispose to both conditions.

The second patient with papillary carcinoma of the thyroid shows an association which has not been previously described. There was no coexistent thyroiditis. This patient also exhibited a segmental proliferative glomerulonephritis and this may have been due to circulating antigen and antibody complexes. It has been described only once before in association with acute T cell leukaemia.[2] The gross hypercalcaemia with normal parathyroid glands strongly suggests that the abnormal lymphoid cells are responsible for secreting a parathyroid hormone-like substance and this association has been described before.[3,4] This patient was, however, unique in showing the changes of advanced osteitis fibrosa cystica. Hypercalaemia is also common in acute T cell lymphoma/leukaemia associated with HTLV-1 infection.

The nature of angioimmunoblastic lymphadenopathy is now believed to be a variety of peripheral T cell lymphoma, but displaying features of a disordered immune system.

Autoantibodies occur in angioimmunoblastic lymphadenopathy but the precise nature of association with thyroiditis is unknown.

It is uncertain whether thyroid carcinoma in case 2 is mere coincidence, but altered immunity might predispose to carcinoma, and malignant disease has been reported in association with angioimmunoblastic lymphadenopathy.

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Prevalence of HTLVI in Zimbabwe: a pilot survey

Researchers have shown that human T cell leukemia/lymphoma virus (HTLVI) infection is common in Japan and among certain black populations in the Caribbean and other well defined areas of the world, and that it is associated with a high incidence of adult T cell leukemia/lymphoma.[1,2] Because of concern with diseases acquired after transfusion, the Blood Transfusion Service carried out a survey to identify the possible presence of HTLVI in Zimbabwe, and to determine the need to do routine HTLVI screening on all blood donations.

Random samples were collected from normal blood donors as well as from a sample of patients with lymphoma and those sent for human immunodeficiency virus (HIV) exclusion because of symptoms. The survey, conducted in December 1987, showed no evidence of HTLVI antibodies in the group with lymphoma or normal blood donors. There was an 0·4% positivity rate found among the patients sent for HIV exclusion (table). The Dupont HTLVI and Serodia ATL test kits were used in this survey. The four positive specimens found using the Dupont test kit were retested using the Serodia ATL kit; only two were found to be positive.

The sample size was adequate to detect a true population prevalence of 1%, but with this prevalence most of the positive tests would be expected to be false positive, even with a test sensitivity and specificity of 99%. As no confirmatory test method is available in Zimbabwe, we are unable to confirm these results. In view of the findings presented above we conclude that screening for HTLVI antibodies is not indicated in Zimbabwe at present, but suggest continued sample

Table *Results of HTLVI testing*

Type of specimen	*No of specimens*	*HTLVI antibodies No (%)*
Normal blood donors	578	0
Patients with lymphoma	26	0
Patients sent for HIV exclusion	296	4 (1·4)
Total	900	4 (0·4)

surveillance to detect any changes in the present pattern.

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Condoms and safe sex

The advice that using a condom reduces the risk of transmission of human immunodeficiency virus (HIV) infection would have to be questioned if latex condoms were found to have the same 5 μm tortuous channels which were recently reported to exist in latex surgical gloves.[1]

Freeze fractured sections of Mates, Durex Featherlite, and Durex Elite condoms were studied at magnifications of up to 10 000 times with scanning electron microscopy. Some fracture artefacts were seen, but no other form of channel was detected. Pendle and Cobbold[2] suggested that the channels described by Arnold *et al*[1] were artefacts produced by cracking of the electrically conductive coating that was applied during the preparation of the sample for scanning microscopy.

There is no evidence that condoms contain channels of greater than 300 nm in diameter.

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Matters arising

Postmortem radiology in children with congenital heart disease

We noted with interest the paper by Russell and Berry describing postmortem radiology in children with congenital heart disease.[1] We would like to add a further case in which radiology proved essential in determining the patency of coronary arteries after anatomical correction for transposition of the great arteries.

Clinical history

The patient was seen at Birmingham Children's Hospital on the day of delivery because of cyanosis. An echocardiogram showed transposition of the great vessels and a patent ductus arteriosus. A balloon septostomy was performed, followed 10 days later by an arterial switch operation. The anatomy of the coronary artery was noted to be abnormal at the time of surgery with the right coronary artery giving rise to the circumflex branch which passed to the left. Coronary artery ostia were mobilised and the aorta switched posteriorly. The left coronary ostium was translocated to the facing sinus of the pulmonary artery. Both ostia seemed to be patent, but there was bleeding from around the left coronary ostium. Although this was controlled, the patient developed an intractable arrhythmia which failed to respond to all measures and subsequently died.

The possibility of a mechanical obstruction of one of the coronary ostia as a cause of death was considered. At necropsy the aorta was opened 1·5 cm above the aortic valve and the distal aorta tied off. Water soluble contrast medium was then injected into the aorta and the specimen examined radiologically. There was filling of both coronary arteries, by contrast, together with filling of the left ventricle with reflux into the pulmonary veins (figure). The latter may have been due to the injection pressure being insufficient to force valve closure.

We were thus able to state that the coronary artery anastomosis was technically sound and that there was no evidence of kinking of the coronary arteries. Moreover, the lumen was patent excluding obstruction from thrombosis or embolism. We would, therefore, agree with Russell and Berry[1] that postmortem radiology is a simple but invaluable technique for assessing vascular anatomy in congenital heart disease, particularly where surgical repair has been performed.

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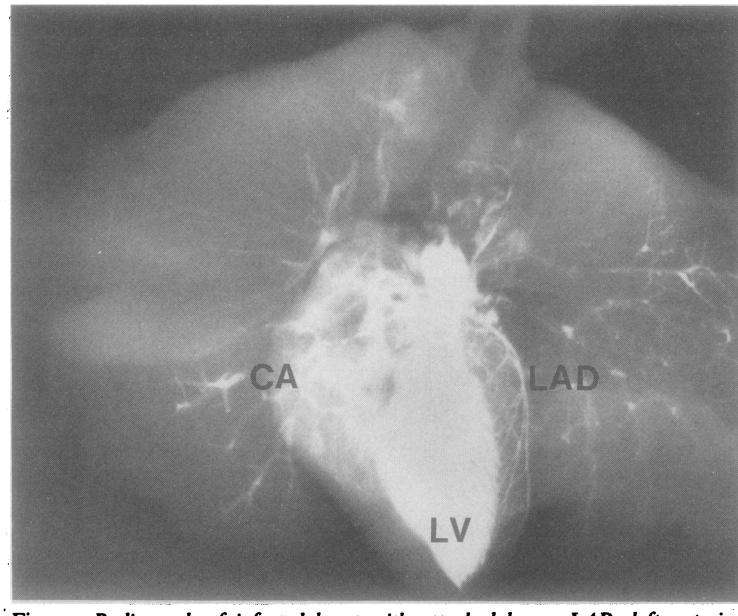

Figure *Radiograph of infected heart with attached lungs. LAD, left anterior descending coronary artery; CA, circumflex coronary artery; LV, left ventricule.*