neutrophils, 1% eosinophils, 64% lymphocytes, and 5% monocytes. Four days later the white cell count was $86 \times 10^9/l$, with 5% neutrophils, 92% small lymphocytes, and 3% monocytes. Bone marrow examination showed an 88% infiltration with small, mature, PAS-negative lymphocytes.

There were no symptoms suggesting whooping cough, and no history of recent vaccination. A chest x-ray film was normal; in particular, there was no suggestion of thymic enlargement. The result of bone marrow cytogenetic analysis and electron microscopy was normal, as was a Paul-Bunnell test. Bacteriological cultures were all sterile, as were all the following antibody titres measured at fortnightly intervals: influenza A, B; parainfluenza I, II, III; adenovirus; ornithosis; psittacosis; respiratory syncytial virus; *Mycoplasma pneumoniae*; Q fever; poliomyelitis I, II, III; herpes; cytomegalovirus; mumps; Coxsackie B1, B2, B3, B4, B5, B6. Immunological investigations showed: IgG 1.120 mg/100 ml, IgA 35 mg/100 ml, IgM 112 mg/100 ml; antibodies to nuclei, smooth muscle, and mitochondria were absent. The lymphocytes were typed by the method of E rosettes for T cells (normal for laboratory $60\% \pm 10\%$) and membrane immunoperoxidase for B cells (normal for laboratory $10\% \pm 5\%$). 78% of the patient's lymphocytes ($56.7 \times 10^9/l$) were T lymphocytes and 5% ($2.9 \times 10^9/l$) were B lymphocytes. Phytohaemagglutinin stimulation showed one-quarter blastic transformation of control. The symptoms resolved rapidly and the lymphocyte count returned to normal in two weeks. The only treatment given was allopurinol to avoid hyperuricaemia. Three months after the infectious episode there were $1.78 \times 10^9/l$ T lymphocytes and $0.22 \times 10^9/l$ B lymphocytes.

Discussion

T lymphocytes play an important part in antiviral immunity and it could be conceived in a genetically predisposed individual that a viral infection might induce a transitory T lymphocytosis. In 1976 lymphocyte typing in a case of acute infectious lymphocytosis was reported² where the authors found equal proportions of T lymphocytes and null lymphocytes. In our case 17% were null lymphocytes and we think that this proportion is insufficient to say there was a "null-cell" proliferation. Indeed, in Sezary's syndrome the median for T cells is 75% and for B cells 3%.³ The same differential exists in our case but is insufficient to postulate the existence of another non-T cell proliferation. If further observations are confirmatory, acute infectious lymphocytosis must be classed among other T immunoproliferative disorders, along with Sezary's syndrome, and or mycosis fungoides, some chronic lymphatic leukaemias, prolymphocytic leukaemias, infectious mononucleosis, and whooping cough.⁴

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Intrathoracic tracheal tumour presenting as asthma

Intrathoracic tracheal stenosis may present a difficult diagnostic problem and is often misdiagnosed as asthma. It was recently suggested¹ that flow volume curves should be the mainstay of diagnosis, and subsequently that direct examination is more helpful.²

Case report

A 53-year-old active amateur football referee was first seen as an outpatient at his local hospital in April 1976. He had been forced to give up refereeing two years previously as he developed dyspnoea with a wheeze and tightness across his chest on running 100 yards. In his own words he was "unable to get his breath out." He was a non-smoker. The result of physical examination was completely normal, as was chest radiography, but spirometry showed forced expiratory volume in one second (FEV₁) 2.69 l (predicted 3.82), forced vital capacity (FVC) 6.58 l (4.9), and FEV₁/FVC ratio 40% (78%). Exertional asthma was diagnosed.

He was lost to follow-up but was admitted to his local hospital in January 1977, with a three-month history of progressive dyspnoea with wheeze and a two-week history of cough with sticky white sputum. He was febrile, dyspnoeic at rest, though not cyanosed, and had generalised high-pitched expiratory rhonchi on auscultation. Pneumococcus was grown from his sputum, and chest radiography showed right basal pneumonia. He responded poorly to treatment and needed ventilation for eight days via a cuffed endotracheal tube. He was finally discharged after three months, when chest radiographs were clear, but spirometry showed FEV₁ 0.50 l, FVC 1.80 l, and FEV₁/FVC ratio 28%, not improved by bronchodilators.

His convalescence in Devon was interrupted by a recurrence of his wheezy dyspnoea after a heavy cold, and in May 1977 he was admitted to this hospital. On admission he was dyspnoeic at rest with pronounced expiratory stridor. He was afebrile, but had a tachycardia of 120 with pulsus paradoxus. There was a generalised expiratory wheeze, loudest over the trachea. He was otherwise normal.

Tracheal stenosis, probably secondary to controlled ventilation via a cuffed endotracheal tube, was provisionally diagnosed. The patient was treated with antibiotics, steroids, and salbutamol and was referred to the otolaryngologist. The cause of the obstruction could not be seen on mirror examination. Tracheal tomograms were considered, but his condition was thought to necessitate urgent tracheostomy. At tracheostomy an annular tumour was seen just above the carina and distal to the tracheostomy tube. An extended tube was passed through the tumour. The histology of the tumour was adenocystic carcinoma (cylindroma) of the trachea.

At thoracotomy five days later the tumour was found to extend down past the carina into both upper main bronchi. Lower tracheal and carinal resection was performed with reconstruction by anastomosis of the right main bronchus to the trachea and the left main bronchus to the side of the right main bronchus. Postoperatively the patient made a good recovery.

Comment

Intrathoracic localised airways obstruction may masquerade as asthma^{3,4} or chronic obstructive airways disease. The interval between presentation with symptoms and correct diagnosis varies between one month and six years.⁴ Yet it is an important diagnosis to consider, as stenosis secondary to trauma from cuffed endotracheal tubes is likely to become more common, and locally invasive tumours may be successfully treated with surgery or radiotherapy if diagnosed early.

Flow-volume loops⁴ and the comparison of maximum inspiratory with maximum expiratory flow rates⁵ have been suggested as diagnostic tools. The case for direct examination has been succinctly stated.² A history of dyspnoea with wheeze or stridor on moderate exertion, the absence of symptoms at rest, and an abnormally low FEV₁ and FEV₁/FVC ratio should arouse suspicion. The definitive investigations then are tracheal tomography and endoscopy.

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