Chronic lymphocytic leukaemia and concomitant relapsing polychondritis: a report on one treatment for the combined manifestation of two diseases

Sir, We report the case of a 60-yr-old patient who suffered from chronic lymphocytic leukaemia (CLL) with concomitant relapsing polychondritis. Polychondritic exacerbations were regularly observed with CLL progression and included bilateral painful swellings of the external ear cartilage and of the trachea, disseminated inflammatory polyarthritis, ocular inflammation and a palatal ulceration (Fig. 1). All manifestations of polychondritis and CLL responded well to chlorambucil/prednisone initially, and to cyclophosphamide during later stages of the disease, while the response to methotrexate was poor and treatment with bendamustin was complicated by tumour lysis syndrome and subsequent acute renal failure. Treatment with cyclophosphamide repeatedly induced remissions for both diseases, but was regularly accompanied by serious infectious complications. Relapsing polychondritis is rare and is generally diagnosed clinically if the patient develops at least three of the following signs: bilateral chondritis of the external ears, inflammatory polyarthritis, ocular inflammation, nasal chondritis, vestibular/auditory malfunction and respiratory tract chondritis. Notably, its 5-yr mortality may be as high as 30%, due to collapse of laryngeal and tracheal cartilaginous supporting structures, or cardiovascular involvement. Mild cases may respond to NSAIDs. More severe cases are usually treated with prednisone and may require additional immunosuppressive agents, e.g. cyclophosphamide [1].

![Fig. 1. Pain and a purple swelling of the external ear are the most common first clinical signs of relapsing polychondritis. The swelling may extend into the ear canal and induce ear infections, hearing loss and balance disturbances with vertigo and vomiting. During later stages of polychondritis, the destruction of supporting cartilaginous tissue may become visible as floppy ears occurring together with auditory and vestibular abnormalities, a flattened nose bridge (saddle nose) or, less frequently, with visual disturbances due to recurrent inflammation, which may lead to blindness. The two pictures at the top (A) show our patient prior to treatment, while the pictures at the bottom (B) show our patient after one cycle of chlorambucil. This figure may be viewed in colour as supplementary data at Rheumatology Online.](image)

The authors have declared no conflicts of interest.

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Adalimumab-induced asthma

Sir, A 51-yr-old lady with a 15-yr history of erosive seropositive rheumatoid arthritis (RA) was treated with adalimumab, having failed multiple DMARDs. Prior to this she had no personal or family history of asthma or atopy and had never smoked.

Within 2 weeks of starting adalimumab she developed a diurnal bronchial wheeze with shortness of breath. This persisted and was reported at review 8 weeks later. Pulmonary function tests (PFT) showed an obstructive pattern, forced expiratory volume in 1 s (FEV1) reduced by 49% of predicted and a significant bronchodilator response of 25% improvement in FEV1 (15% improvement indicates reversible airways disease) (Table 1). A raised transfer coefficient (KCo) of 1.88 mmol/kPa/min/l (117% predicted) was also noted, which is typical of asthma. A full blood count showed a new eosinophilia of 1.0 × 10⁹/l (normal range 0-81).

![Table 1. Pulmonary function test before and after bronchodilator](image)

The chronology of events and absence of previous respiratory disease suggested an adverse reaction to adalimumab. Introducing methotrexate was considered to suppress this presumed immunological side-effect, but due to previous severe rashes with methotrexate this was not pursued. Therefore inhaled beclometasone 200 µg twice daily was added.

Within 3 days her symptoms had improved and within 2 weeks they were completely controlled by beclometasone. Subsequent PFT on beclometasone whilst still on adalimumab were much improved, with a FEV1 of 2.02 l and a FEV1/forced vital capacity (FVC) ratio of 81.8%.

Unfortunately, after an initial good response to adalimumab with a greater than 50% reduction in swollen and tender joint count, the RA persistently flared. Adalimumab was stopped after 5 months of treatment. The patient subsequently stopped...
Letters to the Editor

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Fatal streptococcal toxic shock syndrome in a patient with rheumatoid arthritis treated with etanercept

Sir, We report the case of a 24-yr-old female with a 5-yr history of severe seropositive rheumatoid arthritis (RA) treated only with chloroquine and prednisone. Over the last 2 yr she had been maintained on prednisone 25 mg. Her physical exam revealed Cushingoid features in addition to persistent active joint inflammation in the small joints of the hands and wrists. In view of the severity of her disease it was decided to start her on methotrexate and etanercept. She received her first dose of etanercept 25 mg subcutaneously; the next day she started to complain of nausea, vomiting and diarrhoea associated with fever. She was managed with intravenous fluid and electrolyte replacement. Two days later she presented to the emergency room with fever, hypotension (blood pressure 80/50 mmHg) and generalized lethargy. She reported a history of a fall a few hours before with trauma to her right lower extremity. Her physical exam revealed swelling and erythema over the right knee and thigh. She was managed with fluid replacement and broad spectrum antibiotics. Her condition rapidly deteriorated and she went into shock with further drop in her blood pressure, tachycardia, tachypnoea and anuria. She was intubated, mechanically ventilated and was transferred to the intensive care unit. Her blood pressure did not pick up despite full-dose inotropes and flush fluids. Her right lower extremity rapidly became mottled with sloughing of the overlying skin. She had a cardiac arrest around 12 hr after her admission to the emergency room. Two blood cultures revealed streptococcus group A.

The rapid development of a streptococcal toxic shock syndrome shortly after the initiation of etanercept therapy in a...