young patient with chronic RA raises the question of whether this was a coincidental occurrence or whether the etanercept caused exacerbation of an active subclinical infection. The chronic steroid therapy with 25 mg of prednisone daily should also be considered as an additional contributing factor in masking the infection. In our review of the literature only two cases of fatal sepsis associated with tumour necrosis factor alpha (TNF-α) blockers were reported [1, 2]. Baghai et al. [1] reported a case of fatal pneumococcal sepsis occurring in a 37-year-old woman with rheumatoid arthritis treated with etanercept, and Herrlinger et al. [2] reported the case of a 40-year-old woman who after six infusions of infliximab for peri-anal Crohn’s disease developed staphylococcal pneumonia resulting in fatal adult respiratory distress syndrome. Kroesen et al. [3] reviewed patient charts and records of the infectious disease unit for serious infections in patients with RA in the 2 yr preceding anti-TNF-α therapy and during therapy. Serious infections affected 18.3% of patients treated with infliximab or etanercept. In several cases, only a fewsigns or symptoms indicated the severity of developing infections and sepsis; therefore a high level of suspicion of infection is necessary in patients under anti-TNF-α therapy.

Our experience with TNF-α blockers at the American University of Beirut Medical Center (a tertiary-care teaching hospital, one of the largest medical centres in Lebanon), dates back to October 2000; up to the present time around 90 patients with various rheumatic diseases have received this drug [4]. No serious infections in our series have been reported except for a case of miliary tuberculosis [5]. A thorough screening for any latent infections in our series have been reported except for a case of miliary tuberculosis [5]. A thorough screening for any latent infections in our series have been reported except for a case of miliary tuberculosis [5].

The authors have declared no conflicts of interest.

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Accepted 8 April 2005

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Rheumatology 2005;44:1201–1202
doi:10.1093/rheumatology/keh700
Advance Access publication 7 June 2005

Fatal Mycobacterium peregrinum pneumonia in refractory polymyositis treated with infliximab

Sir, Polymyositis (PM) and dermatomyositis (DM) are systemic inflammatory disorders affecting skeletal muscles and other organs. PM and DM are considered to be associated with high morbidity and mortality rates, particularly related to infectious complications, which have been described in up to 26% of patients [1]. Recently, opportunistic infections have been reported in 12–21% of PM/DM patients. Many factors have been implicated in this apparently increased frequency of infections in PM/DM, especially immunosuppressive drugs [1, 2]. We report a case of fatal Mycobacterium peregrinum pneumonia in a patient with refractory PM, which occurred shortly after institution of anti-tumour necrosis factor α (anti-TNF-α) therapy.

A 68-yr-old man had PM that had evolved since December 2001. Therapy with prednisone was initiated at a dose of 1 mg/kg daily. As both clinical and biochemical status continued to deteriorate gradually, subsequent treatments were started: methotrexate, azathioprine, cyclophosphamide and cyclosporin (cumulative doses of 1080 mg, 31 500 mg, 3 g and 36 000 mg, respectively), which proved ineffective. Intravenous immunoglobulins (1 g/kg/day for 2 days monthly) for 24 months resulted in partial improvement of PM. In January 2004, the patient was admitted for generalized muscle weakness. Muscle power, gauged for eight proximal muscles by a modification of the UK Medical Research Council grading system, was 59/88 points [1]. Serum creatine kinase level was 15 000 IU/l. Pulmonary function tests showed decreased vital capacity (64% of predicted values); lung computed tomography (CT) scan was normal. Other investigations, including abdominal CT scan, gastroscopy, colonoscopy and bronchoscopy, to exclude underlying malignancy, were normal.

As PM was refractory to immunosuppressive therapy, the patient was given anti-TNF-α, as mentioned previously [3]: infliximab (5 mg/kg) at weeks 0, 2 and 6, in addition to prednisone (20 mg daily). Before initiation of infliximab, gastric aspiration product cultures for mycobacteria were performed, which proved negative.

One month after the third infliximab infusion, the patient presented with a 3-week history of fatigue, non-productive cough and progressive dyspnoea. On admission, he was febrile (38°C) and his general condition was poor. Physical examination revealed ‘Velo’ crackles bilaterally. Laboratory findings were as follows: erythrocyte sedimentation rate 90 mm/h, C-reactive protein 142 mg/l, haemoglobin 10.2 g/dl, white blood cell count 9.5 x 10³/l (lymphocytes 9%, CD4 cell count 0.09 x 10⁴/l), and creatine phosphokinase 12 697 IU/l. Chest radiograph revealed diffuse bilateral lung infiltrates. Blood cultures, bacterial (Mycoplasma pneumoniae, Chlamydia pneumoniae, Legionella) and viral (cytomegalovirus, influenza viruses) serologies were negative. Bronchoscopy was normal; analysis of bronchoalveolar lavage fluid (BAL) was negative for Gram and Gomori–Grocott staining and bacterial and fungal cultures. The patient was treated with ceftriaxone (2 g daily) and ciprofloxacin (500 mg/12 h) for 2 days; however, pulmonary clinical manifestations continued to deteriorate. Further microbiological studies (Zielh–Neelsen stain) yielded acid-fast bacilli in sputum, gastric aspiration products and BAL. The patient received combined therapy with rifampin,isoniazid, ethambutol and pirazinamide; despite the use of mechanical ventilation, he died of respiratory failure within 9 days. Cultures of BAL and three samples of sputum and gastric aspiration products grew Mycobacterium peregrinum within 15 days.

Infections are potential complications of anti-TNF-α therapy [4–9]. In patients with rheumatoid arthritis being treated with anti-TNF-α agents, Kroesen et al. [5] have found an elevated incidence of severe infections (0.181 per anti-TNF-α therapy vs 0.008 in the 2 yr preceding anti-TNF-α therapy). In a series of patients receiving anti-TNF-α therapy, Wallis et al. [7] found the incidence rate of opportunistic infections to be as high as 313/100 000 patients; tuberculosis was the most common infection, occurring in 179/100 000 patients. Opportunistic infections due to other pathogenic microorganisms have also been observed in association with anti-TNF-α therapy; e.g. Pneumocystis carinii, Histoplasma, Aspergillus, Candida, Nocardia, Cryptococcus and non-tuberculous mycobacteria [4–9].
We report, to the best of our knowledge, the first case of *Mycobacterium peregrinum* pneumonia in a patient with refractory PM. *Mycobacterium peregrinum* is a non-tuberculous mycobacterium, which may cause disseminated infections in patients with connective tissue disorders. In our patient, several factors favoured *Mycobacterium peregrinum* pneumonia onset: (i) ventilatory insufficiency due to striated muscle weakness; and (ii) immunodeficiency secondary to denutrition, steroids and (mainly) infliximab. *Mycobacterium peregrinum* pneumonia, indeed, occurred shortly after institution of infliximab; our findings confirm previous data demonstrating that 75% of opportunistic infections occurred within 90 days after anti-TNF-α therapy initiation [4–9].

Moreover, in a series of 156 PM/DM patients, we have shown a high prevalence of opportunistic infections (12%) due to, for example, fungi (56% of cases) and mycobacteria (22% of cases; *Mycobacterium avium-intracellulare*, *xenopi*, *marinum* and *tuberculosis*) [10]. Factors predisposing to opportunistic infection onset were thoracic myopathy, oesophageal motor dysfunction and lymphopenia [10]. Interestingly, the present case report therefore highlights that it is questionable to initiate anti-TNF-α therapy in refractory PM/DM patients presenting with predisposing factors, placing them at risk of opportunistic infections. Our data strongly encourage searching for factors predictive of opportunistic infections in PM/DM patients before anti-TNF-α therapy is begun; these patients should undergo investigations (CD4 blood cell count, lung and oesophageal tests) and be monitored closely for onset of opportunistic infection. To date, prophylaxis against *Mycobacterium tuberculosis* infection is recommended for patients with a past history of tuberculosis who are undergoing anti-TNF-α therapy [4–9]; as shown by the variety of pathogenic agents responsible for opportunistic infections in PM/DM [10], it seems difficult to initiate primary prophylaxis in patients exhibiting risk factors for opportunistic infections before initiation of anti-TNF-α agents.

The authors have declared no conflicts of interest.

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Accepted 4 May 2005

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Advance Access publication 7 June 2005

Undergraduate education in rheumatology

Sir, We are encouraged by the recent article by Goh et al. [1], which reviews the need for a rheumatology curriculum. We agree that further progress is necessary to incorporate rheumatology into the core components taught in our medical schools, particularly in view of the high prevalence of rheumatic conditions in the community.

In support of this, the charity the Arthritis Research Campaign (arc) has published an educational strategy (www.arc.org.uk), and has, over the last few years, developed a number of initiatives to address this problem. Through its Teaching the Teachers workshops, arc has been encouraging rheumatologists to develop their skills as clinical teachers. Our belief is that, through direct teaching involvement as well as by becoming champions and role models, teachers and rheumatologists within the specialty can have a significant impact on the undergraduate curriculum. At arc’s national Teaching the Teachers workshop in Northumberland in 2002, emphasis was placed on the development of an extended musculoskeletal clinical examination for undergraduates. More recently, this has led to the development of a regional musculoskeletal examination (REMS) alongside the well-validated GALS screening examination.

At the national Teaching the Teachers workshop in Sheffield in 2003, agreement was reached that musculoskeletal history-taking and musculoskeletal examination were essential core skills for all undergraduates. It was also agreed that this should be achieved through ‘spiral learning’, whereby musculoskeletal learning opportunities as well as associated assessments are provided throughout the undergraduate curriculum rather than simply at one or two time points. At the Sheffield conference, a national collection of extended matching questions and objective structured clinical examination (OSCE) stations in rheumatology was developed and made available to all UK medical schools. This built on the development work in previous are Teaching the Teachers workshops.

It is our belief (and experience) that assessment drives learning, and in May 2004 the most recent Teaching the Teachers workshop focused on competency-based assessments and performance relevant to both undergraduate and postgraduate medical training. The workshop was extended to professions allied to rheumatology, and there were many multiprofessional sessions.

In addition to the promotion of undergraduate electives and self-selective components in rheumatology for undergraduates, the arc has also developed a range of literature and electronic resources for undergraduates. Later in the year, the charity hopes to launch a graduate certificate in rheumatology practice for allied