Cutaneous Mycobacterium chelonae infection in chronic lymphocytic leukaemia

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A 67 year old man was diagnosed with chronic lymphocytic leukaemia (CLL) in 1999. He was also known to have hypogammaglobulinaemia and bronchiectasis. He had been previously treated with 12 cycles of chlorambucil, 2 cycles of fludarabine and cyclophosphamide completed in July 2004, and alemtuzumab 30 milligrams subcutaneously three times a week for 12 weeks between January and March 2005. Twelve months after the completion of alemtuzumab therapy whilst in clinical and histological remission, he presented with a four month history of a purple nodule on the left upper thigh. The clinical differential diagnosis included squamous cell carcinoma, leukaemic infiltrate or Kaposi's sarcoma so an incisional skin biopsy was done. Histological examination showed an infiltration of small lymphocytes in the mid dermis and immunohistochemistry showed these to be positive for CD5 and CD23 consistent with infiltration by CLL. In addition a large number of acid fast bacilli were seen on Ziehl Nielsen staining. The patient was considered to be significantly immunocompromised by virtue of the CLL, hypogammaglobulinaemia and his previous chemo and immunotherapy so he was initially commenced on rifampacin and ethambutol in line with British Thoracic Society guidelines for the management of extra-pulmonary opportunistic mycobacterial infection. Blood cultures and bronchoscopy with alveolar lavage and culture were negative. Skin tissue culture showed an atypical mycobacterium identified as Mycobacterium chelonae. The patient did not improve clinically on this regimen and developed a second adjavent nodulo-ulcerative lesion on the left thigh. He was started on clarithromycin 500mg bd based on previous dermatological experience and review of the literature. Sensitivity of the M. chelonae strain to ciprofloxacin, clarithromycin, rifabutin and amikacin, with resistance to ethambutol and isoniazid was demonstrated *in vitro* so ciprofloxacin 500mg twice daily was added with monthly immunoglobulin replacement. A slow but steady improvement has occurred with partial resolution of the two lesions. Due to further progression of his chronic lymphocytic leukaemia, the patient has since been retreated with alemtuzumab. He has continued with clarithromycin, ciprofloxacin and monthly immunoglobulin replacement without progression of these lesions or development of further skin lesions at 6 months follow up.

Mycobacterium chelonae is a non tuberculous mycobacterium which is a free living ubiquitous organism found in soil, water and dust. The source of contamination is usually from colonized tap water, although iatrogenic infection following environmental contamination of equipment such as sutures and skin marking solu-

Figure 2.

Figure 1.

Figure 3.

tion has been reported. The source in this case is obscure. While it can cause both soft tissue and skeletal infection in healthy patients, disseminated disease occurs almost only in the immunosuppressed. There are a few reports of Mycobacterium chelonae infection in neutropenic patients with other haematological malignancies (acute leukaemias, myelodysplasia and hairy cell leukaemia) but this is the first reported case of Mycobacterium chelonae infection in CLL. Although this patient was neither neutropenic nor lymphopenic, he was hypogammaglobulinaemic and had been heavily pre-treated most recently with alemtuzumab which will have severely impacted on his cell mediated immunity. The patient's CD4 T cell count was not specifically measured.

Skin disease is common in CLL and a spectrum of problems are encountered. Characteristic features are histological appearances worse than clinical features, infiltrate with CLL complicating the rash or tumour but as a bystander phenomenon rather than frank leukaemia cutis.

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