Perianal Tuberculosis: A Rare Case of Skin Ulceration?

Ljubka Miteva and Emil Bardarov

Department of Dermatology and Venereology, Medical University, Sofia, 431 Bulgaria E-mail: mitev@medfac.acad.bg Accepted June 18, 2002.

Sir,

Perianal tuberculosis is an extremely rare disease. We describe a male patient with perianal ulcers associated with active pulmonary tuberculosis. A wide range of differential diagnoses for perianal ulcers might be one reason for a possible delay in establishing this diagnosis. The different aetiopathogenetic, clinical and diagnostic manifestations of perianal tuberculosis are discussed.

CASE REPORT

A 38-year-old heterosexual man presented with painful perianal ulcers of 9-months' duration. Associated symptoms of weakness, night sweats, productive cough and weight loss were reported during the previous 2 years. He was a heavy smoker and drinker and had a past medical history of tuberculous lymphadenitis 19 years earlier, along with frequent pulmonary infections and haemorrhoids.

On admission, he was cachectic, pyrexic and very weak. Coarse breath sounds were heard in the upper lung fields bilaterally. Dermatologic examination revealed a deep and sharply demarcated ulceration about 8 cm in diameter with purulent base (Fig. 1). Inguinal lymph nodes were not enlarged. No abnormality on rectal



Fig. 1. Ulcerated perianal lesions.

examination was found except slightly inflammatory haemorrhoids.

Laboratory findings, including a complete blood count, liver function tests and urinalysis were normal, except for ESR (52/mm). Syphilis serology (VDRL, TPHA), hepatitis B, C and human immunodeficiency virus (HIV) serology were negative. Chest X-ray showed bilateral apical infiltrations with evidence of caviation in both lungs. No evidence of intestinal tuberculosis was found by intestinal X-ray and colonoscopy. Mantoux test (with 10 TU of PPD) was negative.

Histopathologic examination of the biopsy obtained from the ulcer revealed acanthosis with pseudoepitheliomatous hyperplasia overlying granulomatous infiltrate in the dermis consisting of epitheloid cells and a few Langhans giant cells. Focal areas of caseation necrosis were also present. A Ziehl-Neelsen stain showed numerous acid-fast bacilli. M. tuberculosis was cultured from skin biopsy specimen and sputum.

These findings established a diagnosis of perianal tuberculosis with underlying pulmonary tuberculosis. Antituberculosis therapy was started, consisting of isoniazid 5 mg/kg, rifampicin 10 mg/kg, ethambutol 15 mg/kg and pyrazimide 30 mg/kg. The strain was sensitive to all first-line drugs used. Two months after treatment, the ulcers healed with minimal atrophic scarring and the respiratory symptoms resolved. Pyrazimide and ethambutol were discontinued. A complete clinical improvement was noted at the end of 1-year of therapy.

DISCUSSION

The incidence of tuberculosis in Western European countries has decreased in the past few decades (1). Tuberculosis is a well-recognized complication in patients who are HIV-1-positive, suffer from malignancy, or are immunosuppressed (2). Cutaneous tuberculosis is still uncommon in Bulgaria (3-5). We report the first case of perianal tuberculosis in our country.

In extrapulmonary tuberculosis (about 5% of all cases) the anal localization is rare (0.7%) (6). In recent years, because of the AIDS pandemic, tuberculosis has resurrected. However, perianal tuberculosis has not been reported in HIV-infected individuals. Orificial tuberculosis is the tuberculosis of the oral and perianal mucoses resulting from auto-inoculation of the tuber-cle bacilli in patients with advanced pulmonary or gastrointestinal tuberculosis, the skin lesions arise as a consequence of auto-inoculation from swallowed bacilli containing sputum which attack previously traumatized skin (7, 8). Its occurrence by lymphatic, haematogenous

or direct extension of the disease is rare (7). In the present case, the course of the infection could be classified as reactivated tuberculosis because of the past history of tuberculous lymphadenitis established by investigative procedures, followed by specific treatment and asymptomatic period. There were two possible ways for development of perianal ulceration: haematogenous spread or, more likely, ingested mycobacteria from swallowed respiratory secretions.

The most common cutaneous lesions are large painful ulcers with bluish undermined edges and purulent, greyish in colour base. The presentation of perianal tuberculosis may vary from abscesses and fistulas, through vertucous and lupoid to miliary forms (7-9). Cutaneous hypersensitivity to tuberculin in these patients is variable; however, there is absolute consensus that such patients ultimately develop anergy (10). Histological findings are usually nonspecific, showing ulceration and lymphedema. Yet, in most cases, tuberculoid infiltrates with pronounced necrosis are found in deep dermis. Tubercle bacilli may be demonstrated by microscopy (10). Polymerase chain reaction (PCR) can lead to a more accurate diagnosis, but cultures should always be done to confirm PCR results (11).

The following differential diagnoses were discussed in our patient: Crohn's disease, pyoderma gangrenosum, sarcoidosis, veneral diseases, ulcerated haemorrhoids, as well as leishmaniasis, deep mycoses and neoplasms.

In order to eradicate tuberculosis, it is important that the diagnosis be established early in the disease process. This patient's 2-year history of systemic symptoms and perianal ulcers leads us to believe that tuberculosis should be kept in mind in the differential diagnosis, especially in developing countries.

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Exacerbation of Pemphigus Foliaceus after Tetanus Vaccination Accompanied by Synthesis of Auto-antibodies Against Paraneoplastic Pemphigus Antigens

Karsten Korang^{1,3}, Reza Ghohestani², Thomas Krieg¹, Jouni Uitto² and Nicolas Hunzelmann^{*}

Department of Dermatology, University of Cologne, Joseph-Stelzmannstr. 9, DE-50924 Cologne, Germany and Department of Dermatology and Cutaneous Biology, Jefferson Medical College, Philadelphia, USA; Present address: Skin Research Institute, Hopital Saint Louis, Paris, France. *E-mail: nico.hunzelmann@uni-koeln.de Accepted August 6, 2002.

Sir,

Pemphigus folicaeus (PF) is an autoimmune disorder characterized clinically by superficial subcorneal blisters and autoantibodies against desmoglein 1 (Dsg1) (1). It can transform into another variant of the pemphigus group of autoimmune bullous skin diseases, i.e. pemphigus vulgaris (PV), in which patients develop antibodies against desmoglein 3 (Dsg3). In paraneoplastic pemphigus (PNP), which is described as a blistering disorder associated with neoplasms, autoantibodies against Dsg1 and Dsg3 have been described (2). Additionally, autoantibodies are developed against members of the plakin family including envoplakin, periplakin, desmoplakins I and II, plectin and bullous pemphigoid antigen 1 (BPAG1) (3).

We report the case of a 45-year-old woman with a history of PF, first diagnosed in 1994, who presented in January 1999 with exacerbation of the PF disease. Three months earlier she had received a tetanus vaccination. Skin lesions erupted after the first vaccination and the planned second vaccination was cancelled. Nevertheless, the skin lesions increased and topically applied steroid cream had no effect. On physical examination, the patient presented with erythematous scaly plaques on the whole integument, with an emphasis on the trunk, the legs and the face. Additionally, a few small