INTERNATIONAL JOURNAL OF MYCOBACTERIOLOGY 2 (2013) 230-232



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Case Report

Cutaneous leukocytoclastic vasculitis revealing multifocal tuberculosis

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ARTICLEINFO

Article history: Received 21 July 2013 Accepted 29 July 2013 Available online 17 August 2013

ABSTRACT

Cutaneous leukocytoclastic vasculitis (CLV) is an inflammatory vascular disorder rarely reported to be associated with tuberculosis. The following report describes the case of a young man with multifocal tuberculosis revealed by CLV. Diagnosis was confirmed by the presence of tuberculoid granuloma with caseous necrosis on pleural and perianal biopsy, and a rapid improvement in anti-tuberculous quadritherapy.

Although rarely seen, Mycobacterium tuberculosis should be considered as a potential cause of CLV.

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Introduction

Cutaneous leukocytoclastic vasculitis (CLV) is an inflammatory vascular disorder due to deposition of immune complexes in dermal vessels. It can have several causes, including drugs, malignancy, collagen vascular disease, and infection [1]. Among the infectious agents, bacteria are well recognized as causes of CLV, but Mycobacterium tuberculosis is rarely reported to be associated with CLV [2–4].

The following reports the case of a young man with multifocal tuberculosis whose initial presentation was that of a lower limb leukocytoclastic vasculitis. This case seems to be the first case reporting this association and discussing the possible mechanisms.

Observation

A 19-year-old man with a history of perianal swelling for 1 month was admitted to the dermatological department with a 1 week history of erythematous lesions of the lower limbs, fever, abdominal pain, and arthralgia of the knees, ankles and wrists with weight loss. He had no other personal or familial history and had not taken any medications recently. Upon physical examination, the finding was the presence on both legs of several palpable purpuric lesions that coalesced, resulting in vesicles (Fig. 1). Clinical examination also showed a 2 cm anal ulceration and a pleural left effusion.

The blood workup revealed that the blood count, serum creatinine, aspartate aminotransferase and alanine aminotransferase were normal. The erythrocyte sedimentation rate was 32 mm in the first hour, and the level of C-reactive protein was also elevated at 152 mg/l.

Serological markers for HIV, cytomegalovirus, Epstein-Barr virus, hepatitis B and C, and syphilis were all negative. Rheumatoid factor, antinuclear antibody, anti-dsDNA antibody, and anti-neutrophil cytoplasmic antibody (ANCA) were also negative. Complement level was normal.

A chest X-ray showed a cavern in the right superior lobe and a left pleurisy (Fig. 2). A sputum smear microscopy

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Fig. 1 - Purpuric lesions of the dorsum of the foot.

(Ziehl–Neelsen staining) was negative in three consecutive samples and skin testing with purified protein derivative (PPD) was positive (induration of 14 mm).

A skin biopsy of the purpuric lesions was carried out. The pathological skin analysis revealed leukocytoclastic vasculitis (neutrophilic vasculitis of the small superficial vessels) and immunofluorescence did not show the deposit of immunoglobulin or complement. A pleural and perianal biopsy showed tuberculoid granuloma with caseous necrosis. Polymerase chain reaction (PCR) determination of Mycobacterium tuberculosis lesions was not done. A rectoscopy and colonoscopy were normal. There were no signs of vasculitis present in the internal organs.

The patient was diagnosed with CLV associated with multifocal tuberculosis (pleural, pulmonary and anal). He was put under confinement to one's bed and anti-tuberculosis treatment was initiated with isoniazid, rifampin, pyrazinamide and ethambutol for 2 months followed by 7 months of rifampicin and isoniazid, with a good response and tolerance. The skin lesions showed gradual remission until complete resolution after 2 weeks of treatment, the anal lesion healed after 1 months and pleurisy after 4 weeks. During the 6-month course of treatment, there was no recurrence of purpura on the skin. A 2-year follow-up was recommended.



Fig. 2 – A chest X-ray, showing a cavern in the right superior lobe and a left pleurisy.

Discussion

CLV secondary to Mycobacterium tuberculosis is uncommon with less than 20 cases reported currently [2–8].

Three main forms of the combination of tuberculosis and vasculitis were described in literature: pulmonary tuberculosis/Henoch–Schönlein purpura; pulmonary tuberculosis/vasculitis secondary to rifampicin and pulmonary tuberculosis/cutaneous leukocytoclastic vasculitis [8]. In this third category, as in this case, CLV was described in 12 patients, aged between 13 and 61 years old, with a slight male predominance [4–6,9].

In those 12 cases of CLV reported, vasculitis was associated with pulmonary tuberculosis in 7 cases [4–6,9] and with tuberculous lymphadenitis in 4 cases [2,4], while the site of Mycobacterium tuberculosis infection was not found in 1 patient [3]. It is believed that this observation is the first to date describing the association between multifocal tuberculosis and CLV.

Clinically, CLV appears as palpable purpuric lesions on both lower legs which can progressively spread to both thighs or be accompanied by systemic symptoms as in this patient. Purpura can be the first symptom of the disease or it can be part of the overall clinical profile [6].

Histologically, the lesion is an angiocentric inflammatory process associated with leukocytoclasia (neutrophil fragmentation) and fibrinoid necrosis without the presence of Mycobacterium tuberculosis in the small vessel wall, which differentiates it from cutaneous tuberculosis, in which microorganisms are seen in biopsy samples [6].

Even if cutaneous leukocytoclastic vasculitis is rare, it is reported in the literature in young individuals with otherwise normal immunity, with chronic and untreated tuberculosis [8] as was the profile of this patient. The exact pathogenesis of CIV due to Mycobacterium tuberculosis remains unknown, however, several mechanisms have been proposed: direct invasion of vessel walls by tubercle bacilli; immunological reaction involving the deposition of immune complexes; intravascular release of mycobacteria followed by Arthus reaction and delayed type hypersensitivity response; or Rifampicin-dependent antibody and then immune complex formation [8]. In this case, it is believed that an intravascular release of mycobacteria from digestive, pulmonary tuberculous pleural locations, which are highly vascularized, participated to the direct invasion of vessel walls by mycobacterium tuberculosis.

Cutaneous leukocytoclastic vasculitis is treated by treating the underlying disease; as in this case, a symptomatic treatment of cutaneous lesions and anti-tuberculosis medication are always sufficient with no recurrence after therapy [4]. In hypersensitivity vasculitis caused by anti-tuberculosis therapy (for example, rifampicin), discontinuation of the medication and its replacement improve skin lesions [6]. Severe skin manifestations can be treated with a brief course of oral corticosteroids [1].

This report described a case of multifocal tuberculosis associated with CLV confirmed by histology and therapy. Although rarely seen, Mycobacterium tuberculosis should be considered as a potential cause of CLV.

Financial support

None.

Conflict of interest

None declared.

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