CASE REPORT

Erythema induratum of Bazin and Ponçet's arthropathy as epiphenomena of hepatic tuberculosis

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SUMMARY

A 42-year-old black woman presented with fever, polyarthritides, livedo reticularis, subcutaneous calf nodules and hepatomegaly. She had been diagnosed with depression 6 weeks prior. Blood analysis showed anaemia, elevated erythrocyte sedimentation rate and C reactive protein, elevated liver enzymes, and positive antinuclear and antiribonucleoprotein antibodies. Abdominal ultrasound revealed heterogeneous hepatomegaly with necrotic lymphadenopathy around the caeliac trunk and splenic hilum. We considered the following diagnoses: lymphoma, connective tissue disease, tuberculosis and sarcoidosis. Subcutaneous nodule histology was compatible with erythema induratum of Bazin, and liver biopsy evidenced granulomatous hepatitis. Although microbiological examinations were negative in tissue samples, a presumptive diagnosis of hepatic tuberculosis was admitted. Having excluded other causes, erythema of Bazin, livedo reticularis and polyarticular involvement (Ponçet's arthropathy) were accepted as immunological epiphenomena associated with tuberculosis. Empirical antituberculous treatment was started and after 3 weeks the patient improved substantially. This clinical response was a further confirmation of the diagnosis.

BACKGROUND

The protean clinical presentation of tuberculosis varies with the host's immunological status and the natural course of the disease (whether it is a primary infection with or without acute haematogenous spread or a late organ reactivation). Every organ and system can be affected in the disease's initial dissemination, becoming a potential reservoir for later reactivation. In acute systemic haematogenous tuberculosis (miliary tuberculosis), the liver and spleen are affected to the same extent as all the other organs, for example, the lungs (miliary pneumonia) and the eyes (choroidal tubercles). However, the liver and spleen can be the focus of secondary reactivation and localised disease. In this context, the presence of adjacent necrotic lymphadenopathy should raise the suspicion of local lymphatic spread. *Mycobacterium tuberculosis* identification is normally required for diagnostic confirmation. Cultural examination of an organic sample is the gold standard method for the diagnosis of tuberculosis. However, due to its higher sensitivity, nucleic acid amplification test (NAAT) for *M. tuberculosis* is an alternative method, receiving growing acceptance, when applied in the right

clinical context. When microbiological confirmation is not possible, presumptive diagnosis can be carried out in the presence of adequate clinical elements and concordant ancillary medical examinations, after excluding other aetiologies. In this setting, empirical treatment can be used as a diagnostic tool. Some tuberculosis clinical manifestations are not the result of direct virulence of the bacilli but a consequence of immunological distant epiphenomena. These include, among others, livedo reticularis, Poncet's arthropathy and erythema induratum of Bazin. The presence of these phenomena should be interpreted as additional clinical evidence favouring the diagnosis.

CASE PRESENTATION

A 42-year-old black woman from Guinea-Bissau, living in Portugal for the past 8 years, presented with depressed mood, emotional lability, insomnia and myalgias. Her husband had recently migrated to another country, leaving her alone with their two children. We considered that the symptoms could be due to a reactive depression and started mirtazapine 30 mg once a day, orally. Six weeks later, her mood seemed to have improved, but myalgias persisted. Also, the patient mentioned having fever (38–39°C) during the previous week. At this point, we observed the onset of inflammation of several joints—namely, wrists, elbows, knees and ankles—and the following skin findings: livedo reticularis on the thighs, and a nodular rash consisting of 1–2 cm tender erythematous nodules in the back of her lower limbs, extending from the calves to the heels. The physical examination also revealed tender hepatomegaly, and no other remarkable signs.

INVESTIGATIONS

Blood analysis showed haemoglobin 11.5 g/dL; leucocytes $11.40 \times 10^9/L$; platelets $315 \times 10^9/L$; erythrocyte sedimentation rate (ESR)=86 mm/h. C reactive protein (CRP) 96 mg/L (ref value <38 U/L); alkaline phosphatase 496 U/L (ref value 30–120 U/L); positive antinuclear antibodies 1/320 speckled pattern; positive anti-ribonucleoprotein antibodies; negative double stranded DNA, anti-Sm, anti-Ro, anti-La, anticyclic citrullinated peptide antibodies; negative rheumatoid factor; ACE 89 U/L (ref value: 16–85 U/L); negative anti-neutrophil cytoplasm antibodies; negative anti-smooth muscle, antiliver/kidney microsomes and anti-mitochondrial antibodies; and negative

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antiphospholipid antibodies. Serologies for hepatitis B and C virus (HBV, HCV), HIV, *Brucella* species, *Coxiella burnetii*, *Borrelia burgdorferi* and Syphilis were negative. The Interferon Gamma Release Assay (IGRA) was positive. Blood and urine cultures were negative.

Mycobacterial hepatic and lymph node tissue cultures were negative.

NAAT for *M. tuberculosis* on hepatic and lymph node tissues were negative.

Chest radiograph was normal.

Abdominal ultrasound revealed hepatomegaly with a heterogeneous echographic pattern. Normal anatomy of the gallbladder and biliary tree was seen. Lymphadenopathy with central necrosis in the hepatic and splenic hila, and around the caeliac trunk, was evident.

Chest and abdomen CT scan (figure 1) showed normal anatomy of the lung parenchyma and mediastinal structures, and an enlarged liver and a normal sized spleen both expressing heterogeneous contrast enhancement. Multiple instances of necrotic lymphadenopathy (1–2 cm of diameter) were located in the hepatic and splenic hila regions as well as in the lateroaortic and peri-caeliac trunk topography. Normal presentation of the pelvic cavity organs was noted.

Cutaneous nodule histopathology (figures 2 and 3) results showed lobular panniculitis with lymphohistiocytic infiltrates consisting of giant multinucleated cells, neutrophils, eosinophils and extensive lipophagic fat necrosis. There was also infiltration of the lobular vessel walls with lymphocytes and neutrophils. These histopathological findings were compatible with erythema induratum of Bazin.

Hepatic histopathology (figures 4–6) revealed multiple epithelioid granulomas with focal necrosis, and Langhan's giant cells scattered in the periportal spaces and acinar parenchyma. The histopathological diagnosis was granulomatous hepatitis, compatible with a tuberculous aetiology. No acid-fast bacilli or fungi were detected.

Lymph node histopathology showed four lymph nodes with maximum diameter of 2 cm, suggestive of reactive lymphadenitis, of possibly a viral or autoimmune aetiology. There was an absence of lymphoid neoplasia and metastatic tumour infiltration, and no evidence of tuberculous infection. One poorly developed granuloma was found.

Figure 1 Abdominal CT scan: contrast-enhanced hepatic heterogeneity; 1–2 cm multiple necrotic lymphadenopathy in the hepatic and splenic hila.

Figure 2 Cutaneous nodule biopsy (H&E stain): predominantly lobular panniculitis with lymphohistiocytic infiltrate consisting of giant multinucleated cells, neutrophils, eosinophils and extensive lipophagic fat necrosis. There is also infiltration of the lobular vessel walls with lymphocytes and neutrophils. These findings of lobular granulomatous panniculitis with neutrophilic vasculitis are consistent with erythema induratum of Bazin.

TREATMENT

After establishing the presumptive diagnosis of hepatic tuberculosis, empirical treatment was started: 6-month antituberculous treatment, consisting of 4-drug regimen in the first two months—isoniazid 300 mg per day, rifampin 600 mg, pyrazinamide 1500 mg per day and ethambutol 1200 mg per day—followed by 4 months of a 2-drug regimen with isoniazid and rifampin. Clinical improvement was achieved in the first 3 weeks of treatment.

DISCUSSION

The clinical condition of fever, tender hepatomegaly, calf subcutaneous nodules, arthritis and livedo reticularis, suggested a systemic disorder. We considered each sign separately to better evaluate their contribution to the diagnosis of a single nosological entity. The immunological pattern on blood analysis

Figure 3 Cutaneous nodule biopsy (H&E stain): predominantly lobular panniculitis with lymphohistiocytic infiltrate consisting of giant multinucleated cells, neutrophils, eosinophils and extensive lipophagic fat necrosis. There is also infiltration of the lobular vessel walls with lymphocytes and neutrophils. These findings of lobular granulomatous panniculitis with neutrophilic vasculitis are consistent with erythema induratum of Bazin.

Figure 4 Hepatic tissue biopsy: augmented portal space by granulomatous infiltration (periodic acid-Schiff stain).

evoked a connective tissue disease; however, many infectious diseases may also induce false-positive autoantibodies serologies.¹ At this point, our differential diagnosis included: sarcoidosis, lymphoproliferative disorder, tuberculosis, deep fungal disease such as histoplasmosis and autoimmune diseases such as lupus or mixed connective tissue disease. The abdominal CT scan (figure 1) showed heterogeneous liver enlargement and multiple instances of necrotic lymphadenopathy around the celiac trunk; this radiological pattern² is usually seen in association with tuberculosis,³ lymphoma,⁴ Whipple's disease,⁵ metastatic tumours as germ cell tumours and the so called 'mesenteric lymph node cavitation syndrome' of celiac disease.⁶⁻⁷ An abdominal lymph node excisional biopsy was performed in order to exclude malignancy. Considering the radiological necrotic pattern of the lymph nodes, we expected more conclusive histopathological changes in the biopsy sample. Thus, we hypothesised that the surgically removed lymph nodes were not those most affected by the disease, and therefore they were yielding mostly reactive changes. Individual subcutaneous nodules resembled erythema nodosum, which can be related to primary tuberculosis,⁸⁻⁹ sarcoidosis and other diseases.¹⁰⁻¹¹ However, the distal calf location was more consistent with the nodular vasculitis known as erythema induratum of Bazin. Lupus profundus (lupus panniculitis) could also be considered in this case.¹²⁻¹⁴ Cutaneous biopsy, mandatory

Figure 5 Hepatic tissue biopsy: hepatic granulomas evidenced by Chromotrope-Aniline-Blue (CAB) staining.

Figure 6 Hepatic tissue biopsy: a detail of hepatic granuloma (CAB staining).

to distinguish between these entities, suggested erythema induratum of Bazin (figure 2 and 3). Erythema induratum was first identified in 1861, by Bazin, as a nodular eruption that occurred on the lower legs (posterior face) of young women with tuberculosis; in 1945, Montgomery *et al* described the histological hallmarks of this nodular panniculitis, while fully acknowledging its association with tuberculosis.¹⁵ Viable organisms in these skin lesions have not been demonstrated. A hypersensitivity reaction against *M. tuberculosis* antigen dermal deposition is the implicated pathophysiological mechanism, as *M. tuberculosis* DNA fragments are occasionally identified by PCR.¹⁶⁻¹⁷ Liver biopsy was performed for histopathological diagnosis, mycobacterial culture and *M. tuberculosis* NAAT. Mycobacteriological isolation and NAAT were negative in the liver sample, but histology revealed granulomatous hepatitis (figures 4–6). Granulomas in the liver are associated with a myriad of disorders, most commonly sarcoidosis, tuberculosis, primary biliary cirrhosis and drug reactions.¹⁸⁻¹⁹ Deep mycosis should also be included in the differential diagnosis of fever of unknown origin and granulomatous liver disease, according to the epidemiological context. Histoplasmosis can be found worldwide, but is particularly seen in North and Central America, being the most common endemic mycosis in the USA. Liver involvement as a part of disseminated histoplasmosis, which usually originates in the lung, is well known, however, extrapulmonary hepatic histoplasmosis as a primary manifestation is extremely rare.²⁰ Histopathological recovery of microorganisms is usually performed by periodic-acid Schiff or H&E stain, which showed no microorganisms in our liver biopsy.²¹ Paracoccidioidomycosis and blastomycosis are systemic endemic mycotic diseases that have a well-known geographic distribution limited to Central and South America, and North America, respectively. They typically cause asymptomatic pulmonary infection that may evolve into one of two patterns: the chronic form, which represents reactivation of the primary infection, and the acute/subacute form, which can present with fever, lymphadenopathy and hepatosplenomegaly.²²⁻²³ Since Portugal is not an endemic country to these diseases, these diagnoses were not overemphasised; however, they should be kept in mind by clinicians practising in endemic countries. According to our pathologist's report, the type and distribution of granulomas in the liver was highly suggestive of tuberculosis, and no fungal organisms were identified by the appropriate stains. This finding, together with the necrotic pattern of the intra-abdominal lymph nodes, positive IGRA and the

dermatopathological diagnosis of erythema induratum of Bazin, prompted us to assume the diagnosis of tuberculosis. There is no consensual classification of hepatic tuberculosis in the literature; Levine classifies it as miliary tuberculosis, pulmonary tuberculosis with hepatic involvement, primary liver tuberculosis, focal tuberculoma or abscess, or tuberculous cholangitis.²⁴ However, Reed divided it into three forms: tuberculosis of the liver associated with generalised acute miliary tuberculosis, tuberculoma or abscess of the liver and primary miliary tuberculosis of the liver,²⁵ which seems to be the most accurate classification of our patient's condition. Although referred to as 'primary', this latter condition does not represent an acute dissemination of a tuberculosis primoinfection. Instead, it means that the liver is the primary recognised affected organ, with no demonstrable lesions elsewhere. Indeed, this form of tuberculosis represents a secondary dissemination from an unidentified source. When it is not possible to demonstrate pulmonary lesions (even sequelae), some authors suggest that hepatic dissemination can result from an intestinal primary non-identified lesion.²⁶ In this case, periportal distribution of hepatic granulomas can be a clue to support portal venous spreading.^{27–28} Some other infectious diseases that can present with arthritis, fever and hepatic inflammation were excluded by serology (Q fever,²⁹ brucellosis,³⁰ viral hepatitis,^{31–32} HIV,³³ Lyme disease³⁴ and syphilis³⁵). Livedo reticularis is a dermal epiphenomenon associated with all the diseases we considered, including tuberculosis.³⁶ Tuberculous joint infection is usually a monoarthritis.^{37–38} Polyarthritis occurring in the setting of tuberculosis infection is known as Ponçet's arthropathy. It is a rare form of inflammatory reactive arthritis associated with immunological dysregulation, and not a consequence of direct mycobacterial joint infection.^{39–41} It has been hypothesised that after infection, due to systemic immunisation, sensitised CD4 cells and bacterial antigens migrate to the joints, causing arthritis.⁴² Assuming the presumptive diagnosis of hepatic tuberculosis with regional lymph node and possible splenic spread, antituberculous empirical treatment was started, bearing in mind that a positive clinical response would be a confirmation of this diagnosis.⁴³ In fact, a favourable clinical outcome was observed: fever, arthritis and cutaneous lesions resolved within 3 weeks, alongside progressive normalisation of the liver enzymes, ESR and CRP. Mirtazapine was suspended. Three months later, the patient returned to work and to her normal life, fully recovered. As a general rule, the principles used for the treatment of pulmonary tuberculosis also apply to extrapulmonary forms of the disease: a 6-month treatment regimen is recommended—unless the organisms are known or strongly suspected to be resistant to first-line drugs, and also may be warranted for children and immunocompromised hosts. It is known that the current standard four-drug treatment regimen of first-line drugs achieves cure rates of more than 95% of drug-sensitive active tuberculosis cases in 6 months. Since our patient achieved significant clinical improvement early in the course of treatment, we opted for the standard 6-month treatment regimen.⁴⁴ Concerning the initial depression diagnosis, there are reports of clinically apparent depression occurring before a subjacent organic disease manifestation.⁴⁵ That seems to be the case in our patient: her mood and psychological health improved during the antituberculous treatment, even after interrupting antidepressive medication. As the patient became better with the instituted treatment, the possibility of tuberculosis of the central nervous system became low, thus discouraging us from performing a diagnostic lumbar puncture.

Learning points

- ▶ Isolated hepatic tuberculosis represents a local reactivation, following dissemination from a primary source, most commonly the lungs or intestinal tract.
- ▶ Diagnostic confirmation of tuberculosis by microbiological isolation or DNA detection in tissue samples is not always possible, and lack of this should not delay treatment in the appropriate clinical context.
- ▶ Presumptive diagnosis of tuberculosis is acceptable in the setting of a high clinical index of suspicion supported by radiological, laboratorial and histopathological elements.
- ▶ Some epiphenomena classically associated with tuberculosis, such as Ponçet's arthropathy, erythema induratum of Bazin and livedo reticularis, can provide clinical guidance.
- ▶ Favourable clinical outcome under antituberculous therapy is the final confirmatory evidence in establishing the diagnosis.

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