First Report of Splenic Abscesses Due to *Coccidioidomycosis*

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Abstract

Involvement of the spleen by *Coccidioides* is uncommon. It is usually associated only with disseminated infection and manifests as microscopic granulomas in the spleen. We report an immunosuppressed dermatomyositis patient who presented with splenic abscesses demonstrated on a computed tomography (CT) scan which was presumed to be bacterial in origin. At splenectomy the spleen was found to be filled with aggregates of spherules due to *Coccidioides*. Finding large splenic abscesses on CT scan due to *Coccidioides* has not been previously described. We offer a hypothesis for why the abscesses occurred in this unique patient.

Introduction

Involvement of the spleen by coccidioidomycosis is usually associated with disseminated disease, however the development of splenic abscesses has not been reported. Splenic involvement by coccidioidomycosis is usually manifest as microscopic miliary splenic granulomas which have been demonstrated at autopsy in patients with disseminated infection (1,2). We report an immunocompromised dermatomyositis patient who was found to have splenic abscesses due to *Coccidioides* spherules which were diagnosed at splenectomy.

Case Presentation

A 33-year-old Hispanic man with dermatomyositis for five years and a history of disseminated coccidioidomycosis for two years, presented to the emergency room because of left upper quadrant abdominal pain, fever and chills. Treatment of his dermatomyositis was ongoing over the previous five years and included prednisone, azathioprine and courses of intravenous immunoglobulin (IVIG) at doses of 2 g/kg (3). Treatments of his coccidioidomycosis over the previous two years included intravenous liposomal amphotericin B followed by oral fluconazole. The patient would periodically be non-compliant about taking the fluconazole and then experience relapses of his coccidioidomycosis which required additional courses of intravenous liposomal amphotericin B.

Physical Examination and Course: Admission vital signs - temperature 38.4⁰ C; blood pressure 147/81 mmHg; heart rate 106 bpm; respiratory rate 18 breaths/minute and pulse oximetry 90% on room air. There was pigmentation of his face consistent with dermatomyositis, tenderness in the left upper quadrant and significant weakness of all extremities. He was bedridden and could barely move his arms and legs against

gravity. His medications on admission were fluconazole and prednisone. An admission CT scan of the abdomen was performed because of the left upper quadrant tenderness and revealed multiple splenic abscesses (Figure 1).

Figure 1. CT scan of abdomen demonstrating splenic abscesses (arrow).

An admission urine culture grew $>10^5$ colony forming *Klebsiella pneumoniae* which was noted on day two of hospitalization. Blood cultures were negative. It was initially believed that the splenic abscesses were due to a *Klebsiella* infection because of the admitting urine culture results. Prednisone was stopped on admission and the oral fluconazole continued. Piperacillin-tazobactam was started empirically on admission. In addition, IVIG was given for a presumed dermatomyositis exacerbation. On hospital day four his abdominal pain and fevers had not improved. To avoid a splenectomy, a splenic biopsy was performed to determine the cause of the splenic abnormalities. The biopsy was consistent with a *Coccidioides* infection. A laparoscopic splenectomy was then performed on hospital day seven.

The pathology on the removed spleen showed multiple necrotizing granulomatous foci containing numerous aggregated *Coccidioides* spherules (Figure 2).

Figure 2. Pathology of splenic abscesses demonstrating aggregated *Coccidioides* spherules.

Post-operatively, fluconazole was empirically replaced by voriconazole (4) and the patient was restarted on prednisone for his dermatomyositis. The fever and chills eventually resolved and he was discharged. At four months follow-up he had returned to his usual state and was encouraged to not stop taking the voriconazole.

Discussion

This patient illustrates an unusual complication of disseminated coccidioidomycosis. Prior to the advent of CT scans, splenic granulomas were described mainly at autopsy in patients with disseminated infection. Splenic involvement at autopsy was described as granulomas due to the invasion of the *Coccidioides* into the spleen from the blood stream. Usually there was granuloma formation described as microscopic military nodules. Reports of gross *Coccidioides* abscesses in the spleen have not been described.

We considered the potential reasons for the development of splenic abscesses in this unique patient. His dermatomyositis was present for about five years and the coccidioidomycosis, two years. He had received repeated doses of IVIG for flares of his dermatomyositis prior to, and after, his *Coccidioides* infection. Investigating his past medical history revealed that he would develop a febrile illness when off fluconazole - usually due to non-compliance. The clinical presentation was consistent with either a relapse of his *Coccidioides* infection, an exacerbation of his dermatomyositis, or both. The febrile episodes would cause him to be admitted to the hospital, often into the intensive care unit, and then he would receive more IVIG for his dermatomyositis, as well as antifungals. It is known that fungemia occurs in immunosuppressed patients who have significant coccidioidomycosis (5). The fact that he had a large *Coccidioides* burden in his spleen suggests he likely experienced episodes of fungemia, presumably associated with his poor antifungal compliance.

Our hypothesis for why the abscesses formed in the spleen of this patient is illustrated in Figure 3.

Figure 3. Hypothesis of *Coccidioides* abscess formation in the spleen.

We theorized that *Coccidioides* endospores in the blood stream became coated with the gamma globulins when he received the IVIG given for his dermatomyositis (6). The opsonization of the organisms by the IVIG presumably facilitated the spleen to take up viable endospores into the spleen and reticuloendothelial system (Figure 3, part 3). This resulted in the localization of the organisms promoting the formation of an abscess within the spleen (Figure 3, part 4). We suggest that these unusual circumstances of fungemia and IVIG were responsible for facilitating the appearance of abscesses in this patient's spleen.

We believe true splenic abscesses are uncommon with disseminated coccidioidomycosis. The unusual circumstances of this patient's relapsing *Coccidioides* infection with fungemia (due to poor compliance with antifungals) and the repeated IVIG treatments for his dermatomyositis, combined to provide a reasonable explanation for why splenic abscesses occurred in this patient.

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