Case Report

Intra-abdominal abscess of tuberculosis associated thrombocytopenia in a patient with systemic lupus erythematosus

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Tuberculosis is a complex, potentially life-threatening infectious disease. Mycobacterium tuberculosis infection, associated with intra-abdominal abscess formation was not common in systemic lupus erythematosus (SLE) patients. Multiple hematological abnormalities had been reported in tuberculosis infection. However, tuberculosis infection associated with thrombocytopenia was quite rare in patients with SLE. We reported a 28-year-old woman with SLE who developed intra-abdominal abscess of Mycobacterium tuberculosis infection. Thrombocytopenia occurred during active tuberculosis infection and rapidly recovered after effective anti-tuberculosis therapy within two weeks. The intra-abdominal abscess completely resolved after one month of anti-tuberculosis therapy.

Key words: Tuberculosis, thrombocytopenia, systemic lupus erythematosus

Introduction

In past century, tuberculosis infection is potentially life-threatening infectious disease until effective anti-tuberculosis combination therapy in late 1970s. However more reports of new Mycobacterium tuberculosis infection was announced in developed country in recent 10 years. Some new cases are reported in patients with immunosuppressive status such as acquired immunodeficiency syndrome (AIDS). Mycobacterium tuberculosis infection with intra-abdominal abscess was rarely reported in immunosuppressive patients with systemic lupus erythematosus (SLE). Tuberculosis infection associated with thrombocytopenia was even less reported in patients with SLE. Herein, we reported a patient with SLE who developed intra-abdominal abscess of Mycobacterium tuberculosis infection with thrombocytopenia during active stage. Quick recovery of thrombocytopenia was observed after effective anti-tuberculosis therapy.

Case Report

A 28-year-old woman was diagnosed to be SLE, based on initial presentation of malar rash, photosensitivity, oral ulcer, polyarthritis, fever and proteinuria (daily urine protein 2.0 gm), positive antinuclear antibody (ANA) and positive anti-double-stranded DNA (anti-dsDNA) at the age of 22. Pathology of renal biopsy revealed lupus nephritis WHO class IIb. She was treated with oral prednisolone (initial dose 0.6 mg/kg/d). Unfortunately, bizarre behavior, self-crying, self-laughing and auditory hallucination were noted but without episode of seizure. Central nervous system involvement with psychiatric manifestation of SLE was impressed. Methylprednisolone and cyclophosphamide pulse therapy (six times monthly) were administrated.
However, fluctuation of psychological condition (including delusion, auditory hallucination and self-laughing) persisted in spite of oral prednisolone 30 mg daily and mycophenolate mofetil (MMF) 1500 mg/day in divided doses. Nevertheless, serological activity of lupus was in quiescent status. Negative test for antiphospholipid antibody, and anti-platelet antibody or thrombocytopenia were noted in the past.

In January 2001, she suffered from intermittent high spiking fever up to 40-41°C, nausea and abdominal fullness for one week. Physical examination disclosed tenderness over right upper quadrant of abdomen without rebounding pain. There were no Murphy’s sign and Rovig’s sign, which suggested surgical conditions such as cholecystitis or appendicitis. Abdominal vasculitis or intra-abdominal infection was suspected initially. Computer tomography scan of abdomen demonstrated peri-pancreatic head abscess formation (Fig. 1). Three combined antibiotics therapy (including cefazolin, amikacin and metronidazole) was administrated immediately. Laboratory data showed normal white blood count 9400/cumm, normal platelet count 159000/cumm, low C3 at 63.0 mg/dL (normal value 90-180 mg/dL), normal C4 at 30.6 mg/dL (normal value 10-40 mg/dL), high C-reactive protein 6.28 mg/dL (normal value <0.5 mg/dL), negative anti-platelet antibodies (IgM/IgG/IgA), negative antiphospholipid profiles, including mixing activated partial thromboplastin time, lupus anticoagulant, anti-cardiolipin antibodies, and antibody to beta-2-glycoprotein I. Thrombocytopenia (62000/cumm) occurred seven days after hospitalization. Disseminated intravascular coagulation (DIC) was ruled out with normal levels of D-dimers, prothrombin time, and fibrinogen. The plasma level of activated protein C, under activation by the venom of copperhead snake [1] was abnormally low (21%, normal value 70-40%). Percutaneous drainage was performed for peri-pancreatic abscess formation. Smears of aspirate from peri-pancreatic abscess showed positive acid-fast stain (+++). Culture of peri-pancreatic aspirate yielded Mycobacterium tuberculosis. Antibiotics was switched to four-combined anti-tuberculosis therapy, which included ethambutol, isoniazid, rifampin and pyrazinamide. She responded to four-combined anti-tuberculosis therapy rapidly with fever subsided one week later. However, short of breath and exertional dyspnea developed that miliary tuberculosis was shown in chest X-ray film. Profound jaundice, increased plasma level of total bilirubin (4.5 mg/dL, normal value <2.0 mg/dL) and direct bilirubin (2.7 mg/dL, normal value <1.2 mg/dL), increased aspartate aminotransferase (107 U/L, normal value <40 U/L). Rifampin was discontinued and instead streptomycin intra-muscular injection was added under suspicion of rifampin associated hepatitis. Fever subsided again one more week later. Complete resolution of peri-pancreatic abscess was shown by repeated CT scan of abdomen one month later. Serum level of C-reactive protein decreased to 1.23 mg/dL and platelet count increased to 153000/cumm at the same time of peri-pancreatic abscess resolution by successful four-combined anti-tuberculosis therapy. The kinetic changes of platelet counts and C-reactive protein before and after anti-tuberculosis therapy were illustrated in Fig. 2.

![Figure 1. Computed tomography of abdomen. Arrow indicates abscess formation located at peri-pancreatic head.](image1)

![Figure 2. Kinetic changes of platelet counts (■) and C-reactive protein (▲) before and after anti-tuberculosis therapy.](image2)
Abdominal Abscess of Tuberculosis In SLE

Discussion

Mycobacterium tuberculosis infection with intra-abdominal abscess was not common in SLE patients. Normochromic and normocytic anemia were the most commonly reported hematological abnormalities during infection of tuberculosis [2]. However, thrombocytopenia associated with tuberculosis infection was unusual. Hemophagocytic syndrome was rarely reported in SLE patients except association with disseminated tuberculosis infection. Browett et al. [3] reported a 43 year-old man with progressive neutropenia and thrombocytopenia with positive anti-platelet antibodies during disseminated infection of pulmonary tuberculosis. Anti-tuberculosis therapy, hydrocortisone and intra-venous immunoglobulin were given for the treatment of hemophagocytic syndrome caused by mycobacterial infection. DIC was also rare in association with cavitary tuberculosis infection [4]. Eight patients in thirteen patients with DIC were reported association with acute respiratory distress syndrome with high mortality. Possible mechanisms included release of procoagulant substances by tuberculosis or contact of blood with an injured or abnormal surface leading to generation of procoagulant active substances within blood. Fujita et al. [5] reported a 43 year-old man with pulmonary tuberculosis developed DIC due to the interactions between lipopolysaccarides of mycobacterium and thromboplastin derived from tissue necrosis. Majed at al. [6] reported 9 of 846 patients with active tuberculosis presented immune thrombocytopenic purpura (ITP). The platelet count returned to normal value 2-6 weeks after oral prednisolone and anti-tuberculosis drugs. Another case report showed ITP complicating by pulmonary tuberculosis [7]. Attachment of membrane IgG and IgM on patient's platelets was detected by direct immunofluorescence test. Positive IgG anti-platelet antibody on platelet surface but no circulation anti-platelet was found [8]. Our patient did not have episodes of thrombocytopenia, or positive anti-platelet antibodies, positive antiphospholipid antibodies before the tuberculosis infection. In our patient, thrombocytopenia occurred during tuberculosis infection with intra-abdominal abscess formation and rapid recovery of platelet counts after successful treatment with anti-tuberculosis agents. This result suggests that tuberculosis infection may cause thrombocytopenia during tuberculosis infection.

SLE patients may also develop opportunistic infections, especially when treated with immunosuppressive agents. As a high-risk population, identification and treatment of chronic infections such as tuberculosis are important prior to the institution of immunosuppressant to prevent reactivation or exacerbation of the tuberculosis infection. It is important to distinguish a lupus disease flare-up from an acute infection. Judicious use of corticosteroids and cytotoxic drugs is critical in limiting infectious complications. The risk factors associated with susceptibility to infectious disease include severe flare up, active renal disease, treatment with moderate or high doses of corticosteroids and/or immunosuppressive agents. Genetic factors (including complement deficiencies, mannose-binding lectin, Fc-gamma III, granulocyte macrophage colony-stimulating factor (GM-CSF), osteopontin) may predispose certain SLE patients susceptible to infections.

In conclusion, our patient with peri-pancreatic abscess preceded by tuberculosis infection may develop thrombocytopenia. Mycobacterium tuberculosis infection not only causes disease flare-up but thrombocytopenia in patients with SLE.

References

發生於紅斑性狼瘡病人的結核菌性膿瘍併發血小板低下：一臨床病例報告

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結核菌感染是一個複雜且具威脅性的感染性疾病。結核分枝桿菌感染，特別是合併腹腔內膿瘍在全身性紅斑性狼瘡病患身上並不多見。過去文獻曾報告過結核菌感染具有多種血液系統異常表現。然而結核菌感染合併血小板低下甚少被報告發生於全身性紅斑性狼瘡病患。我們報告一位28歲全身性紅斑性狼瘡的女性病患因結核菌感染合併腹腔膿瘍而發生血小板低下。在治療結核菌感染後，血小板數迅速回昇而且腹腔內膿瘍完全消失。

關鍵詞：結核菌、血小板低下、紅斑性狼瘡