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Short Communication

A Case of Splenic Abscess during Treatment of Interstitial Pneumonia

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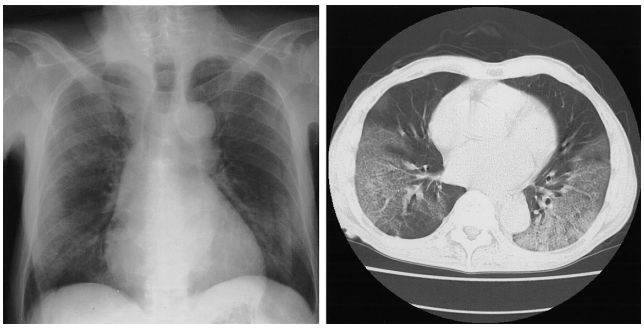
SUMMARY: We experienced a case of a 76-year-old man who developed a splenic abscess while undergoing treatment for interstitial pneumonia. Splenic abscess-like abnormal intensities were accidentally found by the chest computed-tomography (CT) examinations 3 weeks after the initiation of corticosteroids and immunosuppressive treatment for interstitial pneumonia. An ultrasonography-guided percutaneous aspiration test resulted in the isolation of methicillin-resistant *Staphylococcus aureus* (MRSA). Since colonized MRSA had been detected intermittently from sputum after admission and the patient risked bloodstream infection from an indwelling central venous catheter and intubation, we suspected that the organism colonized in the airway had spread into the bloodstream via these devices. Although CT-guided percutaneous drainage followed by postoperative antibiotic therapy are normally required for the treatment of splenic abscess, the patient was successfully treated by the administration of vancomycin without drainage.

Splenic abscess caused by methicillin-resistant *Staphylococcus aureus* (MRSA) is relatively rare and is sometimes diagnosed by autopsy. However, due to the increasing number of immunocompromised patients, splenic abscess cases are becoming a clinical concern (1). We here describe a case of splenic abscess caused by MRSA during immunosuppressive therapy for interstitial pneumonia.

A 76-year-old male was admitted to Izumikawa Hospital because of a 2-week history of low-grade fever with ground glass shadows on chest X-ray films on January 9, 2005. The initial treatment in the outpatient clinic performed prior to inpatient admission, oral levofloxacin followed by intravenous latamoxef, had failed. The patient had a fever but no respiratory symptoms such as cough, sputum, or dyspnea on admission. The patient's vital signs at admission were as follows: body temperature, 37.6°C; heart rate, 84 beats/min and regular rhythm; respiratory rate, 14 breaths/min; and blood pressure, 112/72 mmHg. On physical examination, a fine crackle in the bilateral middle to lower back side was recognized by auscultation. Dynamic aphemia and left semi-numbness due to cerebral infarction caused by chronic atrial fibrillation in 1998 were also observed. Clinical signs of lymphadenopathy, hepatosplenomegaly, and pretibial edema were not observed. Chest X-ray films showed cardiomegaly and interstitial shadows in both lower lung fields (Fig. 1A), and computed-tomography (CT) scan images showed ground glass opacity at the back side of both lower lung fields (Fig. 1B). Laboratory findings on admission were as follows: white blood cells (WBC) count, $8.4 \times 10^3/\mu L$ with a shift to the left (neutrophils: 83.3%); C-reactive protein (CRP), 21.5 mg/dL; erythrocyte sedimentation rate, 105 mm/h; and sodium, 129 mEq/L. All other results were within normal limits. Arterial blood gas (ABG) analysis showed respiratory alkalosis (pH 7.477), hypoxia, and hypocapnia (PaO_2 and $PaCO_2$ were 67.7 and 26.2 Torr at room air, respectively). A microbiological test revealed no causative bacteria but colonized MRSA on the upper respiratory tract on day 11. Rapid antigen tests for *Legionella pneumophilla* and influenza virus were negative. Serum antibody tests for *Mycoplasma pneumoniae* and *Chlamydophila pneumoniae* were also negative. Although there were no positive data allowing us to determine causative pathogens, pneumonia caused by an atypical pathogen was not ruled out, and intravenous ciprofloxacin (CPFX) was administrated on admission. Since the hypoxia progressed rapidly and dyspnea worsened, 1.0 g/day of methyl-predonisolone (mPSL) was added to the CPFX on day 3. In spite of intensive administration of CPFX and mPSL, there was no trend toward improvement by day 7, so the patient was intubated and 150 mg/day of cyclosporine A was added to the treatment regimen. After the administration of cyclosporine A, the patient's hypoxia and the ground glass opacity of chest CT improved slowly, but a low-grade fever remained even after the administration of several antibiotics, including meropenem plus clindamycin, and vancomycin (VCM) plus ceftazidime. On day 19, splenic abscess-like abnormal intensities were accidentally found during a chest CT examination, which was initially performed as a follow-up exam (Fig. 2A). Cyclosporin A was discontinued, and imipenem/cirastacin (IPM/CS) followed by cefzoplan (CZOP) was



(A) Posterior-anterior (B) Computed tomography

Fig. 1. Chest radiography images on admission.

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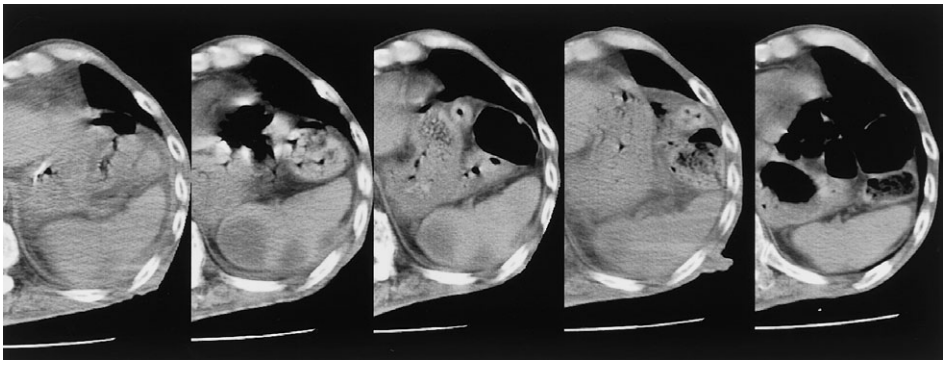
newly administrated to the patient. Although there were frequent attempts made to detect the causative microorganism from the patient's blood, no microorganism was isolated from blood specimens. The follow-up abdominal CT on day 49 revealed low attenuation areas that became very clear and increased in size (diameter was 3 cm at max) (Fig. 2B). Since the patient's low-grade fever and CRP level were not improved, we performed ultrasonography-guided percutaneous aspiration on day 49. A total of 5 ml of a milky-white puslike secretion was pulled out. Gram staining of the pus revealed Gram-positive cocci. Since colonized MRSA had been isolated from the patient's sputum intermittently after admission and there was a risk of infection because of the central venous catheter and intubation, we suspected that the bloodstream had been infected with MRSA via these devices. Because various beta-lactams administrated prior to aspiration were not effective, we started VCM 1.0 g/day intravenously to target MRSA infection. A couple of days after the administration of VCM, the patient's fever began to decrease and his CRP level was becoming normal. CT revealed that the splenic abscess decreased in size after 12 days, and after 23 days of administration of VCM (Fig. 2C and 2D), we discontinued it on March 22 (day 75). During a total of 60 days follow-up with free antimicrobial agents, we observed no relapse of fever, positive inflammatory signs, or abscesslike masses on CT (Fig. 2E). Therefore, the patient was discharged from our hospital on April 27. *S. aureus* was isolated from pus by a cultural test. The drug susceptibility test of this strain showed the same result as that of MRSA isolated from the respiratory tract as a colonization on day 11. These two strains were resistant to all drugs including minocycline, IPM/CS, and other beta-lactams and aminoglycosides except glycopeptides. Splenic abscesses are very rare; the incidence has been reported to be only 0.26 to 0.67% in various autopsy series (2,3). However, the increase of immunocompromised hosts due to intensive chemotherapy, organ transplantation, and HIV infection has led to an increase in the number of splenic abscess cases, and it is becoming a major clinical concern (1). According to several reports (1,4,5), *Staphylococcus* spp., *Salmonella*, and *Eschericia coli* are most frequently isolated bacteria from splenic abscesses, and other *Streptococcus* spp., *Klebsiella* spp., *Pseudomonas* spp., *Enterococcus* spp., *Mycobacterium* spp., and yeasts can also be found. Other lactobacillus, *Brucella* spp., and anaerobic bacteria have also been isolated from splenic abscesses (5,6). Fever is observed in more than 90% of cases. Upper abdominal pain, chilling, left shoulder pain, and general malaise are also reported as major symptoms of splenic abscess (1,4,5,7,8). A total of 50 to 60% of splenic abscess cases are caused by systemic infection or abdominal infection (1,4,5). However, other etiologies such as thrombosis, traumatic injuries, and immnuodeficient status are also reported as causes. The delay of diagnosing splenic abscess leads to a poor outcome, early diagnosis is extensively important (9).

Because of the advanced technology and development of CT and magnetic resonance imaging (MRI), the rate of diagnosing splenic abscess has reached between 90 to 100%, recently (9,10). Many investigators recommend CT-guided percutaneuous catheter drainage for the initial treatment of a splenic abscess (11-15). Liu et al. noted that the administration of antibiotics without drainage could be considered for splenic abscesses smaller than 4 cm (7). Splenectomy is indicated under certain conditions such as, (i) drainage is not effective, (ii) insufficient drainage due to debris of blood, and (iii) multiple abscesses.

In our case, the intensive administration of immunosuppressants such as corticosteroids and cyclosporine A for interstitial pneumonia might have induced the splenic abscess. Although a total of five attempts to isolate the causative microorganisms from the bloodstream were not successful, the MRSA was isolated from pus. Ooi and Leong reported that the positive rate of blood culture is 48.2% and that of spleen pus is 72.9% in splenic abscess cases (1). MRSA might have been selected by the intensive use of various antibiotics before the first isolation on day 11, and the devices such as the central venous catheter and intubation might have provided the site for the bloodstream infection. Since there was no outbreak or increased tendency toward MRSA infection in the ward at that time, the possibility that MRSA transferred and spread via doctors, nurses, or medical devices was faint but not completely denied. As noted previously, CT- or ultrasonography-guided percutaneous drainage is recommended for the treatment of splenic abscess. Although there were multiple abscesses in the spleen in our case, we administrated VCM without leaving a drainage catheter, and clinical signs such as a fever or signs of inflammation were diminished even 30 days after antibiotics were discontinued. We speculated that drainage was not required, since the abscesses were relatively small.

The etiology of this patient's interstitial pneumonia was not determined, since we did not perform a lung biopsy for definite diagnosis. Because of the rapid response to corticosteroid and cyclosporine A, allergic drug induced-pneumonia could be raised as one of the differential diagnoses. Although further examination to detect responsible agents was not performed, levofloxacin, latamoxef, warfarin, and ticlopidine



(A) (B) (C) (D) (E)

Fig. 2. Serial computerized tomography findings of the spleen.

hydrochloride, all of which were administrated prior to admission, are the probable agents for interstitial pneumonia. Collagen diseases were also considered as one of the etiologies; however, there were no positive antibody tests or symptoms indicating collagen diseases (data not shown).

In summary, we have presented a rare case of splenic abscess by MRSA during the administration of immunosuppressant therapy for interstitial pneumonia. The diagnosis of splenic abscess is relatively easy with advanced CT or MRI. Although the drainage of pus is often required as part of the treatment, administration of VCM without drainage was effective in our case.

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