Co-infection of *Pneumocystis jirovecii* and *Aspergillus* in a patient with idiopathic thrombocytopenic purpura

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To the Editor: *Pneumocystis jirovecii* pneumonia (PCP) is a life-threatening opportunistic infection that commonly occurs in patients with human immunodeficiency virus (HIV) infection. PCP can occur in patients without HIV, including those who have undergone organ transplantation, chemotherapy, and/or high-dose corticosteroid therapy. Invasive aspergillosis (IA) is also an opportunistic infection in immunocompromized patients, including those undergoing chemotherapy, steroid treatment, transplantation, and individuals with advanced HIV infection.

PCP and IA co-infection in HIV patients has been reported. However, only nine cases co-infected with PCP and IA co-infection in HIV patients have been reported. Herein, we report a fatal case of PCP and IA co-infection in a patient undergoing high-dose corticosteroid therapy for idiopathic thromocytopenic purpura.

A 75-year-old woman visited our emergency room (ER) after developing dyspnea over a week. One month earlier, she was diagnosed with idiopathic thrombocytopenic purpura and took corticosteroids (prednisolone 60 mg/day and tapered 10 mg/day). On arrival, her vital signs were as follows: blood pressure 156/122 mmHg; heart rate 180 beats/min (irregular); respiratory rate 44 breaths/min; oxygen (O2) saturation 56% (on room air); body temperature 37.7°C. Laboratory results revealed severe inflammation as reflected by increased level of high-sensitivity C-reactive protein (24.29 mg/dL) and white blood cell count (15,340/μL with 89.0% neutrophils). Tests for influenza antigen, respiratory virus, and bacterial polymerase chain reaction (PCR) (16 and 5 types, respectively) were negative. Tests for *P. jirovecii* (SEE AMP; Seegene, Seoul, Republic of Korea) and aspergillosis antigen (V Max Molecular devices, San Jose, CA, USA) were performed during ER stay. Initial chest radiography revealed diffuse ground-glass opacities (GGOs) and infiltration in both lungs [Figure 1A]. Chest computed tomography (CT) revealed diffuse geographic consolidations and GGOs in both lungs as well as two cavitary lesions in both lower lungs [Figure 1B].

She was admitted to the intensive care unit and empirical treatment for community-acquired pneumonia was initiated with intravenous (IV) piperacillin/tazobactam 4.5 g every 8 h and azithromycin 500 mg/day. Considering the possible PCP infection, IV sulfamethoxazole/trimethoprim was administered with 320 mg trimethoprim every 6 h and methylprednisolone 60 mg/day.

On hospital day 3, endotracheal intubation and mechanical ventilator support were started. Antibiotics were switched to meropenem and levofloxacin. On hospital day 7, fraction of inspiration O2 reduced to 45% and the patient exhibited clinical improvement. Initial serology, PCP PCR, and aspergillus antigen tests were positive, and itraconazole (200 mg/day) was initiated to treat possible chronic cavitary aspergillosis, considering the two cavitary lesions. Follow-up non-enhanced chest CT revealed newly formed GGOs in the left upper lung and cavitary changes in the previous consolidation in the right middle lobe [Figure 1C].

On hospital day 9, bedside bronchoscopy and bronchoalveolar lavage (BAL) were performed due to the patient’s unstable condition. PCP PCR and aspergillus antigen tests were performed using BAL fluid collected from the right middle lobe. On hospital day 11, aspergillosis was reported in BAL fluid cytology [Figure 1D]. Considering cytology and CT results, IA was diagnosed, and the antifungal agent was changed to voriconazole (200 mg twice/day).

On hospital day 13, percutaneous dilatational tracheostomy and bronchoscopy with BAL were performed in the portion of the right upper lobe to obtain the evidence of PCP. However, the results of BAL fluid cytology (second) only revealed aspergillus again.
After a few days, her condition deteriorated and her family declined aggressive management, including extracorporeal membrane oxygenation. On hospital day 15, the results of PCP PCR of the BAL (first) fluid specimen were positive but the fungus culture revealed no growth after 1 and 2 weeks. On hospital day 16, the patient died of multi-organ failure and septic shock.

PCP and IA co-infection is rare but can be fatal. To our knowledge, our patient is the tenth such case of PCP and IA co-infection in a non-HIV patient. Five of the six cases, including our case, with co-infection who underwent intubation and mechanical ventilation died from progression to acute respiratory failure (mortality rate, \( \sim 83.3\% \)).

Declaration of patient consent

Written informed consent was obtained from the patient’s family for publication of these case reports and any accompanying images. They understand that neither the patient’s name nor their initial will be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed. A copy of the written consent is available for review by the corresponding author.

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Conflicts of interest

None.

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