

Methylprednisolone/tacrolimus

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Non-HIV *Pneumocystis jirovecii* pneumonia: case report

A 46-year-old man developed non-HIV *Pneumocystis jirovecii* pneumonia during the treatment with methylprednisolone and tacrolimus for membranous nephropathy [dosages and routes not stated; durations of the treatments to reaction onset not stated].

The man presented to a hospital with complaints of fever and shortness of breath. Laboratory tests revealed leukocytes of $10.99 \times 10^9/L$, neutrophils of 87.7%, lymphocytes of 9.6% and C-reactive protein of 252.92 mg/L. CT scan of the chest revealed double lung inflammation. He was admitted to respiratory department with lung infection on 26 July 2021. He had medical history of membranous nephropathy for 1 year and had received treatment with oral tacrolimus capsule and methylprednisolone tablet in recent 2 months. After admission, his body temperature was 39.1°C and his lips were cyanotic, double lung auscultation revealed breathing sounds were thick and wet rales could be heard. After admission, blood gas analysis revealed obvious hypoxia. He had severe pneumonia and was assisted by noninvasive ventilator. Based on the history of membranous nephropathy, long-term oral unspecified hormone and immunosuppressant treatment, he was considered to be the immunosuppressive host.

The man was treated with meropenem, moxifloxacin, oseltamivir [oseltamivir phosphate], cotrimoxazole [trimethoprim-sulfamethoxazole] and caspofungin. Nucleic acid detection tests for influenza A and B viruses, nucleic acid detection for cytomegalovirus and EB virus, 1-3-b-D glucan and aspergillus immune test and acid fast staining results showed no abnormalities. Hence, oseltamivir phosphate was discontinued on day 2. On the same day, his dyspnoea worsened, the oxygen concentration of noninvasive ventilator was raised to 100%. Bedside chest X-ray revealed that the exudation of both lungs was obvious. He was started on invasive ventilator and endotracheal intubation to assist in breathing. According to acute respiratory distress syndrome (ARDS) criteria, he was considered to be severe ARDS. On day 3 of hospitalisation, he received veno-venous extracorporeal membrane oxygenation (VV-ECMO) and then was transferred to ICU under protection of ECMO. The result of acute physiology and chronic health evaluation II was 22, critical nutritional risk (NUTRIC score) was four, sequential organ failure assessment was 10 and venous thromboembolism risk (Padua score) was six. On day 5 of hospitalisation, high-throughput gene detection of pathogenic microorganisms detected sequences of *Pneumocystis jirovecii*, *Acinetobacter baumannii*, *Serratia marcescens* and Elizabeth meningitides were also detected [aetiology not stated]. Serum HIV-antibody was negative. He was diagnosed with non-HIV *Pneumocystis jirovecii* pneumonia, which was determined to be related to methylprednisolone and tacrolimus. Moxifloxacin was discontinued. He received treatment with cotrimoxazole combined with caspofungin to treat *Pneumocystis jirovecii* and meropenem for bacterial infection. Thereafter, the results of alveolar lavage fluid culture were reported intermittently, including *Pseudomonas aeruginosa* (resistant to meropenem) and *Stenotrophomonas maltophilia* (sensitive to cotrimoxazole) on 1 August 2021, Klebsiella pneumonia (resistant to meropenem) and *Pseudomonas aeruginosa* (sensitive to meropenem) on 4 August 2021 and *Stenotrophomonas maltophilia* (resistant to cotrimoxazole) and *Pseudomonas aeruginosa* (sensitive to meropenem) on 6 August 2021. Although some bacteria cultured in lung lavage fluid were resistant to the unspecified antibiotics. His body temperature and oxygenation were improved. Thereafter, he received dexamethasone and immunoglobulin. He had low reflux due to prone position ventilation (PPV), so enteral nutrition support was started by nasal intestinal tube. He had high risk of venous thromboembolism. During the anticoagulation period of ECMO, bilateral lower extremity pneumatic pump treatment were given to prevent thrombosis. After ECMO discontinuation, he received unspecified low molecular weight heparins. After treatment, his oxygenation improved, the peak airway pressure reduced and the pulmonary static compliance increased. On day 15, he was transferred to the respiratory department. After 12 days, he was discharged. His bedside chest X-ray revealed that the right lung lesions were less severe than on day 5 and day 8. Thereafter, his condition was slightly stable, his chest CT scans revealed that the lung lesions gradually improved. During the treatment, his tolerance and compliance were good and no adverse events occurred. At 2 months follow-up, his condition was good.

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