

CASE REPORT

Interstitial pneumonitis related to rituximab therapy for Waldenström's macroglobulinemia

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Key Clinical Message

Rituximab-combined therapeutic regimens have been recommended for treatment of Waldenström's Macroglobulinemia (WM). We report one WM patient who developed interstitial pneumonitis after rituximab therapy.

Keywords

Interstitial pneumonitis, rituximab, treatment, Waldenström's macroglobulinemia.

Rituximab-combined therapeutic regimens have been recommended for treatment of Waldenström's Macroglobulinemia (WM) [1, 2]. We report one WM patient who developed interstitial pneumonitis after rituximab therapy.

The patient was a 58-year-old man who presented with intermittent gingival hemorrhage for 1 year and dizziness for 1 month. Serum detections showed a high immunoglobulin M (IgM) concentration as 111 g/L. A bone marrow biopsy and a phenotypic analysis confirmed existence of abnormal CD20+ B cells; a gene analysis showed an L265P mutation (2.6%) at MYD88 gene locus, findings that were consistent with primary WM. The patient initially received plasma exchange therapy and then R-CHOP regimen (rituximab + cyclophosphamide + doxorubicin + vincristine + prednisone) with rituximab at 375 mg/m² of body-surface area. The patient showed relieved gingival hemorrhage and dizziness with a reduced IgM concentration to 50.5 g/L. However, 3 days after the

third R-CHOP course, the patient presented a high fever of 39°C centigrade with dry cough and worsening dyspnea with remarkable rhonchi in bilateral lungs. Analysis of arterial blood gases confirmed the presence of hypox-

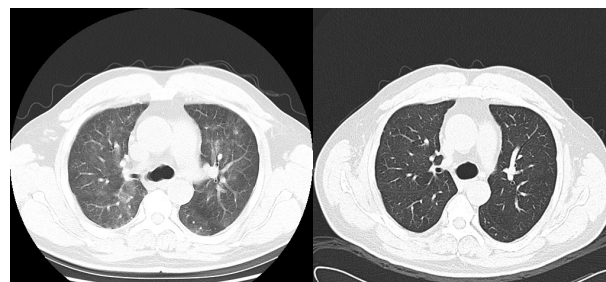


Figure 1. Helical computed tomographic scanning showed ground-glass shadowing in bilateral lungs before prednisone treatment and a recovery at 1 week post-treatment.

emia. Helical computed tomographic scanning showed ground-glass shadowing and pulmonary function tests demonstrated a restrictive pattern and a diffusion deficit, supporting the diagnosis of interstitial pneumonitis.

Laboratory investigations for suspicious causes, including blood and sputum cultures and autoantibodies, antineutrophil cytoplasmic, rheumatoid factor antibodies, and procalcitonin were negative. Empirical treatment with antibiotics for 6 days showed no obvious improvement, and then prednisone therapy was commenced at 60 mg/day. The sustained fever and other symptoms were dramatically relieved within 6 days. A repeated helical computed tomographic scanning confirmed a substantial recovery (Fig. 1) and the restrictive ventilation pattern was reversed.

In this case, the WM patient was receiving rituximab-involved combination therapy. We acknowledge noninfection-related rituximab-induced lung disease is rare, and it is difficult to completely exclude other causes in immunosuppressed patients. Based on the ineffectiveness by empirical antibiotic therapy and negative results from laboratory investigations, the temporal relation indicated rituximab a culprit of interstitial pneumonitis in this patient.

Rituximab-CD20 combination might trigger cytokine release to cause interstitial pneumonitis development. Notably, the rituximab-induced lung disease is a potentially fatal complication [3, 4]. Considering previous rituximab cases [5], early administrations of hormone therapy showed striking outcomes in this patient. Since rituximab is increasingly prescribed for treatment of various disorders, physicians should pay more attention to this complication.

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Conflict of Interest

None declared.

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