## Convalescent-anti-sars-cov-2-plasma/dexamethasone/rituximab

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### Depletion of B-lymphocytes, lack of efficacy and off label treatment: case report

A 37‑year‑old man developed depletion of B lymphocytes during treatment with rituximab for chronic lymphocytic leukaemia. Additionally, he exhibited lack of efficacy during treatment with dexamethasone for progressive respiratory failure and off label treatment of convalescent-anti-SARS-CoV-2-plasma for SARS‑CoV‑2 infection [*routes and dosages not stated*].

The man, who had a history of chronic lymphocytic leukaemia admitted to hospital for dry cough and fever of 40°C since 2 days. He had been treating with 6 cycles of rituximab and cyclophosphamide for chronic lymphocytic leukaemia. His COVID-19 PCR tested positive in June 2020. On admission, she was in good general condition, without features of respiratory and cardiovascular failure. His laboratory tests indicated pancytopenia and a moderate elevation of inflammatory markers. A chest CT and X‑ray revealed extensive bilateral ground‑glass opacities in the lungs, with no features of pulmonary embolism. A Flow cytometry showed a severe depletion of B lymphocytes, which was consistent with prior rituximab therapy for chronic lymphocytic leukaemia. During his hospitalisation, a bacterial coinfection was suspected due to clinical deterioration and increasing levels of inflammatory markers, which prompted initial treatment with ceftriaxone. However, no clinical improvement was noted by switching the regimen to ceftazidime and amikacin, and then to meropenem, levofloxacin and linezolid. Enoxaparin-sodium [enoxaparin] was also initiated. Cotrimoxazole [trimethoprim/sulfamethoxazole], aciclovir and unspecified antifungal therapy were also added to his regimen. Due to decreased neutrophil count and recurrent high fever, neutropenic fever was suspected, and he was treated with granulocyte colony–stimulating factor. He was also treated with dexamethasone for progressive respiratory failure. On day 9, he received passive oxygen therapy. Due to increasing D‑dimer and a high risk of thromboembolism, enoxaparin was initiated. Control chest X‑ray revealed a significant progression of infiltrative lesions in the left lung, and abdominal CT showed hepatosplenomegaly.

Anti–SARS‑CoV‑2 IgA and IgG tests were negative on day 12 and 24, but repeated PCR of nasopharyngeal swab specimens for SARS‑CoV‑2 were positive. After a few days, a sudden clinical deterioration with spontaneous pneumomediastinum, subcutaneous emphysema and progression of lung lesions were noted. A conservative treatment was then initiated. After 2 days, he received off-label treatment of convalescent-anti-SARS-CoV-2-plasma [convalescent plasma] transfusion for SARS‑CoV‑2 infection. However, the man's respiratory failure worsened. He was transferred to ICU, and a high‑flow nasal oxygen therapy was started. After 10 days, he was intubated and mechanical ventilated, but his general condition continued to deteriorate. He then developed sudden asystolic cardiac arrest. Unfortunately, he died.

Stepien A, et al. Impaired humoral immune response in a COVID-19 patient with chronic lymphocytic leukemia complicated by spontaneous pneumomediastinum and hemophagocytic lymphohistiocytosis syndrome. Polskie Archiwum Medycyny Wewnetrznej 131: 16108, No. 12, 22 Dec 2021. Available from: URL: http://doi.org/10.20452/pamw.16108

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