

Multiple drugs

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Pulmonary embolism, deep vein thrombosis and treatment failure: case report

A 46-year-old woman exhibited treatment failure during treatment with abatacept, etanercept, infliximab, methotrexate and tocilizumab for seropositive rheumatoid arthritis (RA). Additionally, she developed pulmonary embolism and deep vein thrombosis during treatment with baricitinib for seropositive RA [*not all dosages stated; routes not stated*].

The woman was diagnosed with seropositive RA in April 2010. She initially received treatment with methotrexate, but methotrexate failed to control the disease activity. Therefore, she received sequent four different biological therapies that included etanercept plus methotrexate, proceeding to infliximab plus methotrexate, tocilizumab plus methotrexate, and abatacept monotherapy. However, all the therapies failed and the disease activity increased.

In March 2020, the woman received high-throughput leukocytapheresis due to treatment failure of previous therapies. Following 5 leukocytapheresis, treatment with baricitinib 4 mg/day was initiated along prednisolone. After eight weeks, low disease activity was achieved. However, 12 weeks following initiation of baricitinib, she developed dyspnea and chest pain that suddenly appeared on lifting heavy objects. Also, a week prior to attack, she developed painless swelling of the left. Due to worsening dyspnea, she was taken to the emergency hospital via ambulance. She was in shock in the emergency room and her respiratory rate was 30 breaths/min and SpO₂ was 90% with reservoir mask oxygen at 7 L/min. Arterial blood gas analysis showed the following: PaO₂ 77 Torr, PaCO₂ 29 Torr, and HCO₃ 19.2 mmol/L. Additionally, serum D-dimer and brain natriuretic peptide were elevated. The electrocardiogram indicated right ventricular strain and transthoracic echocardiography demonstrated a dilated right ventricular dimension, McConnell sign, and decreased tricuspid annular plane systolic excursion. The findings suggested severe right ventricular systolic dysfunction. Her CT scan showed thrombi in both main pulmonary arteries, the left popliteal vein, and the left superficial femoral vein. Based on the investigation, a diagnosis of acute massive pulmonary embolism caused by deep vein thrombosis was confirmed. Both anti-SARS-Cov-2 antibody and anti-phospholipid syndrome-related tests were negative. Her BMI indicated obese class I and no other cardiovascular or venous thromboembolism risk factors were identified. She received treatment with tissue-type plasminogen activator. On day 2 of hospitalisation, her shock resolved and dyspnea improved. No bleeding was observed. Further, she received anticoagulation therapy with rivaroxaban. On day 6, her dyspnea and hypoxia resolved. CT scan showed decreased amounts of thrombi. Also, the findings of right ventricular strain disappeared. On day 10, she was discharged with rivaroxaban. Also, certolizumab pegol [certolizumab] and methotrexate therapy was initiated. After 4 months, the emboli disappeared and low disease activity was achieved.

Mori S, et al. Risk of venous thromboembolism associated with Janus kinase inhibitors for rheumatoid arthritis: case presentation and literature review. [Review]. *Clinical Rheumatology* 40: 4457-4471, No. 11, Nov 2021. Available from: URL: <https://link.springer.com/article/10.1007%2Fs10067-021-05911-4>

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