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A case of hypercalcemia and acute kidney injury caused by *Pneumocystis jirovecii* in an immunocompromised patient with granulomatosis with polyangiitis

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Introduction: We report the case of a patient with a history of granulomatosis with polyangiitis who developed severe *Pneumocystis jirovecii* pneumonia (PJP) with an atypical clinical presentation.

Case description: A 55-year-old male with a history of granulomatosis with polyangiitis (GPA) with lung and kidney involvement, treated with glucocorticosteroids, methotrexate and rituximab, presented to the rheumatology department with symptoms including generalised weakness, loss of appetite, weight loss, oral ulcers, and decline in renal function. Initial examination and laboratory tests revealed no signs of activity of the anti-neutrophil cytoplasmic antibodies (ANCA)-associated vasculitis, however they showed increased levels of serum creatinine (4.92 mg/dl, glomerular filtration rate: 13.1 ml/min/1.73 m^2) and hypercalcemia (total calcium: 3.58 mmol/l; normal range: 2.15–2.55 mmol/l). Symptomatic treatment with fluid therapy, diuretics and hydrocortisone was initiated. A comprehensive diagnostic work-up was performed, revealing low parathyroid hormone levels (11.1 pg/ml; normal range: 15.0–68.3 pg/ml), negative tumour markers, slightly elevated inflammatory marker levels and bilateral nephrocalcinosis on abdominal ultrasound. Patient's condition gradually worsened as he developed respiratory symptoms with simultaneous deterioration of pulmonary imaging. Due to rapid-onset severe pneumonia, the patient was temporarily transported to the intensive care unit. *Pneumocystis jirovecii* pneumonia was confirmed, and trimethoprim-sulfamethoxazole (TMP-SMX) treatment was initiated, resulting in both clinical and biochemical improvement. Rituximab treatment was temporarily discontinued after considering the risk-benefit balance, and chronic secondary prevention with TMP-SMX was implemented. The patient was discharged in good condition. A case of hypercalcemia secondary to *P. jirovecii* infection was concluded.

Conclusions: To our knowledge, this is the second reported case of hypercalcemia in a non-HIV, non-transplant immunosuppressed patient with ANCA-vasculitis. Hypercalcemia preceding clinical presentation of PJP in immunosuppressed patients is documented in medical literature and, along with this case study, underlines the importance of extended diagnostic work-up after the most common causes of laboratory hypercalcemia are excluded. Furthermore, considering that patients with rheumatic diseases, especially those receiving immunosuppressive therapy, are more prone to the *P. jirovecii* infection, an individual decision regarding chronic chemoprophylaxis needs to be made.