Title: Acute bilateral pulmonary embolism in a patient with B12 deficiency

Authors: Phillip Lim, MD; Rafael Nigri, MD; Mirela Feurdean, MD

Affiliation: Rutgers New Jersey Medical School, Department of Medicine

Introduction
Vitamin B12 deficiency typically presents with anemia and neurologic manifestations but can also present with uncommon clinically challenging manifestations. We report the case of a patient who presented with unprovoked bilateral pulmonary embolism (PE) and was subsequently diagnosed with severe vitamin B12 deficiency.

Case
A 60-year-old male with a history of hypertension presented with dyspnea for one week. The patient reported lightheadedness but denied fever, chills, cough, night sweats, or chest pain. He denied recent travels, surgery, or immobilization. Vitals were as follows: T 98.6°F, BP 128/70 mmHg, HR 86 bpm, RR 18 bpm, and SpO2 94% on RA. Physical examination was normal except bilateral lower extremity edema. Labs were as follows: Hgb 5.2 g/dL, MCV 110 fL, MCH 36%, platelets 107,000/µL, D-dimer 2,637 ng/mL, B12 150 ng/mL, homocysteine 158 µmol/L, and methylmalonic acid 9,691 µmol/L. Chest x-ray was within normal limits. Doppler showed deep vein thrombosis (DVT) in the right popliteal and gastrocnemius veins. Computed tomography chest revealed PE in the distal, left interlobar pulmonary artery and in the junction between the segmental pulmonary arteries in the right lower lobe. The patient was given intramuscular B12, started on enoxaparin, and bridged to warfarin. Three months later, his symptoms resolved with labs showing a B12 level of 355 ng/mL and a homocysteine level of 11.2 µmol/L.

Discussion
Prior reports have linked B12 deficiency with venous thromboembolism (VTE) but in patients with recent surgery, trauma, pregnancy, or states of endothelial dysfunction such as metabolic syndrome. Our patient did not have typical risk factors for VTE, highlighting the need for an increased index of suspicion for vitamin B12 deficiency in the setting of unprovoked DVT.

Conclusion
Vitamin B12 deficiency is a treatable cause of PE and should be investigated in all patients diagnosed with PE, especially in those with no typical risk factors for VTE.

References: