Title- Provoked Extensive PE and DVT In a Patient With B-Thalassemia Minor- A Rare Presentation

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Introduction: Thromboembolic events are well recognized in β-thalassemia major and intermedia patients; however, they are less studied in β-thalassemia minor patients. Evidence shows a prevalence of VTE in 29% of β-thalassemia intermedia patients in a 10-year large splenectomized cohort, compared with 1–4% in β-thalassemia major patients in similar cohorts. There are case reports and small studies reporting VTE in β-thalassemia minor patients. In β-thalassemia major and intermedia, chronic hemolysis, splenectomy, and endothelial dysfunction contribute to high VTE risk. In contrast, β-thalassemia minor is considered hematologically benign, with a lower risk of VTE. However, limited data suggest that VTE can occur, warranting further study. We present a case of a 74-year-old female with β-thalassemia minor who presented with shortness of breath and was found to have provoked extensive pulmonary embolism (PE) and deep vein thrombosis (DVT), with no other risk factors except history of β-thalassemia minor,hypercoagulable workup was negative.

Case Presentation: A 74-year-old female with β-thalassemia minor presented to a community hospital with a two-day history of progressively worsening shortness of breath. CT imaging revealed moderate-to-large PEs in the right and left main pulmonary arteries extending into the lower lobes. She was transferred to our ICU for further management. On presentation, vital signs were stable (BP 143/94 mmHg, HR 86 bpm, RR 15/min). Labs showed hemoglobin 9.5 g/dL, hematocrit 33.8%, MCV 78 fL, MCH 22 pg, RDW 25%, WBC 8.7×10^9/L, and platelets 34×10^9/L. Echocardiogram revealed elevated right atrial pressures consistent with right heart strain. Bilateral lower extremity ultrasound showed acute left femoral and popliteal DVT.

She was started on IV bivalirudin due to severe thrombocytopenia and received platelet transfusions. Once platelet counts stabilized, she underwent mechanical thrombectomy for extensive PE and was transitioned to oral anticoagulation. Hypercoagulable workup revealed no clear cause; thrombophilia testing showed mildly elevated IgM anticardiolipin antibody, which is nonspecific and can occur in inflammation, infection or thrombosis. She reported starting kimchi supplementation one week prior; literature does not support a link to VTE.

Discussion: β-thalassemia is a disorder of hemoglobin synthesis caused by defective or absent beta-globin production. Major phenotypes include major, intermedia, and minor. VTE is a well-recognized complication in thalassemia intermedia and major. In intermedia, hypercoagulability arises from chronic hemolysis, which releases free hemoglobin and heme that scavenge nitric oxide and activate platelets and the endothelium. Splenectomy increases the risk of VTE even further as it increases circulating abnormal RBCs and platelets. Abnormal RBC morphology also contributes to coagulation activation. Whereas in β-thalassemia major, regular transfusions partially mitigate hypercoagulability due to dilution of abnormal RBCs. Although splenectomy, older age, and iron overload remain risk factors. In β-thalassemia minor, most carriers are asymptomatic with mild anemia, and VTE is rare. Nonetheless, case reports suggest that thrombosis can occur, particularly with additional

prothrombotic factors such as surgery, immobility, pregnancy, or inherited thrombophilia. Larger studies are needed to clarify VTE risk and potential surveillance strategies in β-thalassemia minor patients.

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