Mobile aortic mural thrombus in a patient with small-cell lung cancer receiving cisplatin-based chemotherapy

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A 55-year-old man with right-sided small-cell lung cancer (SCLC) undergoing chemotherapy with cisplatin and etoposide was admitted to the hospital with a balloting thrombus located in a non-aneurysmal, non-atherosclerotic aortic arch.

The patient's SCLC (T4N3M0-IIIC) was diagnosed 2.5 months before admission with a Khorana score of 2, classified as intermediate-risk for thromboembolism, with the unclear benefit of routine anticoagulation in this subset of patients at the time. The oncology team did not start thromboprophylaxis in the absence of absolute indications. Past medical history was only significant for a 60 pack-year smoking history. On admission, laboratory tests revealed thrombocytosis (408×10^3/ul), and elevated D-dimer (625 ng/ml). Physical examination showed a significant reduction of vesicular sounds in the upper field of the left lung. Transthoracic echocardiographic (TTE) examination revealed the presence of a normoechogenic longitudinal balloting structure, 18 mm long and 5 mm wide, located at the base of the aortic arch (Figure 1, Supplementary material, *Videos S1* and *S2*). TTE also showed an enlarged left atrium and slight myocardial hypertrophy. No segmental left ventricular wall motion or valvular abnormalities were reported.

Immediately after confirmation of the presence of the aortic thrombus, the patient was anticoagulated with a therapeutic dose of enoxaparin 80 mg twice a day subcutaneously. Control TTE after 4 and 7 days of anticoagulation showed only a slight reduction of thrombus size (to 15 × 5 mm). No symptoms of embolism were reported by the patient during treatment. He was discharged on enoxaparin 80 mg twice a day, which resulted in complete clot resolution after 29 days of uninterrupted anticoagulation, followed by a continuation of treatment until the next chemotherapy.

The association between malignancy and hypercoagulability, leading to thromboembolic events such as venous thromboembolism (VTE) and arterial thromboembolism (ATE), is well recognized and extensively described in the literature. Aortic mural thrombus (AMT) is a rare form of ATE, where approximately 25% of patients have documented hypercoagulable states, and 10% were previously diagnosed with malignancy [1].

Currently, there are no explicit guidelines regarding the management of AMT in cancer patients receiving chemotherapy, with treatment strategies guided by evidence-based guidelines for venous thrombosis in cancer patients or analyses of the few previously described cases. Treatment modalities include pharmacological anticoagulation, thoracic endovascular aortic repair (TEVAR), and open aortic surgery. Pharmacological options for management of patients with cancer-associated VTE range from monotherapy with low molecular weight heparin (LMWH), unfractionated heparin (UFH), rivaroxaban, apixaban, or fondaparinux, and strategies based on a combination of warfarin with LMWH, UFH or fondaparinux, or combination of edoxaban with LMWH or UFH [2]. Treatment of AMT following cisplatin-based chemotherapy in the current literature differed between patients. All described cases implemented

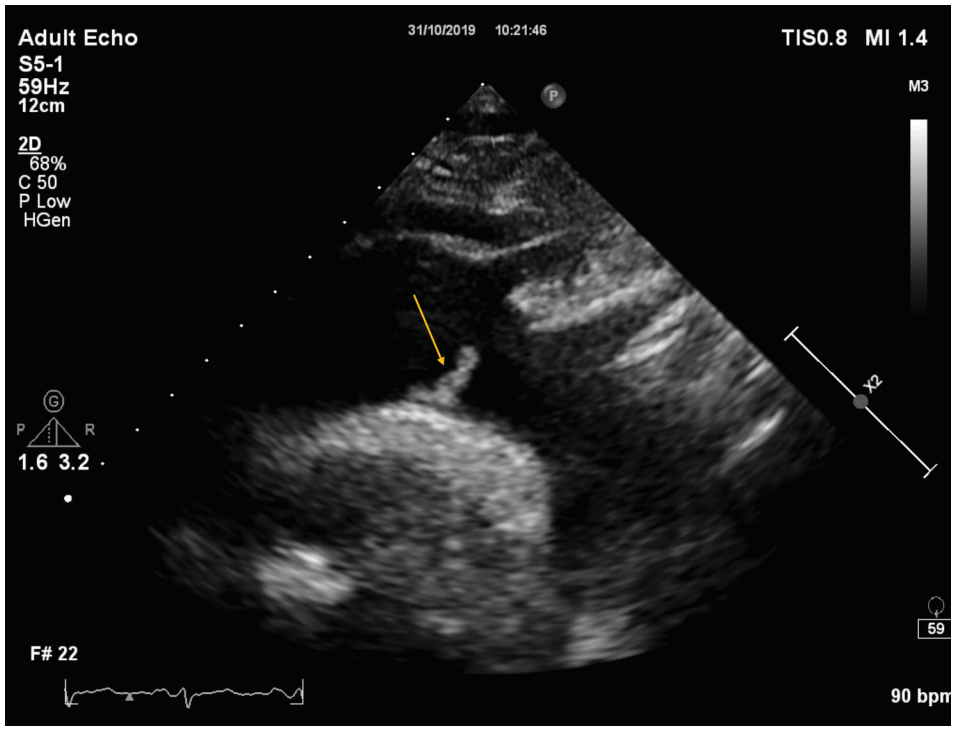

Figure 1. Normoechogenic longitudinal balloting structure at the base of the aortic arch on transthoracic two-dimensional echocardiography (the yellow arrow)

conservative management, varying from LMWH with acetylsalicylic acid followed by vitamin K antagonist (VKA) [3], UFH followed by VKA [4], to therapy with subsequent administration of UFH, LMWH, and rivaroxaban [5].

In conclusion, clinicians should be aware of the risk of AMT as a rare, but potentially fatal, complication of chemotherapy in cancer patients receiving chemotherapy. As no clinical guidelines are currently available, choosing the most appropriate treatment modality presents a challenge and should be individualized. In our case, the thrombus was successfully treated with enoxaparin, showing that monotherapy with LMWH could be a safe and effective strategy. However, further studies are needed to reach a definitive conclusion.

Supplementary material

Supplementary material is available at https://journals.viamedica.pl/kardiologia_polska

Article information

Conflict of interest: None declared.

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