In rod c ion

Pulmonary thromboembolism was considered as the result of clinical evaluation. ere were no risk factors such as age, smoking, trauma, immobilization, surgery, heart disease, and genetic risk factors to explain pulmonary embolism. In this case we see that the pulmonary embolism was associated with quetiapine. We should be more careful about pulmonary thromboembolism [1]. Venous romboembolism has been associated with risk factors such as smoking, trauma, immobilization, surgery, pregnancy, use of combined oral contraceptives, malignant disorders, and certain cardiac and haemostatic disorders, including factor Leiden mutation. In our case there was no family history of hypercoagulable state, nor any past surgical or chronic systemic medical history. He did not have any risk factors for pulmonary embolism. We strongly suspected that quetiapine might have contributed to his pulmonary thromboembolısm on the basis of published reports [2]. e biological mechanism explaining the relation between antipsychotic drugs and venous thromboembolism is unknown. Many biological mechanisms have been proposed to explain this relationship until this time. Previous studies in the literature have shown that antipsychotics increase platelet aggregation, especially due to the e ects on 5-hydroxy tryptamine [3]. A second possible explanation is about anti-cardiolipin antibodies. Anti-cardiolipin antibodies are associated with increased risk of venous or arterial thrombosis and it has been observed that anti-cardiolipin antibodies are increased in patients using chlorpromazine [4]. At the same time, no relationship has been found between venous thromboembolism and antipsychotic drug use in those in whom anti-cardiolipin antibodies were detected. Patients treated with low-potency antipsychotic drugs have the side e ect sedation much more o en. A third hypothesis is that venous stasis can be aggravated by sedation and this can increase the risk of thrombosis. Also it is thought that putting on weight, high body mass index, and sedative life style developing with the use of these drugs could be the risk factors [5]. Pulmonary thromboembolism is o en misdiagnosed as sudden cardiac death. Only in necropsy in psychiatric patients of idiopathic, fatal pulmonary embolism were diagnosed [6]. ere is an association between sudden cardiac death and antipsychotic drug use which has been described by the spontaneous reports. However, evidence to explain the causal relationship between antipsychotic drugs and venous thromboembolism is still insu cient [7]. Venous romboembolism, which includes pulmonary embolism and deep-