

Short Review

Short review for “Venlafaxine-Induced Thrombocytopenia: A Case Report and Literature Review”

Shi Lijun*, Ma Zhongrui, Wei Li, Feng Lei, Jiang Wei, Yu Xia and Pan Yaning

Department of Hematology, Chengdu Fifth People's Hospital, Chengdu, China

In this study, we present a case involving a patient with anxiety who developed severe thrombocytopenia and subcutaneous hemorrhaging following one week of treatment with a venlafaxine-containing regimen. Drug-induced thrombocytopenia (DITP) was considered as a diagnosis after excluding other secondary causes. Notably, the patient's platelet count returned to normal following treatment with glucocorticoids in combination with recombinant human thrombopoietin. Subsequently, anti-anxiety medication excluding venlafaxine was administered to continue the patient's treatment.

Venlafaxine is classified within the category of serotonin (5-HT) and noradrenaline (NA) reuptake inhibitors (SNRIs), which are frequently prescribed for the treatment of depression, anxiety, and various other psychiatric disorders [1]. Although venlafaxine is rarely associated with Drug-Induced Thrombocytopenia (DITP), potential mechanisms have been proposed [2-6]. One proposed mechanism involves the direct antagonism of the serotonin transporter, which inhibits platelet aggregation and subsequently reduces serotonin levels within platelets. Another proposed mechanism involves the mediation of platelet count reduction by drug-dependent antibodies.

The existing guidelines for platelet monitoring in the drug instructions are evidently insufficient. Instances of platelet reduction induced by venlafaxine have highlighted systemic deficiencies in the safety monitoring protocols for psychiatric medications. In the context of precision medicine, the platelet reduction linked to venlafaxine serves as a reminder that even well-established drugs may harbor

complexities that remain incompletely understood. Achieving an optimal balance between the benefits and risks of antidepressant therapy necessitates ongoing pharmacological research, stringent clinical monitoring, and open interdisciplinary communication.

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***Corresponding author:** Shi Lijun, Department of Hematology, Chengdu Fifth People's Hospital, Chengdu, China, E-mail: shilj0729@163.com

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