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Case Report

An unusual case report of chlorpheniramine-induced severe thrombocytopenia

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ABSTRACT

A rare but fatal illness is drug-induced thrombocytopenia (DITP). Severe thrombocytopenia is not frequently linked to antihistamines, especially first-generation H1 blockers like chlorpheniramine. We describe a rare instance of a 31-year-old man who used chlorpheniramine for rhinitis and thereafter got severe thrombocytopenia (platelet count=0×10^9/L). The patient had subcutaneous petechiae, bilateral conjunctival hemorrhage, and bleeding gums. Other causes were ruled out by laboratory tests, such as bone marrow analysis and peripheral blood smears. After receiving intravenous immunoglobulin (IVIG) and corticosteroids, the patient's platelet counts gradually recovered. This instance emphasizes how crucial it is to identify DITP as a side effect of chlorpheniramine so that early diagnosis and treatment may be carried out.

Keywords: Drug-induced thrombocytopenia, Chlorpheniramine, Antihistamine, IVIG, Platelet recovery

INTRODUCTION

Immune-mediated platelet destruction or reduction of platelet synthesis can lead to DITP, a rare but dangerous illness. Heparin, quinine, and antibiotics including vancomycin and sulfonamides are the medications most commonly linked.[1] Severe thrombocytopenia is a rare side effect of antihistamines, especially chlorpheniramine. A typical treatment for allergic rhinitis and other allergic disorders is chlorpheniramine, a first-generation H1 antihistamine. Even while leukopenia and other moderate hematological side effects have been documented, life-threatening thrombocytopenia is still very uncommon.[2] We report a rare instance of a young male patient who took chlorpheniramine and experienced severe thrombocytopenia, which resulted in mucocutaneous hemorrhage but responded favorably to IVIG and corticosteroid treatment.

CASE REPORT

Patient data and background

A male patient, age 31, who had no notable medical history, arrived at the emergency room with bilateral conjunctival hemorrhage, several petechial lesions across his extremities, and spontaneous gum bleeding. Three days after he started taking chlorpheniramine 12 mg BID, He received five doses each one 8 mg for allergic rhinitis plus paracetamol 500 mg q6h, before the symptoms

appeared. He had no history of autoimmune disorders, recent infections, immunizations, or chronic drug usage.

Clinical examination and investigations

The patient was hemodynamically stable upon physical examination. Mucosal bleeding, bilateral subconjunctival hemorrhages (Figure 3, green arrow), and extensive petechiae on the upper and lower extremities were the main findings (Figure 2, blue arrows). Neither lymphadenopathy nor hepatosplenomegaly was present.

Initial laboratory investigations revealed:

Complete blood count (CBC)

Platelet count: 0×10^9/L, hemoglobin: 13.2 g/dL and white blood cell count: 7.1 $\times10^9$/L

Peripheral blood smear

Normal morphology and no schistocytes or abnormal cells.

Coagulation profile

Prothrombin time (PT), activated partial thromboplastin time (aPTT), and fibrinogen levels were within normal limits

Bone marrow aspiration and biopsy done after platelet count improved as postponed avoiding high risk procedure: Normocellular marrow with normal megakaryocytes, ruling out bone marrow failure or malignancy.

Autoimmune panel and viral screening

Negative for antinuclear antibodies (ANA), anti-phospholipid antibodies, hepatitis B/C, HIV, and Epstein-Barr virus

These findings confirmed isolated severe thrombocytopenia, strongly suggestive of DITP induced by chlorpheniramine.

Management and outcome

The patient was immediately admitted to the hematology unit. Chlorpheniramine was discontinued, and treatment was initiated with-IVIG 1 g/kg/day for 4 days, intravenous methylprednisolone (1 mg/kg) starting on day 4, platelets transfused 2 doses on admission. The patient's platelet count showed a gradual recovery (Table and Figure 1). On day 5, platelets rose to 45×10^9/L, reaching 100×10^9/L by day 7, allowing for discharge with a hematology follow-up. The patient remained stable during follow-up, with no recurrence of thrombocytopenia after 2 months.

Table 1: Platelet count trend over time.

Day	Platelet count (×10⁹/L)
1	0
2	1
3	1
4	1
5	45
6	80
7	100

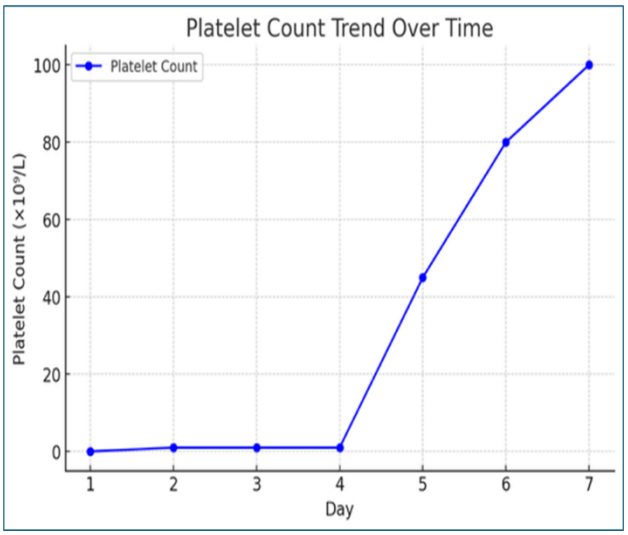

Figure 1: Platelet count trend over time showing platelet recovery over 7 days.

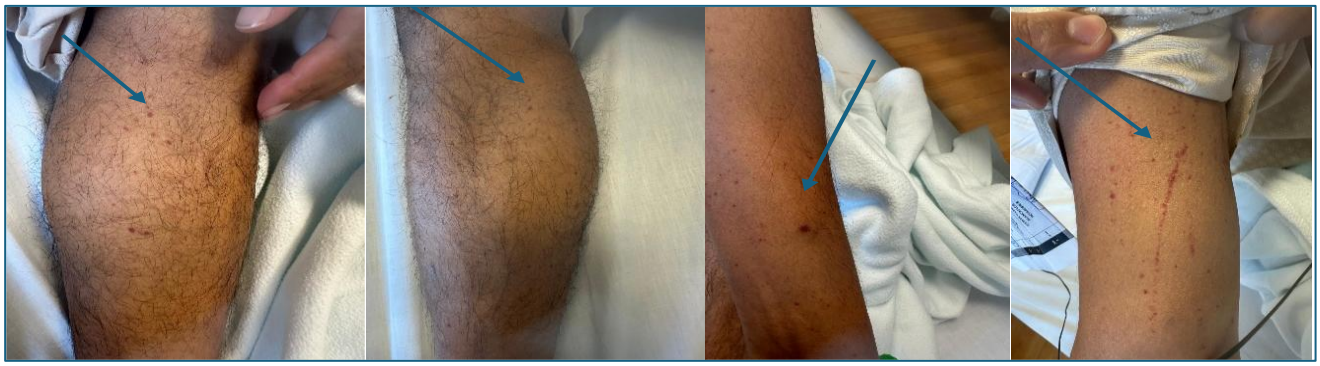

Figure 2: Lower and upper limbs show extensive petechiae on the upper and lower extremities (blue arrows).

Figure 3: Conjunctival hemorrhage (green arrow).

DISCUSSION

Drug-dependent antibodies frequently cause the immune system to destroy platelets in DITP, an uncommon but dangerous illness. There is little clinical awareness of the role of antihistamines, especially first-generation H1 blockers like chlorpheniramine, even though common offenders like quinine, NSAIDs, and beta-lactam antibiotics are well-established.[1,2]

Mechanisms of chlorpheniramine-induced thrombocytopenia, either immune-mediated platelet destruction or direct inhibition of platelet production are involved in the pathophysiology of DITP. By producing drug-dependent antibodies that target platelets, chlorpheniramine is thought to cause thrombocytopenia and speed up platelet clearance through the reticuloendothelial system.[3] This process is comparable to other well-known causes of DITP, like quinine, in which complement pathways are activated by drug-dependent antibodies, resulting in platelet lysis.[4] Furthermore, by changing the expression of glycoprotein IIb/IIIa, some medications may cause platelets to be destroyed, which would then trigger immune identification and platelet clearance. Studies indicate that similar mechanisms might be at work, despite the paucity of study on this effect unique to chlorpheniramine.[5]

Clinical presentation and diagnosis, As was the case with our patient, who experienced acute and severe thrombocytopenia only three days after beginning chlorpheniramine, DITP frequently manifests this condition. The clinical signs of severe thrombocytopenia are consistent with the presence of petechial lesions, bilateral conjunctival hemorrhage, and mucocutaneous bleeding.[6] Other potential reasons, including primary immune thrombocytopenia (ITP), bone marrow problems, viral infections, and autoimmune diseases, must be ruled out in order to diagnose DITP. The absence of autoimmune markers, negative viral screening results, and normal bone marrow findings in our patient all supported the diagnosis of drug-induced thrombocytopenia as opposed to underlying hematologic disease.[7]

Results and management, the cornerstone of managing DITP is stopping the offending substance right away. After receiving IVIG and corticosteroids, our patient's platelets recovered in just one week. While corticosteroids lessen inflammation and immunological activation, IVIG works by modifying Fc receptor-mediated platelet destruction.[8] Although platelet transfusion is occasionally tried for patients with life-threatening bleeding, it is not always successful because circulating drug-dependent antibodies have the potential to quickly destroy transfused platelets.[9]

Review of related cases and literature

According to a comprehensive analysis of cases of drug-induced thrombocytopenia, significant platelet depletion has infrequently been linked to chlorpheniramine. The necessity for greater clinical awareness is highlighted by the occasional studies that suggest possible hematologic side effects.[10] Although chlorpheniramine was not one of the most often implicated medications, research by Reese et al examined FDA adverse drug reaction records and found that a number of antihistamines may be responsible for rare incidences of thrombocytopenia.[11] According to a case series by Chong et al the majority of DITP patients recover in 1-2 weeks after stopping their medication, which is consistent with our patient's recovery period.[12] This emphasizes the significance of early detection and timely treatment to avoid serious consequences like cerebral bleeding, which can happen in severe situations.

Future perspectives, since chlorpheniramine-induced thrombocytopenia is uncommon, more investigation is necessary to pinpoint any immunological or genetic predispositions and clarify the precise pathophysiological pathways. Additionally, prompt diagnosis and treatment of DITP can be facilitated by raising clinician awareness of the hematological adverse effects of routinely used drugs.

CONCLUSION

This case highlights a rare but life-threatening adverse effect of chlorpheniramine. Physicians should be aware of DITP as a potential cause of unexplained thrombocytopenia, even with commonly used medications. Early recognition and prompt treatment with IVIG and corticosteroids can result in favorable outcomes. Further studies are needed to explore the exact mechanism of chlorpheniramine-induced thrombocytopenia.

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