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MUCOCUTANEOUS BLEEDING IN A DIABETIC PATIENT: AN ATYPICAL PRESENTATION OF ITP

General Medicine

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ABSTRACT

Immune thrombocytopenia (ITP) is an acquired autoimmune disorder marked by isolated thrombocytopenia due to autoantibody-mediated platelet destruction and impaired platelet production. It typically presents with mucocutaneous bleeding, including petechiae, ecchymosis, epistaxis, and gingival hemorrhage. Diagnosis is based on exclusion of secondary causes such as infections, autoimmune disorders, and malignancy. First-line treatment includes corticosteroids and intravenous immunoglobulin, with therapy tailored to bleeding severity and platelet count.[1 2] We present the case of a 54-year-old diabetic female who developed spontaneous gum bleeding and petechiae, with a platelet count of 8,000/µL. Following exclusion of secondary causes, a diagnosis of ITP was made, and corticosteroid therapy initiated, resulting in clinical improvement and rising platelet counts. This case underscores the importance of timely recognition and management of ITP to prevent hemorrhagic complications.[3]

KEYWORDS

ITP, Gum bleeding, mucosal bleeding in Diabetes

INTRODUCTION

Immune Thrombocytopenic Purpura (ITP), formerly termed idiopathic thrombocytopenic purpura, is an autoimmune hematological disorder characterized by isolated thrombocytopenia (platelet count <100 × 10^9/L) in the absence of other identifiable causes[1]. The pathogenesis of ITP involves the production of autoantibodies targeting platelet surface glycoproteins, leading to accelerated platelet destruction predominantly in the spleen[12]. Additionally, impaired platelet production due to megakaryocyte suppression and T-cell–mediated cytotoxicity contributes to the thrombocytopenia observed in ITP patients[14].

Clinically, ITP presents with a spectrum ranging from asymptomatic thrombocytopenia to severe mucocutaneous bleeding manifestations such as petechiae, ecchymoses, epistaxis, menorrhagia, and, in rare cases, life-threatening hemorrhages[12]. The diagnosis is primarily one of exclusion, necessitating the elimination of other potential causes of thrombocytopenia, including but not limited to viral infections (e.g., HIV, hepatitis C), autoimmune disorders (e.g., systemic lupus erythematosus), bone marrow pathologies, and drug-induced thrombocytopenia[124]. Laboratory evaluation typically reveals isolated thrombocytopenia with normal white blood cell and hemoglobin levels, and a peripheral blood smear devoid of schistocytes[2]. Bone marrow examination, though not routinely required, may be considered in atypical cases or in patients over 60 years of age to exclude myelodysplastic syndromes[24].

Management strategies for ITP are tailored based on the severity of thrombocytopenia and bleeding risk[12]. First-line therapy often involves corticosteroids, such as prednisone or dexamethasone, aiming to suppress autoantibody production[1]. Intravenous immunoglobulin (IVIG) serves as an adjunct or alternative, particularly in cases necessitating rapid platelet count elevation[12]. For patients refractory to initial treatments, second-line options include rituximab, a monoclonal antibody targeting CD20-positive B cells, and thrombopoietin receptor agonists (TPO-RAs) like eltrombopag and romiplostim, which stimulate platelet production[24]. Splenectomy remains a consideration for chronic, treatment-resistant cases[2].

Recent advancements have expanded the therapeutic landscape of ITP[14]. Emerging treatments under investigation encompass agents targeting novel pathways implicated in ITP pathophysiology[4]. Sutimlimab, a monoclonal antibody inhibiting the classical complement pathway component C1s, has demonstrated efficacy in patients with complement-mediated platelet destruction[4]. Bruton tyrosine kinase (BTK) inhibitors, such as rilzabrutinib, have shown promise in early-phase trials by modulating B-cell receptor signaling and attenuating autoantibody production[4]. Efgartigimod, an antagonist of the neonatal Fc receptor (FcRn), facilitates the degradation of pathogenic IgG autoantibodies, thereby reducing platelet clearance[4]. Additionally, recombinant human ADAMTS13 (rADAMTS13) has been explored in thrombotic thrombocytopenic purpura (TTP), a related disorder, offering potential insights into future ITP therapies[3]. Gene editing technologies, such as CRISPR-Cas9, and cellular therapies, including chimeric antigen receptor (CAR) T-cell therapy, are being investigated for their applicability in refractory ITP cases[4].

In the context of these developments, we present a case of a 54-year-old female patient with a 4-day history of spontaneous mucocutaneous bleeding and severe thrombocytopenia (platelet count of 5,000/µL, declining to 3,000/µL). Comprehensive evaluation excluded secondary causes, including negative dengue serology, unremarkable antinuclear antibody profile, and normal erythrocyte sedimentation rate. Initiation of corticosteroid therapy with intravenous dexamethasone led to a gradual improvement in platelet counts, reaching 11,000/µL at discharge. The patient was subsequently managed with an oral prednisone tapering regimen[13].

CASE REPORT

54-year-old female with a known history of type 2 diabetes mellitus for the past five years, controlled on oral hypoglycemics, presented with a 4-day history of spontaneous gum bleeding and diffuse petechial rashes across her body, particularly over the lower limbs. She denied any fever, myalgia, headache, recent infections, trauma, or medication changes. Clinical examination revealed widespread petechiae and discoloration, especially over the legs. Extraoral examination showed no signs of conjunctival hemorrhage or ecchymosis. Intraoral inspection revealed bleeding gums, although there was no evidence of local periodontal disease that could account for spontaneous hemorrhage.

Initial laboratory investigations demonstrated severe thrombocytopenia with a platelet count of 5,000/µL, which rapidly declined to 3,000/µL. Peripheral blood smear confirmed marked thrombocytopenia without atypical cells. Other hematological parameters including WBC, RBC, and hemoglobin were within normal limits. Biochemical investigations revealed normal liver and renal function. Immunologic tests including HBsAg, Anti-HBs, Anti-HCV, Anti-HIV, Anti-cardiolipin IgM/IgG, and Anti-phosphatidylserine IgM/IgG were all negative. Inflammatory markers such as ESR were normal. Coagulation profile including INR, fibrinogen, and PTT was also within normal range. Imaging revealed no hepatosplenomegaly or lymphadenopathy.

With secondary causes ruled out, a diagnosis of primary immune thrombocytopenia (ITP) was confirmed. The patient was admitted and treated initially with intravenous dexamethasone twice daily for three days, later tapered to once daily. Platelet transfusion with six units of platelet concentrate was administered during the acute phase due to the high bleeding risk. She was also started on methylprednisolone 1

mg/kg/day for 10 days and received supportive therapy including intramuscular vitamin B12 (1000 µg daily for five days) , oral vitamin C and oral vitamin D supplementation.

Following initiation of treatment, her platelet count showed marginal improvement to 11,000/µL on day 7. By day 15, hematological reassessment demonstrated a significant increase in platelet count to ,1,32,000/µL, with hematocrit of 48.06%. The petechial lesions gradually resolved, and the spontaneous gingival bleeding ceased. She was discharged on a tapering dose of oral prednisolone (Wysolone 40 mg once daily, tapered by 10 mg every 5 days), along with a continued course of vitamin B12 and vitamin D.

At two-week post-discharge, her platelet count further improved to 132,000/µL . At follow-up, the patient remained clinically stable, and no recurrence of bleeding was noted.

Laboratory Findings

Table 1. Serial Monitoring Of Parameters

Days	Day 1	Day 3	Day 7	Day 15	Normal Range
HB	14.2	13.8	13.4	14	13-17 g/dl
WBC	7800	7200	6900	8400	4.5 - 11 x 103/ul
PLT	5000	3000	11000	132000	130- 400 x 103/ul
HCT	32	30	38	48	% 36-46

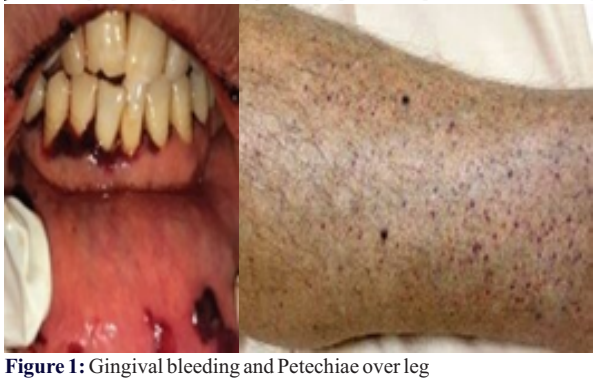

Figure 1: Gingival bleeding and Petechiae over leg

DISCUSSION

Immune thrombocytopenia (ITP) represents a complex autoimmune disorder characterized by isolated thrombocytopenia resulting from both increased platelet destruction and impaired production[12]. Our case of a 54-year-old diabetic female presenting with spontaneous gingival bleeding and profound thrombocytopenia (5,000/µL) underscores several critical aspects of ITP diagnosis and management that merit discussion.

Diagnostic Challenges

The diagnosis of ITP remains one of exclusion, requiring meticulous elimination of alternative causes of thrombocytopenia. In our patient, the absence of fever, negative serology for dengue and autoimmune markers, and normal peripheral smear findings were pivotal in differentiating ITP from infection-related thrombocytopenia or other hematologic disorders. This aligns with established diagnostic criteria where ITP is confirmed only after excluding secondary causes such as viral infections (HIV, HCV), drug-induced thrombocytopenia, and lymphoproliferative disorders[12]. The rapid platelet decline (8,000 to 5,000/µL) observed in our case further highlights the dynamic nature of ITP, where platelet counts can fluctuate dramatically without obvious triggers[1].

Therapeutic Considerations

First-line therapy with corticosteroids (dexamethasone followed by prednisolone taper) elicited a prompt but partial response in our patient, consistent with literature reporting that 70–80% of adults achieve initial platelet count improvement with steroids[3]. However, the modest rise to 11,000/µL at discharge emphasizes the variability in treatment responses, particularly in severe cases[1]. While intravenous immunoglobulin (IVIG) or thrombopoietin receptor agonists might have been considered for faster platelet recovery, our patient's gradual improvement on steroids alone supported the decision to avoid more aggressive interventions[24]. This aligns with current guidelines recommending individualized therapy based on bleeding risk rather than platelet count alone[2].

Oral Manifestations and Dental Implications

Our case exemplifies how ITP may first manifest with oral bleeding—a finding reported in <10% of cases but critical for early diagnosis[2]. Unlike typical gingivitis, spontaneous gingival hemorrhage in ITP occurs without local inflammation, a distinction that should alert clinicians to systemic etiology[2]. The absence of mucosal petechiae in our patient contrasts with classic ITP presentations but reinforces the disease's heterogeneous clinical spectrum[12]. For dental practitioners, this underscores the need to consider ITP in unexplained oral bleeding, especially when accompanied by cutaneous signs like petechiae[2].

Interdisciplinary Management

The successful outcome in this case hinged on timely hematologic referral and collaborative care. ITP patients requiring dental procedures pose unique challenges; even routine interventions like scaling warrant platelet counts >50,000/µL to minimize bleeding risk[1]. Our patient's post-treatment scaling was deferred until hematologic stabilization, illustrating the importance of coordinating care with hematologists[2]. Additionally, the use of soft-bristle toothbrushes and chlorhexidine rinses post-recovery highlights preventive strategies to mitigate oral bleeding risks in chronic ITP[2].

Broader Implications

This case adds to the limited literature on ITP diagnosed through oral bleeding, emphasizing dentists' role in identifying systemic disorders[2]. The patient's diabetes may have compounded her bleeding risk, as microvascular changes in diabetes can exacerbate thrombocytopenic hemorrhage[3]. Future studies should explore such comorbidities' impact on ITP presentation and outcomes.[5]

In conclusion, ITP remains a diagnosis of exclusion with variable therapeutic responses[1]. Spontaneous gingival bleeding, though rare, demands systemic evaluation, and interdisciplinary collaboration is essential for optimal outcomes. This case reinforces the need for heightened clinical vigilance among both dental and medical providers managing unexplained mucocutaneous bleeding.

CONCLUSION

Immune thrombocytopenia (ITP) is a multifaceted autoimmune disorder requiring a high index of suspicion for timely diagnosis, especially in atypical presentations such as isolated oral bleeding. The condition remains a diagnosis of exclusion, underscoring the importance of comprehensive evaluation to rule out secondary causes. Our case of a middle-aged diabetic female with profound thrombocytopenia and gingival bleeding highlights both the diagnostic complexity and therapeutic variability of ITP. Corticosteroids remain a cornerstone of first-line treatment, but individualized management strategies are critical, especially in severe or refractory cases. Moreover, the interdisciplinary approach involving dental and hematologic care plays a pivotal role in reducing bleeding risk and optimizing patient outcomes. This case reinforces the need for vigilance among clinicians and dental professionals alike in recognizing the systemic implications of unexplained mucocutaneous bleeding.

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