



Recurrent Hodgkin's Lymphoma with Bleeding: A Case Report

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Abstract

Introduction: One of the rare platelet dysfunction, which is impairment of receptor GPIIb/IIIa and platelet aggregation defect, is acquired Glanzmann's thrombasthenia. A common cause of this thrombasthenia is an autoantibody or plasma protein inhibitor against a normal GPIIb/IIIa glycoprotein.

Case Presentation: In this case report, a 28-year-old female with a history of treated Hodgkin's lymphoma presented with a menometrorrhagia and ecchymosis. Before that, she had no bleeding history and there was no family history bleeding tendency. Laboratory findings revealed bleeding time > 10 minutes, normal partial thromboplastin time (APTT), prothrombin time (PT), fibrinogen, and von Willebrand factor. Platelet-aggregation studies showed no aggregation to adenosine diphosphate (ADP), collagen, and a normal response to ristocetin. The clinical history, medical history, and laboratory findings supported a diagnosis of acquired Glanzmann's thrombasthenia. Imaging revealed significant lymph nodes was compatible with recurrent disease. The patient's menorrhagia and bleeding tendency were controlled by systemic chemotherapy.

Conclusions: This report shows the association of acquired thrombasthenia with the recurrent Hodgkin's lymphoma, which can be controlled by remission induction chemotherapy.

Keywords: Glanzmann's Thrombasthenia, Hodgkin's Lymphoma, Acquired Thrombasthenia

1. Introduction

Glycoprotein GPIIb/IIIa (integrin α IIb β 3) as the receptor for fibrinogen, von Willebrand's factor, and the adhesive plasma legends, is saved and expressed by platelet. Once activated, binding of plasma proteins to platelet GPIIb/IIIa causes platelet-platelet adhesion and aggregation is necessary for hemostasis. Qualitative or quantitative impairment of the GPIIb GPIIIa receptors on the platelet membrane can result in a rare hemorrhagic disorder as Glanzmann's thrombasthenia (1). Acquired Glanzmann's thrombasthenia was reported with autoimmune (systemic lupus erythematosus (SLE), idiopathic thrombocytopenic purpura (ITP)) (2) and lymphoproliferative disorders (3) that is associated with autoantibodies directed against this receptor (4). Prolonged bleeding time (BT) with normal platelet count and morphology as well as normal partial thromboplastin time (APTT) and prothrombin time (PT) characterized this thrombasthenia (5).

In this study, we report a patient with a history of treated Hodgkin's lymphoma, who presented with a hemorrhagic diathesis resembling thrombasthenia.

2. Case Presentation

A 28-year-old white female presented in May 2002 with right upper cervical and supraclavicular adenopathy.

Spiral computed tomography (CT) scan with intravenous (IV) and oral contrast revealed the left-sided moderate pleural effusion with partial lung collapse. After lymph node biopsy and staging, the patient was diagnosed with Hodgkin's disease, Stage A, nodular sclerosis (grade in BNLI classification).

The patient was treated with adriamycin, bleomycin, vinblastine, and dacarbazine (ABVD) every 2 weeks. No transfusions were required during her chemotherapy and her chemotherapy sessions were completed in June 2002. Imaging study after 6 cycles of ABVD showed complete remission; so, she was on follow-up evaluation every 3 months.

After 5 years, she presented with the left cervical adenopathy (8 × 6 × 1). Abdominal-pelvic spiral CT scan with IV and oral contrast was unremarkable. The biopsy showed recurrent disease; thus, the patient was treated with 9 cycles of cyclophosphamide, vincristine, procar-

bazine, prednisone (C-MOPP) followed by mantle field radiotherapy.

Approximately, 3 years after the chemotherapy completion, the patient returned to Shahid Modaress Hospital in Tehran with complaints of spontaneous ecchymosis on lower extremities and menorrhagia. Before that, the patient had no history of increased bleeding tendency. She did not take any medications including aspirin and non-steroidal anti-inflammatory agents. Also, she was not pregnant. Apparently her family history of abnormal bleeding was unremarkable. Upon admission for evaluation, she had normal platelet count, normal liver function tests, lactate dehydrogenase (LDH), Thyroid-stimulating hormone (TSH), normal prothrombin time and partial prothrombin time, negative direct and indirect Coombs test, and a negative human chorionic gonadotropin (BHCG). Her bleeding time was prolonged by the template method. antinuclear antibody (ANA), anti-SM, anti-RNP, anti-SSA (RO), and anti-SSB (LA) were also negative.

Platelet aggregation studies revealed normal aggregation with ristocetin, but absent aggregation with collagen, adenosine diphosphate (ADP), and arachidonic acid (Table 1).

Table 1. Agonist- Induced Platelet Aggregation Using Patient Platelets/RIPA

Agonist	Pre-Treatment	Reference/Unit, (%)
ADP 20.0 μ M/L	0	50 - 82
ADP 10.0 μ M/L	0	44 - 78
Arachidonic acid 0.5 mg/mL	17.0	50 - 85
Collagen 2.0 μ g/mL	1	43 - 85
Ristocetin 1.5 mg/mL	87	50 - 150

Thoracic and abdominal-pelvic CT scan with the contrast showed mediastinal lymph node measuring up to 16 \times 8 mm and multiple oval mass lesions in epigastric region (celiac trunk and superior mesenteric vessels the largest measuring 41 \times 27 mm in diameter to the level of inferior renal segment of aorta and multiple smaller lymph nodes in para-aortic area).

The patient did not require any transfusions including packed red cells or platelets. Retreatment began due to the relapse of her disease with GCD regimen (combination of gemcitabine, dexamethasone, and cisplatin). Three weeks after the commencement of treatment, the patient's bleeding tendency was improved. Chest, abdominal, and pelvic CT scan revealed complete response. At the final follow-up, the patient was in good health and her disease was in remission. Informed consent was taken from the patient.

3. Discussion

According to the results of the platelet functional studies and her medical history, acquired Glanzmann's thrombasthenia is the most appropriate diagnosis. She did not have any history of transfusion; thus, alloantibody have not been responsible for the platelet dysfunction.

With regard to previous case studies on association between acquired Glanzmann's thrombasthenia and Hodgkin lymphoma; the first one was a case of Hodgkin lymphoma and autoantibody against GPIIb/IIIa with disappearance of bleeding symptoms after prednisolone treatment (6). The second one was a probable lymphoid cancer in colon, which was associated with autoantibody that was cured after hemicolectomy (1). Two other cases with lymphoma were cured by lymphoma treatment.

In this case, the patient's menorrhagia and ecchymosis clearly indicated more investigation. She did not have any lymph node enlargement in examination despite of previous case reports.

However, for patients presenting with generalized bleeding, who do not have enough localized symptoms or signs, either an occult or obscure lymphoid lesion perhaps exists. As patients with a history of a lymphoproliferative disorder present with a hemorrhagic diathesis were thrombasthenia, imaging studies should be ordered promptly to identify lymphoid masses to rule out the relapse of lymphoma.

This case clearly demonstrates an occurrence of reversible acquired thrombasthenia, which can occur some months after the successful treatment of Hodgkin's lymphoma and can be a sign of relapse. An awareness of its occurrence and reversibility should help in its clinical management.

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Footnotes

Authors' Contribution: None declared.

Conflict of Interests: None declared.

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