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Recurrent Hemarthrosis Revealing Acquired Platelet Dysfunction in a Patient With Chronic Lymphocytic Leukemia: A Case Report and Literature Review

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Abstract

Hemorrhagic manifestations in chronic lymphocytic leukemia (CLL) are generally attributed to thrombocytopenia, autoimmune cytopenias, coagulation abnormalities, or treatment-related complications. Acquired platelet dysfunction remains a rare and underdiagnosed cause of bleeding.

We report a case of a 79-year-old female with Binet stage A CLL who was under active surveillance and developed recurrent spontaneous hemarthrosis of the right knee associated with rectal bleeding. Laboratory investigations revealed persistent lymphocytosis, macrocytic anemia, and moderate thrombocytopenia (111-143 × 10^9/L). Coagulation tests were normal. Platelet function test, performed twice by platelet aggregometry, demonstrated acquired platelet dysfunction characterized by major abnormalities in aggregation induced by arachidonic acid and ristocetin, with variable responses between the two studies. Acquired von Willebrand syndrome was considered unlikely. The patient was receiving dabigatran; however, hemarthrosis persisted despite discontinuation of anticoagulation, suggesting an underlying qualitative platelet dysfunction.

This case highlights acquired platelet dysfunction as a rare but clinically relevant cause of bleeding in patients with CLL, whether receiving treatment or under surveillance. In patients presenting with unexplained bleeding despite preserved or only moderately reduced platelet counts and normal coagulation tests, platelet function testing should be considered. Recognition of this underdiagnosed complication may improve diagnostic accuracy and guide appropriate therapeutic management.

Categories: Hematology, Oncology
Keywords: acquired platelet dysfunction, chronic lymphocytic leukemia, hemarthrosis, platelet aggregometry, platelet disorder

Introduction

Chronic lymphocytic leukemia (CLL) is a lymphoproliferative hematologic malignancy characterized by the clonal accumulation of mature B lymphocytes with functional abnormalities and dysregulation of immune responses. This immune dysfunction may predispose patients to infectious and autoimmune complications, particularly autoimmune hemolytic anemia (AIHA) and immune thrombocytopenic purpura (ITP) [1,2].

Bleeding manifestations in CLL are primarily related to thrombocytopenia, autoimmune cytopenias, and, in some patients, the use of antithrombotic therapies. However, accumulating evidence suggests that CLL itself may be associated with qualitative platelet dysfunction, independently of platelet count [3,4]. In patients with untreated CLL, abnormalities in platelet function have been demonstrated by whole-blood aggregometry, with reduced responses to several agonists, including adenosine diphosphate (ADP), ristocetin, thrombin receptor-activating peptide (TRAP), and collagen. However, these abnormalities were not consistently reproduced by light transmission aggregometry using platelet-rich plasma, highlighting the influence of the experimental setting on their detection [3]. Furthermore, reduced expression of platelet membrane receptors, particularly glycoprotein Ib alpha (GPIbα) and glycoprotein VI (GPVI), has been reported in a small cohort of patients with untreated CLL, suggesting a potential involvement of platelet adhesion and activation pathways [4]. These findings support the hypothesis that qualitative platelet dysfunction may contribute to bleeding risk in some patients with CLL, including those with only moderate thrombocytopenia.

The introduction of Bruton tyrosine kinase (BTK) inhibitors, particularly ibrutinib, has substantially changed the therapeutic management of CLL. However, their use, especially ibrutinib, is associated with an increased risk of bleeding, ranging from generally mild mucocutaneous manifestations to rare major

hemorrhagic events [5]. This risk is related to interference with several signaling pathways involved in platelet function, including BTK-dependent pathways downstream of GPVI and GPIb-IX-V, as well as integrin αIIbβ3 outside-in signaling [6,7]. The antiplatelet effects of ibrutinib may be further enhanced by concomitant antithrombotic therapy [5,6].

Bleeding manifestations associated with ibrutinib are most commonly low-grade, such as bruising and petechiae, although major bleeding events may also occur [3,7]. Studies have also shown that some platelet function abnormalities may be present before initiation of BTK inhibitor therapy. McGregor et al. reported platelet function abnormalities in patients with CLL that were further impaired by ibrutinib or acalabrutinib [3]. Similarly, abnormalities in platelet aggregation have been observed in patients with CLL before exposure to ibrutinib, suggesting that the disease itself may contribute to platelet dysfunction [7-9]. Barrientos et al. reported that, among patients with CLL or indolent non-Hodgkin lymphoma treated with idelalisib, concomitant use of anticoagulants or antiplatelet agents was not associated with a significant increase in bleeding events, with bleeding risk being influenced by thrombocytopenia and underlying comorbidities [10].

The management of patients with CLL may be complicated by cardiovascular comorbidities requiring anticoagulant or antiplatelet therapy. These treatments may increase bleeding risk, particularly when combined with BTK inhibitors [9,11,12]. This issue is particularly relevant in patients with concomitant platelet dysfunction. Therefore, in patients presenting with unexplained or disproportionate bleeding relative to their platelet count and conventional coagulation tests, evaluation for acquired platelet dysfunction may contribute to a more comprehensive assessment of bleeding risk and help guide therapeutic management.

Platelet disorders usually cause mucocutaneous bleeding, whereas hemarthrosis more often points to a coagulation factor deficiency. Recurrent hemarthrosis as the presenting feature of platelet dysfunction in untreated, early-stage CLL is therefore unusual and, to our knowledge, rarely reported. We report a case of a 79-year-old female with Binet stage A CLL who had not received CLL-specific therapy and who presented with recurrent spontaneous hemarthroses associated with acquired platelet dysfunction demonstrated by two sequential assessments of platelet function. Through this case and a focused review of the literature, we discuss the available evidence regarding CLL-associated platelet dysfunction, its potential pathophysiological mechanisms, its diagnostic implications, and its consequences for the management of bleeding manifestations.

Case Presentation

A 79-year-old female with Binet stage A CLL was followed in the hematology department for approximately 40 months under an active surveillance strategy. The diagnosis of CLL was established by flow cytometry, with a Matutes score of 5/5. Throughout the follow-up period, she remained clinically asymptomatic, with no constitutional symptoms, lymphadenopathy, hepatomegaly, or splenomegaly.

Serial complete blood counts demonstrated persistent lymphocytosis associated with mild cytopenias (Table *1*). At the beginning of follow-up, laboratory investigations revealed a hemoglobin level of 9.8 g/dL, a mean corpuscular volume (MCV) of 105.6 fL, a white blood cell count of 55.26 × 10^9/L, an absolute lymphocyte count of 52.31 × 10^9/L, and a platelet count of 142 × 10^9/L.

Date	Hb (g/dL)	MCV (fL)	WBC (×10⁹/L)	Lymphocytes (×10⁹/L)	Platelets (×10⁹/L)	Other laboratory findings
Reference range	12–16	80–100	4–10	1–4	150–400	LDH: 120–250 U/L; Reticulocytes: 25,000–100,000/mm³; Folate: >4 ng/mL; Ferritin: 15–150 ng/mL
Nov 2022	9.8	105.6	55.26	52.31	142	LDH: 364.1 U/L; Reticulocytes: 37,320/mm³; Ferritin: 255.2 ng/mL; Folate: 3.9 ng/mL
Mar 2023	9.8	105	46.36	41.63	111	LDH: 255 U/L
July 2023 (bleeding episode)	8.7	108.4	32.3	27.7	143	Ferritin: 111 ng/mL; Folate: 2.3 ng/mL
Aug 2023	9.9	105.4	51.8	46.8	143	Persistent platelet aggregation abnormalities (second study)

TABLE 1: Evolution of hematological and laboratory parameters during follow-up.

Hb: hemoglobin; MCV: mean corpuscular volume; WBC: white blood cells; LDH: lactate dehydrogenase.

Given the persistent macrocytic anemia, an etiological workup was performed. The reticulocyte count was 37,320/mm³, serum ferritin was 255.2 ng/mL, lactate dehydrogenase (LDH) was 364.1 U/L, haptoglobin was 0.97 g/L, folate was 3.9 ng/mL, and vitamin B12 was 297 pg/mL. Serum protein electrophoresis showed no evidence of hypogammaglobulinemia. Bone marrow aspiration did not reveal features suggestive of myelodysplastic syndrome. The macrocytic anemia was attributed to folate deficiency rather than to CLL-related bone marrow failure, and the disease was therefore classified as Binet stage A.

After several months of follow-up, the patient remained asymptomatic. Laboratory evaluation revealed anemia, with a hemoglobin level of 9.8 g/dL, a white blood cell count of 46.36 × 10^9/L, an absolute lymphocyte count of 41.63 × 10^9/L, and a platelet count of 111 × 10^9/L. The LDH level was 255 U/L.

In July 2023, the patient presented with spontaneous painful swelling of the right knee, without a history of significant trauma. Arthrocentesis confirmed hemarthrosis, requiring joint drainage. She also reported rectal bleeding that had developed over the preceding two weeks. Her medical history was notable for probable heart failure, treated with amlodipine, furosemide, and carvedilol. She was also receiving dabigatran.

Additional biochemical investigations showed no significant abnormalities in renal or hepatic function. Serum creatinine was 6.7 mg/L, aspartate aminotransferase (AST) was 18 U/L, alanine aminotransferase (ALT) was 10 U/L, and potassium was 3.7 mmol/L. Haptoglobin was 1.63 g/L, and ferritin was 111 ng/mL (reference range: 15-150 ng/mL). Vitamin B12 was 268 pg/mL, whereas folate was decreased to 2.3 ng/mL, consistent with folate deficiency.

Conventional hemostasis tests showed no significant abnormalities. The prothrombin time was 87% (reference range: 70-100%), the international normalized ratio (INR) was 1.09, the activated partial thromboplastin time (aPTT) ratio (patient/control) was 1.02, and fibrinogen was 3.4 g/L. These findings did not suggest any significant coagulation factor deficiency.

Given the occurrence of spontaneous bleeding despite a relatively preserved platelet count and normal coagulation parameters, platelet function testing was performed. Light transmission aggregometry on platelet-rich plasma revealed markedly impaired aggregation responses to adenosine diphosphate (ADP) (13.88%), collagen (21.93%), and ristocetin (24.40%), with a low response to arachidonic acid (21.14%), consistent with acquired platelet dysfunction (Table *2*).

Agonist	First aggregation (%)	Second aggregation (%)	Reference range (%)	Interpretation
ADP (10 µM)	13.88	43.14	50-100	Marked (first), moderate (second)
Collagen (5 µg/mL)	21.93	40.67	50-100	Marked (first), moderate (second)
Arachidonic acid (0.5 mg/mL)	21.14	4.14	50-100	Low (first), marked (second)
Ristocetin (0.5 mg/mL)	24.4	11.7	50-100	Marked (both studies)

TABLE 2: Results of the two platelet aggregation studies (light transmission aggregometry on platelet-rich plasma).

Grading relative to the lower reference limit: marked, <50%; moderate, 50-80%; borderline, at or just above the limit.

ADP: adenosine diphosphate.

A second platelet aggregation study showed the persistence of the platelet functional defect, although the degree of impairment varied between the two assessments. Aggregation responses were 43.14% with ADP, 40.67% with collagen, 4.14% with arachidonic acid, and 11.70% with ristocetin, corresponding to moderate abnormalities in ADP- and collagen-induced aggregation and marked abnormalities in arachidonic acid- and ristocetin-induced aggregation (Table *2*). The platelet-rich plasma contained 210 × 10^3 platelets/µL, while von Willebrand factor (VWF) activity was 187%, making acquired von Willebrand syndrome unlikely. VWF antigen, factor VIII level, and multimer analysis were not performed.

Despite discontinuation of dabigatran, the patient continued to experience recurrent spontaneous hemarthrosis of the right knee. Given the persistence of clinically significant bleeding despite moderate thrombocytopenia and the persistent abnormalities on two consecutive platelet aggregation studies, the case was discussed at a multidisciplinary meeting. Hospitalization was planned for a comprehensive geriatric assessment, nutritional optimization, and multidisciplinary therapeutic decision-making regarding both the acquired platelet dysfunction and the management of the underlying CLL.

Discussion

CLL is classically associated with bleeding manifestations that may be related to thrombocytopenia, autoimmune cytopenias, and antineoplastic or antithrombotic therapies [1,2]. However, evidence suggests that CLL may also be associated with qualitative platelet dysfunction, independently of the degree of thrombocytopenia [4,8,13]. Abnormalities in platelet function have been reported in patients with CLL, including altered responses to various agonists and changes in the expression of specific platelet receptors [4,8,13]. Our case illustrates this situation in a patient with CLL who had not received CLL-specific therapy and who developed recurrent spontaneous hemarthroses despite moderate thrombocytopenia and unremarkable conventional coagulation tests.

In our patient, the moderate thrombocytopenia, with no evidence supporting immune thrombocytopenic purpura (ITP) or massive bone marrow infiltration, was insufficient to fully explain the recurrent hemarthroses. The persistence of bleeding manifestations after discontinuation of dabigatran, together with the persistence of the platelet aggregation abnormalities in two samples, was consistent with acquired platelet dysfunction, particularly involving responses to arachidonic acid and ristocetin. The markedly reduced ristocetin-induced aggregation despite preserved or elevated VWF activity suggests an abnormality involving platelet function rather than an evident VWF deficiency.

Dmitrieva et al. demonstrated that CLL may be associated with platelet function abnormalities before initiation of ibrutinib, particularly reduced ADP-induced aggregation. During ibrutinib therapy, 28 of 50 patients (56%) developed bleeding manifestations, mainly petechiae and ecchymoses [13]. Kazianka et al. showed that ibrutinib significantly reduced ristocetin-induced platelet aggregation, and that this reduction was associated with bleeding manifestations and was more pronounced in patients receiving concomitant antithrombotic therapy [14]. These findings suggest that platelet dysfunction associated with CLL may contribute to bleeding risk, potentially in combination with thrombocytopenia and the antiplatelet effects of ibrutinib (Table *3*) [13,14].

Author	Study population	Treatment	Bleeding manifestations	Platelet function abnormalities
Barton et al. (2025) [8]	Patient with CLL	Ibrutinib	Hemorrhagic pericardial effusion	Platelet dysfunction associated with CLL and exacerbated by ibrutinib (no aggregation response to collagen or ADP)
Emelina et al. (2021) [11]	Patients with CLL and atrial fibrillation	Ibrutinib + DOAC	Epistaxis, hematuria, other bleeding events	Platelet function was not directly assessed; platelet count and platelet-count variability were not significantly associated with bleeding
Gendlin et al. (2019) [12]	Patients with CLL	Ibrutinib + antithrombotic therapy	Bruising, epistaxis, hematuria	No significant differences in platelet parameters were observed between patients with and without bleeding. DOAC discontinuation was mainly related to thrombocytopenia (<100 × 10^9/L)
Georgantopoulos et al. (2019) [9]	24,198 patients with CLL	Pre-BTK inhibitor era	Major hemorrhage	Not assessed
McGregor et al. (2018) [3]	Functional platelet study in patients with CLL	With or without BTK inhibitors	Not reported	Broad platelet-function defect involving ADP, ristocetin, TRAP, and collagen
Dmitrieva et al. (2020) [13]	50 patients with CLL + 16 with MCL	Before and during ibrutinib	Bleeding in 28/50 (56%) of CLL patients, mainly petechiae and ecchymoses	Before ibrutinib: reduced ADP-induced platelet aggregation; during ibrutinib: changes in ADP- and collagen-induced aggregation and other platelet activation parameters
Present case (2026)	1 patient with Binet stage A CLL	No specific CLL treatment + DOAC	Recurrent spontaneous hemarthrosis	Two platelet aggregation studies showing persistent abnormalities across multiple agonists (ADP, collagen, arachidonic acid, ristocetin), with variable responses between assessments

TABLE 3: Comparison of the main published studies on bleeding manifestations and platelet dysfunction in chronic lymphocytic leukemia with the present case.

ADP: adenosine diphosphate; BTK: Bruton tyrosine kinase; CLL: chronic lymphocytic leukemia; DOAC: direct oral anticoagulant; MCL: mantle cell lymphoma; TRAP: thrombin receptor-activating peptide.

McGregor et al. also demonstrated that untreated CLL may be associated with impaired platelet function, particularly in response to ADP, ristocetin, TRAP, and collagen, mainly detected by whole-blood multiple electrode aggregometry (MEA). This dysfunction was more pronounced in patients receiving ibrutinib, suggesting an additive effect of treatment on a pre-existing functional abnormality [3]. These findings support the possibility of CLL-associated platelet dysfunction that is not solely explained by platelet count.

The pathophysiological mechanisms underlying platelet dysfunction in CLL remain incompletely understood. Abnormalities involving several pathways implicated in platelet adhesion and activation have been reported in patients with untreated CLL. Elaskalani et al. reported reduced platelet responses to collagen, ristocetin, and ADP, together with decreased GPVI expression, in patients with untreated CLL [15]. These abnormalities could contribute to the functional impairment observed in our patient, although GPVI and the related signaling pathways were not directly investigated in our case.

The role of dabigatran should also be considered. Anticoagulant and antiplatelet therapies may increase bleeding risk in patients with CLL, particularly when combined with BTK inhibitors (Table 3) [9,11,12]. In a large study of 24,198 patients with CLL conducted before the BTK inhibitor era, the incidence of major hemorrhage was 1.9 events per 100 person-years. Concomitant anticoagulant and antiplatelet therapy, anticoagulant therapy alone, and antiplatelet therapy alone were each associated with an increased risk of major hemorrhage [9]. In our patient, dabigatran may have contributed to the severity of the bleeding manifestations. However, their persistence after discontinuation, together with the persistence of the platelet aggregation abnormalities, is compatible with concomitant platelet dysfunction. Nevertheless, based on this single observation, it cannot be determined whether dabigatran was merely an aggravating factor or a major contributor to the bleeding episodes.

Our observation highlights that qualitative platelet dysfunction may contribute to bleeding manifestations

in patients with CLL, including those who have not received CLL-specific therapy and who have only moderate thrombocytopenia. In our patient, the persistence of hemarthroses after discontinuation of dabigatran and the persistence of platelet aggregation abnormalities in two successive samples are consistent with acquired platelet dysfunction, possibly related to CLL, although a direct causal relationship cannot be established. In patients with unexplained or disproportionate bleeding relative to platelet count and conventional coagulation tests, assessment of platelet function may therefore be considered. Our report has limitations. Including unavailability of the exact timing of each bleeding episode relative to dabigatran withdrawal, a standardized bleeding score, factor XIII assay, a complete von Willebrand panel, and follow-up, so other contributors to bleeding cannot be fully excluded. Larger prospective studies are needed to determine the frequency, underlying mechanisms, and clinical relevance of these functional abnormalities in CLL.

Conclusions

Our case suggests that acquired platelet dysfunction may contribute to bleeding in patients with CLL, even in the early stages of the disease, in the presence of only moderate thrombocytopenia and without CLL-directed treatment. In our patient, hemarthroses persisted after dabigatran discontinuation, and platelet aggregation abnormalities were reproducible in two separate samples, supporting the presence of a qualitative platelet defect. When bleeding in patients with CLL remains unexplained or appears disproportionate to the platelet count and routine coagulation results, platelet function testing may be warranted. Such abnormalities have been reported in CLL both before and during treatment with BTK inhibitors. Further prospective studies are needed to establish the prevalence, underlying mechanisms, and clinical significance of platelet dysfunction in CLL.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Disclosures

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