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Original Research Article

MedPeer Publisher
Abbreviated Key Title: MedPeer
ISSN : 3066-2737
homepage: https://www.medpeerpublishers.com

Acquired Hemophilia Following Bullous Pemphigoid in an Elderly Patient: A Case Report and Literature Review

DOI: 10.70780/medpeer.000QGTM

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ABSTRACT

Acquired hemophilia A (AHA) is a rare autoimmune bleeding disorder caused by autoantibodies directed against coagulation factor VIII, leading to spontaneous or trauma-related hemorrhage. Unlike congenital hemophilia, AHA predominantly affects the elderly and may be associated with autoimmune diseases, malignancies, pregnancy, or medications. Bullous pemphigoid (BP), a subepidermal autoimmune blistering disorder, has been reported as a potential trigger for AHA due to persistent immune dysregulation.

We report the case of an 84-year-old woman with a history of bullous pemphigoid treated successfully with topical corticosteroids eight months prior, who presented with a rapidly progressive purpuric plaque and uncontrolled bleeding following minor trauma. Laboratory evaluation revealed isolated prolongation of activated partial thromboplastin time (aPTT: 78 seconds) with normal prothrombin time. Mixing studies failed to correct the aPTT, suggesting the presence of an inhibitor. Further workup confirmed markedly reduced factor VIII activity (1%) and the presence of factor VIII inhibitors (18 Bethesda units), establishing the diagnosis of acquired hemophilia A. The patient was treated with recombinant activated factor VII (rFVIIa) for acute bleeding control and immunosuppressive therapy with prednisone and cyclophosphamide for inhibitor eradication. Complete remission was achieved at 12 weeks, with normalization of factor VIII levels and disappearance of inhibitors.

This case highlights the importance of considering acquired hemophilia A in elderly patients presenting with unusual bleeding manifestations, particularly in the context of prior autoimmune disease. The association between bullous pemphigoid and AHA, though rare, underscores the need for vigilance regarding immune-mediated complications in patients with autoimmune blistering disorders.

KEYWORDS :

Dermatology, hematology, acquired hemophilia A; bullous pemphigoid; factor VIII inhibitors; autoimmune disease; elderly; bleeding disorder

MAIN ARTICLE

INTRODUCTION

Acquired hemophilia A (AHA) is a rare but potentially life-threatening autoimmune bleeding disorder characterized by the development of autoantibodies (inhibitors) directed against coagulation factor VIII (FVIII).[1] Unlike congenital hemophilia, which is an X-linked recessive disorder affecting males from birth, AHA occurs sporadically in individuals with no prior personal or family history of bleeding disorders, with an estimated incidence of 1.5 per million per year.[1][2]

AHA predominantly affects two distinct populations: young women in the peripartum period and elderly individuals, with a median age at diagnosis of approximately 75 years.[1][2] In approximately 50% of cases, AHA is associated with an underlying condition, including autoimmune diseases (systemic lupus erythematosus, rheumatoid arthritis, inflammatory bowel disease), malignancies (solid tumors, lymphoproliferative disorders), pregnancy, and medications (penicillins, sulfonamides, interferon).[1][3] The remaining cases are classified as idiopathic.

Bullous pemphigoid (BP) is the most common autoimmune subepidermal blistering disorder, predominantly affecting elderly individuals with a mean age of onset of 80 years.[4] BP is characterized by autoantibodies directed against hemidesmosomal proteins BP180 and BP230, leading to subepidermal blister formation.[4] Importantly, BP has been associated with various autoimmune phenomena and immune dysregulation, which may predispose affected individuals to other autoimmune conditions, including AHA.[5][6]

The association between BP and AHA, though rare, has been documented in several case reports, suggesting that the persistent immune activation characteristic of BP may trigger the development of FVIII inhibitors.[5][6][7] Recognition of this association is clinically important, as delayed diagnosis of AHA can lead to severe, potentially fatal hemorrhagic complications.

We report a case of AHA occurring in an elderly woman eight months after successful treatment of BP, and provide a comprehensive review of the literature on this rare association.

CASE PRESENTATION

An 84-year-old woman presented to the emergency department with a rapidly progressive red, edematous plaque on her right upper limb following a fall down a flight of stairs 24 hours earlier. The lesion was associated with persistent bleeding from a superficial wound that had not responded to local pressure.

Past medical history

The patient had a history of type 2 diabetes mellitus controlled with metformin and bullous pemphigoid diagnosed eight months prior. The BP had presented with tense bullae on erythematous bases involving the trunk and extremities. Diagnosis was confirmed by skin biopsy showing subepidermal blistering with eosinophilic infiltrate, and direct immunofluorescence demonstrating linear IgG and C3 deposits along the basement membrane zone. Serum anti-BP180 antibodies were positive (>200 U/mL). The patient was treated with superpotent topical corticosteroids (clobetasol propionate 0.05% cream) with complete resolution of lesions within six weeks. She had been in clinical remission without maintenance therapy for the past six months.

The patient had no prior personal or family history of bleeding disorders. She denied any recent medication changes, including anticoagulants or antiplatelet agents. There was no history of malignancy, recent infections, or other autoimmune diseases.

Clinical examination

On presentation, the patient was afebrile (temperature 36.8°C) with stable vital signs: blood pressure 135/78 mmHg, heart rate 82 beats per minute, respiratory rate 16 breaths per minute, and oxygen saturation 97% on room air.

Dermatological examination of the right upper limb revealed a large, well-demarcated purpuric plaque extending from the dorsum of the hand to the mid-forearm, measuring approximately 15 × 8 cm (Figure 1). The plaque was non-blanching on diascopy, consistent with extravasated blood. Marked edema was present, and the skin was tense but not warm. A superficial laceration (approximately 2 cm) on the dorsal hand continued to ooze blood despite local pressure. No fluctuance or crepitus was noted. Examination of other sites revealed scattered ecchymoses on the lower extremities and trunk, which the patient reported had appeared spontaneously over the preceding two weeks without significant trauma.

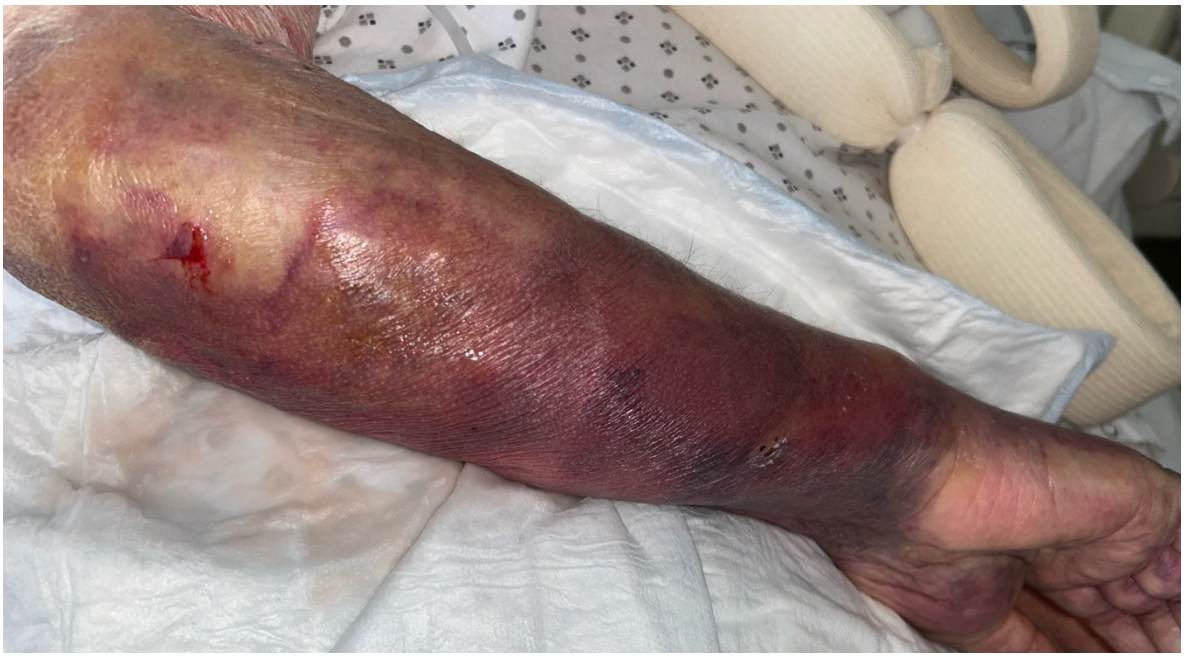

Figure 1 : Large, sharply demarcated purpuric and ecchymotic plaque involving the dorsum of the hand and extending proximally, associated with marked edema and tense skin. Focal superficial hemorrhagic erosions are present within the lesion.

There was no lymphadenopathy, hepatosplenomegaly, or joint swelling. No mucosal bleeding, hemarthrosis, or signs of deep tissue hematoma were observed. The remainder of the physical examination was unremarkable.

Initial clinical impression

Given the erythema, edema, and recent trauma, the initial clinical suspicion included erysipelas or cellulitis. However, the absence of fever, the non-blanching purpuric nature of the lesion, and the persistent bleeding from a minor wound raised concern for an underlying bleeding disorder. Dermatology consultation was requested.

Laboratory investigations

Initial laboratory evaluation revealed the following:

- Complete blood count: Hemoglobin 10.2 g/dL (normal 12-16), white blood cell count 8,400/µL with normal differential, platelet count 245,000/µL (normal)

- Coagulation studies:

  - Prothrombin time (PT): 12.5 seconds (normal 11-13.5 seconds)

  - International normalized ratio (INR): 1.0

  - Activated partial thromboplastin time (aPTT): 78 seconds (normal 25-35 seconds) —

markedly prolonged

- Fibrinogen: 320 mg/dL (normal 200-400 mg/dL)

- D-dimer: 0.8 µg/mL (mildly elevated)

- Liver function tests: Within normal limits

- Renal function: Creatinine 1.1 mg/dL, eGFR 52 mL/min/1.73m²

The isolated prolongation of aPTT with normal PT, platelet count, and fibrinogen prompted further investigation for intrinsic pathway abnormalities.

Mixing study

A mixing study was performed by combining patient plasma with normal pooled plasma in a 1:1 ratio:

- Immediate aPTT (0 hours): 52 seconds (partial correction)

- Incubated aPTT (2 hours at 37°C): 68 seconds (failure to correct)

The failure of the aPTT to correct after incubation was highly suggestive of a time- and temperature-dependent inhibitor, characteristic of factor VIII inhibitors.

Factor assays and inhibitor quantification

- Factor VIII activity: 1% (normal 50-150%) — **severely reduced**

- Factor IX activity: 85% (normal)

- Factor XI activity: 92% (normal)

- Factor XII activity: 78% (normal)

- von Willebrand factor antigen: 145% (normal)

- von Willebrand factor activity (ristocetin cofactor): 138% (normal)

- Factor VIII inhibitor (Bethesda assay): **18 Bethesda units (BU)** — confirming high-titer inhibitor

Additional workup

Given the association of AHA with underlying conditions, additional investigations were performed:

- Antinuclear antibodies (ANA): Negative

- Anti-double-stranded DNA antibodies: Negative

- Rheumatoid factor: Negative

- Serum protein electrophoresis: No monoclonal gammopathy

- Computed tomography of chest, abdomen, and pelvis: No evidence of malignancy or lymphadenopathy

- Anti-BP180 antibodies: 45 U/mL (previously >200 U/mL, indicating partial serological remission of BP)

Diagnosis

Based on the clinical presentation of spontaneous and trauma-induced bleeding, isolated prolonged aPTT, severely reduced factor VIII activity, and presence of high-titer factor VIII inhibitors, a diagnosis of **acquired hemophilia A** was established. The temporal association with prior bullous pemphigoid suggested BP-associated immune dysregulation as the likely trigger.

Management

The patient was admitted to the hematology unit for management. Treatment was initiated according to established guidelines for AHA:[1][2]

Acute bleeding control:

- Recombinant activated factor VII (rFVIIa, NovoSeven): 90 μg/kg intravenously every 2-3 hours until bleeding cessation

- Local hemostatic measures: Pressure dressing, topical tranexamic acid

- Transfusion support: Two units of packed red blood cells for symptomatic anemia

Inhibitor eradication (immunosuppressive therapy):

- Prednisone: 1 mg/kg/day (75 mg daily) orally

- Cyclophosphamide: 1.5 mg/kg/day (100 mg daily) orally, initiated on day 3

- Proton pump inhibitor and calcium/vitamin D supplementation for corticosteroid-related prophylaxis

- Pneumocystis jirovecii prophylaxis with trimethoprim-sulfamethoxazole

Clinical course

The bleeding from the superficial wound was controlled within 24 hours of initiating rFVIIa therapy. The purpuric plaque gradually resolved over the following two weeks, leaving residual ecchymotic discoloration. No new bleeding episodes occurred during hospitalization.

Serial monitoring of coagulation parameters showed gradual improvement:

- Week 2: aPTT 55 seconds, FVIII activity 8%, inhibitor titer 12 BU

- Week 4: aPTT 42 seconds, FVIII activity 25%, inhibitor titer 4 BU

- Week 8: aPTT 38 seconds, FVIII activity 48%, inhibitor titer 1.2 BU

- Week 12: aPTT 32 seconds, FVIII activity 72%, inhibitor titer 0.6 BU (below the detection threshold of the Bethesda assay)

Complete remission, defined as normalization of FVIII activity (>50%) and inhibitor titer below the detection threshold (0.6 BU), was achieved at 12 weeks. Prednisone was tapered gradually over the subsequent 8 weeks, and cyclophosphamide was discontinued after a total treatment duration of 6 weeks.

Follow-up

At 6-month follow-up, the patient remained in complete remission with no recurrence of bleeding. FVIII activity was 95%, and no inhibitors were detectable. There was no clinical or serological relapse of bullous pemphigoid. The patient continues to be monitored with periodic coagulation studies.

DISCUSSION

This case illustrates the rare but clinically significant association between bullous pemphigoid and acquired hemophilia A, highlighting the importance of considering AHA in elderly patients with autoimmune diseases who present with unusual bleeding manifestations.

Acquired hemophilia A: Clinical features

AHA is characterized by a distinct bleeding phenotype that differs from congenital hemophilia.[1][2] While patients with congenital hemophilia typically present with hemarthroses and deep muscle hematomas, patients with AHA more commonly develop extensive subcutaneous bleeding (ecchymoses, purpura), soft tissue hematomas, and mucosal bleeding.[1] Hemarthroses are rare in AHA. This difference in bleeding pattern is thought to reflect the typically lower residual FVIII activity in congenital hemophilia compared to the variable levels seen in AHA.[2]

The bleeding in AHA can be severe and life-threatening, with mortality rates historically reported as high as 20-30%, although contemporary management has improved outcomes significantly.[1][8] Common causes of death include intracranial hemorrhage, gastrointestinal bleeding, and complications of treatment.

Association between bullous pemphigoid and acquired hemophilia A

The association between BP and AHA, though rare, has been documented in multiple case reports.[5][6][7][9][10] Table 1 summarizes the published cases of AHA associated with BP.

Table 1. Published cases of acquired hemophilia A associated with bullous pemphigoid

Author (Year)	Age/Sex	BP treatment	Interval BP to AHA	FVIII activity	Inhibitor titer (BU)	AHA treatment	Outcome	Reference
Soria et al. (2007)	74/M	Systemic CS	Concurrent	<1%	110	CS + AZA + rFVIIa + IVIG	Remission	[5]
Qiu et al. (2012)	60/F	Systemic CS	3 months	<1%	NR	CS + CYC + IVIG	Remission	[6]
Siah & Harries (2014)	NR/NR	Topical CS	NR	NR	NR	CS + RTX	Remission	[9]
Fakprapai & Wattanakrai (2019)	68/F	Systemic CS	11 months	<1%	High	CS + CYC + rFVIIa	Remission	[10]
Binet et al. (2017)	NR/NR	NR	NR	NR	NR	CS + methylprednisolone	Remission	[11]
Fu et al. (2022)	NR/NR	NR	Post-vaccine	NR	NR	CS	Remission	[12]
Liu & Xu (2025)	91/F	NR	Concurrent	NR	NR	CS + CYC	Remission	[13]
Present case	**84/F**	**Topical CS**	**8 months**	**1%**	**18 BU**	**CS + CYC + rFVIIa**	**Remission**	**—**

[5][6][7][9][10][11][12]

CS: corticosteroids; CYC: cyclophosphamide; IVIG: intravenous immunoglobulin; RTX: rituximab; AZA: azathioprine; BU: Bethesda units

Review of the literature reveals several important observations:

1. **Temporal relationship**: AHA typically develops 3-12 months after the diagnosis of BP, often after apparent clinical remission of the blistering disease.[5][6][7]

2. **Elderly predominance**: All reported cases occurred in patients over 70 years of age, reflecting the shared demographic of both conditions.[5][6][7]

3. **Treatment independence**: AHA has been reported following various BP treatments, including topical corticosteroids, systemic corticosteroids, and dapsone, suggesting that the association is related to the underlying immune dysregulation rather than specific therapy.[5][6][7]

4. **Favorable prognosis**: Most patients achieved remission with immunosuppressive therapy, although one fatal case has been reported.[5][6][7]

Pathophysiology

The pathophysiological link between BP and AHA remains incompletely understood, but several mechanisms have been proposed:[5][6][13]

1. **Epitope spreading**: The chronic autoimmune response in BP may lead to epitope spreading, whereby the immune system develops reactivity against additional self-antigens, including FVIII.[13]

2. **Shared immunological milieu**: Both BP and AHA are characterized by Th2-predominant immune responses and the production of pathogenic IgG autoantibodies. The immunological environment that permits anti-BP180/BP230 antibody production may also facilitate anti-FVIII antibody development.[5][6]

3. **B-cell dysregulation**: Elderly patients with autoimmune diseases may have underlying B-cell dysregulation that predisposes to multiple autoantibody production.[13]

4. **Genetic susceptibility**: Certain HLA haplotypes have been associated with both BP and AHA, suggesting shared genetic risk factors.[5]

Diagnosis

The diagnosis of AHA should be suspected in any patient presenting with:[1][2]

- New-onset bleeding without prior personal or family history of bleeding disorders

- Isolated prolongation of aPTT with normal PT

- Failure of aPTT to correct on mixing study (especially after incubation)

- Severely reduced FVIII activity with detectable FVIII inhibitors

The Bethesda assay quantifies inhibitor titer and guides treatment decisions. Inhibitors are classified as low-titer (5 BU) or high-titer (≥5 BU), with high-titer inhibitors requiring bypassing agents for acute bleeding control.[1][2]

Differential diagnosis

The differential diagnosis of isolated aPTT prolongation includes:[1]

Condition	**Key distinguishing features**
Acquired hemophilia A	Bleeding symptoms, low FVIII, FVIII inhibitor positive
Lupus anticoagulant	Usually no bleeding (thrombotic tendency), positive LA tests
Factor deficiency (VIII, IX, XI, XII)	Corrects on mixing study, specific factor low
von Willebrand disease	Low VWF antigen/activity, may have low FVIII
Heparin contamination	Corrects with heparinase, thrombin time prolonged

[1][2]

Treatment

Management of AHA involves two parallel strategies:[1][2][8]

1. Acute bleeding control:

For patients with active bleeding, hemostatic therapy is essential. Options include:

- **Bypassing agents**: Recombinant activated factor VII (rFVIIa, 90 μg/kg every 2-3 hours) or activated prothrombin complex concentrate (aPCC, FEIBA, 50-100 U/kg every 8-12 hours)

- **Recombinant porcine FVIII (susoctocog alfa)**: May be effective if cross-reactivity with porcine FVIII is low

- **Emicizumab**: A bispecific antibody mimicking FVIII function, increasingly used in AHA

2. Inhibitor eradication:

Immunosuppressive therapy is essential to eliminate the autoantibody and restore normal hemostasis. First-line options include:[1][2][8]

- **Corticosteroids alone**: Prednisone 1 mg/kg/day (for low-titer inhibitors or frail patients)

- **Corticosteroids plus cyclophosphamide**: Prednisone 1 mg/kg/day + cyclophosphamide 1.5-2 mg/kg/day (preferred for high-titer inhibitors)

- **Corticosteroids plus rituximab**: Prednisone 1 mg/kg/day + rituximab 375 mg/m² weekly × 4 (alternative for high-titer inhibitors or cyclophosphamide contraindication)

In our patient, the combination of prednisone and cyclophosphamide achieved complete remission at 12 weeks, consistent with reported response rates of 70-80% with this regimen.[1][8]

Prognosis

With contemporary management, the prognosis of AHA has improved significantly. Complete remission is achieved in approximately 70-80% of patients, with a median time to remission of 5-8 weeks.[1][8] However, relapse occurs in 15-20% of patients, necessitating long-term surveillance.[1] Mortality remains significant, primarily due to bleeding complications (3-8%) and treatment-related adverse events (infections, thrombosis).[1][8]

Clinical implications

This case has several important implications for clinical practice:

1. **Vigilance in autoimmune disease**: Patients with autoimmune blistering disorders, particularly BP, should be monitored for signs of bleeding that may indicate the development of AHA.

2. **Recognition of atypical bleeding**: Elderly patients presenting with extensive purpura, soft tissue hematomas, or uncontrolled bleeding from minor wounds should be evaluated for AHA, even in the absence of prior bleeding history.

3. **Laboratory evaluation**: Isolated aPTT prolongation in a bleeding patient should prompt mixing studies and factor assays to exclude AHA.

4. **Multidisciplinary management**: Collaboration between dermatologists and hematologists is essential for optimal management of patients with BP who develop AHA.

5. **Long-term follow-up**: Patients who achieve remission should be monitored for relapse, with periodic assessment of FVIII activity and inhibitor titers.

CONCLUSIONS

This case emphasizes that acquired hemophilia A should be suspected in elderly patients presenting with rapidly progressive purpura and uncontrolled bleeding, especially in the context of prior autoimmune disease such as bullous pemphigoid. The characteristic laboratory findings of isolated aPTT prolongation, severely reduced FVIII activity, and detectable FVIII inhibitors are diagnostic.

The association between BP and AHA, though rare, underscores the importance of vigilance regarding immune-mediated complications in patients with autoimmune blistering disorders.

Prompt recognition and treatment with hemostatic and immunosuppressive therapy are essential to prevent severe hemorrhagic complications and achieve remission.

Increased awareness of this association among dermatologists, hematologists, and emergency physicians may facilitate early diagnosis, appropriate management, and improved patient outcomes.

DECLARATIONS

Ethics statement: Written informed consent was obtained from the patient for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal.
Conflict of interest: The authors declare no conflicts of interest.
Funding: This research received no specific grant from any funding agency.
Author contributions: All authors contributed to patient care, data collection, manuscript drafting, and critical revision. All authors approved the final version.

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