A Large Spontaneous Abdominal Bruise in a 60-Year-Old Woman: A Case Report

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Key Words
Acquired hemophilia A • Coagulation disorder • Factor VIII • Spontaneous bruising

Abstract
Objective: To report a case of severe abdominal bruising successfully diagnosed using systematic hemostatic investigations. Clinical Presentation and Intervention: A 60-year-old woman developed a large spontaneous nontraumatic, painless bruise over the right lower abdominal wall. Computed tomographic scan of the abdomen showed a subcutaneous hematoma beneath the site of the obvious bruise diagnosed as a hematoma. Laboratory tests revealed an activated partial thromboplastin time of 53 s and a factor VIII level of <1%, consistent with the diagnosis of acquired hemophilia A. The patient was treated with steroids and immunosuppressants and her factor VIII levels gradually normalized. Conclusion: This case report showed that whenever there is spontaneous bruising, factor VIII deficiency should be considered as one of the important differential diagnosis.

Case Report
A 60-year-old woman developed a large spontaneous, nontraumatic, painless bruise over the right lower abdominal wall. The lesion started as a small knot and progressively increased in size (fig. 1). Past medical history revealed fibromyalgia, spinal stenosis, bilateral hip bursitis, and hypercholesterolemia. Surgical history included tonsillectomy, temporomandibular joint sur-
Thromboplastin time (APTT) was 53 s (normal). Converting enzyme and rheumatoid factor were not consistent. Sjögren’s syndrome antibodies (anti-SSA, anti-SSB), angiotensin-antibodies, cytoplasmic antineutrophil cytoplasmic antibodies, C3, C4, total complement, peripheral antineutrophil cytoplasmic extractable nuclear antigen, anticardiolipin, C-reactive protein, cancer with a chest X-ray, computed tomography scan, and bone marrow biopsy, all of which were normal. After the treatment, the APTT levels normalized within a few weeks and her factor VIII activity increased from 1 to >21% with no additional bleeding episodes. She was followed up weekly, then monthly as bleeding symptoms decreased and factor VIII levels normalized.

**Discussion**

Acquired hemophilia A is a rare disease associated with severe bleeding complications. It is typically caused by a congenital deficiency of factor VIII, but an acquired form due to inhibitors to factor VIII occurs later in life [3]. The term ‘acquired hemophilia’ is derived from the fact that the inhibitors to factor VIII create a functional coagulation factor VIII deficiency in individuals who have no prior history of bleeding [4]. Unlike congenital hemophilia A, where hemarthrosis is a typical feature, the bleeding pattern in acquired hemophilia A is different in the sense that these patients experience hemorrhages into the skin, muscles or soft tissues, and mucous membranes, as presented in our case. Moreover, there is no known genetic inheritance pattern and thus this condition occurs equally in men and women [5, 6]. The incidence of acquired hemophilia increases with age (the median age at presentation is somewhere between 60 and 67 years) as in the case of the 60-year-old woman. An underlying pathology like autoimmune disorders, malignancy, or pregnancy is observed in 50% of cases, but since all these workups were negative, our case falls into the remaining 50% of cases that are idiopathic [4]. The timely detection of this condition is critical, since early therapy directed towards achieving hemostasis and inhibitor eradication is life-saving [3]. The diagnosis of acquired hemophilia A in a patient with no previous personal or family history of bleeding is typically based on two factors: prolonged APTT and a normal prothrombin time indicating an abnormality in the early part of the intrinsic coagulation cascade. In mixing studies, correction of APTT implies a deficiency, whereas persistence of an abnormally prolonged APTT suggests the presence of an inhibitor [7]. In our case, the patient’s APTT was 53 s with factor VIII levels of <1% indicating acquired hemophilia A. Treatment of acquired hemophilia is aimed at controlling hemorrhage and ameliorating autoantibody formation [8]. Therapy includes immunosuppression with high-dose corticosteroids, and cyclophosphamide, or with factor VIII supplements [8]. The patient in our case was treated with prednisone 40 mg/day and Cytoxan 50 mg/day until the bleeding resolved and her factor VIII levels normalized.

**Fig. 1.** Spontaneous bruising in the anterior abdominal wall.
level normalized. During the acute phase, patients with low-titer inhibitors can be treated using human factor VIII concentrates, whereas in patients with high titer it is not possible to overcome the inhibiting activity by raising the factor VIII level. Therefore, factor VIII bypassing agents, such as activated prothrombin complex concentrates or recombinant activated factor VII, are used [4].

**Conclusion**

This report showed a case of acquired factor VIII deficiency presenting as a single large ecchymosis. It also demonstrated that awareness of how hemophilia A presents, and the initiation of a thorough and timely workup saved her life.

**References**