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CASE REPORT

A Case of Hemorrhagic Shock Due to Abdominal Hemorrhage in Hemophilia A: The Role of Laboratory Medicine in Multidisciplinary Team

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ABSTRACT

Background: **Spontaneous abdominal hemorrhage due to hemophilia A is one of the most lethal complications, progressing rapidly and potentially leading to hemorrhagic shock or death.**
Methods: **We report a case of a 38-year-old male patient with hemophilia A who presented with massive spontaneous abdominal hemorrhage, leading to a large hematoma and severe hemorrhagic shock. Upon admission, the patient showed unstable vital signs and a significant decline in hemoglobin levels, indicating an extremely perilous condition that required immediate multidisciplinary collaborative intervention.**
Results: **A multidisciplinary team was assembled, including specialists from emergency medicine, intensive care, hematology, cardiothoracic surgery, laboratory medicine, and radiology. The team quickly diagnosed active abdominal hemorrhage and performed successful catheter-directed arterial embolization. Despite ongoing hemoglobin decline after the procedure, the multidisciplinary team employed targeted laboratory monitoring and assessments to identify underlying causes and adjust coagulation factor replacement and transfusion strategies, effectively stabilizing hemoglobin levels. During recovery, the patient developed progressive jaundice, which was determined to be due to the absorption of the large hematoma, thus preventing unnecessary invasive procedures.**
Conclusions: **This case underscores the importance of a multidisciplinary approach in managing complex critical conditions and highlights the vital role of laboratory medicine in accurate diagnosis, decision-making, and management of complications.**
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KEYWORDS

hemophilia A, abdominal hemorrhage, hemorrhagic shock, multidisciplinary team, laboratory medicine

INTRODUCTION

Hemophilia A is an X-linked recessive disorder caused by a factor VIII (FVIII) deficiency, typically leading to bleeding in joints and muscles [1]. Spontaneous abdominal hemorrhage is a rare but life-threatening complication that can rapidly progress to hemorrhagic shock with high mortality [2]. Managing these cases is complex due to the coexistence of bleeding and coagulopathy, necessitating a multidisciplinary team (MDT) for effective treatment [3]. This report details the successful MDT management of a patient with hemophilia A who

presented with a massive abdominal hematoma and shock. We describe the entire clinical course - from diagnosis and intervention to managing complications like postoperative bleeding and jaundice - to highlight the pivotal roles of the MDT and laboratory medicine, offering a reference for similar critical cases.

CASE PRESENTATION

General information

A 38-year-old male was admitted to the hospital on January 2, 2025, at 01:10, with a chief complaint of abdominal pain lasting four hours. The patient reported a sudden onset of diffuse abdominal pain, primarily localized to the right side, without any identifiable precipitating factors. The pain was continuous and accompanied by dizziness and general weakness. He denied any bowel movements, passage of gas, nausea, or vomiting, and showed no signs of fever, chills, or gastrointestinal bleeding.

Prior to admission, he sought care at an outside facility, where an abdominal ultrasound indicated a large heterogeneous mass in the right abdomen, with further evaluation pending.

The patient had a known history of hemophilia A and had not undergone regular treatment.

Physical examination on admission

Upon examination, vital signs were as follows: temperature 36.8℃, pulse 98 bpm, respiratory rate 15 breaths/minute, and blood pressure 95/53 mmHg. The abdominal examination revealed distension and tense abdominal muscles, with a hard mass palpable in the right abdomen, measuring approximately 15 cm x 16 cm. No subcutaneous ecchymosis or petechiae were observed on the abdominal wall or back.

Laboratory tests and imaging studies

Initial laboratory tests (January 2)

Complete Blood Count (CBC): White Blood Cell Count (WBC): 13.97 x 10^9/L (↑), Neutrophil Percentage (NEUT%): 86.0% (↑), Lymphocyte Percentage (LYMPH%): 8.2% (↓), Red Blood Cell Count (RBC): 3.7 x 10^{12}/L (↓), Hemoglobin (HGB): 103 g/L (↓), Hematocrit (HCT): 31.6% (↓).

Coagulation profile: Activated Partial Thromboplastin Time (APTT): 74.1 seconds (↑↑), significantly prolonged, consistent with the coagulation dysfunction characteristic of acute bleeding in hemophilia A.

Other laboratory indicators: Procalcitonin (PCT): 0.076 ng/mL (↑), Lactate (LAC): 3.1 mmol/L (↑), Creatine Kinase (CK): 560.00 U/L (↑), Lactate Dehydrogenase (LDH): 263 U/L (↑), Myoglobin (MYO): 367.0 ng/mL (↑).

Imaging studies (January 2)

Emergency CT revealed a large (approx. 184 x 124 x 122 mm), slightly hyperdense hematoma in the abdominal cavity and right psoas muscle, causing significant displacement of adjacent bowel loops and the right kidney, accompanied by surrounding exudation and a small amount of ascites.

Subsequent CTA confirmed the findings and demonstrated scattered contrast extravasation within the hematoma, primarily near the inferior vena cava, suggestive of active bleeding. The pancreatic head and uncinate process were closely associated with the hematoma and showed hypoperfusion. Mass effect with significant compression of the inferior vena cava and right renal vein was noted, along with a slight increase in peritoneal fluid.

Diagnosis and differential diagnosis

Hypovolemic Shock: Evidenced by hypotension, dizziness, weakness, elevated lactate, and a marked decline in hemoglobin.

Retroperitoneal Hematoma (right psoas origin): Physical exam revealed a tense abdomen with a tender, rigid 15 x 16 cm right-sided mass. CT confirmed a large (184 x 124 x 122 mm) hyperdense hematoma in the right psoas.

Hemophilia A: Established based on the patient's known history and lack of regular prophylaxis, supported by a significantly prolonged APTT.

Gastrointestinal Perforation: Ruled out as CT imaging showed no evidence of free air.

Solid organ rupture (liver/spleen): Effectively excluded by the absence of clear rupture signs on CT.

Treatment progress

The patient with hemophilia A presented in critical condition with a massive abdominal hematoma and hemorrhagic shock. An immediate multidisciplinary rescue was initiated. The management strategy included urgent FVIII infusion to correct the coagulopathy and aggressive fluid resuscitation to reverse shock. A full abdominal aortic CTA was promptly performed to localize the active bleeding site. Adjuvant measures included proton pump inhibitor administration for gastric protection and intensive monitoring of vital signs, hemoglobin levels, and abdominal girth.

Phase 1: Emergency rescue and MDT investigation and management of the bleeding cause (January 2)

Following admission, the patient received immediate transfusion support and FVIII replacement. By the next morning, the APTT had corrected to 36.5 seconds; however, the hemoglobin (HGB) dropped precipitously from 103 g/L to 83 g/L, and further to 76 g/L by 14:23, indicating significant ongoing hemorrhage.

Integrated with the emergency CT and CTA findings, which confirmed active intra-abdominal bleeding, it was determined that factor replacement and transfusions alone were insufficient. A multidisciplinary consensus was reached to proceed with immediate transcatheter lumbar artery embolization.

That evening, the patient successfully underwent the procedure. Angiography confirmed a bleeding lumbar

artery, which was subsequently embolized. The patient was then transferred back to the ICU for postoperative care, including continuous monitoring, oxygen therapy, infection control, and fluid resuscitation.

Phase 2: Investigation and management of progressive hemoglobin decline (January 3 to January 4)

January 3

On the first postoperative day, the patient's vital signs remained stable. Blood transfusion was completed at 2:30; however, the morning complete blood count revealed that HGB had decreased from 76 g/L to 73 g/L, indicating the possibility of ongoing bleeding despite the embolization or potential hemolysis due to factors such as hematoma compression or shock. Treatment involved continued dynamic monitoring, with additional transfusions administered as necessary. FVIII was also provided, initially at a dose of 40 IU/kg, followed by a maintenance dose of 20 - 25 IU/kg every 8 to 12 hours.

January 4

The laboratory reported a critical HGB value of 48 g/L at 7:17. Immediate transfusion was performed to correct anemia, and symptomatic hemostatic treatment was initiated.

The follow-up CTA results for the patient indicated a reduction in the size of the abdominal and psoas hematoma compared to January 2, measuring 182 mm x 120 mm x 122 mm. The average density of the hematoma had increased; however, scattered contrast agent leakage was still observed within it, suggesting the possibility of minor ongoing bleeding.

Based on the cardiothoracic surgery consultation, the reduction in hematoma size confirmed the efficacy of the prior embolization. Any residual bleeding was attributed to minor collaterals or the hematoma surface. Given the high risks and uncertain benefits of re-intervention, conservative management was advised. The hematology team stressed maximizing coagulation support due to the persistent bleeding risk.

Through MDT consensus, a conservative treatment plan was implemented. This included increasing the FVIII dosage to 4,000 U every 12 hours, continuing transfusions, and closely monitoring FVIII activity and inhibitor levels.

Phase 3: Hemorrhage control and differential diagnosis of complications (January 5 to January 14)

The patient's active bleeding was ultimately controlled through dynamic laboratory monitoring and MDT-guided management, with hemoglobin levels rising and stabilizing above 100 g/L.

Concurrently, factor VIII supplementation entered a precision-control phase: dosing was dynamically adjusted based on real-time FVIII activity levels, with administration paused once the target (> 100%) was achieved to avoid thromboembolic risk from overtreatment. The absence of FVIII inhibitors (0.00 BU) ruled out inhibitor-related treatment complications.

As the patient's vital signs stabilized, progressive jaundice developed. At admission, Total Bilirubin (TBiL) levels were unremarkable; however, on January 5, TBiL began to rise significantly, reaching 45.2 μmol/L. By January 6, it increased to 75.6 μmol/L, and on January 8, it reached 155.3 μmol/L, peaking at 220.5 μmol/L on January 9. Clinically, the patient exhibited pronounced yellowing of the skin and sclera, along with intermittent vomiting. Both Direct Bilirubin (DBiL) and Indirect Bilirubin (IBiL) levels were elevated, with IBiL peaking at 62.3 μmol/L, suggesting a complex etiology of jaundice that likely included both hemolytic and hepatocellular factors.

Differential diagnosis of jaundice

Biliary Obstruction: Effectively ruled out by a January 9 liver ultrasound showing no intra- or extrahepatic bile duct dilation.

Hepatitis (hepatocellular jaundice): Unlikely, as comprehensive viral and autoimmune hepatitis panels returned negative.

Drug-induced/hypoxic ischemic liver injury: Supported by elevated transaminases (AST/ALT > 2), γ-GGT, and ALP, consistent with shock-induced and/or transfusion-related hepatocellular damage.

Hemolytic jaundice: A major contributing factor, driven by the resorption of the large hematoma and multiple transfusions, leading to a significant bilirubin overload.

The MDT concluded that the patient's jaundice was primarily due to hemolytic jaundice resulting from the absorption of the large hematoma, compounded by mixed hyperbilirubinemia due to hepatocellular injury following shock. Consequently, no specific intervention was required; the focus remained on hepatoprotection, supportive care, and promoting hematoma absorption.

Subsequent laboratory results confirmed this assessment: as the hematoma gradually absorbed, bilirubin levels began to decline steadily after peaking on January 9, leading to an improvement in jaundice symptoms.

Recovery

On January 14, the patient's condition improved, with stable circulation, cessation of bleeding, and correction of anemia (HGB at 102 g/L). Jaundice had also resolved. Following a thorough assessment, the patient met the discharge criteria and was discharged successfully.

DISCUSSION

Hemophilia A, an X-linked recessive disorder, can lead to life-threatening spontaneous bleeding, with abdominal hemorrhage being particularly dangerous. The pathophysiology involves multiple factors: the fundamental issue is coagulation dysfunction, where FVIII deficiency impairs the intrinsic pathway, reducing thrombin generation and stable fibrin clot formation [4]. Vascular abnormalities from repeated factor infusions may also increase bleeding risk [5]. The abdomen's vascularity, pressure fluctuations, and capacity as a large potential

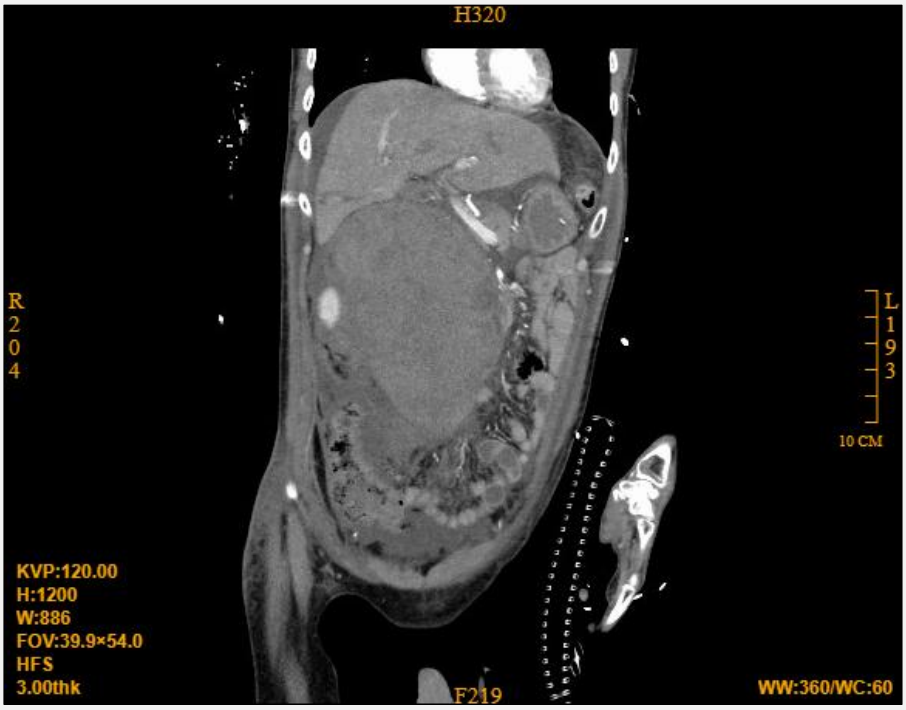

Figure 1. CTA enhanced scan image on January 2.

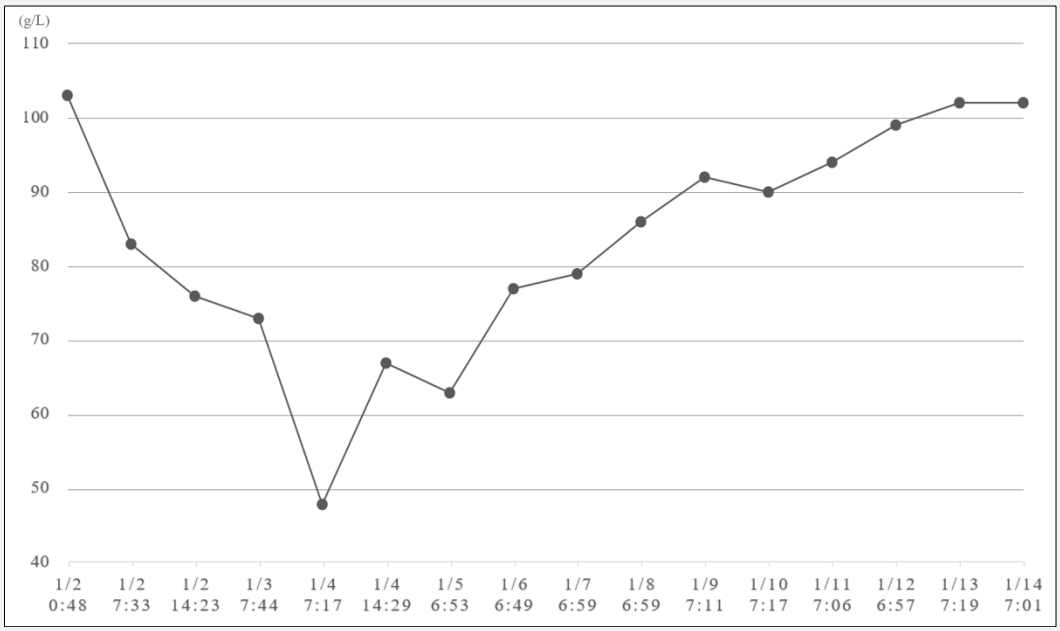

Figure 2. Line graph of patient HGB monitoring trends.

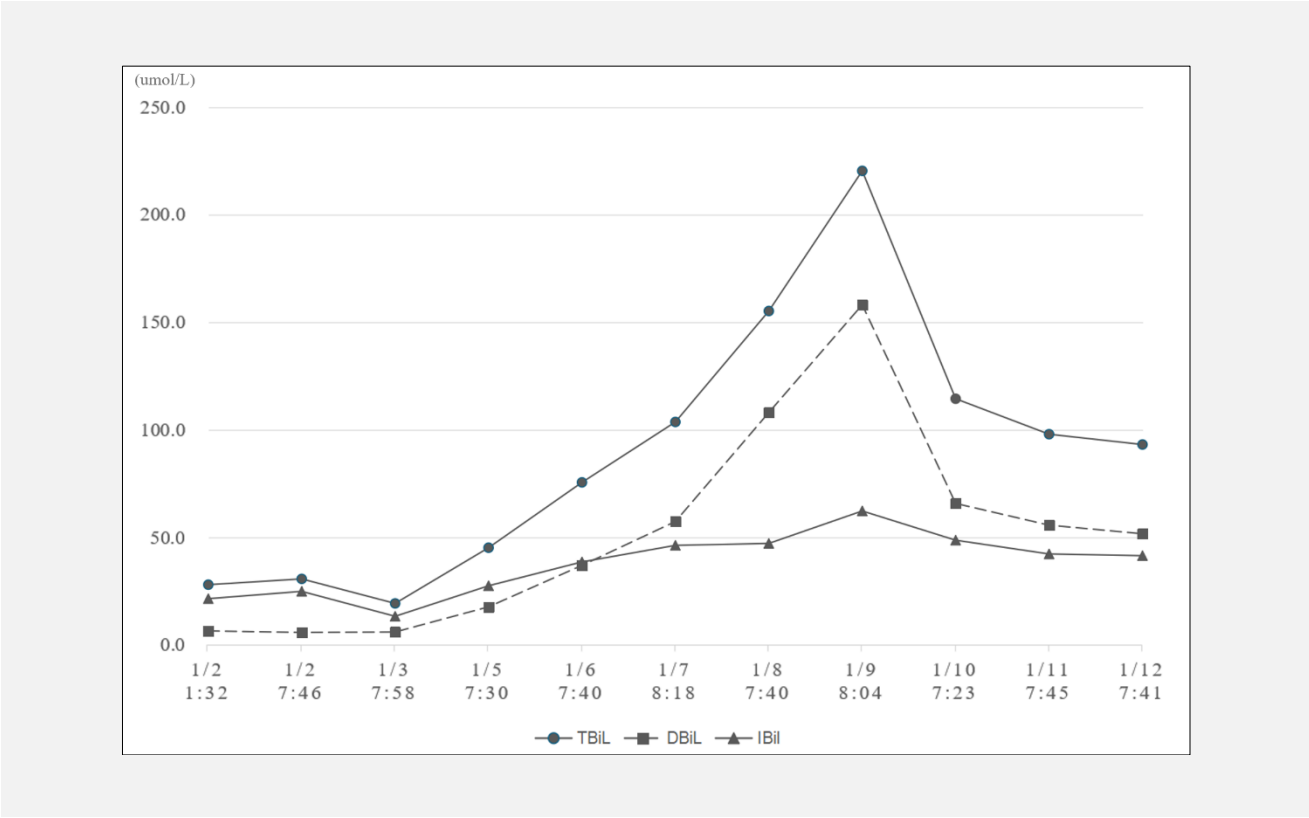

Figure 3. Line graph of patient bilirubin monitoring trends.

space allow significant blood accumulation without early signs, often delaying diagnosis [6]. Trauma or surgery, without adequate coagulation correction, are common triggers [7]. The development of FVIII inhibitors further complicates treatment. Mortality for abdominal arterial bleeding with an unclear source is as high as 50%, compared to 8.6% when the source is identified [8]. Therefore, early identification of the bleeding source and initiating multidisciplinary collaboration are crucial for successful management [9].

This case demonstrates the efficacy of multidisciplinary collaboration in integrating specialized expertise. The emergency, laboratory, and imaging departments worked in concert to rapidly assess the patient, provide critical metrics, and localize the bleeding focus via CTA. This coordination within hours guided subsequent transfusion, factor replacement, and intervention, securing a critical rescue window. Postoperatively, as HGB declined, the team promptly reassessed and adjusted treatment. In managing later complications, the MDT analyzed clinical, laboratory, and imaging findings to accurately diagnose hematoma-related hemolytic jaundice, avoiding the risks of invasive procedures and highlighting the diagnostic and therapeutic value of integrated care.

In the multidisciplinary collaboration framework, the laboratory played a crucial role in monitoring key data and assessing treatment efficacy. In this case, the HGB trend became a pivotal decision-making reference throughout the treatment process. Starting from an initial level of 103 g/L, HGB progressively declined, indicating active bleeding, eventually reaching a critical value of 48 g/L. The laboratory's continuous involvement in treatment decision-making and adjustments was vital, leading to a steady recovery of HGB and successful hemorrhage control. This continuous monitoring system provided an irreplaceable objective basis for clinical decision-making, highlighting the guiding role of critical laboratory indicators in the management of acute and critical illnesses.

In hemophilia management, coagulation factor supplementation must adhere to the principle of precision, as excessive infusion may cause thrombotic complications [3]. In this case, dynamic monitoring of APTT, FVIII activity, and FVIII inhibitor levels enabled precise FVIII dosing. This strategy maintained FVIII activity above 100% while ruling out inhibitor-related resistance, achieving an optimal balance between hemostasis and safety.

Bilirubin typing and dynamic monitoring provided key evidence for etiological differentiation: rising indirect bilirubin indicated hemolysis, while elevated direct bilirubin alongside increased ratio of AST/ALT, γ-GGT, and ALP supported liver injury. Combined with imag-

ing showing no biliary obstruction, the MDT identified hematoma-related hemolysis and hepatocellular injury as core mechanisms. Continuous monitoring of creatinine, electrolytes, and lactate further established a multi-parameter assessment system, supporting fluid management, shock evaluation, and renal injury warnings, demonstrating the collaborative value of laboratory indicators in critical care.

CONCLUSION

The successful rescue of this patient with hemophilia A complicated by life-threatening spontaneous abdominal hematoma and hemorrhagic shock highlights the critical importance of rapid multidisciplinary response and decisive clinical decision-making. The laboratory provided essential support through timely and precise dynamic monitoring of key parameters and prompt critical value reporting, enabling real-time data-driven decisions across all treatment phases. Through close MDT collaboration, integrated expertise facilitated comprehensive optimization of the entire clinical process-from rapid diagnosis and precise intervention to meticulous perioperative management and complication control. This case demonstrates the vital role of multidisciplinary integration and high-quality laboratory medicine within modern healthcare systems.

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Declaration of Generative AI in Scientific Writing:
The authors declare that no Artificial Intelligence (AI) or AI-assisted technologies were used in the preparation of this article.

Declaration of Interest:
The authors declare that there is no conflict of interests.

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