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Anticoagulation-Associated Spontaneous Retroperitoneal Hemorrhage Complicated by Abdominal Compartment Syndrome and Multiorgan Failure: A Case Report

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Abstract

Spontaneous retroperitoneal hemorrhage (SRH) complicated by abdominal compartment syndrome (ACS) presents a significant management challenge in anticoagulated patients. We report the case of a 52-year-old woman with complex valvular heart disease, atrial fibrillation, previous cerebrovascular accident, and chronic warfarin therapy who was admitted with acute decompensated heart failure. Warfarin was withheld following detection of a markedly supratherapeutic international normalized ratio (INR) of approximately 8. Vitamin K was not administered because of a documented phytonadione allergy. Therapeutic enoxaparin was initiated when the INR decreased to approximately 2.1 and continued for approximately five days. The patient subsequently developed abdominal and flank pain, progressive abdominal distension, severe anemia, and hemorrhagic shock. Computed tomography (CT), performed approximately seven hours after the final enoxaparin dose, revealed a massive right retroperitoneal hematoma measuring 26.3 × 15.7 × 12.6 cm with multifocal active arterial extravasation. Hemostatic resuscitation, protamine administration, transcatheter arterial embolization (TAE), and placement of a right external iliac covered stent achieved angiographic hemostasis. However, refractory shock and multiorgan dysfunction persisted. After intervention, transvesical intra-abdominal pressure (IAP) was 23 mmHg and increased to 26 mmHg on repeat measurement the following day. These findings, accompanied by new-onset anuria and worsening respiratory and circulatory failure, were consistent with ACS. The surgical team recommended conservative management because of profound hemodynamic instability requiring maximal vasopressor support and concern for recurrent hemorrhage following surgical decompression. The team maintained this recommendation on reassessment the following day. Despite continued intensive care support, the patient died from refractory multiorgan failure. This case highlights that successful angiographic hemostasis does not necessarily translate into physiological recovery and emphasizes the importance of assessing IAP and persistent organ dysfunction following endovascular intervention.

Categories: Emergency Medicine, Internal Medicine, Cardiac/Thoracic/Vascular Surgery

Keywords: abdominal compartment syndrome, anticoagulation, enoxaparin, hemorrhagic shock, multiorgan failure, spontaneous retroperitoneal hemorrhage, transcatheter arterial embolization

Introduction

Spontaneous retroperitoneal hemorrhage (SRH) is defined as bleeding into the retroperitoneal space without preceding trauma, surgery, or invasive intervention [1,2]. The retroperitoneum can accommodate substantial blood loss before overt clinical manifestations develop. Consequently, the patients may initially present with nonspecific symptoms before progressing to severe anemia, hemorrhagic shock, and multiorgan dysfunction [1-4]. Anticoagulant and antiplatelet therapies are frequently associated with SRH [1,3,4]. Other associated conditions include coagulopathies, hemodialysis, vascular abnormalities, malignancy, and renal or adrenal disease. The underlying pathophysiology remains incompletely understood but may involve rupture of vulnerable vessels or minor vascular injury in the setting of impaired hemostasis [1,3]. Clinical manifestations include abdominal, flank, back, groin, or hip pain, progressive anemia, hypotension, and otherwise unexplained clinical deterioration [1,2]. Contrast-enhanced computed tomography (CT) is the main way to look at the extent of bleeding and identify active arterial extravasation. Management depends on hemodynamic stability, transfusion requirements, ongoing bleeding, anticoagulant exposure, and imaging findings. Patients with persistent hemodynamic instability or active arterial hemorrhage may require endovascular or surgical intervention [2,4,5]. Transcatheter arterial embolization (TAE) enables angiographic localization and control of bleeding vessels. However, successful angiographic hemostasis does not necessarily result in physiological recovery, particularly when severe shock and organ dysfunction have already developed [5]. Large retroperitoneal hematomas may increase intra-abdominal pressure (IAP), impairing venous return, renal perfusion, diaphragmatic excursion, and splanchnic circulation. Abdominal compartment syndrome (ACS) is defined as sustained IAP above 20 mmHg associated with new organ dysfunction or failure [6,7]. We report a case of massive anticoagulation-associated SRH complicated by ACS despite successful angiographic hemostasis. This case highlights the difficult balance between surgical decompression to relieve elevated IAP and the risk of recurrent

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hemorrhage following recently achieved hemostasis.

Case Presentation

A 52-year-old woman was admitted with acute decompensated heart failure and concomitant upper-limb cellulitis, presenting with pulmonary and peripheral congestion. Her cardiac history included bioprosthetic mitral valve replacement in 2004, followed by redo mitral and tricuspid valve replacements with tissue prostheses in 2009. During the current admission, cardiac evaluation revealed severe degenerative prosthetic mitral stenosis, severe tricuspid stenosis, and moderate tricuspid regurgitation. She had been accepted for repeat mitral and tricuspid valve surgery. Her medical history also included atrial fibrillation following ablation, a previous cerebrovascular accident with residual hemiplegia, permanent pacemaker implantation, a solitary functioning left kidney, obesity, hypothyroidism, and anxiety. Her actual body weight was 95 kg, and her height was 160 cm (body mass index, 37.1 kg/m²). Her medical record listed phytonadione as an allergy; however, it provided no information on the nature, severity, or timing of the previous reaction. During initial treatment, she received antimicrobial therapy for cellulitis and intensified diuresis for volume overload. Warfarin was withheld after a markedly supratherapeutic international normalized ratio (INR) of approximately 8 was detected. Vitamin K was not administered because of the documented allergy. Following warfarin discontinuation, the INR decreased to approximately 2.1. Given her atrial fibrillation and previous cerebrovascular accident, the treating team considered her at elevated thromboembolic risk and initiated therapeutic enoxaparin at 90 mg subcutaneously every 12 hours (approximately 0.95 mg/kg per dose), while warfarin remained withheld. At enoxaparin initiation, serum creatinine was approximately 100 µmol/L (1.13 mg/dL). Cockcroft-Gault estimates of creatinine clearance varied according to the weight descriptor used because of obesity, ranging from approximately 48 mL/min using ideal body weight to higher values using adjusted or actual body weight. All estimates exceeded 30 mL/min, the threshold for therapeutic enoxaparin dose reduction specified in the prescribing information. Nevertheless, uncertainty in estimating renal clearance in obesity complicates the assessment of potential drug exposure. Anti-factor Xa activity was not measured; therefore, residual low-molecular-weight heparin (LMWH) activity at hemorrhage onset could not be quantified. Therapeutic enoxaparin was administered for approximately five days. After approximately five days of treatment, the patient developed nausea and vomiting, followed by right lower abdominal and flank pain beginning at approximately 3:00 PM. Her symptoms progressed to abdominal distension, tenderness, and guarding. No gastrointestinal bleeding was reported. Recurrent hypotension developed, with a mean arterial pressure of approximately 50-55 mmHg, and the abdomen became tense and markedly distended. Hemoglobin decreased to approximately 60 g/L, and lactate was 2.9 mmol/L. General surgery was consulted at approximately 11:00 PM and requested a CT angiogram. Imaging was performed at approximately 3:00 AM the following morning, and interventional radiology was consulted immediately after reviewing the imaging findings. The final enoxaparin dose had been administered approximately seven hours before CT confirmation of the hemorrhage. The patient was urgently transferred to the cardiac critical care unit, where ultrasound-guided arterial and central venous access were established, and a massive transfusion protocol was activated. Progressive hemodynamic instability and impending cardiorespiratory collapse necessitated endotracheal intubation and invasive mechanical ventilation. During ongoing hemorrhage and resuscitation, severe acquired coagulopathy developed. Laboratory investigations demonstrated a prothrombin time (PT) of 56.7 seconds, an INR of 5.1, an activated partial thromboplastin time (aPTT) of 116 seconds, a fibrinogen concentration of 0.8 g/L, and a platelet count as low as $41 \times 10^9/L$ (Table 1).

Parameter	Baseline	Initial post-deterioration results	Normal reference range	Interpretation
Hematology				
Hemoglobin (g/L)	108	60	120-160	Severe anemia
Hematocrit (L/L)	0.339	0.187	0.36-0.46	Marked reduction
WBC (×10 ⁹ /L)	6.6	26.3	4-11	Leukocytosis
Platelets (×10 ⁹ /L)	205	41	150-450	Severe thrombocytopenia
Coagulation				
PT (s)	25.0	56.7	11-13.5	Prolonged
INR	2.0	5.1	0.8-1.2	Prolonged
aPTT(s)	43	116	25-35	Prolonged
Fibrinogen (g/L)	-	0.8	2.0-4.0	Severe hypofibrinogenemia, subsequently corrected.
Biochemistry				
Creatinine (μmol/L)	100	120	45-90	Worsening renal function
BUN (mmol/L)	10.5	11.1	2.5-7.1	Elevated BUN
Alanine aminotransferase (U/L)	11	-	7-56	The result is within normal limits, but no follow-up measurement is available.
Total bilirubin (μmol/L)	41	58	3-21	Hyperbilirubinemia
Albumin (g/L)	18	32	35-50	Hypoalbuminemia
Sodium (mmol/L)	135	148	135-145	Mild hyponatremia
Potassium (mmol/L)	2.7	4.2	3.5-5.0	Hypokalemia, subsequently corrected.

TABLE 1: Laboratory investigations.
WBC: white blood cell count; PT: prothrombin time; INR: international normalized ratio; aPTT: activated partial thromboplastin time; BUN: blood urea nitrogen

Protamine was administered because of recent therapeutic enoxaparin exposure, recognizing that it only partially reverses enoxaparin-associated anti-factor Xa activity [6]. Hemostatic resuscitation included packed red blood cells, fresh-frozen plasma, platelets, prothrombin complex concentrate, fibrinogen replacement, and tranexamic acid. We could not determine the relative contributions of residual anticoagulation, coagulation factor consumption, dilution, and shock-associated coagulopathy. Multiphasic contrast-enhanced CT of the abdomen and pelvis demonstrated a large right retroperitoneal hematoma measuring approximately 26.3 × 15.7 × 12.6 cm, with an approximate ellipsoid volume of 2.7 L. Multiple foci of active arterial extravasation were identified within the hematoma and adjacent right psoas muscle, with right lumbar arterial branches suspected as potential bleeding sources. Additional findings included extensive retroperitoneal hemorrhage and mild hemoperitoneum. The inferior vena cava was markedly collapsed, consistent with intravascular volume depletion in the setting of hemorrhagic shock. The celiac artery origin was poorly opacified, although the distal hepatic and splenic branches remained patent. Gastric pneumatosis raised concern for ischemic injury, while reduced splenic enhancement suggested hypoperfusion or possible ischemia (Figure 1).

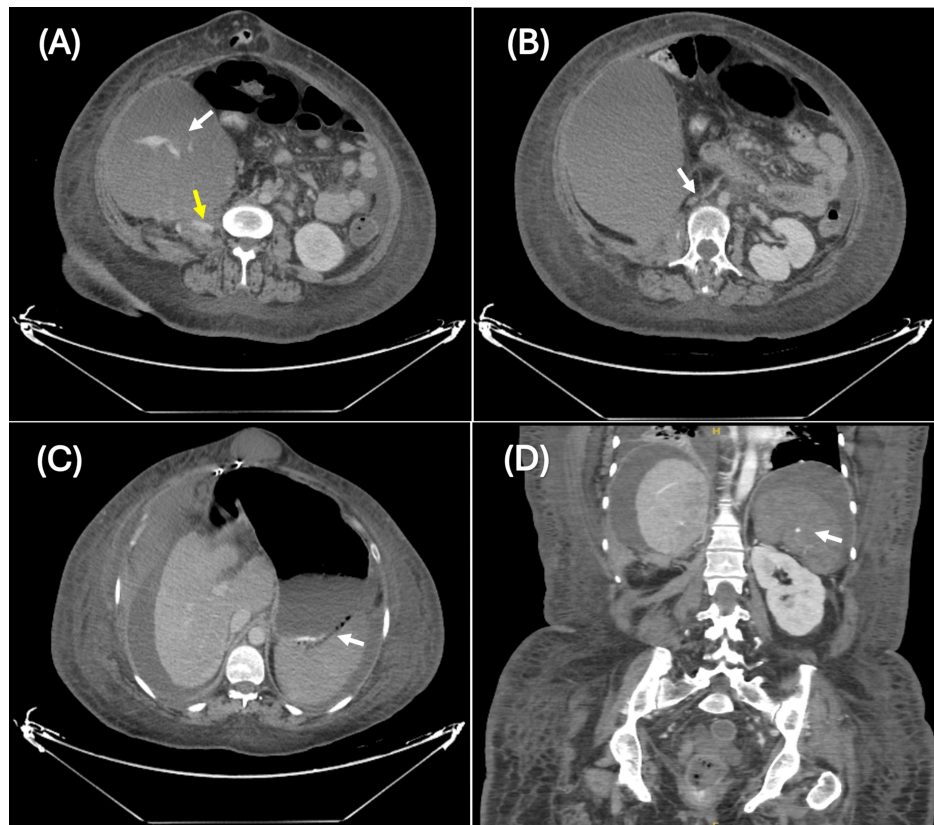


FIGURE 1: Contrast-enhanced computed tomography findings during acute hemorrhagic shock.

(A) Axial contrast-enhanced CT of the abdomen demonstrating a large right-sided retroperitoneal hematoma with active contrast extravasation (white arrow), with additional active extravasation within the right psoas muscle (yellow arrow), indicating ongoing hemorrhage. (B) Marked collapse of the inferior vena cava, consistent with severe intravascular volume depletion in the setting of acute hemorrhagic shock (white arrow). (C) Gastric pneumatosis, concerning for ischemic gastric injury (white arrow). (D) Coronal contrast-enhanced CT demonstrating diffusely decreased splenic enhancement, consistent with splenic hypoperfusion (white arrow). In the setting of profound circulatory shock, the findings in panels C and D raised concern for global splanchnic hypoperfusion with possible ischemic injury.

Other findings included diffuse anasarca, moderate abdominopelvic free fluid, bilateral pleural effusions with passive atelectasis, absence of the right kidney, and cortical scarring of the left kidney. Following continued resuscitation and attempts at hemodynamic stabilization, endovascular intervention was initiated at approximately 10:00 AM. Catheter angiography identified active bleeding from branches of the right lower lumbar, internal iliac, and external iliac arteries. The lumbar and internal iliac bleeding sites were embolized using polyvinyl alcohol particles and Gelfoam® absorbable gelatin sponge (Pharmacia & Upjohn Company LLC; MI, USA). A GORE VIABAHN endoprosthesis (W. L. Gore & Associates, Inc., Flagstaff, Arizona, USA) was deployed across the right external iliac arterial lesion. Completion angiography demonstrated cessation of contrast extravasation, with preserved patency of the right external iliac and common femoral arteries (Figure 2).

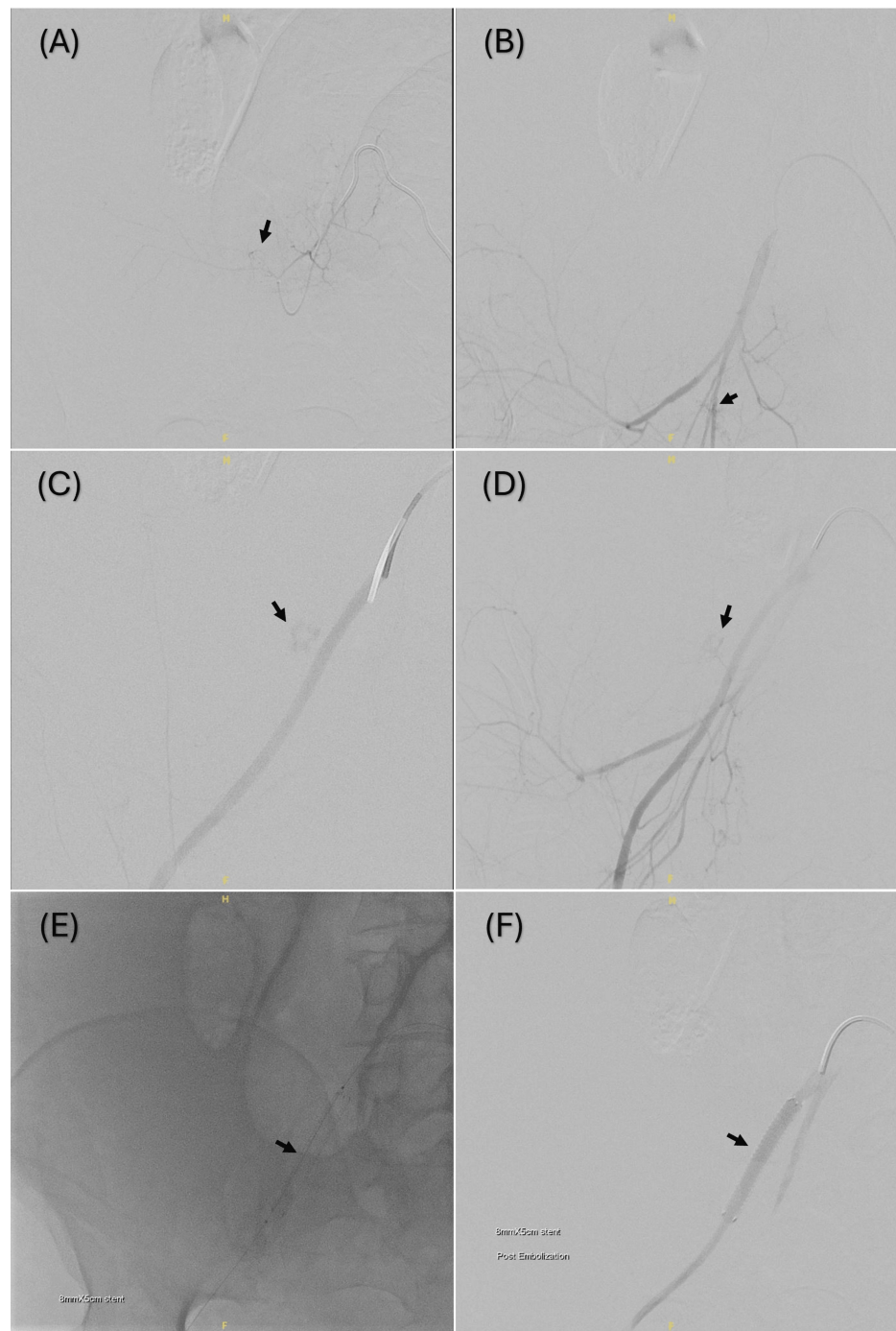


FIGURE 2: Emergency catheter angiography and endovascular treatment of active retroperitoneal hemorrhage.

(A) Active contrast extravasation from the branches of the right lower lumbar arteries (arrow). (B) Active contrast extravasation from a branch of the right internal iliac artery (arrow). (C, D) Angiographic views demonstrating active contrast extravasation from the right external iliac artery (arrow). (E) Completion angiography following transcatheter arterial embolization of the lumbar and internal iliac arterial branches and covered stent placement in the right external iliac artery, demonstrating successful hemostasis (arrow). (F) Additional angiographic view demonstrating a patent right external iliac artery stent without residual contrast extravasation (arrow).

No immediate procedural complications were identified. Despite angiographic hemostasis and aggressive resuscitation, profound circulatory shock persisted, requiring norepinephrine, epinephrine, and vasopressin. Venous blood gas analysis demonstrated a pH of 7.20 and bicarbonate concentration of 15.1 mmol/L (Table 2).

Parameter	Post-deterioration VBG	Post-hemostatic intervention VBG	Normal reference range	Interpretation
pH	7.45	7.20	7.31-7.41	Severe acidemia
PvCO ₂ (kPa)	6.0	5.1	5.5-6.8	Inadequate compensation
HCO ₃ ⁻ (mmol/L)	31.3	15.1	22-29	Metabolic acidosis
Base excess (mmol/L)	+6.7	-12.1	-2 to +2	Severe base deficit
Lactate (mmol/L)	2.9	17	0.5-2.0	Severe hyperlactatemia
Ionized calcium (mmol/L)	1.08	0.96	1.12-1.32	Worsening Hypocalcemia
Hemoglobin (g/L)	70	65	120-160	Severe anemia associated with acute hemorrhage and hemodilution during resuscitation

TABLE 2: Venous blood gas analysis.

VBG: venous blood gas; PvCO₂: venous partial pressure of carbon dioxide; HCO₃⁻: bicarbonate

Hypoxemic respiratory failure progressed despite invasive mechanical ventilation, with reported PaO₂/FiO₂ ratios below 100. Following the endovascular intervention, the abdomen remained tense and markedly distended. The initial transvesical IAP, measured after the procedure, was 23 mmHg, increasing to 26 mmHg on repeat measurement the following day. These repeated elevations, together with new-onset anuria, persistent vasopressor-dependent shock, worsening respiratory failure, and severe metabolic acidosis, were consistent with ACS [7]. The surgical team reassessed the patient following endovascular hemorrhage control and recommended conservative management rather than immediate decompressive laparotomy. On repeat evaluation the following day, the team maintained this recommendation because of profound hemodynamic instability requiring maximal vasopressor support and concern for recurrent hemorrhage following surgical decompression. The team applied nonoperative measures, including deep sedation, neuromuscular blockade, nasogastric decompression, bladder drainage, fluid-balance optimization, and IAP monitoring. Anuric acute kidney injury developed in the setting of hemorrhagic shock, systemic hypoperfusion, elevated IAP, and limited renal reserve associated with a solitary kidney and cortical scarring. Continuous venovenous hemodiafiltration was initiated for renal support and the management of severe metabolic derangements, with fluid removal adjusted according to hemodynamic tolerance. Following the angiographic procedure, bilateral fixed pupillary dilation prompted urgent neurological evaluation. Brain CT and CT angiography of the head and neck revealed no acute intracranial hemorrhage, major territorial infarction, significant cerebral edema, herniation, or large-vessel occlusion. Hypoxic-ischemic or metabolic encephalopathy was considered in the context of profound shock and metabolic derangement. Magnetic resonance imaging and electroencephalography were planned if clinical stability permitted. Broad-spectrum empirical antimicrobials were administered because concomitant infection could not initially be excluded; blood cultures remained negative. The pharmacological anticoagulation was withheld following the hemorrhage, and intermittent pneumatic compression was used for venous thromboembolism prophylaxis. Despite angiographic hemorrhage control, extensive transfusion and hemostatic support, invasive mechanical ventilation, continuous renal replacement therapy, and treatment with three vasopressors, refractory multiorgan failure persisted. The patient subsequently experienced cardiac arrest. Resuscitation was unsuccessful, and death was pronounced.

Discussion

Anticoagulant exposure and hemorrhagic risk

The hemorrhage occurred amid complex anticoagulant exposure. The patient initially had a markedly supratherapeutic INR of approximately 8 while receiving warfarin. Although warfarin was discontinued, therapeutic enoxaparin was subsequently initiated while the INR remained approximately 2.1. Enoxaparin was given subcutaneously at a dose of 90 mg every 12 hours for about five days. The final dose was given approximately seven hours before CT confirmed the hemorrhage. This temporal correlation substantiates the classification of the event as an anticoagulation-associated SRH. However, it does not establish enoxaparin as the sole cause or quantify the contribution of residual warfarin activity. Long-term therapeutic anticoagulation was indicated for atrial fibrillation with a history of cerebrovascular accident

rather than for the presence of bioprosthetic valves alone [8,9]. The decision to initiate enoxaparin during warfarin interruption reflected the assessment of thromboembolic risk by the treating team and should not be interpreted as guideline-mandated bridging. Importantly, the INR remained approximately 2.1 at initiation, indicating persistent anticoagulant activity from warfarin. Potential alternatives included deferring parenteral anticoagulation while monitoring the INR or using intravenous unfractionated heparin if short-term therapeutic anticoagulation was considered necessary. Unfractionated heparin has a shorter half-life, allows more rapid dose titration, and is more completely reversible with protamine than LMWH. However, these pharmacological advantages do not establish that an alternative strategy would have prevented hemorrhage or provided a superior clinical outcome in this patient. Enoxaparin is substantially cleared through the kidneys. Although serum creatinine was approximately 100 µmol/L, obesity introduced uncertainty into estimates of creatinine clearance using the Cockcroft-Gault formula, depending on the weight descriptor used. Estimated clearance remained above 30 mL/min using the reported methods, but actual drug exposure could not be determined. Conventional coagulation tests, including PT/INR and aPTT, do not reliably quantify LMWH activity. Anti-factor Xa activity was not measured, which prevented assessment of the residual enoxaparin effect. The 2018 American Society of Hematology guidelines suggest against routine anti-factor X-guided LMWH dose adjustment in patients with obesity or severe renal dysfunction treated for venous thromboembolism; however, these recommendations do not directly address anticoagulation for atrial fibrillation [10]. The significant coagulation abnormalities noted during resuscitation cannot be solely ascribed to anticoagulant exposure. An INR of 5.1, aPTT up to 116 seconds, fibrinogen concentration of 0.8 g/L, and platelet count as low as $41 \times 10^9/L$ were consistent with severe acquired coagulopathy associated with massive hemorrhage, consumption, dilution, and profound shock. Phytonadione was documented as an allergy without details of the previous reaction. This documentation influenced reversal decisions, although the nature of the reported hypersensitivity could not be confirmed.

Diagnosis and endovascular hemorrhage control

SRH may remain clinically occult until substantial blood loss has occurred. Abdominal, flank, back, or groin pain, rapidly declining hemoglobin, hypotension, or unexplained organ dysfunction should raise suspicion, particularly in anticoagulated patients [1,2]. In this case, acute abdominal and flank pain, progressive distension, severe anemia, and circulatory shock prompted contrast-enhanced CT, which demonstrated a massive retroperitoneal hematoma with multifocal active arterial extravasation. Inferior vena cava collapse was consistent with severe intravascular volume depletion, while gastric pneumatosis and reduced splenic enhancement raised concern for splanchnic hypoperfusion or ischemic injury. However, these imaging findings alone could not establish irreversible ischemia. Management of severe SRH requires simultaneous hemodynamic resuscitation, the interruption or reversal of relevant anticoagulant effects, correction of hemorrhage-associated coagulopathy, and timely definitive hemorrhage control. We administered protamine because of recent therapeutic enoxaparin exposure, although it incompletely neutralizes LMWH-associated anti-factor Xa activity [6]. Active arterial extravasation and persistent hemodynamic instability supported urgent endovascular intervention [2,4,5]. Catheter angiography identified bleeding from branches of the right lumbar, internal iliac, and external iliac arteries. TAE and covered stent placement achieved cessation of visible extravasation while preserving major arterial patency. In a 2024 systematic review and meta-analysis involving six retrospective studies and 141 patients with SRH treated by TAE, technical success was reported as 100%, whereas clinical success ranged from 56.3% to 89.5% [5]. These findings highlight the distinction between successful angiographic hemorrhage control and subsequent clinical recovery, although the retrospective evidence cannot predict individual outcomes. Despite successful endovascular intervention, the patient remained in profound shock, requiring three vasopressors, and developed metabolic acidosis, respiratory failure, and anuric acute kidney injury. These findings demonstrate that cessation of angiographically visible arterial bleeding does not necessarily reverse the physiological consequences of severe tissue hypoperfusion, acquired coagulopathy, and elevated IAP.

Abdominal compartment syndrome and the decompression dilemma

The most consequential complication following hemorrhage control was ACS. The World Society of the Abdominal Compartment Syndrome (WSACS) defines intra-abdominal hypertension as sustained or repeated IAP of at least 12 mmHg. In contrast, ACS is defined as sustained IAP above 20 mmHg associated with new organ dysfunction or failure [7]. In this patient, the initial transvesical IAP measurement after endovascular intervention was 23 mmHg, increasing to 26 mmHg on repeat measurement the following day. These repeated elevations, accompanied by new-onset anuria, persistent circulatory shock, and worsening respiratory failure, were consistent with ACS. Although IAP was elevated on consecutive days, we could not establish exact measurement times or the continuous duration of elevation. Metabolic acidosis was also present but was nonspecific in the setting of preceding hemorrhagic shock. Elevated IAP can impair venous return, increase renal venous pressure, reduce renal perfusion, restrict diaphragmatic excursion, and compromise splanchnic circulation [7]. These mechanisms may have contributed to the patient's progressive organ dysfunction. However, the relative contributions of elevated IAP and preceding hemorrhagic shock could not be reliably distinguished. Also, the large residual retroperitoneal hematoma may have continued to exert a mass effect despite successful angiographic hemostasis. Initial nonoperative management included deep sedation, neuromuscular blockade, nasogastric decompression, bladder drainage, fluid-balance optimization, and IAP monitoring. Continuous renal replacement therapy was initiated to manage renal failure and severe metabolic disturbances but did not constitute definitive treatment for ACS. The WSACS recommends decompressive laparotomy for overt ACS, although this recommendation is based on very low-

quality evidence (GRADE 1D) [7]. The surgical team assessed the patient following endovascular hemorrhage control and recommended conservative management. On reassessment the following day, this recommendation was maintained because of profound hemodynamic instability requiring maximal vasopressor support and concern for recurrent hemorrhage following surgical decompression. Decompressive laparotomy is recommended for overt ACS; however, the anticipated operative risks were substantial in this patient because of refractory shock, severe coagulopathy, and recently controlled multifocal arterial bleeding. The decision to continue conservative management reflected an individualized assessment of competing risks and should not be interpreted as evidence against surgical decompression in other patients with overt ACS. Abdominal decompression without retroperitoneal hematoma evacuation should be distinguished from direct retroperitoneal exploration or hematoma evacuation, as the potential risk of disrupting tamponade may differ. Nevertheless, the hemodynamic consequences of surgery in this critically unstable patient remained a major concern. Tully et al. described a case of retroperitoneal hemorrhage complicated by ACS in which the endovascular hemorrhage control was followed by operative hematoma evacuation because of persistent compartment physiology [11]. This report illustrates that hemorrhage control and abdominal decompression may represent sequential therapeutic objectives. However, a single case cannot establish the optimal timing, patient selection, or procedural risks associated with decompression. Similarly, the fatal outcome in our patient does not establish that decompressive surgery would have improved survival. By the time ACS was suspected, profound circulatory shock, severe coagulopathy, suspected splanchnic hypoperfusion, and multiorgan dysfunction were already present. Although decompression might have improved abdominal perfusion and organ function, it also carried potential risks of recurrent hemorrhage and further cardiovascular collapse.

Clinical implications and limitations

This case emphasizes the importance of considering SRH in anticoagulated patients presenting with unexplained abdominal or flank pain, rapidly declining hemoglobin, abdominal distension, hypotension, or sudden clinical deterioration. Contrast-enhanced CT helps establish the diagnosis and identify active arterial bleeding, while persistent hemorrhage or hemodynamic instability may require definitive endovascular or surgical intervention [2,4,5]. Following successful angiographic hemostasis, continued assessment should extend beyond evidence of bleeding control to include urine output, respiratory mechanics, abdominal examination, vasopressor requirements, lactate, and acid-base status. Persistent or worsening organ dysfunction should prompt evaluation for elevated IAP and ACS. Several limitations warrant consideration. First, this single case cannot establish a causal relationship between enoxaparin and hemorrhage or determine whether an alternative anticoagulation strategy would have prevented the event. Anti-factor Xa activity was not measured, and the relative contributions of residual warfarin activity, enoxaparin exposure, renal clearance, and subsequent acquired coagulopathy remain uncertain. Also, obesity complicated estimation of renal function using the Cockcroft-Gault equation. Second, the contributions of hemorrhagic shock, underlying structural cardiac disease, elevated IAP, and possible infection to multiorgan failure could not be independently quantified. CT findings suggested possible gastric and splenic ischemic injury, but irreversible ischemia was not confirmed. Neurological deterioration also remained incompletely characterized because further investigations could not be completed. Finally, although completion angiography demonstrated successful hemorrhage control, subsequent occult or recurrent bleeding could not be definitively excluded. The decision to continue conservative management reflected the competing risks of decompression and recurrent hemorrhage rather than evidence that surgical intervention would have been ineffective. Overall, this case illustrates the complex relationship between technical hemorrhage control, persistent compartment physiology, and progressive multiorgan dysfunction. It highlights the need for individualized, multidisciplinary decision-making when balancing decompression for suspected ACS against preservation of recently achieved retroperitoneal hemostasis.

Conclusions

Massive anticoagulation-associated SRH can progress to hemorrhagic shock, severe acquired coagulopathy, ACS, and refractory multiorgan failure. Early recognition, prompt resuscitation, contrast-enhanced CT, appropriate management of anticoagulant exposure, and timely hemorrhage control are essential. However, successful angiographic hemostasis does not necessarily translate into physiological recovery. Persistent organ dysfunction following endovascular intervention should prompt reassessment for elevated IAP and the potential mass effect of residual retroperitoneal hematoma. Although decompressive laparotomy is recommended for overt ACS, the risks of surgical intervention must be weighed against the potential for recurrent hemorrhage and further hemodynamic deterioration in critically unstable, coagulopathic patients. This case emphasizes the necessity of early multidisciplinary assessment and individualized decision-making when balancing abdominal decompression against preservation of recently achieved hemostasis.

Additional Information

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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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