Retroperitoneal Hematoma Following Elective Abdominal Aortic Aneurysm Repair: A Case Report

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Retroperitoneal hematoma formation following elective open abdominal aortic aneurysm (AAA) repair may be occult. We report a case of recurrent hypotensive episodes in the postanesthesia care unit (PACU) that were temporarily treated successfully with fluid, blood products, and vasopressors. At reoperation, active bleeding was excluded; however, upon reopening the bovine pericardial patch closure of the retroperitoneum, hematoma between the aneurysmal sac and inferior vena cava (IVC) had caused IVC compression. Evacuation of the hematoma rapidly restored venous return and hemodynamics. This report describes a case of retroperitoneal hematoma formation and highlights challenges associated with diagnosing bleeding in this compartment. (A&A Practice. 2020;14:e01241.)

GLOSSARY

AAA = abdominal aortic aneurysm; **CT** = computed tomography; **CVP** = central venous pressure; **FFP** = fresh-frozen plasma; **Fibrin** = fibrinogen; **INR** = international normalized ratio; **IVC** = inferior vena cava; **OR** = operating room; **PACU** = postanesthesia care unit; **POCUS** = point-of-care ultrasound; **PRBC** = packed red blood cell; **PTT** = partial thromboplastin time; **SDU** = step-down unit; **U** = units

Postoperative hypotension following open abdominal aortic surgery has a broad differential diagnosis. Maintaining hemodynamics while excluding causes of hypotension remains the cornerstone of management postoperatively. Ultimately, if hemodynamic instability is unresponsive to resuscitation, the patient requires urgent surgical reexploration.

Hemodynamic instability within 24 hours postoperatively may indicate surgical bleeding. However, bleeding may not be clinically apparent on physical examination given the retroperitoneal location of the abdominal aorta. Retrospective reviews have demonstrated a low rate of reoperation, 1.0%–1.7%, for postoperative bleeding in elective open abdominal aortic aneurysm (AAA) repairs.^{1,2} Relaparotomy has been associated with higher first operation blood loss and need for packed red blood cell (PRBC) transfusion.^{1,2} Patients requiring relaparotomy are at increased risk of morbidity and mortality.²

We present a case of retroperitoneal bleeding causing hematoma formation and postoperative shock 13 hours after an open AAA repair to highlight challenges associated with diagnosing bleeding in this compartment.

We obtained written consent from the patient to publish this report.

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CASE DESCRIPTION

A 77-year-old man with a 5.5-cm asymptomatic infrarenal AAA and bilateral iliac aneurysms presented for an elective open aortobiliac repair. His medical history was unremarkable. Preoperative exercise sestamibi myocardial perfusion scan and transthoracic echocardiogram were normal. His preoperative vital signs and laboratory testing (hemoglobin 116 g/L, platelets 177×10^9 /L, creatinine 66 μ mol/L) were unremarkable.

Preoperative computed tomography (CT) angiography demonstrated severe angulation of the aortic neck and proximal aneurysm with deviation of the aorta toward the right side with associated inferior vena cava (IVC) compression by the aneurysm (Figures 1 and 2). No aortocaval fistula was present on imaging. Because of the patient's excellent functional status and angulated aortic anatomy, an open surgical repair was selected over an endovascular repair.

In the operating room (OR), an intravenous access, an arterial line, and a thoracic level 8 of 9 epidural were placed uneventfully. Following induction of general anesthesia, central venous access was obtained. Anesthesia was maintained with sevoflurane, hydromorphone intravenous boluses, and epidural ropivacaine 0.2% at 5 mL/h. Norepinephrine was started at 0.05 μ g/kg/min 2 hours into the operation, and the patient remained hemodynamically stable. Lactate remained <1 mmol/L, and urine output was >0.5 mL/kg/h.

The 6-hour operation was technically challenging, with an estimated blood loss of 4 L. Fluid replacement included 1.7 L from the cell saver, 2 units fresh-frozen plasma (FFP), 1 L 5% albumin, and 3 L crystalloid solution. Laboratory investigation at hour 4 showed hemoglobin 109 g/L, platelets 118×10^9 /L, international normalized ratio (INR) 2.6, and fibrinogen 1.1 g/L. The coagulopathy corrected to an INR 1.3 following transfusion of 2 units FFP (Table). At the

Figure 1. Preoperative CT abdomen: coronal images demonstrating a large infrarenal aortic aneurysm with severe angulation toward the IVC. AAA indicates abdominal aortic aneurysm; CT, computed tomography; IVC, inferior vena cava.

Figures 2. Preoperative CT abdomen: axial image demonstrating a large infrarenal aortic aneurysm with severe angulation toward the IVC. AAA indicates abdominal aortic aneurysm; CT, computed tomography; IVC, inferior vena cava.

end of the operation, heparin was reversed with protamine. The activated clotting time returned to baseline.

To facilitate retroperitoneal closure and reduce risk of aortoduodenal fistula, a bovine pericardial patch was sutured to the peritoneal edges of the open retroperitoneum through which the graft was exposed. The abdomen was closed, and the patient was taken to the postanesthesia care unit (PACU), intubated, and sedated due to the operation duration and transfusion requirement.

On arrival to the PACU, the patient had several hypotensive episodes, with the lowest blood pressure of 70/50 mm Hg. These hypotensive episodes were responsive to 1 L crystalloid solution, 500 mL 5% albumin, norepinephrine infusion (0.05–0.15 µg/kg/min), and phenylephrine boluses. Postoperative laboratory testing revealed hemoglobin 89 g/L, platelets 88×10^9 /L, INR 1.3, and fibrinogen 1.2 g/L. Propofol sedation (75 µg/kg/min) was stopped, hemodynamics stabilized, and the patient was allowed to wake up and then extubated uneventfully. To facilitate abdominal examination and rule out epidural induced hypotension, the epidural infusion was lowered to 3 mL/h of 0.2% ropivacaine. Epidural analgesia was excellent, with no reported pain and lower limb motor function intact. The surgical team reexamined the patient, finding a nontender abdomen with no distension.

During the PACU stay, the patient's systolic blood pressure dropped intermittently <90 mm Hg. His heart rate remained 90–100 beats per minute and pulse oximetry $\geq 98\%$ on 3 L/min by nasal prongs. The decreased hemoglobin concentration to 77 g/L 1.5 hours postoperatively was attributed to aggressive volume resuscitation in the PACU (Table).

Signs of ongoing bleeding included central venous pressure (CVP) <5 mm Hg, hypotensive episodes, and inadequate response to PRBC transfusion. The ongoing need for volume resuscitation occurred over an 11-hour PACU stay. Hemoglobin increased by 7, 2, and 6 g/L in response to the first 3 PRBC units (Table). After the fourth PRBC unit at hour 9 in the PACU, the hemoglobin increased from 81 to 91 g/L, and no further hypotensive episodes occurred. Additional signs of clinical stability were lactate <2.2 mmol/L, urine output >0.5 mL/kg/h, and a soft, nondistended abdomen. The patient was subsequently transferred to the step-down unit (SDU).

Two hours after arrival to the SDU and 13 hours after surgery completion, the patient's blood pressure dropped to 70/50 mm Hg, and abdominal distension with tenderness was noted. The hemoglobin decreased from 91 to 85 g/L. The patient was resuscitated and urgently taken back to the OR for presumed bleeding. In the OR, the patient's abdomen was prepped and draped, then anesthesia was induced uneventfully. During the SDU resuscitation and reexploration laparotomy, the patient was transfused 4 units PRBC, 1 unit platelets, 2 units FFP, 10 units cryoprecipitate, and 1 L crystalloid solution (Table).

Intraoperatively, the abdomen revealed no bleeding sources. When the bovine pericardial patch was taken down and the retroperitoneum opened to examine the aortic graft anastomoses, a large hematoma (~1000 mL) was discovered between the IVC and aneurysm sac. No active bleeding from the aortic anastomoses was identified. Following hematoma removal, rapid restoration of hemodynamics occurred with CVP recovery to >8 mm Hg enabling vasopressor downtitration. Following this procedure, the patient was transferred to the cardiovascular intensive care unit and extubated uneventfully 6 hours later.

The patient developed atrial fibrillation on postoperative day 3, was started on rivaroxaban, and was discharged from hospital on postoperative day 8. At the 1-month

Table. Postoperative Bloodwork and Transfusion Requirement: Postoperative Bloodwork Demonstrating Temporary and Inadequate Hemoglobin Increases to PRBC Transfusions Nos. 1–3

Hours postoperative	Hemoglobin (g/L)	Platelet ($\times 10^9$ /L)	INR	PTT (s)	Fibrin. (g/L)	Transfusion
0.5	89	88	1.3	...	...	
1.5	77	72	...	...	1.2	1U PRBC (No. 1)
2.5	84	...	...	...	...	
4.5	73	73	...	...	...	1U PRBC (No. 2)
5.5	75	82	...	...	...	1U PRBC (No. 3)
7.5	81	77	1.5	33.2	...	1U PRBC (No. 4)
9	91	76	1.4	32.0	...	
13	85	86	1.5	...	...	
OR No. 2						
14	69	79	1.4	28.3	2.21	4U PRBC, 2U FFP
16	97	71	1.3	30.4	2.51	10U cryoprecipitate,
16.5	104	75	1.3	...	2.87	1U platelet

Following PRBC transfusion No. 4 at hour 7.5, there was an adequate hemoglobin increase of 10 g/L from 81 to 91 g/L.

Abbreviations: FFP, fresh-frozen plasma; Fibrin., fibrinogen; INR, international normalized ratio; OR, operating room; PRBC, packed red blood cell; PTT, partial thromboplastin time; U, unit.

postoperative visit, the patient was recovering well with no additional complications.

DISCUSSION

The significance of this case involving diagnostic uncertainty is 2-fold: (1) to advocate for the use of postoperative diagnostic imaging and (2) to increase awareness of how cognitive biases can lead to diagnostic error. Our patient developed 2 sources of hemodynamic instability postoperatively: first, occult bleeding into the retroperitoneum over a prolonged 11-hour PACU stay, and second, localized retroperitoneal hematoma causing critical IVC compression and sudden hemodynamic collapse at hour 13 postoperatively.

Point-of-care ultrasound (POCUS) examination may have led to an earlier diagnosis. Bedside echocardiography can assess myocardial function and can demonstrate under-filled cardiac chambers and proximal IVC, which could have corroborated that our patient's low CVP was due to hypovolemia.

Adapted from trauma protocols, POCUS of the postoperative abdomen can detect free fluid.³ However, there is no postoperative standard free fluid scoring system to delineate normal from abnormal.³ Nevertheless, POCUS can screen for retroperitoneal hematoma with a sensitivity of 50%–62%.⁴ Sonographically, retroperitoneal hematomas can appear as well-defined fluid collections, have heterogeneous echogenicity, or have 2 layers of low- and high-density material in dependent areas known as a hematocrit sign.⁴ Distinguishing the peritoneal from retroperitoneal space can be challenging on ultrasound, but assessing the pararenal space can identify retroperitoneal hematoma.^{4,5} If there is a high index of suspicion for retroperitoneal hematoma despite a negative POCUS scan, CT imaging is required.⁴

Specific risk factors for retroperitoneal hematoma development in our patient may have been severe aneurysmal angulation and proximity to the IVC requiring close dissection. Additionally, retroperitoneal closure may have created a watertight compartment, which retained the hematoma causing IVC compression.

In uncertain situations, such as the one described in this case report, cognitive bias can lead to errors known as

decisional short cuts.^{6,7} Our anchoring cognitive bias (ie, the misassumption that we maintained) was that the patient was underresuscitated from the OR.^{7–10} We committed a search satisficing error (ie, we prematurely stopped considering other diagnostic possibilities) when we did not pursue additional testing for bleeding when the patient's hemoglobin failed to appropriately increase with PRBC transfusion.^{7,8,10} We assumed that the patient was adequately resuscitated when his hemoglobin finally increased 10 g/L with the fourth PRBC transfusion and, mistakenly, did not perform CT imaging to rule out retroperitoneal bleeding. As a result of our confirmation bias (ie, our focus on data that supported the original diagnosis), we undervalued evidence for occult retroperitoneal bleeding, such as multiple transfusions, low CVP, ongoing norepinephrine requirement, and normal abdominal examination.^{7–10} Strategies to mitigate these cognitive errors include awareness of potential cognitive biases, metacognition (ie, thinking about the decision-making process), and deliberate reconsideration of objective facts.^{7–11}

Our case demonstrates the difficulty of diagnosing postoperative hypotension caused by occult retroperitoneal bleeding and retroperitoneal hematoma formation underneath a watertight retroperitoneal closure. This complication created critical IVC compression, impaired venous return, and ultimately required urgent operative management. This case highlights the potential for significant blood loss into the retroperitoneum and advocates for POCUS and CT imaging. Additionally, this case is a reminder to be aware of cognitive biases informing medical care decisions and to utilize specific strategies to mitigate cognitive errors. ■■

DISCLOSURES

Name: Spencer Holowachuk, MD.

Contribution: This author helped care for the patient intraoperatively, gather information, review the literature, and write the manuscript draft.

Name: Martin Ma, MD.

Contribution: This author helped care for the patient postoperatively, review the literature, and edit the manuscript.

Name: George Oreopoulos, MD, MSc.

Contribution: This author helped care for the patient perioperatively, review the literature, and edit the manuscript.

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