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Case Report

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Atraumatic Splenic Rupture Associated with Long-Term Dual Antiplatelet Therapy: A Case Report and Surgical Decision-Making Perspective

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Abstract

Background: Atraumatic splenic rupture is a rare but potentially life-threatening cause of hemoperitoneum. Drug- or treatment-related cases have been reported in patients receiving antiplatelet or anticoagulant therapy, but evidence guiding management outside trauma cohorts remains limited.

Case presentation: We describe a 62-year-old man with coronary artery disease and six prior coronary stents who had received long-term aspirin and clopidogrel for approximately two years. He presented with an 11-hour history of positional, non-traumatic abdominal pain. Initial contrast-enhanced computed tomography showed hemoperitoneum without an identifiable bleeding source. Despite withholding antiplatelet therapy and supportive treatment, his hemoglobin declined over the following two days. Repeat CT angiography demonstrated focal capsular irregularity of the posterosuperior spleen, consistent with a contained splenic capsular injury. Following multidisciplinary discussion of splenic artery embolization versus splenectomy, he underwent laparoscopic splenectomy and evacuation of the hemoperitoneum. Histopathology showed splenic laceration with subcapsular congestion and hemorrhage, preserved remaining parenchyma, reactive hilar lymph nodes, and no malignancy, inflammation, infarction, thrombus, or atypia. Recovery was uneventful, and dual antiplatelet therapy was resumed on postoperative day 3.

Conclusion: This case demonstrates that atraumatic splenic hemorrhage should remain in the differential diagnosis of unexplained hemoperitoneum in patients receiving long-term dual antiplatelet therapy, even when the initial contrast CT does not identify a source. It also highlights the value of serial hemoglobin assessment, repeat dedicated CT angiography when clinical concern persists, and individualized selection between embolization and splenectomy when early antiplatelet resumption is required.

Keywords: atraumatic splenic rupture; hemoperitoneum; dual antiplatelet therapy; laparoscopic splenectomy; splenic artery embolization; case report

Introduction

Splenic rupture is classically a consequence of blunt abdominal trauma. Atraumatic, or "spontaneous," splenic rupture (ASR) is far less common and was historically defined by the Orloff and Peskin criteria, which require the absence of trauma and of an underlying splenic disease sufficient to explain rupture on its own. In the largest systematic review of the condition, covering 845 patients reported between 1980 and 2008, only 7% of cases were truly idiopathic; among the pathological cases, drug- or treatment-related causes accounted for roughly 9% of identified aetiological factors, behind neoplastic, infectious, and non-infectious inflammatory causes [1]. Antithrombotic therapy is increasingly represented within that drug-related category. A growing number of case reports describe ASR occurring within days of percutaneous coronary intervention (PCI) in patients newly loaded onto dual antiplatelet therapy (DAPT) [2] [3], with low on-treatment platelet reactivity proposed as a contributing mechanism [3], and a further case describes splenic rupture identified incidentally during work-up for unstable angina in a patient loaded with aspirin, clopidogrel, and heparin [8]. A parallel literature describes ASR in patients on oral anticoagulants, including apixaban, rivaroxaban, and dabigatran [9] [10] [11], where impairment of the coagulation cascade rather than primary platelet-mediated hemostasis is the presumed operative mechanism.

Almost all previously reported antiplatelet-associated cases share a common temporal pattern: rupture within days of percutaneous

coronary intervention or an acute loading dose, usually during an acute coronary syndrome. Our patient differed because he had received stable maintenance dual antiplatelet therapy for approximately two years, with no recent coronary intervention, acute coronary syndrome, or other recognized precipitant. The absence of underlying splenic disease on final histopathology strengthens the classification as atraumatic splenic rupture, although a causal relationship with chronic dual antiplatelet therapy cannot be proven from a single case. The case therefore raises the possibility that long-term antiplatelet therapy may contribute to clinically significant splenic bleeding outside the immediate post-PCI period.

From a surgical standpoint, the more consequential gap in the literature concerns management rather than mechanism. Evidence guiding the choice between splenic artery embolization (SAE) and splenectomy is drawn almost entirely from blunt trauma cohorts, graded by trauma injury-severity scales, in stable patients without a competing indication for early resumption of antithrombotic therapy. Data specific to non-traumatic, antiplatelet-associated splenic hemorrhage — where the trigger for operative intervention is a stepwise hemoglobin decline rather than a graded injury on a trauma scan, and where prompt, reliable resumption of dual antiplatelet therapy for coronary stent patency is itself a competing clinical priority — are comparatively sparse. We report this case, prepared in accordance with the CARE reporting guidelines [6] and, given its surgical nature, informed by the SCARE 2025 framework [7], both to compare our diagnostic and operative course directly against previously published cases and to offer a surgical decision-making perspective on this less-represented clinical scenario.

Case Presentation

Patient information

The patient was a 62-year-old man with a history of coronary artery disease and six previously placed coronary stents. The most recent stent had been placed approximately two years before this presentation, and a subsequent angiogram approximately six months later showed no indication for further intervention. His medical history also included asthma managed with inhaled therapy, heart failure with preserved ejection fraction, prior varicose vein surgery, lumbar spine surgery for disc herniation and spinal stenosis approximately nine months earlier, and a motor vehicle collision approximately three years earlier that did not result in a diagnosed injury or hospital admission. He was maintained on chronic dual antiplatelet therapy with aspirin and clopidogrel, together with a proton-pump inhibitor. There was no history of oral anticoagulant use, known splenomegaly, hematologic malignancy, recent febrile illness, recent invasive abdominal procedure, or preceding trauma.

Clinical findings

He presented with an 11-hour history of generalized abdominal pain that was progressively worsening, positional (maximal in the supine position), and aggravated by coughing and straining, with an associated sensation of bloating. He specifically denied palpitations, syncope, vomiting, diarrhea, or fever. On evaluation, he was hemodynamically stable and afebrile (blood pressure 113/61 mmHg, heart rate 79 beats per minute, sinus rhythm), alert and fully oriented (Glasgow Coma Scale 15), with mild bilateral lower-limb pitting edema. The abdomen was soft and distended, with mild tenderness on superficial palpation in the left iliac fossa and normal bowel sounds; there were no peritoneal signs.

Timeline

Timepoint	Event
Day 1	Onset of generalized, positional abdominal pain (11 hours before presentation); no preceding trauma
Day 1 (ED)	Non-contrast CT: moderate hemoperitoneum, left flank/pelvis; source unclear; incidental bilateral inguinal hernias containing blood
Day 1 (ED, later)	Contrast-enhanced CT: no active extravasation; dual antiplatelet therapy held; ICU admission
Day 1–2	ICU monitoring; hemoglobin every 4–6 hours (11.4 → 9.7 → 10.0 → 9.2 g/dL); IV hydration, mechanical thromboprophylaxis; blood products cross-matched
Day 2 (evening)	Hemoglobin 8.4 g/dL; IV tranexamic acid 1 g given; plan for CT angiography the following morning
Day 3	CT angiography: focal capsular irregularity, posterosuperior spleen, consistent with a contained splenic capsular injury as the bleeding source; incidental proximal superior mesenteric artery stenosis
Day 3	Multidisciplinary discussion (cardiology, vascular surgery, interventional radiology); splenic artery embolization versus splenectomy discussed with patient; 2 units PRBC transfused; patient elected splenectomy
Day 4	Laparoscopic splenectomy with evacuation of pelvic hemoperitoneum; on-table endoscopy showed incidental gastric polyps, no gastric injury
Day 5 (POD1)	Vitally stable; drain output ~135 mL, serosanguinous
Day 6 (POD2)	Tolerating soft diet, passing flatus; urinary catheter removed; drain output declining
Day 7 (POD3)	Aspirin and clopidogrel resumed after surgical hemostasis was judged secure
Day 7–8	Drain removed; discharged on a short course of oral antibiotic prophylaxis
Final pathology (reported 17/06/2026)	Splenic laceration with subcapsular congestion and hemorrhage; remaining parenchyma unremarkable; two reactive hilar lymph nodes; no inflammation, infarction, thrombus, atypia, or malignancy
~Day 18	Outpatient follow-up: recovering well; mild incisional swelling (?hematoma) prompting ultrasound; referral to hematology for post-splenectomy immunization

Diagnostic assessment

Initial laboratory studies (Table) showed a normal coagulation profile (INR 1.09), mild normocytic anemia with a progressively falling hemoglobin, a normal platelet count, and mildly elevated inflammatory markers; cardiac biomarkers were not suggestive of an acute coronary event, and renal function was mildly reduced.

Test	Result	Reference range
Hemoglobin (g/dL)	11.4 → 9.7 → 10.0 → 9.2 → 8.5 → 8.4 (serial)	13.0–17.0
Hematocrit (%)	35.4	40.0–50.0
INR	1.09	—
Partial thromboplastin time (s)	39.3	—
Platelet count (×10³/µL)	257	150–410
White blood cells (×10³/µL)	8.94	4.0–10.0
CRP (mg/L)	7.92	—
D-dimer (µg/mL)	0.40	—
Creatinine (mg/dL)	1.28	—
Troponin T hs (ng/L)	18.5 → 18.0	—
CK-MB (ng/mL)	4.46	—

Non-contrast CT of the abdomen and pelvis performed in the emergency department demonstrated a moderate volume of free, high-density fluid consistent with hemoperitoneum, predominantly in the left flank and lower abdomen, with no clear bleeding source; bilateral inguinal hernias containing blood were also incidentally noted. Contrast-enhanced CT performed shortly afterward confirmed a similar, unchanged volume of hemoperitoneum around the spleen and in the lower abdomen, without free air, organ laceration, contusion, or contrast extravasation — findings that, taken alone, would not have identified the spleen as the source.

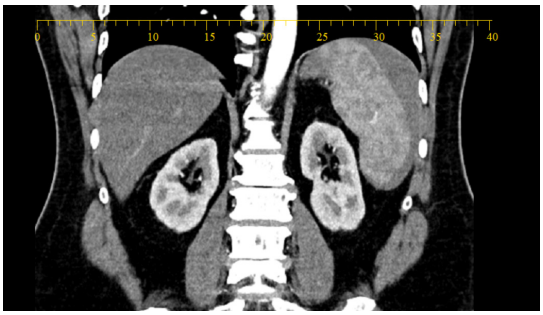

Figure 1: Contrast-enhanced CT of the abdomen and pelvis demonstrating moderate hemoperitoneum surrounding the spleen, without evidence of active contrast extravasation.

Given a serial decline in hemoglobin over the following two days despite supportive measures, CT angiography was repeated and identified a focal area of abnormal enhancement with capsular irregularity involving the posterosuperior aspect of the spleen, best seen on arterial-phase imaging, consistent with a splenic capsular injury as the source of the hemoperitoneum. This sequence — an initial contrast CT without extravasation followed by a positive dedicated CT angiogram only once hemoglobin trends declined further — is, as detailed in the Discussion, slower than the diagnostic course of most previously reported antiplatelet-associated cases, in which active extravasation was typically visible on the first contrast study [2] [3]. An incidental focal, moderate-to-severe stenosis of the proximal superior mesenteric artery and mild aortoiliac atherosclerotic disease were also noted.

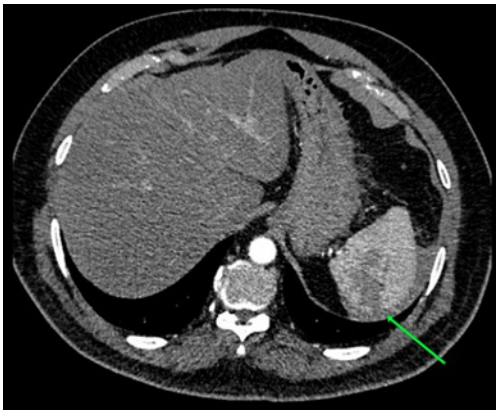

Figure 2: CT angiography (arterial phase) showing a focal capsular irregularity along the posterosuperior aspect of the spleen, consistent with a contained splenic capsular injury as the source of hemorrhage.

Therapeutic intervention

Dual antiplatelet therapy was discontinued on admission. The patient was managed in the intensive care unit with intravenous hydration, mechanical thromboprophylaxis, and hemoglobin monitoring every 4–6 hours, with blood products cross-matched and held on standby. When hemoglobin fell to 8.4 g/dL, intravenous tranexamic acid was administered.

Once CT angiography localized a contained splenic capsular injury, the case was discussed among cardiology, vascular surgery, and interventional radiology, and two management options were presented to the patient: SAE, with a quoted risk of approximately 15% for subsequent splenic abscess given the absence of a discrete extravasating branch vessel to target, or diagnostic laparoscopy with splenectomy. Trauma-derived series suggest SAE in hemodynamically stable patients is associated with lower mortality, shorter intensive care stays, and fewer early infectious complications than splenectomy [12] [13], but embolization failure requiring delayed, higher-risk conversion to splenectomy has been reported in roughly 5–20% of cases [14], and that failure rate — together with this patient's need to resume dual antiplatelet therapy promptly and reliably for stent patency — weighed against a strategy whose durability was uncertain. Even in hemodynamically unstable trauma patients, upfront splenectomy and SAE have shown comparable 30-day mortality [15], suggesting splenectomy remains a reasonable primary strategy when a competing need for early antithrombotic resumption is present. After counselling on these risks and benefits, and after discussing the need for post-splenectomy immunization, the patient elected splenectomy; informed consent was obtained.

He underwent laparoscopic exploration and splenectomy under general anesthesia. Entry was gained through the left Palmer point to avoid the perisplenic hemoperitoneum and any adhesions from prior abdominal or pelvic surgery, with pneumoperitoneum established under direct vision before additional ports were placed. On initial laparoscopic survey, a large volume of hemoperitoneum was encountered, concentrated predominantly around the spleen and left upper quadrant with extension into the pelvis; no other bleeding source was apparent on inspection, and the perisplenic clot was partially evacuated to allow safe orientation before proceeding with splenic mobilization.

The splenocolic ligament was divided first using a vessel-sealing energy device to mobilize the lower pole, and the inferior polar vessels were individually isolated, clipped, and transected — a stepwise, distal-to-hilar approach intended to keep the field relatively bloodless before

the hilum itself was addressed. The splenic hilum was then dissected to expose the splenic artery and vein, which were controlled with a combination of locking clips and endoscopic clips and transected only once adequate exposure and proximal/distal control were confirmed, given that this was a non-trauma dissection in a friable, hemorrhagic field rather than a clean elective splenectomy. The gastrosplenic ligament and short gastric vessels were then divided, followed by the splenophrenic and splenorenal attachments, at which point a posterosuperior capsular interruption corresponding to the CT angiography findings was directly visualized on the mobilized specimen. A Pfannenstiel incision was used for specimen extraction and to allow thorough evacuation and irrigation of pelvic hemoperitoneum under direct vision, after which pneumoperitoneum was re-established for a final laparoscopic hemostasis check. On-table endoscopy was performed to exclude an occult gastric injury during short gastric vessel division — a step worth emphasizing to trainees given how close this dissection plane runs to the greater curvature — and incidentally incidentally identified multiple gastric polyps for subsequent endoscopic assessment. A drain was left tracking from the left upper quadrant to the pelvis.

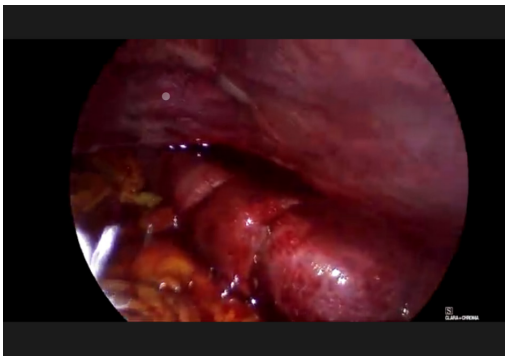

Figure 3: Intraoperative laparoscopic view demonstrating hemoperitoneum concentrated in the perisplenic space at initial exploration.

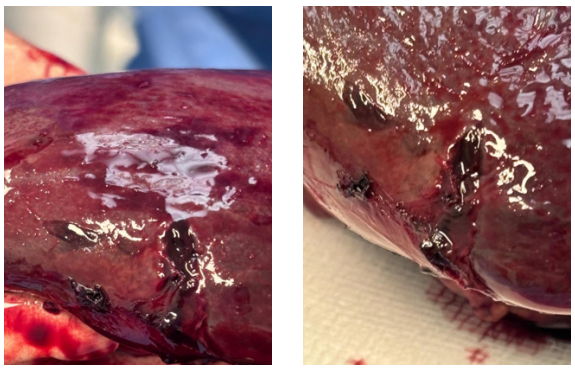

Figure 4: Gross specimen of the resected spleen following splenectomy, demonstrating the posterosuperior capsular interruption corresponding to the injury.

Histopathology

Gross examination showed a spleen measuring 15 x 9 x 5 cm and weighing 416 g, with a 3.5 x 2 cm focal raw capsular area and no solid lesion. Microscopically, the capsular lining was focally stripped, with subcapsular congestion and hemorrhage extending into the splenic parenchyma. The remaining red and white pulp was preserved, with only mild congestion. Two hilar lymph nodes were reactive. There was no acute inflammation, infarction, thrombus, atypia, or malignancy. The final diagnosis was splenic laceration with subcapsular congestion and hemorrhage, with otherwise unremarkable splenic parenchyma.

Follow-up and outcomes

The postoperative course was uneventful. On the first postoperative day the patient was vitally stable with serosanguinous drain output; by the second postoperative day he was tolerating a soft diet, passing flatus, and his urinary catheter was removed without difficulty. Aspirin and clopidogrel were resumed on the third postoperative day once the surgical team judged hemostasis to be secure. The drain was removed after output had declined, and he was discharged home on a short course of an oral antibiotic. At outpatient follow-up approximately two and a half weeks after surgery he was recovering well, with only mild swelling around the incision prompting an ultrasound to exclude a collection. He was referred for post-splenectomy immunization against encapsulated organisms.

Discussion

In the largest systematic review, drug- or treatment-related causes accounted for approximately 9% of pathological atraumatic splenic rupture [1]. Final histopathology was important in this case: it confirmed a capsular laceration with subcapsular congestion and hemorrhage but showed preserved splenic parenchyma and no malignancy, acute inflammation, infarction, thrombus, or atypia. These findings make an underlying pathological splenic process less likely and support classification as atraumatic splenic rupture. They do not, however, establish dual antiplatelet therapy as the cause; long-term platelet inhibition may have contributed to the onset, persistence, or clinical severity of bleeding. To place the case in context, we compared the antiplatelet- and anticoagulant-associated reports identified in our targeted literature search (Table).

Two features distinguish this case from the published antiplatelet-associated reports we identified. First, the comparator cases occurred after an acute trigger, usually a new antiplatelet loading dose or recent PCI for acute coronary syndrome [2,3,8]. In contrast, our patient had been on stable maintenance dual antiplatelet therapy for approximately two years, with no recent intervention or acute cardiac event. Together with the absence of underlying splenic pathology, this temporal pattern raises the possibility that chronic dual antiplatelet therapy may be associated with atraumatic splenic hemorrhage outside the immediate post-PCI period. This remains an association rather than proof of causation, and an unrecognized minor traumatic event cannot be completely excluded.

Second, the diagnostic course differed. In the comparator antiplatelet cases, the bleeding source was generally evident on the initial contrast-enhanced CT and prompted early splenectomy [2,8]. In this patient, the first contrast study showed hemoperitoneum without extravasation, and the splenic source became apparent only on repeat dedicated CT angiography after a continued hemoglobin decline. The findings were consistent with a contained, relatively low-flow capsular injury. Clinically, the case supports repeat imaging when unexplained hemoperitoneum and falling hemoglobin persist despite a negative initial contrast study.

The more central issue for a surgical audience is decision-making once a splenic source is identified outside the trauma setting. The evidence favoring SAE over splenectomy in stable patients [12] [13] is derived from graded blunt trauma injuries and does not directly address a scenario in which the bleeding is drug-related rather than structural, the injury is not gradable on a standard organ injury scale, and the patient has a separate, time-sensitive indication to resume antiplatelet therapy. In this

patient, three factors specifically favored definitive splenectomy over embolization: the reported embolization failure rate of 5–20% [14], the quoted per-patient abscess risk of the embolization approach given the lack of a discrete target vessel, and the clinical cost of any delay in resuming dual antiplatelet therapy in a patient with six coronary stents. Among our comparator cases, DAPT resumption ranged from the first postoperative day to as late as the seventh [2], and the anticoagulant-associated cases generally deferred resumption pending prolonged hematology input [9] [10] [11]; our patient's course — definitive splenectomy followed by confident resumption of DAPT on the third postoperative day once hemostasis was judged secure — sits toward the faster end of that range and may reflect the more contained, capsular nature of an antiplatelet-associated (rather than anticoagulant-associated) injury once the source is surgically controlled. We would argue this competing-priority framework — durable hemostasis versus rapid, unimpeded return to antithrombotic therapy — deserves more explicit weight in future reports and, ideally, dedicated studies of non-traumatic, antithrombotic-associated splenic bleeding, since it is largely absent from trauma-based decision algorithms and yet is often the deciding factor in practice.

Report	Age/sex	Antithrombotic & trigger	Time to source identification	Management	Antithrombotic resumption	Outcome
Present case	62 M	Chronic DAPT (aspirin + clopidogrel), approximately 2 years after last stent; no recent procedure; no underlying splenic disease on histopathology	3 days (serial Hb decline; initial contrast CT negative, CTA positive)	Laparoscopic splenectomy	POD3	Recovered, discharged
Int J Surg Case Rep 2021;88:106578 [2]	61 F	New DAPT, day 1 after PCI for STEMI	Same day (contrast CT with extravasation)	Emergency splenectomy	POD7 (ticagrelor from POD1)	Recovered
PMC6262137 [3]	Not specified	New DAPT (aspirin + ticagrelor) after PCI for ACS	Days after PCI (active bleed on imaging)	Splenectomy	Restarted after risk-benefit review	Recovered
SAGE Open Med Case Rep 2024;12 [8]	49 M	Aspirin + clopidogrel + heparin, loaded for unstable angina	Same presentation (found incidentally during angina work-up)	Splenectomy	Not specified	Recovered
Int J Surg Case Rep 2023;110:108748 [9]	Not specified	Apixaban (oral anticoagulant)	Not specified	Splenectomy	Deferred, hematology input	Recovered
Cureus 2023;15(5):e38992 [10]	Not specified	Rivaroxaban (oral anticoagulant)	Not specified	Reported non-operatively / per source	Deferred	Recovered
Clin Med (Lond) 2018;18(5):406-408 [11]	Not specified	Dabigatran (oral anticoagulant)	Not specified	Splenectomy	Not resumed at report	Not specified in available source

Two intraoperative points are worth highlighting for surgical trainees managing similar cases. First, the sequence of vascular control matters more in a non-trauma, actively bleeding field than in an elective splenectomy: mobilizing the lower pole and controlling the inferior polar vessels before committing to hilar dissection kept the operative field relatively clean in this case and is a reasonable default approach when the exact bleeding point has not been directly visualized preoperatively. Second, routine on-table endoscopy during short gastric vessel division, while sometimes omitted in elective cases, provided an added margin of safety here and identified an incidental finding (gastric polyps) that will inform this patient's future endoscopic surveillance.

A minor incidental finding on initial imaging in this patient was blood tracking into pre-existing bilateral inguinal hernias, a recognized but uncommon pattern that has occasionally been described as a source of confusion with hernia-related bleeding in other contexts [16]; it did not alter management here and is mentioned only for completeness.

This report has several limitations. It describes a single patient, and the association between long-term dual antiplatelet therapy and splenic laceration cannot establish causation. Although final histopathology excluded malignancy, acute inflammation, infarction, thrombus, atypia, and other obvious structural disease, an unrecognized minor traumatic event or a transient lesion not represented in the sampled tissue cannot be completely excluded. Platelet-function testing was not performed. The comparator cases were identified through a targeted rather than systematic literature search, and the patient perspective was not formally collected.

Conclusion

Key teaching points:

- Unlike most previously reported antiplatelet-associated cases clustered after PCI or acute loading doses, this case shows that atraumatic splenic hemorrhage may also occur during stable long-term dual antiplatelet therapy. The association is clinically relevant but does not prove causation.
- An initial contrast-enhanced CT without evident extravasation does not exclude an evolving splenic capsular source; a falling hemoglobin trend should prompt repeat, dedicated CT angiography rather than reassurance from one negative study.
- Outside the graded blunt-trauma setting, the choice between splenic artery embolization and splenectomy should consider the likelihood of durable hemostasis, embolization-related failure or abscess, and the patient-specific need for timely resumption of antithrombotic therapy.
- During laparoscopic splenectomy in a hemorrhagic field, stepwise distal-to-hilar vascular control can facilitate exposure. On-table endoscopy may be used selectively when gastric injury is a concern during division of the short gastric vessels.

Patient Perspective

A formal patient perspective was not collected for this report.

Informed Consent

Written informed consent for publication of this case report and the accompanying clinical images was obtained from the patient.

Declarations

Funding: none declared. Conflicts of interest: none declared. Author contributions: Dr. Hussam Trabulsi, Dr. Tala Muassess and Dr. Arfa Iqbal Pasha managed the patient's clinical and surgical care and conceived the report; Dr. Arfa Iqbal Pasha acquired the data and drafted the manuscript, and Dr. Hussam Trabulsi critically revised it for important intellectual content.

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