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Atraumatic splenic rupture after administering aspirin, clopidogrel, and heparin to a patient with unstable angina: A case report

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Abstract

It is a rare clinical phenomenon when a normal spleen ruptures spontaneously without any prior trauma. We present a 49-year-old male patient who was discovered to have a splenic rupture after he was referred to our facility as a case of unstable angina for cardiac catheterization based on nonspecific electrocardiogram (ECG) abnormalities and symptoms of chest discomfort coupled with abdominal pain and shortness of breath. He received aspirin, clopidogrel, and heparin before arriving at our emergency department. A splenectomy was performed for the patient, and he recovered well. Despite the rarity of spontaneous splenic rupture, physicians must consider it while evaluating elderly patients who are experiencing abdominal pain while on anticoagulants. Splenic rupture should always be considered, and early diagnosis is essential for a better outcome.

Keywords

Spontaneous splenic rupture, atraumatic splenic rupture, splenectomy, abdominal pain, unstable angina, case report

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Introduction

A splenic rupture can be traumatic or atraumatic. About 93% of individuals with atraumatic splenic rupture have histological abnormalities within the spleen. The remaining 7% are classified as atraumatic-idiopathic splenic ruptures.[1] The following are the diagnostic criteria for spontaneous splenic rupture: no trauma history; lack of disease in other organs known to have an adverse impact on the spleen; no evidence of splenic adhesions or scarring; a histological examination should reveal normal splenic tissue.[2]

In this case, a 49-year-old male patient arrived with a picture of unstable angina associated with abdominal pain. He received medications in accordance with the angina protocol. However, upon further assessment, it came to light that he had a splenic rupture with no history of trauma. We provide this case report to highlight the potential link between aspirin, clopidogrel, and heparin usage and atraumatic splenic rupture, as well as the need to address splenic injury in individuals reporting the abrupt onset of severe abdominal pain.

Patient was admitted and managed at a non-profit medical and academic institution. This case report has been reported in line with the SCARE Criteria.[3]

Case report

A 49-year-old heavy smoker man, with a past medical history of coronary artery disease and a drug history of regular use of aspirin once daily, referred to our tertiary hospital as a case of unstable angina for cardiac catheterization. He presented initially to the emergency department at a peripheral hospital with active chest pain and shortness of breath associated with abdominal pain. He was evaluated there and managed as a case of unstable angina. He was planned to be

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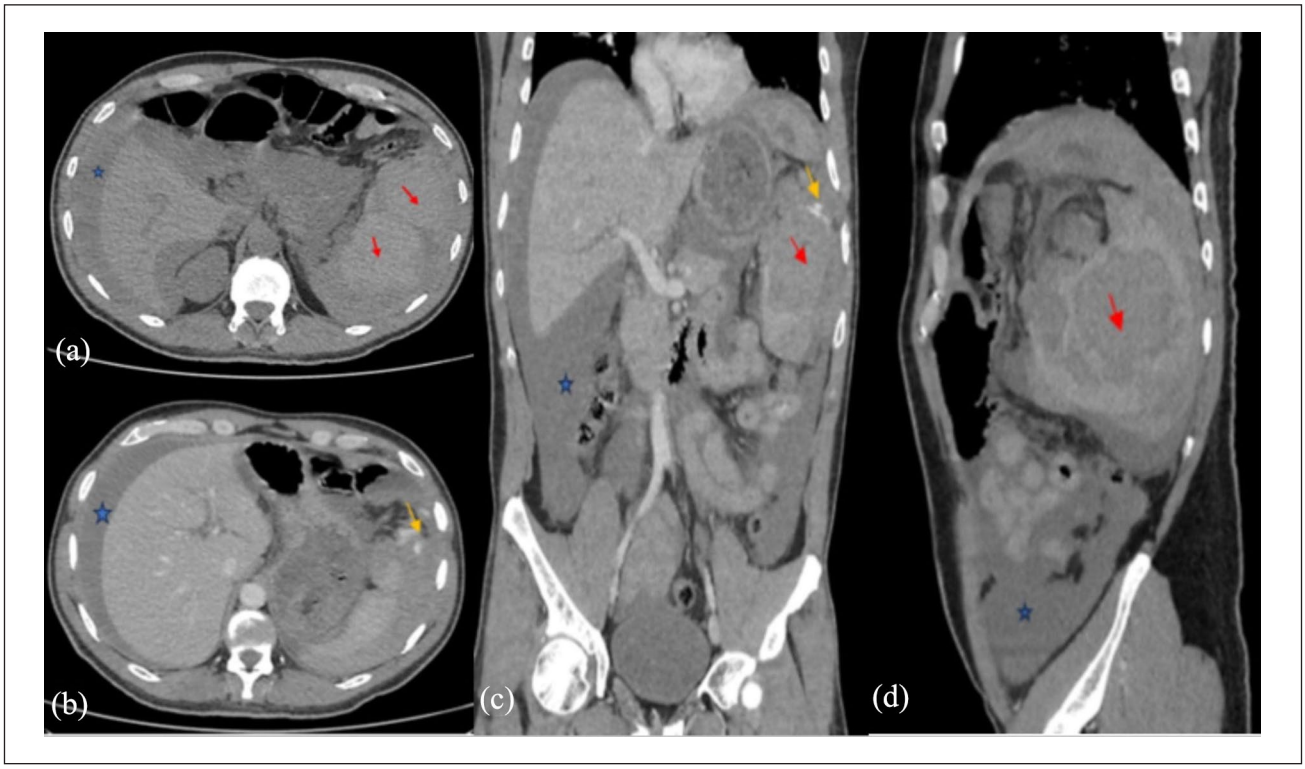

Figure 1. Chest, abdomen, and pelvic CT scan without and with IV contrast. (a) Non-contrast axial CT image shows a significant amount of dense abdominopelvic-free fluid consistent with hemoperitoneum (blue star). Multiple intraparenchymal and perisplenic hematomas (red arrows). (b)–(d) Axial, coronal, and sagittal images, respectively, show a distorted shape of the spleen with a loss of its normal appearance, replaced by multiple large intraparenchymal and perisplenic hematomas (red arrows). Multifocal areas of active extravasation are indicative of active bleeding (yellow arrows). Significant amounts of dense abdominopelvic-free fluid are consistent with hemoperitoneum (blue stars).

referred to our hospital for cardiac catheterization and given 300 mg of aspirin, 300 mg of clopidogrel, and 5000 IU of heparin according to angina protocol before arrival and before the onset of his abdominal pain. Upon arrival at the emergency department, he was complaining of severe, vague abdominal pain with a gradual onset, associated with progressive shortness of breath with mild chest discomfort, which was severe enough to cause him to be unable to complete a sentence.

It is important to note that the patient has a history of chronic abdominal pain and constipation, with chronic use of nonsteroidal anti-inflammatory drugs (Ibuprofen). He is a heavy smoker of about 60 pack years. There was no history of intercurrent or preceding illness.

His vitals initially included a heart rate of 90 beats per minute, a BP of about 140/94 mmhg, an O2 saturation of about 95% on a 4 L per minute nasal cannula and no fever. He was initially seen in the emergency department by the medical team, but due to abdominal pain, a surgical consultation was obtained. On examination, he had moderate abdominal tenderness, mainly in the periumbilical and epigastric areas, on both superficial and deep palpation, Kehr sign was negative and there was no abdominal or flank bruising. An upright chest x-ray was difficult as the patient was unable to stand, but a portable one was obtained which was unremarkable. The decision was to do a chest, abdomen, and pelvic computed tomographic scan with IV contrast (Figure 1), which revealed a grade 5 splenic injury according to the American Association for the Surgery of Trauma Splenic Injury Scale (Table 1).

About 20 min after obtaining the CT scan, he became tachycardic with a heart rate of about 115 beats per minute and a blood pressure of about 90/60 mmHg. The hemoglobin was 7.3 g/dL, the White blood cell count was 19.9 k/µl, the platelets were 223 k/µL, and the troponin was 6.9 ng/L (Table 2). The ECG showed sinus tachycardia with no ischemic changes.

He was admitted emergently to the operating room, and a splenectomy was done through a midline laparotomy (Figure 2), and bleeding was initially controlled by packing the spleen, then by clamping the splenic vessels at the hilum, then by proceeding to splenectomy. He was transferred postoperatively to the intensive care unit intubated. He was extubated after 2 days and transferred to the ward on postoperative day four and discharged home on postoperative day seven.

Table 1. American association for the surgery of trauma splenic injury scale.[4]

Grade	Injury description	
I	Hematoma	Subcapsular, < 10% surface area
	Laceration	Capsular tear, < 1 cm parenchymal depth
II	Hematoma	Subcapsular, 10–50% surface area Intraparenchymal, < 5 cm diameter
	Laceration	1–3 cm parenchymal depth not involving a parenchymal vessel
III	Hematoma	Subcapsular, > 50% surface area or expanding Ruptured subcapsular or parenchymal hematoma Intraparenchymal hematoma > 5 cm
	Laceration	> 3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration of segmental or hilar vessels producing major devascularization (> 25% of spleen)
V	Laceration	Completely shatters spleen
	Vascular	Hilar vascular injury which devascularized spleen

Table 2. Initial laboratory results upon current admission.

Parameter	Result	Reference range
White blood cell count	9.9	4–9 k/µl
Hemoglobin	7.8	13.7–17.2 g/dL
Platelets	315	140–450 k/µl
Lipase	10.1	13–60 U/L
Amylase, serum	22	28–100 U/L
Aspartate aminotransferase	13.7	15–40 U/L
Alanine transaminase	7.7	10–33 U/L
Sodium	138	135–155 mEq/L
Potassium	4.06	3.5–4.8 mEq/L
Chloride	103	98–107 mEq/L
Creatinine	1.65	0.7–1.2 mg/dL
Prothrombin time	18.1	11–14 s
International normalized ratio	1.42	0.8–1.2
Activated partial thromboplastin time	86.6	25–40 s
Troponin T	6.98	0–22 ng/L

Histopathology examination

Histological examination confirmed the non-pathological aspect of a decapsulated spleen (Figure 3).

Final diagnosis

Atraumatic spontaneous splenic rupture.

Personal and family history

The patient denied any personal and family history of illness.

Outcome and follow-up

The patient returned to the clinic 1 week later, and the wound had healed well. He was pleased and grateful with the outcome of the surgery. The patient received pneumococcal, meningococcal, and hemophilus vaccinations 14 days after the surgery, and he was discharged on a 1-year course of oral prophylaxis with amoxicillin.

Discussion

The spleen is located in the left upper quadrant of the abdominal cavity. It is a soft, friable organ that is dark purple in color with a smooth surface in healthy individuals. The spleen is variable in size, shape, and weight. It measures roughly 12 cm long, 7 cm wide, and 3 cm thick. It weighs about 150 g on average.[5]

A splenic rupture can be traumatic or atraumatic. The definition of "atraumatic-pathological splenic rupture" refers to the fact that 93% of individuals with atraumatic splenic rupture have histological abnormalities within the spleen. The remaining 7% are classified as atraumatic-idiopathic splenic ruptures; it is possible they have been misclassified as a result of an unnoticed trauma or an unrecognized illness within the spleen, the evidence of which may have been destroyed during the surgical removal of the spleen or by the rupture itself.[1]

Because intra-abdominal bleeding can be tolerated with no signs of shock, the diagnosis of splenic rupture should be taken with caution.[6] Only 1 of the 28 cases with spontaneous rupture was accurately identified preoperatively in Orloff and Peskin's study.[7] In our situation, the patient arrived with stable vitals and stayed so for a long time until he experienced hypotension after obtaining imaging to rule out intra-abdominal pathology, which was not particularly provisional splenic rupture. Upper or left-sided abdominal pain, tachycardia, and hypotension are the most prominent symptoms, followed by malaise, vomiting, diffuse abdominal tenderness and peritonism, and progressive hemodynamic shock.[8]

Various anticoagulant medicines have been linked to splenic rupture, as in our case. Several articles have linked heparin and its derivatives to this entity. A few case reports have recently been reported that link direct oral anticoagulants to spontaneous splenic rupture.[9–11] Another one reported association with dual antiplatelet therapy, aspirin and ticagrelor.[12] While the cause of spontaneous splenic rupture is unknown in many cases, a particular theory suggests that a spleen with a prior mild injury may rupture when placed under different hematic conditions, such as those seen in anticoagulant, thrombolytic, or anti-fibrinolytic medications.[13]

Although splenic rupture is frequently associated with trauma, it can occur for reasons other than trauma and have a devastating outcome. Early detection, resuscitation, and intervention are critical for saving these individuals. We think that such a scenario may mandate the use of imaging early, such as ultrasonography or computed tomography scans, after proper resuscitation. In this instance, we

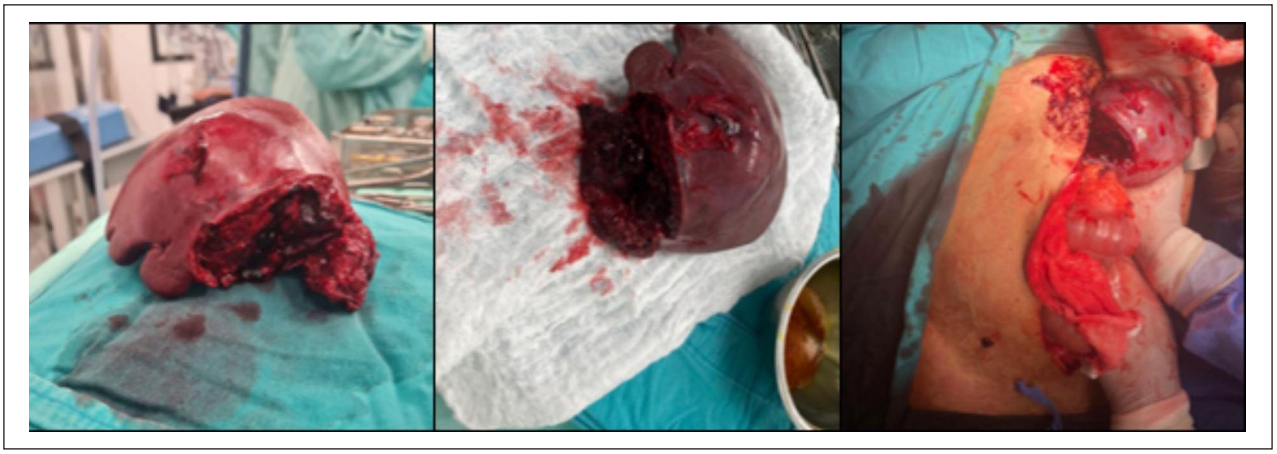

Figure 2. Intraoperative images of the spleen.

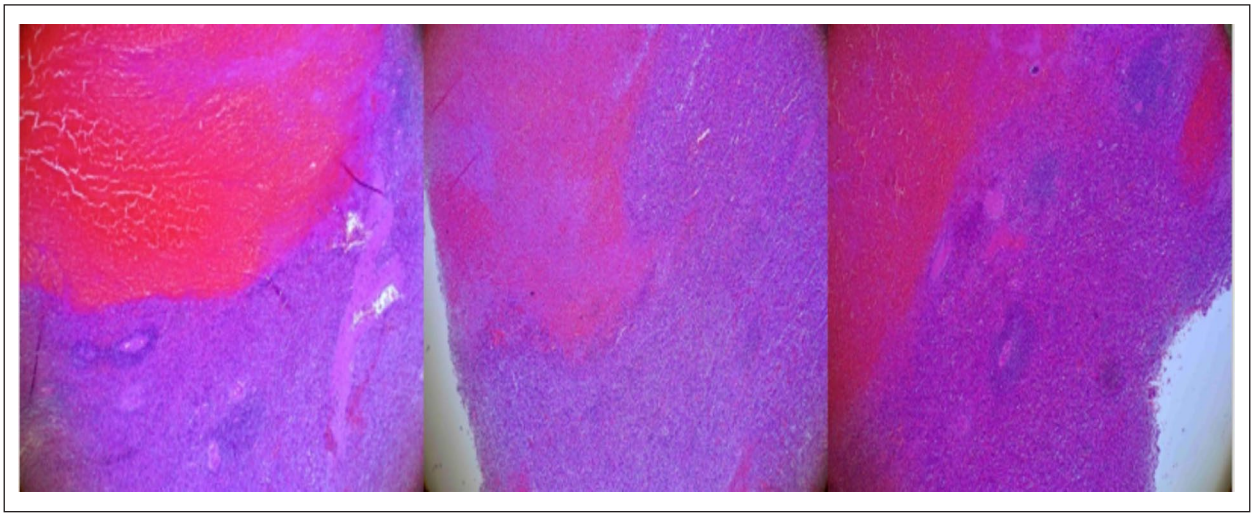

Figure 3. A three microscopic images of the splenic rupture in 4× resolution.
The spleen shows capsular laceration in microscopic morphology. Prominent parenchymal hemorrhage is present. The surrounding spleen is unremarkable, with maintained white pulp and slightly congested red pulp.

attempted to demonstrate the value of including splenic rupture in the differential diagnosis of patients with peritoneal signs and symptoms, particularly if they were on anticoagulants or antiplatelet therapy.

Conclusion

In circumstances where an adult patient complains of sudden-onset of significant abdominal pain, especially if they are taking anticoagulants or antiplatelets, splenic injury should be taken into consideration.

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Author contributions

I.M., L.D., and M.E. contributed to study concept or design; I.M., L.D., and M.E. contributed to writing the manuscript; I.M. and L.D. contributed to review & editing the manuscript; A.A. and H.Q. contributed to imaging interpretation; M.A. contributed to histopathology interpretation; L.D. and M.E. contributed to data collection.

Declaration of conflicting interests

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Ethical approval

All aspects of the study protocol, including access to and use of the patient's clinical information, will be authorized by the An-Najah National University Institutional Review Boards and the local health authorities.

Informed consent

Written informed consent was obtained from the patient(s) for their anonymized information to be published in this article.

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