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Case Report

A rare case of sequential visceral artery aneurysm rupture associated with multifocal vascular abnormalities

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ABSTRACT

Visceral artery aneurysms (VAAs) are rare but potentially fatal, with rupture representing their most serious complication. We report the case of a previously healthy 35-year-old woman who presented to the emergency department with acute abdominal pain caused by rupture of a splenic artery aneurysm, resulting in massive hemoperitoneum. She underwent emergency laparotomy with splenectomy, but approximately one hour post-operatively developed recurrent hemorrhagic shock, necessitating a second surgical intervention with proximal splenic artery ligation. Postoperative CT revealed multiple visceral aneurysms, including rupture of a hepatic artery aneurysm, along with renal and hepatic ischemia. The patient died before angiographic evaluation could be performed.

Autopsy revealed sequential rupture of the splenic and hepatic artery aneurysms, accompanied by approximately 400 mL of blood-stained fluid within the peritoneal cavity. The right kidney showed two aneurysmal dilatations involving the hilar and proximal arterial segments. Examination of the left anterior descending coronary artery demonstrated a parietal dissection with intramural hemorrhage. Death was attributed to cardiac arrest secondary to hemorrhagic shock caused by the sequential rupture of the splenic and hepatic aneurysms, compounded by hemodynamic instability related to right renal artery dissection. Additionally, the pre-existing coronary artery dissection may have aggravated the outcome by limiting myocardial oxygen reserve during the critical period of aneurysm rupture. This case underscores the diagnostic difficulty of visceral artery aneurysms, particularly in young individuals without traditional risk factors, and highlights how multifocal vascular wall abnormalities can exacerbate clinical outcomes, even in the absence of overt rupture in all affected vessels.

1. Introduction

Visceral artery aneurysms (VAAs) represent an uncommon vascular pathology, with an estimated prevalence ranging between 0.01% and 0.2% [1]. An aneurysm is defined as a permanent, localized dilatation of an arterial segment, characterized by at least a 50% increase in its diameter, resulting from structural weakening of the vessel wall that typically involves partial disruption of the elastic and muscular layers [2]. The splenic and hepatic arteries are the most commonly involved, followed in decreasing order of frequency by the celiac, superior mesenteric, gastric and gastroepiploic, intestinal, pancreaticoduodenal, gastroduodenal, and inferior mesenteric arteries [3,4].

The etiology of VAAs is heterogeneous. In elderly individuals, atherosclerosis is the predominant cause, whereas in younger patients, inherited connective tissue disorders—such as Marfan syndrome, Ehlers–Danlos syndrome, Loeys–Dietz syndrome, and fibromuscular dysplasia—and certain acquired conditions, including liver cirrhosis and pregnancy [5], should be considered [6].

With the increasing availability and sensitivity of advanced imaging modalities, asymptomatic VAAs are now more frequently identified incidentally. However, rupture may still represent the initial clinical manifestation, typically presenting with acute abdominal pain. Once hemorrhagic shock develops, the condition rapidly becomes life-threatening [7].

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Here, we report the case of a 35-year-old Caucasian woman who died due to sequential rupture of aneurysms in the splenic and hepatic arteries, occurring in the context of multiple vascular wall abnormalities, with additional involvement of the renal and coronary arteries.

2. Case report

2.1. Case history

A 35-year-old woman was admitted to the emergency department with acute abdominal pain and signs of shock, including hypotension (blood pressure 65/40 mmHg) and bradycardia (heart rate 45 bpm). Her medical and family history was unremarkable.

Initial laboratory tests revealed a marked drop in hemoglobin to 10.3 g/dL, and an urgent blood transfusion was immediately performed. Abdominal ultrasonography demonstrated a moderate amount of free fluid in the perihepatic, perisplenic, peritoneal, and pelvic cavities. A subsequent laboratory reassessment showed a further hemoglobin decrease to 7.3 g/dL, despite transfusion, prompting an urgent exploratory laparotomy.

During surgery, abundant blood clots were found within the peritoneal cavity. Inspection revealed rupture of the spleen involving both the capsule and the parenchyma near the hilum, for which a splenectomy was performed.

Approximately one hour after completion of the procedure, the patient experienced a sudden deterioration with recurrent shock (blood pressure 50/30 mmHg; heart rate 140 bpm). Oxygen was administered via Ventimask at 5 L/min, and a central venous catheter was placed for hemodynamic support. Persistent and copious bleeding through the surgical drains necessitated a return to the operating room. A second laparotomy was performed, and the splenic artery was ligated proximally to the previous surgical site.

Computed tomography (CT) revealed multiple visceral aneurysms: one arising from the right renal artery (1.5 cm), one from the superior mesenteric artery, two from the celiac artery (<1 cm), and a probable small aneurysm at the mesenteric root. At the hepatic hilum, a 4-cm round vascular structure was identified, consistent with rupture of the hepatic artery aneurysm. CT also showed hepatic and renal ischemia. An angiographic examination was planned, but before it could be performed, the patient experienced a first episode of cardiac arrest, successfully resuscitated with restoration of sinus rhythm. Approximately one hour later, a second cardiac arrest occurred, and despite prompt advanced life support with adrenaline and atropine, resuscitation attempts were unsuccessful.

2.2. Autopsy findings

A complete autopsy was performed five days after death. Tissue samples collected during the procedure were fixed in 10% neutral-buffered formalin, embedded in paraffin, and processed using standard hematoxylin and eosin (H&E) staining techniques.

At internal examination, approximately 400 mL of blood-stained fluid was found in the peritoneal cavity, and the wall of the superior mesenteric artery showed hemorrhagic infarction (Fig. 1A). The liver

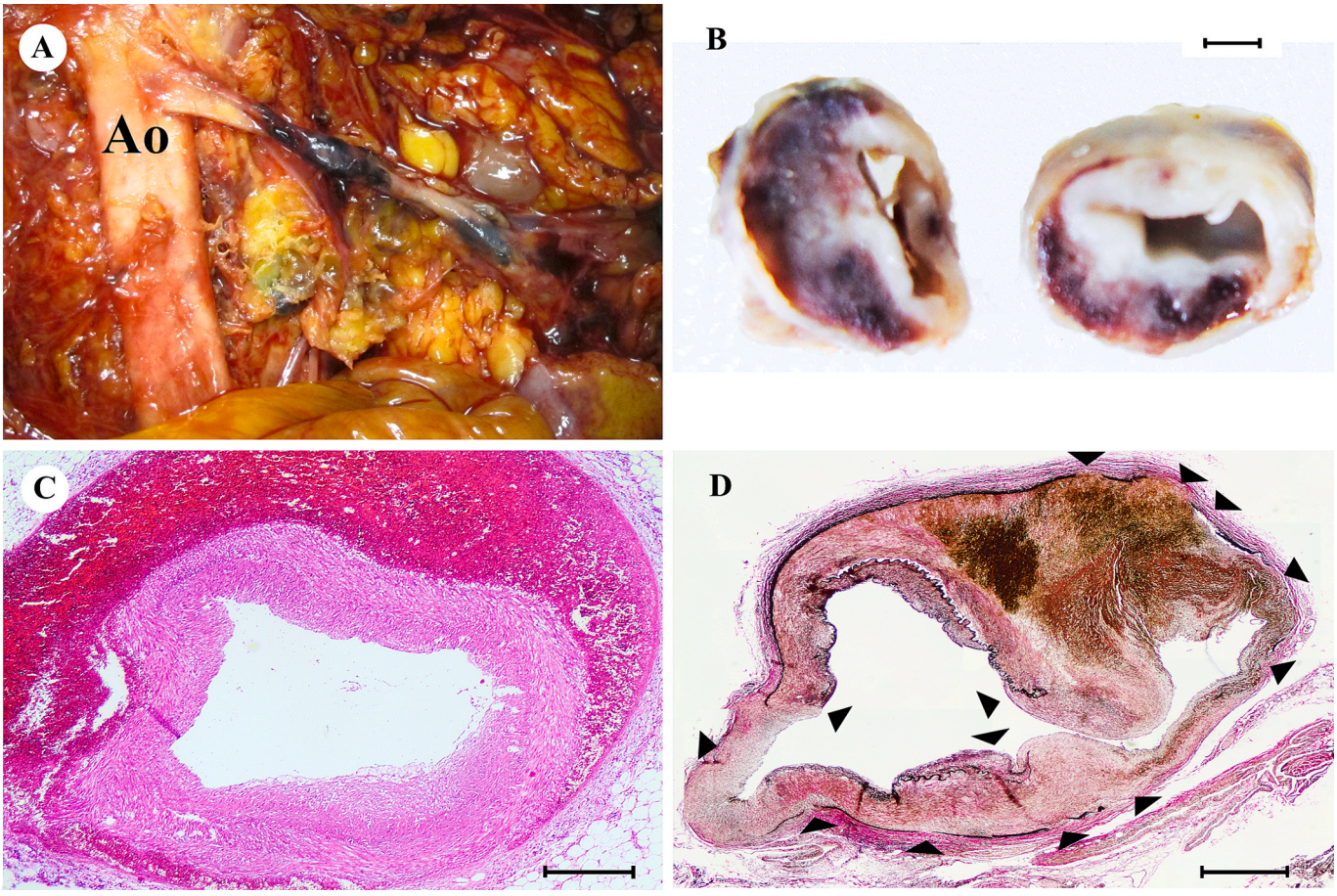

Fig. 1. A. Superior mesenteric artery. The wall shows hemorrhagic infarction. B. Left coronary artery (descending branch, macroscopic sample). Transverse sections show parietal dissection with intramural hemorrhage. C. Left coronary artery (descending branch). Hematoxylin and eosin (HE) staining shows an intramural hematoma reducing the lumen. D. Right renal artery. Elastic-van Gieson (EVG) staining reveals wall deformation due to wide dissection and multiple breaks of both internal and external elastic laminae (arrowheads). All scale bars = 0.1 cm.

appeared pale and mottled, consistent with impaired perfusion. Approximately 300 g of adherent blood clots forming a hematoma were found at the hepatic hilum. The right kidney showed two aneurysmal dilatations involving the hilar and proximal arterial segments. The heart was grossly unremarkable, except for the left anterior descending coronary artery, where transverse sections revealed parietal dissection associated with intramural hemorrhagic infiltration (Fig. 1B). Histopathological examination revealed an intramural hematoma and dissection of the left coronary artery in the absence of inflammatory or atherosclerotic changes (Fig. 1C). The renal parenchyma exhibited a variegated appearance, with extensive areas of infarction interspersed with small foci of preserved cortico-medullary tissue (glomeruli and tubules). The right renal artery demonstrated a wide dissection with an intramural hematoma leading to luminal narrowing (Fig. 1D). The superior mesenteric artery showed an intramural hematoma associated with surrounding granulation tissue and ectatic capillaries, consistent with a previous dissection event (Fig. 2.A–D). The residual portion of the splenic artery after splenectomy displayed a saccular aneurysm (Fig. 2. E–G).

3. Discussion

The typical clinical presentation of VAAs dissection is abdominal pain localized in the same region as the affected artery, often associated with nausea or vomiting [8]. However, atypical manifestations have been reported, depending on the vascular territory involved — for example, jaundice in cases of hepatic artery involvement [9].

Diagnosis relies primarily on computed tomography (CT) angiography, which provides detailed visualization of vascular anatomy and allows assessment of the extent of arterial involvement, luminal stenosis, and collateral circulation [10]. Characteristic radiological features of arterial dissection include a double lumen, intimal flap, short-segment narrowing, and irregular or tapered stenosis. The advent of advanced imaging techniques, such as three-dimensional (3D) multidetector CT, has significantly improved the sensitivity of detection and the ability to differentiate acute from chronic lesions [11]. Indications for surgical or endovascular intervention include aneurysms exceeding 2 cm in diameter, rapid aneurysmal growth, or the presence of symptoms. In particular, the occurrence of symptoms — such as abdominal pain, signs of compression, or bleeding — represents an absolute indication for intervention, regardless of the aneurysm's size [12]. More recently, therapeutic approaches have evolved to include endovascular techniques such as coil embolization and stent-graft repair, as well as minimally invasive laparoscopic procedures for arterial ligation [13,14]. The choice of treatment should be individualized, based on the specific artery involved, the aneurysm's location and morphology, the urgency of intervention, and the surgeon's expertise. In selected cases, combined surgical and endovascular approaches have been successfully adopted [15].

In our case, the absence of identifiable risk factors and the lack of

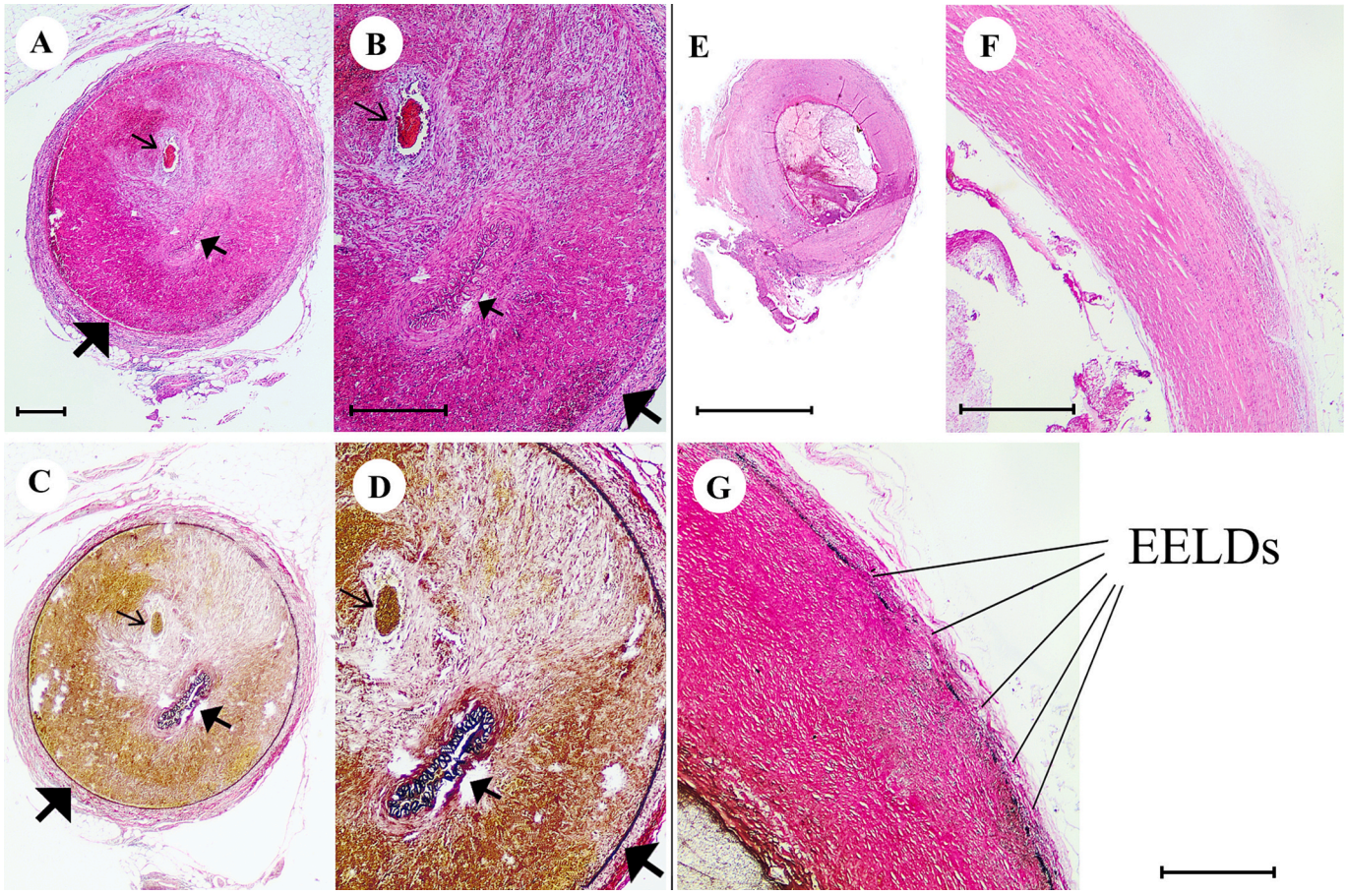

Fig. 2. A–D. Superior mesenteric artery (HE in A and C; EVG in B and D). Transverse sections show complete circular wall dissection with early hematoma organization (granulation tissue). In A, the internal elastic lamina surrounding a compressed lumen is indicated by the intermediate arrow; the outer elastic lamina by the largest arrow; the smallest arrow indicates a small secondary lumen suggestive of hematoma recanalization. Elastic fibers appear black in EVG-stained sections (B–D). All scale bars = 0.1 cm. E–G. Splenic artery (residual portion after splenectomy). Panoramic view of the cross section (E) shows intraluminal thrombotic material without wall dissection (HE; bar = 0.5 cm). At higher magnification (F), wall delamination, possibly artefactual, and replacement of the media by fibrous tissue are observed, with no other major lesions (HE; bar = 0.125 cm). EVG staining (G) highlights defects in the external elastic lamina (EELDs), indicative of fragmentation (bar = 625 μm).

atherosclerotic or inflammatory changes on histology suggest a congenital origin of the vascular abnormalities. Conditions such as inherited connective tissue disorders — including Marfan syndrome, Ehlers-Danlos syndrome type IV, Loeys-Dietz syndrome [16] — as well as certain vasculitic or autoinflammatory diseases, can lead to diffuse arterial fragility and predispose to the formation of multiple aneurysms.

Considering that neither the clinical history nor the autopsy revealed specific signs of vasculitis, one of the most plausible explanations is that a congenital or genetic disorder underlay the fatal outcome in this young woman. The suspicion of an inherited connective tissue disorder typically begins with the assessment of the patient's phenotype, as these conditions often present with distinctive craniofacial or skeletal features. Marfanoid habitus, for instance, is characterized by tall stature, long and disproportionately slender limbs, arachnodactyly, and chest wall deformities such as pectus excavatum or carinatum, often accompanied by scoliosis [17]. Patients with Ehlers–Danlos syndrome type IV and Loeys–Dietz syndrome may likewise exhibit recognizable craniofacial traits [18,19]. In the present case, the external inspection performed prior to autopsy did not reveal any morphological features suggestive of these disorders.

Family history was not investigated; nonetheless, given that such conditions often follow dominant or recessive inheritance patterns, genetic counseling for first-degree relatives would be advisable to reduce the risk of recurrence of similar catastrophic events. In this context, postmortem genetic testing — also referred to as molecular autopsy — could play a crucial role in identifying previously unrecognized hereditary arteriopathies, providing valuable information both for diagnostic clarification and for family risk assessment [20].

Another condition that remains compatible with the observed findings is segmental arterial mediolysis (SAM) [21]. This disorder is not linked to genetic mutations or autoimmune mechanisms but is instead thought to arise from acute alterations in vascular tone due to sympathetic overactivation, resulting in injury to the arterial media and subsequent development of multiple vascular abnormalities, including dissections and aneurysms. Differential diagnosis between SAM and an inherited connective tissue disorder ultimately requires genetic testing. Based on the elements available and reasoning retrospectively, if genetic testing had returned negative results, SAM would have represented the most likely diagnosis.

In investigating the cause of death and the sequence of events leading to the fatal outcome, we reconstructed the following progression, which is of particular interest given the multifocal distribution of the vascular lesions. When the patient presented to the emergency department, the splenic artery aneurysm was likely already distended, with an initial contained rupture forming a subcapsular hematoma within the lesser sac. Progressive bleeding subsequently led to splenic capsular rupture, massive intraperitoneal hemorrhage, and hypovolemic shock — a sequence consistent with the so-called "double rupture phenomenon" [22].

In this case, it is plausible that rupture of the splenic artery aneurysm at the hilar region was triggered by an acute hypertensive surge acting on an enlarging aneurysmal sac. A similar hypertensive episode may have precipitated the subsequent rupture or fissuring of the hepatic hilar aneurysm. The acute hypertensive surge hypothesized to have triggered the splenic artery rupture may have resulted from several concurrent mechanisms. Renal ischemia resulting from right renal artery dissection—occurring prior to splenic aneurysm rupture and subsequent shock—likely activated the renin–angiotensin–aldosterone system, contributing to the transient hypertensive surge [23]. In addition, pain-induced sympathetic activation and the hemodynamic impact of intra- and postoperative fluid resuscitation could have further contributed to the transient rise in mean systemic pressure and venous return [24]. In patients with underlying connective tissue fragility, even modest hypertensive peaks may precipitate aneurysmal rupture.

Based on the above considerations, the autopsy findings allowed attribution of death to cardiac arrest secondary to hemorrhagic shock, resulting from the sequential rupture of two visceral aneurysms — splenic and hepatic — in the context of hemodynamic instability caused by right renal artery dissection. Moreover, the documented dissection of the left coronary artery may have further contributed to the fatal outcome by reducing myocardial oxygen reserve at a critical moment, coinciding with aneurysm rupture.

From a medicolegal perspective, no evidence of professional negligence or diagnostic error was identified. The clinical decisions made by the healthcare team — including prompt imaging, timely surgical intervention, and appropriate management of postoperative complications — were considered consistent with current standards of care.

The only theoretical alternative that might have improved the patient's chances of survival would have required the information provided by a preoperative angio-CT, potentially enabling an early endovascular approach and precise localization of all bleeding sites requiring correction. However, such a strategy is feasible only in the presence of adequate hemodynamic stability [10]. In this case, the patient presented in profound shock, making immediate laparotomy the only viable life-saving intervention.

4. Conclusion

This case report describes an exceptionally rare presentation of sequential rupture of two visceral artery aneurysms in the setting of multiple vascular abnormalities, ultimately resulting in the patient's death despite appropriate medical and surgical management. The aneurysms were clinically silent and only became apparent following catastrophic complications, underscoring the challenges of early diagnosis, as previously reported in the literature [25].

The case highlights the importance of including visceral artery aneurysms in the differential diagnosis of unexplained acute abdominal pain, even in young individuals without conventional risk factors. Moreover, it illustrates how multifocal arterial involvement can worsen the clinical outcome, even in the absence of overt rupture — as observed in this case, where renal and coronary artery dissections likely contributed to hemodynamic instability and cardiac failure. Finally, this report aims to draw attention to the importance of performing postmortem genetic studies in cases of rare and fatal vascular disorders. Although such analyses were not conducted in this case, we emphasize that they could provide crucial insights into the underlying genetic conditions responsible for vascular fragility and the fatal outcome, and should be considered in similar future cases.

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Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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