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Hypertensive emergency presenting with an isolated celiac artery dissection: A rare case study

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ABSTRACT

INTRODUCTION: To our knowledge the most recent article on celiac dissection was published in 2015 and reported 24 known cases of spontaneous isolated celiac trunk dissection [2]. While some of those cases reported hypertension as a risk factor, no other case presents as hypertensive emergency with an isolated celiac artery dissection.
PRESENTATION OF CASE: A 43 year-old man with a past medical history of uncontrolled hypertension, for which he had reportedly been non-compliant with follow-up, presented with complaints of severe, sudden-onset epigastric pain which was non-radiating and constant for 1 hour prior to arrival. On CT an intimal flap was noted within the celiac trunk, starting at the origin and extending into the left gastric, splenic, and the common hepatic arteries.
DISCUSSION: The most common symptom in patients with celiac artery dissection is acute or chronic epigastric or abdominal pain [2,4,9,11]. The crux of the diagnosis of this condition relies on contrast enhanced CT. The superiority of the CT scan is because of the contrast tracking capability [11]. The two most common risk factors for celiac artery dissection are hypertension followed by vasculitis. Patients can be managed nonoperatively or with one of a few operative procedures. Conservative treatment consists of anticoagulants, antihypertensives, and antiplatelet therapy [2].
CONCLUSION: To the best of our knowledge, we present the 25th case of isolated celiac artery dissection. This is the first case of hypertensive emergency induced spontaneous isolated celiac trunk dissection in literature. Our patient was managed primarily with a labetalol drip.

1. Introduction

1.1. Rationale

To our knowledge the most recent article on celiac dissection was published in 2015 and reported 24 known cases of spontaneous isolated celiac trunk dissection [2]. In our literature review we did not identify a single case of isolated celiac artery dissection that presented during a hypertensive emergency. We present a case of hypertensive emergency induced spontaneous isolated celiac trunk dissection, which was managed nonoperatively with a labetalol drip.

1.2. Background

The definition of an arterial dissection is the cleavage of the arterial wall by an intramural hematoma located between two elastic layers [2,4]. As we are reporting the 25th known case of celiac artery dissection, it remains a rare entity. Many of the other reported cases involve other arteries such as the common hepatic, splenic, superior mesenteric, and gastroduodenal [1,2,4,9–12]. Additionally, while some of those cases reported hypertension as a risk factor, no other case presents as hypertensive emergency with an isolated celiac artery dissection. The first case of visceral artery dissection involved the superior mesenteric artery (SMA) and was reported in 1947, while the first celiac artery dissection was not reported until 1959 [2]. More commonly, arterial dissections occur in the carotid and renal arteries [4]. When they occur in the visceral arteries, the most common location is within the SMA [1,4]. Celiac artery dissection

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has been described in blunt abdominal trauma, but it only accounts for 1–2 percent of all visceral vascular lesions [10].

2. Case study

A 43 year-old man with a past medical history of uncontrolled hypertension, for which he had reportedly been non-compliant with follow-up, presented with complaints of severe, sudden-onset epigastric pain which was non-radiating and constant for 1 h prior to arrival. The patient denied associated nausea, vomiting, fevers, or chills. He denied chest pain, shortness of breath, lightheadedness, and dizziness. He reported moving his bowels prior to the onset of symptoms, and noted passing flatus after the onset of symptoms. He denied issues with urination. The patient denied a history of recent abdominal trauma. On initial examination his vital signs consisted of a temperature of 98.1° Fahrenheit, heart rate of 78, respiratory rate of 16, blood pressure of 234/144, and 99% oxygen saturation on room air. His hypertensive emergency in the setting of severe abdominal pain necessitated an emergent vascular surgery consultation. The patient was alert and oriented, and in distress. His lungs were clear to auscultation bilaterally and his respirations were non-labored. His heart rate and rhythm were regular. Examination of the abdomen revealed a soft, non-distended, and obese abdomen. The patient was diffusely tender to palpation, with maximum tenderness overlying the epigastric region. There was no evidence of voluntary guarding or rebound, and he did not display peritoneal symptoms. Pulse examination was positive for 2+ radial, femoral, dorsalis pedis, and posterior tibial pulses bilaterally. There was a well-healed, vertical, surgical scar in the left groin from an unspecified procedure secondary to a remote history of traumatic stab wound. Laboratory analyses (complete blood count, coagulation parameters, comprehensive metabolic panel, lactic acid, and troponin I) were all within normal limits.

Contrast enhanced (oral and intravenous) computed tomography of the chest and abdomen/pelvis demonstrated normal course and caliber of the great vessels, ascending aorta, descending aorta, and abdominal aorta without aneurysmal dilatation. There was no evidence of displaced intimal calcification, intramural hematoma, or dissection flap to suggest an aortic dissection. An intimal flap was noted within the celiac trunk, starting at the origin and extending into the left gastric, splenic, and the common hepatic arteries. The true and false lumens of the celiac trunk appeared well perfused. From the level of the bifurcation of the celiac trunk, perfusion of the true lumen of the left gastric, splenic, and common hepatic artery was noted. The superior mesenteric artery, bilateral renal arteries, inferior mesenteric artery, common iliac arteries, and internal/external iliac arteries were patent without aneurysmal dilatation. There were no signs of ischemic change of the liver and stomach, and the pancreas was of normal size and contour. The spleen appeared heterogeneous. The patient was treated with 8 mg of morphine with only slight relief from his symptoms. The patient was also treated with 10 mg labetalol IV and his systolic blood pressure improved into the 140s. An additional 6 mg of morphine was administered for persistent abdominal pain and an additional 10 mg labetalol was prescribed.

The patient was admitted to the Cardiac Intensive Care Unit for aggressive blood pressure control and was initiated on a labetalol drip to maintain systolic blood pressure less than 120 mm Hg. With aggressive blood pressure control throughout the evening of hospital day 0 and hospital day 1, the patient's abdominal pain decreased in intensity. He was then started on a regular diet, which he consumed without nausea, vomiting, or exacerbation of his pain. On hospital day 1, the patient was weaned off of the labetalol drip, and his blood pressure medication regimen was transitioned to a by mouth route of hydralazine, labetalol, and amlodipine. He was subsequently downgraded to the General Medical Floor with telemetry monitoring.

3. Discussion

The most common symptom in patients with celiac artery dissection is acute or chronic epigastric or abdominal pain [2,4,9,11]. In many of the case studies the patient presented to the emergency department with sudden onset epigastric pain [2,9,16]. Another cardinal symptom for patients with chronic pain is weight loss [4]. Other manifestations include obstructive jaundice, pancreatitis, intestinal angina, tachycardia, hemorrhage, and hypertension [4,10,13]. Interestingly, patients do not usually present with nausea, vomiting, or peritoneal symptoms [4,11,16]. Another case study had a patient present with hematemesis, melena, and postprandial pain [14]. One of the more unique presentations was in a patient who had a history of long-term energy drink consumption and intense exercise routines [7]. Patients with celiac artery dissection may also have a self-limited course of these aforementioned symptoms [9]. The majority of patients with this pathology are middle aged men [4]. Our patient fits this most commonly noted demographic.

One case series elucidated many of the complications of celiac artery dissection [4]. Extensive of the dissection may occur into adjacent arterial walls, including those of the splenic artery and proximal hepatic arteries. Renal arteries and the splenic artery may also infarct in the first week after celiac dissection. The hepatic artery may develop an aneurysm. Additional case studies report other complications. One case study noted that the dissection extended into the common hepatic artery [9]. Another patient presented with upper gastrointestinal hemorrhage as a complication [14]. There was one patient who experienced a sequential SMA dissection in the subsequent week [8] During his hospital stay our patient did not experience any of these complications..

The crux of the diagnosis of this condition relies on contrast enhanced CT (CT). Other modalities that can be used are CT angiography (CTa), magnetic resonance imaging (MRI), magnetic resonance angiography (MRA), and Doppler ultrasonography (US) [9]. CTa is used for definitive diagnosis as it allows precise determination of collateral circulation [11,13]. US is of some efficacy because one can assess areas of abnormal flow in the proper habitus [11]. One of the key findings on CT is an intimal flap [4,9]. Other cases have found infiltration of the fat surrounding the celiac artery or celiac artery aneurysm [4,9]. The stranding of the adjacent soft tissue is suggestive of focal hemorrhage [10]. Additional CT findings include intramural thrombus formation, splenic infarctions, segmental stenosis [2,9]. Intramural hematoma has been identified on CT and may lead to moderate narrowing of the vessel [11]. Dissection length is variable. One study describes a celiac artery dissection of 14 millimeters [2]. Another reported a length of 8–12 millimeters [13]. In the one patient that presented with upper gastrointestinal hemorrhage CT showed enhancement of perigastric and gastric intramural vascular collaterals due to chronic ischemia secondary to celiac artery dissection [14].

The superiority of the CT scan is because of the contrast tracking capability [11]. Additionally, follow up CT studies can be compared to the original one. Follow up imaging is recommended in the management of patients with celiac dissection. One proposed protocol has follow up imaging performed at 1 week and 2–6 months [4]. Another case study repeated the CT scan as early as 12 h later [11]. Yet another performed it 3 days after admission [13]. Regardless of the timeline chosen, it is imperative to have serial CT scans to monitor for the potential serious complications of celiac artery dissection.

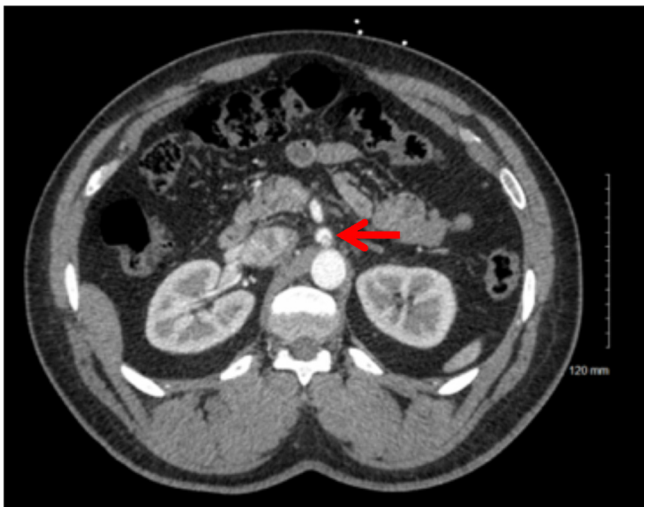

Fig. 1. Contrast Enhanced Axial CT of the Proximal Celiac Artery Dissection.

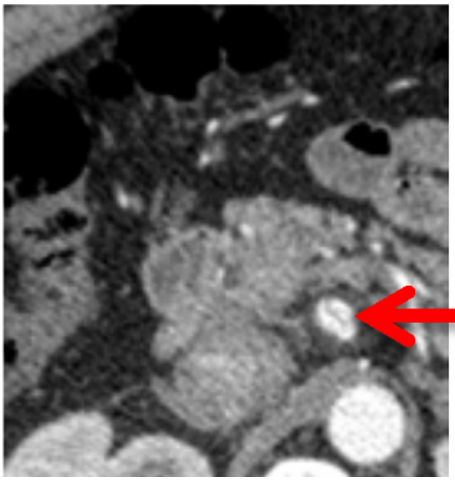

Fig. 3. Contrast Enhanced Axial CT of the Smaller True Lumen and Larger False Lumen.

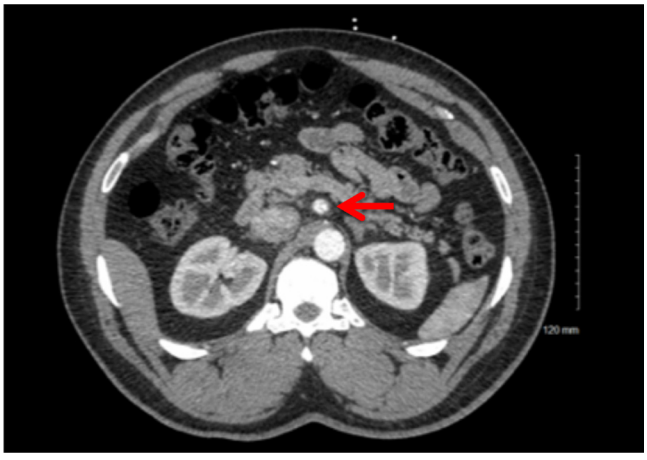

Fig. 2. Contrast Enhanced Axial CT of the Celiac Artery Dissection with Dual Lumens.

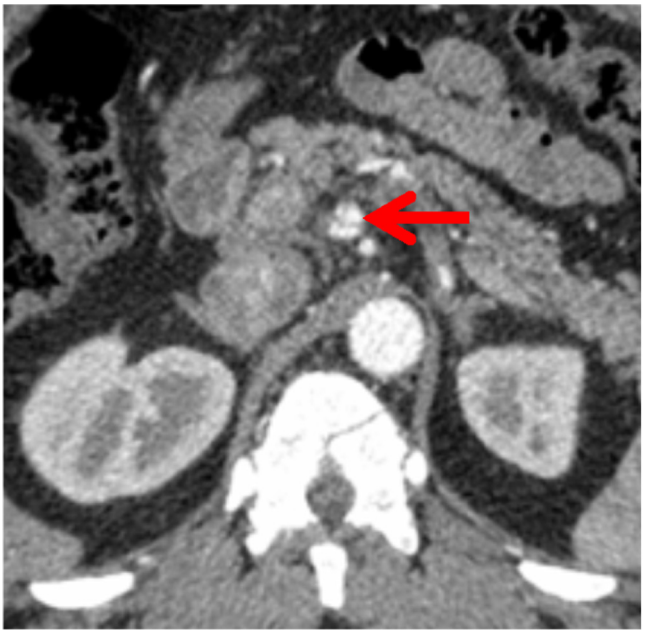

Fig. 4. Contrast Enhanced Axial CT of the Dual Lumens at Distal Celiac Artery.

There are many identifiable and accepted risk factors for celiac artery dissection [2–4,6,7,10,13]. Hypertension is an identified risk factor, though celiac artery dissection as the presenting symptom for hypertensive emergency has yet to be described until now. In addition to hypertension, the other known risk factors are atherosclerotic disease, abdominal aortic aneurysm, cystic medial necrosis, fibromuscular dysplasia, connective tissue disorders such as Ehlers-Danlos syndrome, trauma, previous abdominal surgery, stenosis or occlusion of a major artery, syphilis, polyarteritis nodosa, and pregnancy [2,4,10,11,13]. One case series noted that the two most common risk factors for celiac artery dissection were hypertension followed by vasculitis (particularly polyarteritis nodosa) [6].

Patients can be managed nonoperatively or with one of a few operative procedures. Conservative treatment consists of anticoagulants, antihypertensives, and antiplatelet therapy [2]. Anti-inflammatory drugs and steroids may be indicated to treated underlying conditions such as polyarteritis nodosa [4]. The duration of anticoagulant and antiplatelet therapy is variable but it is thought 3–6 months is sufficient. [4,8] For anticoagulation heparin or lovenox bridging to warfarin is recommended [11,13]. Aspirin dosed at 81 milligrams has been used for antiplatelet therapy [10]. Anticoagulation is essential in order to prevent thromboembolic complications [9,10]. In order to reduce the risk of propagation of the dissection and rupture, cardiac risk factors must be modified [4]. Antihypertensive therapy is thus a critical component of nonoperative management [4,9,10]. If liver damage is suspected, serial liver function tests should be ordered [10]. One retrospective review of 19 patients with celiac artery dissection, published in 2015, notes that long term anticoagulation is not needed in patients undergoing endovascular stenting [5].

Complications such as occlusive lesions, aneurysm formation, arterial rupture, hemodynamic instability, persistent pain, uncontrolled hypertension, ischemia, end organ damage, or dissection extensive are indications for operative treatment [2,4,9,13]. When surgical intervention is necessary, a vascular surgeon should be consulted emergently [4]. Operative treatment may entail resection and anastomosis, prosthetic bypass, or endovascular stenting [4,5,15]. Successful transcatheter embolization has also been reported in the literature [11,13].

Follow-up for the patient population with celiac artery dissection should include CT scan with IV contrast at 3- and 6-month intervals to evaluate for stability of the dissection [15]. Earlier imaging may be obtained should patients become increasingly symptomatic on a more accelerated timeline. In patients with con-

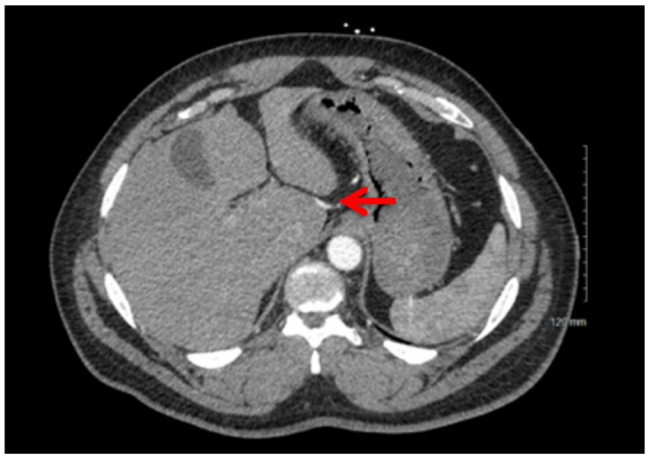

Fig. 5. Contrast Enhanced Axial CT of the Common Hepatic Artery.

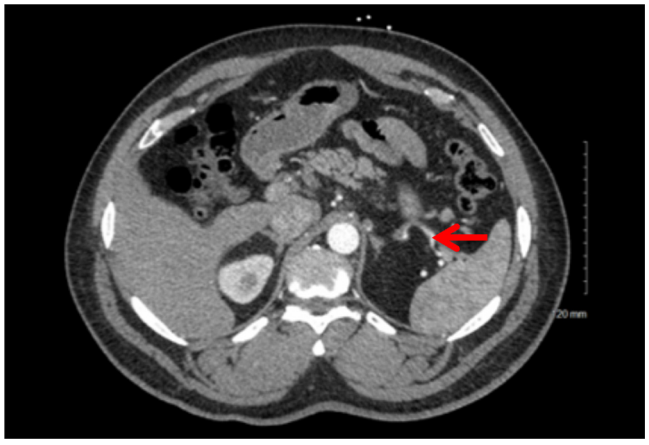

Fig. 6. Contrast Enhanced Axial CT of the Contrast Filled Splenic Artery.

traindications to IV contrast injection, ultrasound in conjunction with MRI may suffice as an alternative option.

It is reported that celiac artery dissection has a poor prognosis [4]. This prognosis, though, depends on the extent of involvement of subsegmental branches [4,9]. Involvement of the renal, hepatic, or splenic arteries can cause detrimental end-organ damage. Our patient had isolated celiac artery involvement. Going forward his prognosis will ultimately depend on control of his blood pressure. In the event that his symptoms recur, he will be a surgical candidate (Fig. 1–6).

4. Conclusion

To the best of our knowledge, we present the 25th case of isolated celiac artery dissection. This is the first case of hypertensive emergency induced spontaneous isolated celiac trunk dissection in literature. Our patient was managed primarily with a labetalol drip. Visceral artery dissection is a rare commodity, and celiac artery dissection is less common than dissection in the SMA. It is a difficult diagnosis to make on history and physical alone, necessitating contrast enhanced CT imaging. In patients who present with acute onset abdominal pain in the setting of hypertension, we recommend an immediate contrast enhanced CT scan. In patients with contraindications to contrast, ultrasound combined with MRI is an alternative.

Conflicts of interest

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Ethical approval

No research conducted on patient and he has given approval - Steven D. Kozusko on behalf of all authors.

Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images - Steven D. Kozusko on behalf of all authors.

Author contribution

Study concept or design – Dr. Swergold
Data collection – Dr. Swergold
Data analysis or interpretation – Dr. Swergold
Writing the paper – Dr. Kozusko
Editing – Dr. Rivera
Oversight – Dr. Sturt
Steven D. Kozusko on behalf of all authors.

Guarantor

Steven D. Kozusko

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