Celiac dissection after blunt abdominal trauma complicated by acute hepatic failure: Case report and review of literature

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Injuries of the abdominal visceral vessels are uncommon but devastating entities resulting in extremely high rates of mortality. The most common cause of abdominal vascular injuries is penetrating trauma, accounting for 90% to 95% of these injuries. In contrast, blunt trauma accounts for 5% to 10% of all abdominal vascular lesions. Although traumatic injury to the celiac artery is among the rarest of all vascular injuries, mortality can be as high as 75%. We report a 66-year-old patient who sustained multiple injuries in a motor vehicle crash. The initial whole-body computed tomography (CT) scan revealed a combination of severe brain injury and bilateral thoracic lesions. On day 6 after the accident, the patient's clinical situation deteriorated rapidly. At this time, the abdominal arterial CT scan showed a dissection of the celiac artery. Therapeutic anticoagulation was not feasible because of the intracranial hemorrhage. Also the patient's clinical situation worsened so rapidly that interventional therapy, including surgical and endovascular treatment, could not be performed. Finally, the patient died of fulminant hepatic failure, therefore not surviving a potentially treatable injury. The diagnosis of celiac artery dissection in this patient was significantly delayed because the initial trauma CT protocol did not include an arterial phase of the abdominal vessels. (J Vasc Surg 2007;46:576-80.)

Injuries to the abdominal visceral vessels are uncommon but devastating entities resulting in extremely high rates of mortality.[1] Because these injuries are quite rare, trauma surgeons do not gain proper experience managing this injury pattern. The small amount of literature gives evidence to this fact.

The most common cause of abdominal vascular injury is penetrating trauma such as gunshot injuries. Blunt abdominal vascular injuries occur less frequently, with an incidence of approximately 5% to 10% of all abdominal vascular injuries.[1] The most frequent location of these injuries is the superior mesenteric artery (SMA); in contrast, an involvement of the celiac artery seems to be extremely rare. Only few cases of celiac artery dissection in trauma patients are described in literature. Possible mechanisms of injury in restrained passengers could be the sudden increase of intra-abdominal pressure with diaphragm elevation and compression of the celiac artery by the median arcuate ligament (MAL) or a deceleration injury of the celiac artery against the MAL.[2,3] Despite the rarity of celiac artery dissection, awareness of this pathology is important because of its life-threatening complications.

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We report a case of celiac artery dissection caused by blunt abdominal trauma that was complicated by development of acute hepatic failure and discuss the etiology, diagnosis, and therapy of this rare traumatic lesion based on current literature.

CASE REPORT

A 66-year-old man was involved in a frontal motor vehicle crash as a restrained front seat passenger. In the field, the patient's systolic blood pressure was 90 mm Hg, pulse was 110 beats/min, and the Glasgow Coma Score was 13. Immediately after admission to the trauma shock unit of our level 1 trauma center, the patient was intubated because of rapid neurologic deterioration.

A whole-body computed tomography (CT) scan was performed in accordance with our trauma resuscitation protocol. The cranial CT revealed bifrontal brain parenchyma contusion, bleeding, and subarachnoid hemorrhage. The chest CT scan showed multiple bilateral rib fractures along with a bilateral hematopneumothorax. Abdominal findings were described as normal. The patient underwent immediate placement of chest tubes on both sides as well as insertion of a cerebral ventricular catheter for continuous intracranial pressure monitoring.

The patient was transferred to the surgical intensive care unit, where he had ongoing blood loss from the chest tubes, being hemodynamically unstable. With mass transfusion of packed red cells (35 units), fresh frozen plasma (36 units), thrombocyte concentrates (9 units), and fibrinogen concentrate (9 grams), the patient's hemodynamic situation stabilized within the first 24 hours after the trauma. Intracranial pressure ameliorated to levels of <15 mm Hg under conservative brain pressure therapy.

On day 5 after the trauma, a follow-up CT scan revealed no progression of hemorrhage or indirect signs of brain pressure. For the first time, the abdominal CT scan raised suspicion of a dissec-

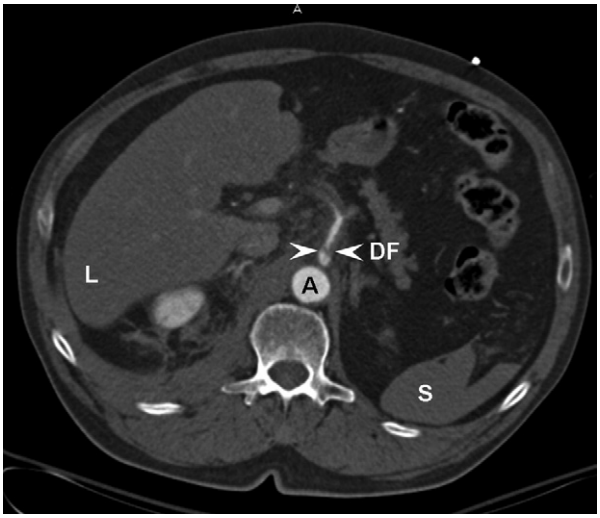

Fig 1. Arterial phase computed tomography scan image of the abdomen performed on day 5 after trauma. For side orientation, the liver *(L)* and spleen *(S)* are marked. The image shows the dissection flap *(DF)* within the celiac artery, originating from the aorta *(A)*. The dissection is characterized by the presence of an intimal flap with concomitant wall hematoma.

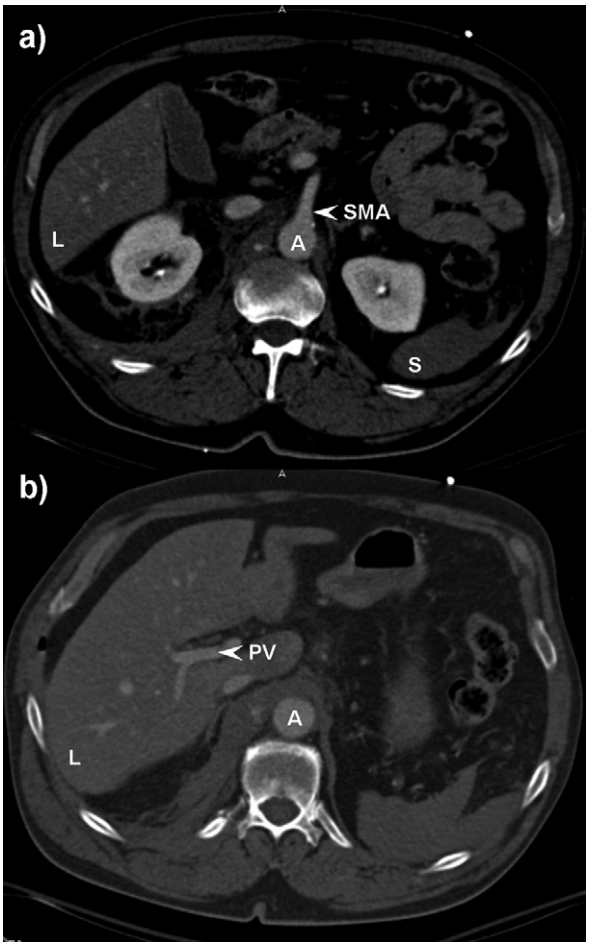

Fig 2. a, Follow-up abdominal native computed tomography (CT) scan on day 7 after trauma depicts complete thrombotic obliteration of the hepatic artery, originating from the dissection of the celiac artery *(CD, white arrows)*. The liver *(L)* presents with large multilocular perfusion deficits *(PD)* within the right liver lobe *(white arrows)*. **b,** A CT scan of the abdmen on the same time point as **(a)** shows further perfusion defects within the liver *(PD, white arrows)*.

tion of the celiac artery without alteration of visceral perfusion (Fig 1). One day later, signs of acute liver failure developed, with an increase of total bilirubin to 35 mg/dL (normal, <1.3 mg/dL), alanine aminotransferase (ALAT) to 6.200 U/L (normal, <40 U/L), aspartate aminotransferase (ASAT) to 2.300 U/L (normal, <40 U/L), γ-glutamyltransferase (γ-GT) to 760 U/L (normal, <40 U/L) and ammonia to 130 μmol/L (normal, <50 μmol/L). All potentially hepatotoxic medications were eliminated, frequent glucose monitoring was set up, and dobutamine therapy was started at a dosage of 10 μg/(kg · min) to improve visceral perfusion. Inquiry of the family revealed that the patient was a chronic abuser of alcohol but had no known history of liver disease.

On day 7, the patient's situation deteriorated rapidly. He became hemodynamically unstable, developing a fulminant hepatic failure with a total bilirubin of 40 mg/dL, ALAT of 23.000 U/L, ASAT of 5.600 U/L, and γ-GT of 980 U/L. Hepatic synthesis of clotting factors collapsed, leading to generalized coagulopathy and hemorrhagic diathesis.

A repeat abdominal CT scan now demonstrated complete obliteration of the hepatic artery, originating from the dissection of the celiac artery. The liver showed large multilocular perfusion deficits (Fig 2). There was neither an injury of the superior mesenteric artery (SMA) seen nor any evidence for compromise of the portal venous flow (Fig 3). A few hours later the patient died of fulminant hepatic failure with subsequent coagulopathy and hemodynamic instability.

Autopsy affirmed dissection of the celiac trunk (Fig 4) with consecutive thrombosis of the hepatic artery over a length of 1.5 cm (Fig 5). The SMA was inconspicuous, without signs of occlusive disease. The liver presented diffuse large necrotic areas. No bowel or spleen malperfusion and no other unknown injuries were reported. Thus, autopsy findings confirmed that death was due to hepatic failure and liver necrosis after dissection of the celiac artery.

DISCUSSION

Penetrating trauma is the most common cause of abdominal vascular injuries, accounting for 90% to 95% of these injuries. Visceral vascular injury after blunt abdominal trauma occurs less frequently and accounts for 5% to 10% of visceral vascular lesions.[4] Traumatic injury to the celiac artery is among the rarest of all vascular injuries reported, and a only few such injuries caused by blunt trauma are reported.[1,2,5,6] The incidence of celiac artery injury in blunt abdominal trauma is estimated to be 1% to 2% among all visceral vascular lesions, but the mortality rate is as high as 75%.[5,7,8]

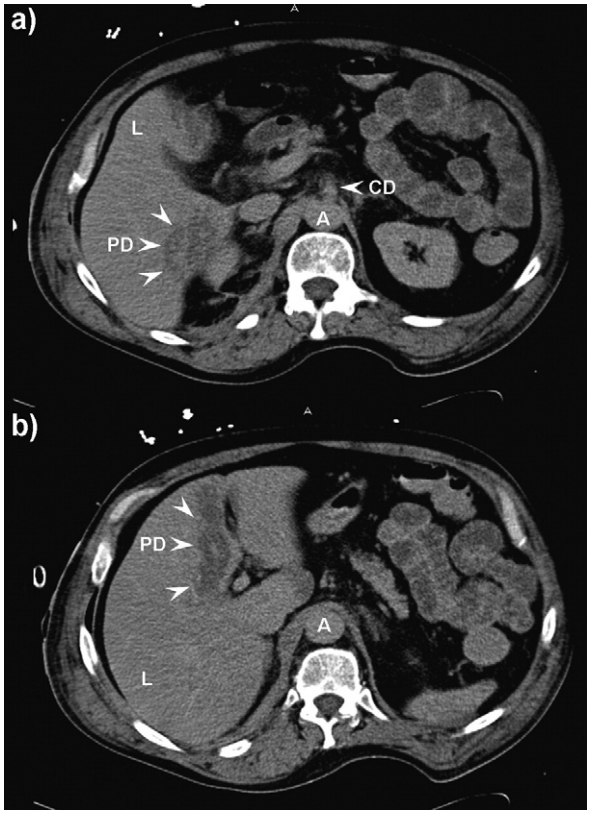

Fig 3. a, Follow-up abdominal excretion phase computed tomography (CT) scan on day 7 after trauma shows the superior mesenteric artery *(SMA, white arrow)*, showing inconspicuous findings. *L*, Liver; *S*, spleen. **b,** A CT scan of the abdomen on the same time point in portal venous phase shows the portal vein *(PV, white arrow)*, also showing patency and normal flow.

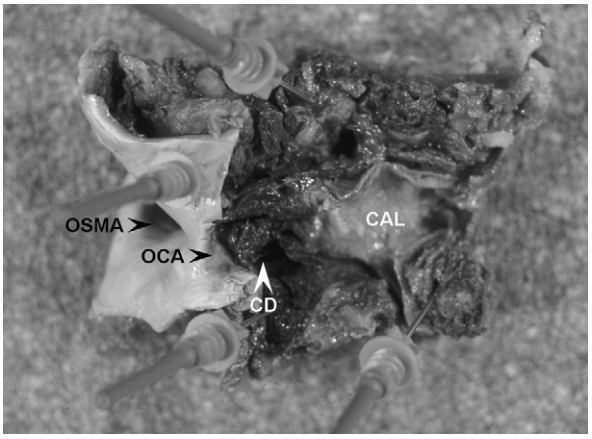

Fig 4. This macroscopic view shows the ostium of the superior mesenteric artery *(OSMA)* on the left side and the ostium of the celiac artery *(OCA)*. Within the region of the celiac trunk, the thrombotic conclusion of the dissection of the celiac artery *(CD, white arrows)* is visible. On the right side, the true lumen of the celiac artery *(CAL)* is shown.

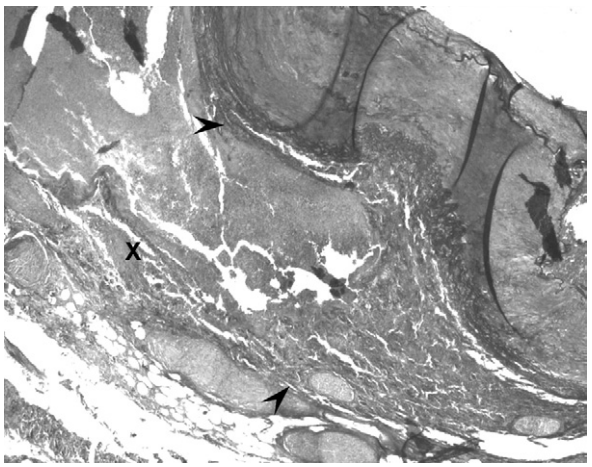

Fig 5. A section of the celiac artery (original magnification ×100, van Gieson staining), shows dissection *(black arrows)* with numerous erythrocytes and fibrin *(X)* within the external elastic layer.

Anatomically, the celiac artery arises from the anterior aorta, just below the aortic hiatus of the diaphragm at the level of the 12th thoracic vertebra, usually measuring between 1.0 and 1.5 cm in length. The splenic, left gastric and common hepatic arteries emanate from the celiac artery.

The anatomic relation of the celiac artery to the MAL might play an important role in the pathogenesis of blunt celiac artery injury. Anatomic studies have shown that in MAL anomaly, compression of the celiac artery can result during expiration (celiac artery compression syndrome).[9,10] Concerning abdominal trauma, Linuma et al[2] reported one case of celiac artery intimal injury in a patient after abdominal compression. Thus, they hypothesized that sudden increase of intra-abdominal pressure with compulsory elevation of the diaphragm might have led to a compression of the celiac artery by the MAL and consecutive intimal celiac artery injury.[2] In a high-energy car accident, however, a deceleration injury of the celiac artery against the MAL, as known from aortic dissections leading to shear forces and tearing of the intima, also might be considerable.[3] Both theories might explain the dissecting injury in the presented case, because belt injury is characterized by sudden increase of intra-abdominal pressure as well as sudden deceleration.[3,11] Predisposing anatomic or pathologic abnormalities have been described for spontaneous celiac artery dissection as rare and nonspecific.[12]

Although only 35% of the hepatic arterial blood flow is derived from the hepatic artery and the rest is from the portal vein, >50% of the hepatic oxygen supply originates from the hepatic artery.[13,14] It has been shown that a healthy liver can partly tolerate an isolated interruption of the arterial influx at the level of the celiac artery.[15] In case of pre-existing hepatic impairment, the portal perfusion decreases due to the increased portal pressure and hepatic resistance. A decreased portal perfusion can be compensated partially by an increase of hepatic artery flow, which is,

however, insufficient to restore normal liver perfusion. In contrast, the portal vein cannot compensate for perfusion deficits originating from reduced hepatic artery flow.[8]

Development of collateral circulation by phrenic, subcostal, and gastric arteries within a few days after occlusion of the celiac artery has been described as a second mechanism for restoration of arterial blood supply.[16,17] However, Graham et al[7] reported a case of acute liver necrosis after hepatic artery ligation, and Fujisawa et al[18] described a case of celiac artery compression syndrome with absence of collateral pathways, leading to transient liver dysfunction.

For diagnostic work-up of multiple trauma patients, CT has become increasingly valuable because it allows for a sensitive and specific evaluation of a wide range of injuries, including vascular lesions.[19] Recent prospective studies on radiologic diagnosis of diseases of the aortoiliac system demonstrated sensitivity of CT angiography of 91% up to 97% and specificity between 95% and 99% compared with digital subtraction angiography (DSA).[20-22] These studies were performed on four-row CT scanners just like the one used in the presented case.

According to our trauma CT protocol, abdominal scanning is performed 70 seconds after the start of intravenous contrast agent administration. This allows for a precise examination of the parenchymal organs, although one drawback is the missing evaluation of dissecting abdominal arterial injury due to a lack of intravascular contrast enhancement.[23] Therefore, in a complex clinical setting or in case of nonspecific findings of the initial scan, a follow-up CT examination after 6 to 8 hours should be performed.[24,25] Especially in high-energy trauma, the performance of a biphasic abdominal contrast-enhanced scan is obligatory. In our case, the dissection was only recognized in a follow-up CT, which was performed as late as 5 days after trauma.

In case of assumed or proven dissecting injury, precise anatomic delineation and evaluation of collateral circulation can only be obtained by angiography.[26,27] Moreover, angiography is essential for the further planning of therapeutic strategy.[28,29]

Conservative nonoperative treatment might be possible in patients with limited dissection in whom serial examinations have demonstrated no evidence of rupture or expansion. Hashimoto et al[30] described successful, conservative treatment in a 28-year-old woman with extensive celiac artery dissection. Franque et al[31] described a case of posttraumatic liver necrosis after portal vein and hepatic artery thrombosis. In contrast to Hashimoto et al, Franque et al emphasized the value of anticoagulation therapy; however, in multiple traumatized patients with concomitant brain injury, the risk of anticoagulation needs to be considered carefully.

Concerning surgical and interventional therapy of traumatic celiac artery injuries, most strategies refer to penetrating injury with major hemorrhage. The reported injuries were either treated with primary arteriorrhaphy, ligation, or end-to-end anastomosis.[1,7,8] The gold standard for treatment of acute occlusive mesenteric ischemia has been open surgical repair for decades, although mortality rates of 40% to 60% have been reported.[32] With the advent of endovascular techniques, the treatment options have been expanded. Patients with comorbidities that prohibit open repair should now be considered for endovascular strategies.[33]

In contrast to occlusive mesenteric ischemia, only a few authors have reported endovascular treatment of traumatic mesenterial vessel injury, although endovascular management of vascular trauma seems particularly appealing in the management of blunt truncal injuries, especially in the setting of severe concomitant organ lesions, such as brain and lung injury.[34] In the context of spontaneous celiac dissection, several authors reported treatment using vascular or endovascular repair. Chaillou et al[35] reported a case of placement of a polytetrafluoroethylene prosthetic graft for reconstruction of the celiac artery. So et al[36] presented a case of iatrogenic celiac artery dissection being recanalized with balloon fenestration of the intimal flap.

CONCLUSION

The optimal therapeutic strategy in blunt traumatic celiac artery dissection remains undetermined, but treatment is mandatory and revascularization must be considered, especially if occlusive lesions jeopardize liver or bowel function.

The clinical course in the presented case was complicated by the coincidence of several drawbacks. At first, the diagnosis was delayed obviously due to the discussed technical properties of our trauma CT protocol. Second, the pattern of severe traumatic brain injury prevented us from establishing anticoagulant therapy betimes. Third, immediately after the diagnosis of the dissection had been confirmed, the patient's clinical course worsened so rapidly that interventional procedures seemed no more promising.

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