Case Series

Pancreaticoduodenal arcades as salvage route for transarterial embolization of life-threatening hepatic hemorrhage in patients with severe celiac axis stenosis: Case series

Ali Barah a,*, Ahmed Omar b, Ayman El-Menyar c, Omran Almokdah a, Ahmed Sayedin a, Ala Alsherbini a, Ahmed Almuzrakshi a, Hatem Khalaf b, Hassan Al-Thani c

a Clinical Imaging Department, Hamad Medical Corporation, Doha, Qatar
b Hepato-Biliary Surgery Department, Hamad Medical Corporation, Doha, Qatar
c Trauma and Vascular Surgery Department, Hamad Medical Corporation, Doha, Qatar

A R T I C L E   I N F O

Article history:
Received 22 January 2018
Received in revised form 21 March 2018
Accepted 23 March 2018
Available online 11 April 2018

Keywords:
Celiac axis stenosis
Pancreaticoduodenal arcades endovascular management

A B S T R A C T

INTRODUCTION: Various transarterial embolotherapies for different hepatic etiologies are performed through the celiac axis (CA). However, this pathway is not always patent due to the extensive stenosis or occlusion of the origin of CA. In such situations, the pancreaticoduodenal arcades (PDAs) catheterization is the main alternative to gain access to the hepatic arteries as demonstrated in clinical studies. PRESENTATION OF CASE: We report two cases of life-threatening hepatic hemorrhage indicated for emergency transarterial embolization (TAE).

DISCUSSION: The massive hemorrhage was due to spontaneous rupture of hepatocellular carcinoma (HCC) in the first case and due to post liver blunt trauma in the second case. Owing to severe stenosis of the origin of CA, PDAs were used as a salvage alternative route for emergency TAE of hepatic arteries.

CONCLUSION: Endovascular management of massive hepatic hemorrhage in cases of inaccessibility to hepatic arteries through CA is a highly challenging situation in which the technical success depends on the operator experience, choice of the material and anatomical knowledge of hepatic arterial collateral supply.

© 2018 The Authors. Published by Elsevier Ltd on behalf of IJS Publishing Group Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

Transarterial hepatic interventions through celiac axis (CA) are very common nowadays for many liver treatments including embolization for hemorrhage, chemoembolization, radioembolization and hepatic arterial infusion chemotherapy. However, the direct catheterization of the hepatic arteries through CA is not always possible due to the extensive stenosis or occlusion of the CA [1]. Atherosclerosis, pancreatitis, tumor invasion, CA agenesis, iatrogenic arterial trauma due to catheterization or surgery and compressive median arcuate ligament (cMAL) are all suggested as causes of CA stenosis or occlusion [2,3]. In such situations, PDAs catheterization via the superior mesenteric artery (SMA) are the main alternative options to get access to the liver arteries [4]. Here we present 2 cases of life-threatening hepatic hemorrhage indicated for emergency TAE. In both cases, the cause of the CA occlusion was confirmed to be cMAL by performing contrast-enhanced CT (CECT) and TAE of the hepatic arteries was successfully performed by choosing PDAs route.

2. Presentation of cases

2.1. Case 1

A 46-year-old male known to have Hepatocellular carcinoma (HCC) presented to the emergency at our institute with hemoperitoneum and early signs of hypovolemic shock. The patient underwent urgent ultrasound screening, which demonstrated exophytic right lobe focal hepatic lesion associated with hyperechoic free perihepatic fluid raising the possibility of spontaneous rupture of the tumor with active hemorrhage. Contrast-enhanced CT scan revealed an extravasation of contrast in the arterial phase from the HCC and interruption of the capsule surrounding the lesion confirming the diagnosis of spontaneous rupture (Fig. 1a). The same CT demonstrated non-calcified, short localized stenosis with a characteristic superior hook shape sign at the origin of CA suggesting cMAL.
Fig 1. Multiplanar reformatted images of abdominal CECT: (a) The coronal plan showing ruptured subcapsular HCC (Arrow) and hemoperitoneum (arrowhead). (b) sagittal plan showing tight stenosis at the origin of CA (arrow) due to eMAL.

(Fig. 1b). The patient was then referred to the angio-suite for an urgent TAE. The SMA angiogram showed retrograde opacification of the hepatic arteries through PDAs confirming the severe stenosis of CA origin (Fig. 2a). Despite several attempts to cross the CA stenosis, the direct catheterization of the liver arteries remained unsuccessful. PDAs were then selectively catheterized using 2.7F microcatheter and the angiogram showed hypervascular HCC in the right liver lobe (Fig. 2b). The same microcatheter was used to perform selective TAE of the right hepatic artery using gelatin sponge.

Fig 2. (a) Selective SMA angiogram demonstrating retrograde opacification of the liver arteries through PDA (white arrow) (b) Selective catheterization of the right hepatic artery through the PDA showing hypervascular HCC lesion (white arrow) (c) Post-embolisation angiogram demonstrating successful occlusion of the right hepatic artery.
2.2. Case 2

A 45-year-old female presented to the emergency, with hypovolemic shock signs, after having a car crash trauma inducing active hepatic hemorrhage. The focused assessment by sonography for trauma (FAST) was performed in emergency department and revealed liver injury with perihepatic free fluid confirming hepatic active hemorrhage. Owing to the hemodynamically unstable conditions of the patient, she was referred emergently to the operating room for exploratory laparotomy where a massive hemoperitoneum and a grade 4 liver injury of the right liver lobe were found. The bleeder artery could not be detected during the surgery; therefore an initial hemostasis was achieved by perihepatic packing. Few hours later, the patient collapsed again and referred immediately to angio-suite for TAE. Hepatic angiogram was done and showed occlusion of the origin of the CA. The angiogram of SMA was then performed and demonstrated retrograde opacification of the hepatic arteries through PDAs (Fig. 3a). A selective catheterization of the proper hepatic artery was then successfully obtained using 2.7F microcatheter and the angiogram showed extravasation of the contrast media from a peripheral branch of the right hepatic artery (Fig. 3b,c). TAE was then performed using gelatin sponge until arterial occlusion obtained (Fig. 3d). After embolization, the patient remained hemodynamically stable and did not require further transfusion. A follow-up CECT was performed 5 days later and showed no more extravasation of contrast media and confirmed the presence of CA stenosis by cMAL (Fig. 4a,b). This case series has been reported in line with the PROCESS criteria [5] and has been registered at ResearchRegistry.com (researchregistry3605).

3. Discussion

Active hepatic hemorrhage is a life-threatening condition for which emergency intervention is required either by surgery or TAE. There are several causes of hepatic hemorrhage such as tumor rupture, aneurysm, iatrogenic injury and hepatic trauma [6]. Surgical intervention is associated with a high rate of mortality in patients with liver trauma; moreover, the rate of rebleeding after hepatic hemostasis with packing could reach up to 60% in some case series [7,8].
The utilization of CECT and ultrasonography in the emergency trauma protocols has shifted the paradigm of hepatic hemorrhage management towards the TAE across the last 2 decades [9–11]. The efficacy of liver TAE through CA has been well established with a success rate ranging from 85 to 100% [12]. The main limiting factor of liver artery catheterization through this route is a CA stenosis or occlusion with an incidence of 12.5–24% in Western populations [1,13–15]. According to Kwon et al., cMAL presents the most common cause of CA stenosis [3]. In the typical hepatic arterial anatomy; PDAs and the dorsal pancreatic artery form the main collateral pathway connections between the CA and SMA. While in patients with aberrant hepatic arterial anatomy, other collateral pathways may exist [16].

PDAs are formed by anterior and posterior branches, bring together the dividing branches of the gastroduodenal artery (GDA) and the inferior pancreaticoduodenal artery (IPDA), forming the natural anastomoses between the CA and the SMA [17]. PDAs are not usually visible in the absence of CA or SMA stenosis. In case of occlusion or severe stenosis of CA, the entire blood supplying the liver is provided by the retrograde arterial flow through PDAs, which induces arterial enlargement leading to prominence of the PDAs on SMA angiogram [18].

Many reports in the literature asserted the importance of PDAs route for different elective transarterial liver treatments in patients having CA stenosis or occlusion, such as chemoembolization, radioembolization and hepatic arterial infusion [2,3,19]. To our knowledge, PDAs are rarely reported as an alternative route in the management of acute hepatic hemorrhage. The appropriate catheterization technique is one of the key parameters for technical success of PDAs catheterization. In order to avoid long procedure time and potentially ineffective manipulations with risk of complications, the direct hepatic artery access through CA should be initially attempted for the cMAL to maintain a potential residual lumen of the CA [3].

This is more truthful in the setting of emergency TAE where more complications may occur owing to the vasospasm that can be precipitated by the catheter manipulation in addition to the coexistence hypotensive status. If this approach fails, attempts should be made to catheterize the hepatic artery through PDAs to achieve TAE [20]. In our institution, we regularly perform liver ultrasound screening and CECT prior to any intervention in hemodynamically stable patients suspected to have hepatic hemorrhage. In case of hemodynamic instability, a surgical management is preferred. In case of failed surgical hemostasis, the patient should be transferred to the angio-suite for angiogram and embolization of hepatic arteries. This angiogram helps to give further information about the hepatic arterial anatomy, the portal vein status and the direction of flow through PDAs. A direct catheterization of hepatic arteries is then attempted through the stenotic CA by using SF catheter over hydrophilic 0.035-inch guide wire. When the CA is impassable, a selective catheterization of the hepatic arteries could be attempted through SMA using a coaxial system composed by SF catheter and hydrophilic 2.7F microcatheter.

Because of the small caliber, severe vasospasm of PDAs, and hypovolemic condition in both patients, the SF catheter was left at the origin of SMA to avoid any arterial complications. Then the 2.7F hydrophilic microcatheter was advanced gently over micro-wire through PADS to the hepatic arteries. Being as selective as possible, is a prerequisite for the success of the TAE procedure for many reasons. Although the liver parenchyma has dual arterial and portal vein blood suppliers, parenchymal necrosis and liver decompensating are the potential risks of underlying hepatic disease in case of unsuccessful selective TAE. Moreover, unsuccessful selective embolization of the targeted hepatic artery leaves the possibility of collaterals to continue to supply the lacerated and extravasating artery along a retrograde flow which might increase the risk of rebleeding [21]. Finally, unsuccessful distal hepatic artery catheterization in case of inability to reach the proper hepatic artery via PDAs and GDA, greatly increase the risk of major complications, such as pancreatitis, duodenal ulcer or splenic infraction [4].

Despite the successful catheterization of the right hepatic artery through long tortuous and small sized PDAs, we have faced 2 major problems in our cases. In case 1, even with a dedicated manipulation of hydrophilic microcatheter, the catheterization of PDAs was complicated by a limited arterial dissection at midway to the target (Fig. 3b). This embarrassing situation was overcome by reattempting catheterization of the PDAs after administration of 100–μg of nitroglycerin through the microcatheter at the site of the dissection. The second major problem was the inability to advance the microcatheter beyond of the right hepatic artery and consequently, failure to achieve a superselective catheterization of the distal

Fig. 4. (a) Five days post embolization Abdominal CECT showing shattered right liver lobe with no contrast extravasation. (b) Sagital plan of multiplanar reformatted images demonstrating severe stenosis of the CA (white arrow) due to cMAL.
bleeder arteries in both cases. This is due to loss of torquability and pushability of the microcatheter after having traversed long and tortuous pathway through the PDAs. In addition, the acute angle between GDA and the proper hepatic artery and the compression effect of hematoma caused by hepatic hemorrhage on the PDAs have made the hepatic TAE more challenging with a higher risk of failure [3]. Although the sandwich technique by deploying coils on both sides of the artery supplying the extravasation is considered as the best approach to achieve selective occlusion of the arterial feeders. The embolic agent used in our cases was gelatin sponge. The reason of this choice was as result of the difficulty to obtain a superselective catheterization of the distal hepatic branches. Moreover, in urgent situation, gelatin sponge is relatively easy and fast to manipulate and induces less damage to normal liver parenchyma after embolization [22].

In conclusion, endovascular management of massive hepatic hemorrhage in case of inaccessibility of hepatic arteries through CA is a highly challenging situation in which the technical success depends on the operator experience, choice of the materials and anatomical knowledge of hepatic arterial collateral supply.

Conflict of interest
The authors have no conflict of interest.

Sources of funding
None.

Ethical approval
This case series was approved by the Medical Research Center (IRB#17163/17) with waiver consent.

Consent
Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contribution
All the following authors have contributed study concept or design, data collection, data analysis or interpretation, writing the paper: Ali Barah, Ahmed Omar, Ayman El-Menyar, Hatem Khalaf, Hassan Al-Thani, Ahmed Sayedin, Omran Almokdad, Aila Alsherbini, Ahmed Al-Muzrkchi.

Registration of research studies
Researchregistry 3605.

Guarantor
Ali Barah

References
[1] J.R. Derrick, H.S. Pollard, R.M. Moore, The pattern of arteriosclerotic narrowing of the celiac and superior mesenteric arteries. Ann. Surg. 149 (1959) 684–689.
[2] O. Ikeda, Y. Tamura, Y. Nakasone, Y. Yamashita, Celiac artery stenosis/occlusion treated by interventional radiology, Eur. J. Radiol. 71 (2010) 369–377.
[3] J.W. Kwon, J.W. Chung, S.-Y. Song, H.G. Lim, J.S. Myung, Y.H. Choi, et al., Transcatheter arterial chemoembolization for hepatocellular carcinomas in patients with celiac axis occlusion, J. Vasc. Interv. Radiol. JVIR 13 (2002) 689–694.
[4] M. Okazaki, H. Hibagashira, H. Ono, F. Koganemaru, R. Fujimoto, Y. Mizuma, et al., Chemoembolization for hepatocellular carcinoma via the inferior pancreaticoduodenal artery in patients with celiac artery stenosis, Acta Radiol. Stockh. Swed. 34 (1993) 20–25.
[5] R.A. Agha, A.J. Fowler, S. Rammohan, I. Barai, D.P. Orgill, the PROCESS Group, The PROCESS statement: preferred reporting of case series in surgery, Int. J. Surg. 36 (2016) 319–323.
[6] R. Andersson, K.G. Tranberg, S. Bengmark, Hemoperitoneum after spontaneous rupture of liver tumor: results of surgical treatment, HPB Surg. World J. Hepatic Pancreat. Biliary Surg. 1 (1998) 81–83.
[7] J.W. Johnson, V.H. Gracias, R. Gupta, O. Guilamondegu, P.M. Reilly, M.B. Shapiro, et al., Hepatic angiography in patients undergoing damage control laparotomy, J. Trauma 52 (2002) 1102–1106.
[8] T.S. Misselbeck, E.J. Teicher, M.D. Gipolle, M.D. Pasquale, K.T. Shah, D.A. Dangleben, et al., Hepatic angiobolization in trauma patients: indications and complications, J. Trauma 67 (2009) 769–773.
[9] J. David Richardson, G.A. Franklin, J.K. Lukas, E.H. Carrillo, D.A. Spain, F.B. Miller, et al., Evolution in the management of hepatic trauma: a 25-year perspective, Ann. Surg. 232 (2000) 324–330.
[10] N. Battula, D. Tsapralis, A. Takhar, C. Coldham, D. Mayer, J. Isaac, et al., Aorto-pathogenesis and the management of spontaneous liver bleeding in the West: a 16-year single-centre experience, HPB 14 (2012) 382–389.
[11] V. Monnin, C. Sengel, F. Thony, I. Bricault, D. Voirin, C. Letoublon, et al., Place of arterial embolization in severe blunt hepatic trauma: a multidisciplinary approach, Cardiovasc. Intervent. Radiol. 31 (2008) 875–882.
[12] B. Mohan, H.S. Bhopad, N. Aslam, H. Kaur, S. Chhabra, N. Sood, et al., Hepatic vascular injury: clinical profile, endovascular management and outcomes, Indian Heart J. 65 (2013) 59–65.
[13] S. Hori, E. Inoue, Y. Narumi, M. Fujita, K. Kadowaki, Hepatic arterial embolization in cases of extensive celiac artery stenosis, Radiology 178 (1991) 353–355.
[14] S.R. Reuter, T. Olin, Stenosis of the celiac artery. Radiology 95 (1965) 616–627.
[15] K.M. Bron, H.C. Redman, Splanchnic artery stenosis and occlusion: incidence, arteriographic and clinical manifestations, Radiology 92 (1969) 323–328.
[16] S.-Y. Song, J.W. Chung, J.W. Kwon, J.H. Joh, S.J. Shin, H.B. Kim, et al., Collateral pathways in patients with celiac artery stenosis: angiographic-spiral CT correlation, Radiogr. Rev. Publ. Radiol. Soc. N. Am. Inc. 22 (2002) 881–893.
[17] R. Kallamadi, M.A. Demoya, S.P. Kalva, Inferior pancreaticoduodenal artery aneurysms in association with celiac stenosis/occlusion, Semin. Intervent. Radiol. 26 (2009) 215–223.
[18] S.P. Kalva, C.A. Athanassoulis, A.J. Greenfield, C.-M. Fan, M. Curvelo, A.C. Walmant, et al., Inferior pancreaticoduodenal artery aneurysms in association with celiac artery stenosis or occlusion, Eur. J. Vasc. Endovasc. Surg. 33 (2007) 670–675.
[19] R.J. Lewandowski, K.T. Sato, B. Atassi, R.K. Ryu, A.A. Nemeck, L. Kulik, et al., Radioembolization with 90Y microspheres: angiographic and technical considerations, Cardiovasc. Intervent. Radiol. 30 (2007) 571–592.
[20] J. Cazejust, C. Garcia-Alba, N. Colignon, O. Planche, S. El Mouhadi, Y. Menu, Chemoembolization and the arcuate ligament: how to manage it? Diagn. Interv. Imaging. 95 (2014) 1105–1107.
[21] A.H. Hardy, H. Phan, P. Khanna, T. Nolan, P. Dong, Transcatheter treatment of liver laceration from blunt trauma, Semin. Intervent. Radiol. 29 (2012) 197–200.
[22] R.A. Schwartz, G.P. Teitelbaum, M.D. Katz, M.J. Fentecost, Effectiveness of transcatheter embolization in the control of hepatic vascular injuries, J. Vasc. Intervent. Radiol. JVIR 4 (1993) 359–365.

Open Access
This article is published Open Access at sciencedirect.com. It is distributed under the IJSCS Supplemental terms and conditions, which permits unrestricted non commercial use, distribution, and reproduction in any medium, provided the original authors and source are credited.