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ORIGINAL ARTICLE

Iatrogenic Pseudoaneurysms of the Extrahepatic Arterial Vasculature: Management and Outcome

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Abstract

Background. Pseudoaneurysms of the extrahepatic arterial vasculature are relatively uncommon lesions following surgery and trauma. In this report we analyze the presentation, management and outcomes of these vascular lesions. Of the related surgical procedures, the reported incidence is highest following laparoscopic cholecystectomy. We hereby analyze the literature on this subject and report our experience, specifically with extrahepatic pseudoaneurysms, drawing an important distinction from intrahepatic pseudoaneurysms. *Methods*. From September 1995 until July 2004, six patients, including three males and three females with a mean age of 67 years, were treated for seven extrahepatic arterial pseudoaneurysms. Patients were evaluated by endoscopy, ultrasound, computerized tomography, and angiography. Management included coil embolization or arterial ligation and/or hepatic resection. *Results*. The mean pseudoaneurysm size was 4.9-cm (range 1.0–11.0-cm) and the locations included the right hepatic artery (n = 5), inferior pancreaticoduodenal artery (n = 1), and gastroduodenal artery (n = 1). All six patients had prior surgical or percutaneous procedures. Median latency period between the original procedure and treatment of pseudoaneurysm was 17 weeks (range one month–16 years). Clinical features ranged from the dramatic presentation of hypotension secondary to intraperitoneal aneurysmal rupture to the subtle presentation of obstructive jaundice secondary to pseudoaneurysm mass effect. The range of patient presentations created diagnostic challenges, proving that accurate diagnosis is made only by early consideration of pseudoaneurysm. Management was ligation of the right hepatic artery (n = 4) and embolization of the pseudoaneurysms (n = 2). Post-treatment sequelae included liver failure requiring liver transplant (n = 1), intrahepatic biloma requiring percutaneous drainage (n = 1) and cholangitis with right hepatic duct strictures requiring right lobectomy and biliary reconstruction (n = 1). These complications followed arterial ligation, with no complications resulting from embolization. All six patients are alive and well after a mean follow-up of 53 months. *Conclusions*. Our six patients demonstrate the diversity and unpredictability with which a pseudoaneurysm of the extrahepatic arterial vasculature may present in terms of initial symptoms, prior procedures, and the latency period between presentation and prior procedure. Through our experience and an analysis of the literature, we recommend a diagnostic and management approach for these patients.

Introduction

The initial descriptions of hepatic arterial pseudoaneurysms (PsA) were most often associated with hepatic trauma; however, the increased use of percutaneous procedures and laparoscopic surgeries has broadened the etiological scope of this disease [1–5]. Pseudoaneurysms are associated with laparoscopic cholecystectomy [6–13], thoracocentesis [14], percutaneous liver biopsy [15,16], pancreaticoduodenectomy [17–19], and liver transplantation [20–22]. Iatrogenic injuries now account for approximately twice as many PsA as accidental trauma with the majority located intrahepatically [5,7].

Most PsA are intrahepatic and present with hemobilia. Extrahepatic PsA may present with abdominal pain, gastrointestinal hemorrhage, and hemobilia [23,24]. Many PsA of the hepatic arterial vasculature, however, may be asymptomatic prior to rupture. Depending upon the presentation and the time from the inciting factor, diagnosis may be difficult and management of extrahepatic arterial PsA is not as well delineated as for that of the intrahepatic type. Options include embolization, stent-grafting, surgical ligation, and resection.

We describe the clinical features and outcomes for six patients at our institution with iatrogenic PsA of the extrahepatic arterial vasculature. Combining our

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experience with an analysis of the literature, we make recommendations for the approach to the diagnosis and management of suspected PsA of the extrahepatic arterial vasculature.

Patients and Methods

From September 1995 through July 2004, six patients presented to the Hepatobiliary Surgery and Abdominal Organ Transplantation Division at University Hospital, University of Southern California with seven PsA of the extrahepatic arterial vasculature. A retrospective chart review was performed, with long-term follow-up obtained by direct telephone contact.

Six patients (3 male/3 female) with mean age 67 years (range: 48–80 years) presented with seven extrahepatic PsA with a mean size of 4.9-cm (range 1.0–11.0-cm). Locations included right hepatic artery (n = 5), inferior pancreaticoduodenal artery (n = 1) and gastroduodenal artery (n = 1). All six patients had one or more prior surgical procedures including liver transplantation (n = 1), percutaneous biopsy of the pancreatic head (n = 1), and open or laparoscopic cholecystectomy with (n = 3) or without (n = 1) additional biliary tract procedures. Three of the four patients with prior cholecystectomies had biliary tract procedures within one month (n = 2) and at nine months (n = 1) of the cholecystectomy for biliary tract complications sustained at the time of cholecystectomy. The time from the original surgery to presentation ranged from one month to 16 years. Details of the presentation and diagnostic evaluation of each patient is delineated in Table I.

Presentations

Presentations varied and at times made the diagnosis difficult (Table I). Patient 1 presented with acute abdominal pain ten days and hypotension twelve days following biliary tract reconstruction. The predominant presenting symptoms of Patients 2, 3 and 4 were gastrointestinal bleeding four to eight weeks following cholecystectomy with biliary tract complications (n = 2) and liver transplantation (n = 1). Patients 5 and 6 presented with less urgent symptoms including jaundice, pruritis and pancreatitis.

Diagnosis

Diagnostic modalities reflected the urgency status of each patient and included endoscopy, computerized tomography (CT), angiography and laparotomy. Patient 1 initially presented with abdominal pain raising suspicion of a bile leak and prompting a CT scan which demonstrated a 2.5-cm PsA with infarction of the right lobe of the liver (Figures 1 and 2). While there was no evidence of intraabdominal blood on the CT scan, he promptly developed abdominal distension and hypotension requiring emergent surgery. Patient 2 was properly diagnosed by CT scan following a futile attempt to locate the bleeding source endoscopically. Patient 3 was diagnosed by angiography (Figures 3 and 4) after a failed attempt to control bleeding from a presumed diagnosis of anastomotic bleeding after prior biliary reconstruction. Patient 4 underwent multiple diagnostic procedures including CT scan and angiography with embolization of a 5-cm PsA of the inferior pancreaticoduodenal artery (IPDA). Continued bleeding with massive uncontrollable bleeding into the duodenum noted on endoscopy, prompted a pancreaticoduodenectomy for presumed rupture of the IPDA at which time the diagnosis of a ruptured 2-cm PsA of the right hepatic artery into the common bile duct was made. Both patients 5 and 6 were diagnosed with CT scan followed by angiography for better delineation to determine therapeutic options (Figure 5).

Table I. Location, Size, and Presentation of Extrahepatic Pseudoaneurysm.

Patient	Site	Size (cm)	Presentation	Prior procedure	Latency period
1	RHA	2.5	Pain, anemia, hypotension	1. Open cholecystectomy	9 months
				2. Biliary reconstruction	10 days
2	RHA	5.0	Pain, gastrointestinal bleeding	1. Laparoscopic cholecystectomy	1 month
				2. Biliary stent placement for cystic duct leak	5 days
3	RHA	1.0	Gastrointestinal bleeding, elevated hepatic chemistries	1. Open cholecystectomy; hepaticojejunostomy for biliary injury	6 weeks
				2. Revision hepaticojejunostomy and enteroenterostomy for presumed bleeding	2 weeks
				3. Embolization PsA from RHA	1 week
4	1. RHA	5.0	Pain, gastrointestinal bleeding, hypotension, pancreatitis	1. Liver transplantation	8 weeks
	2. IPDA	2.0			
5	RHA	11	Jaundice, pruritis	1. Open cholecystectomy	16 years
6	GDA	8.0	Pain, nausea, vomiting, anemia, pancreatitis	1. Percutaneous biopsy of pancreatic head	6 months

RHA – Right hepatic artery, IPDA – Inferior pancreaticoduodenal artery, GDA – Gastroduodenal artery.

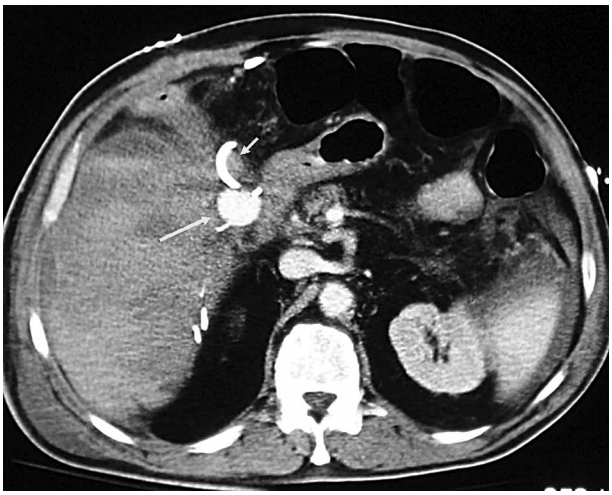

Figure 1. Contrast enhanced CT demonstrating 2.5-cm hepatic artery aneurysm (long arrow) and adjacent biliary stent (short arrow). The patient is status post bile duct resection and biliary reconstruction.

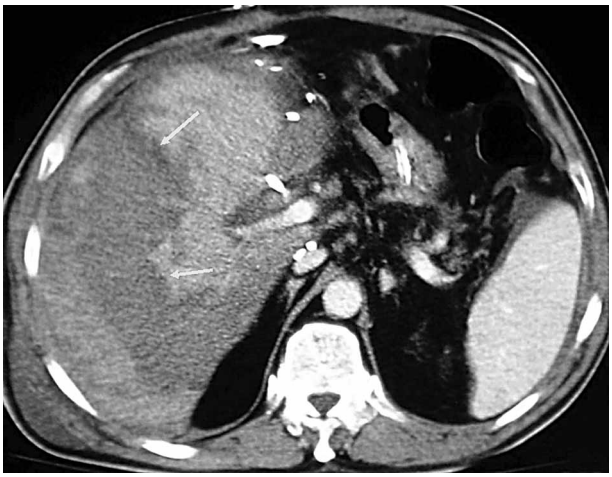

Figure 2. Contrast enhanced CT demonstrating large area of hepatic infarction (arrows).

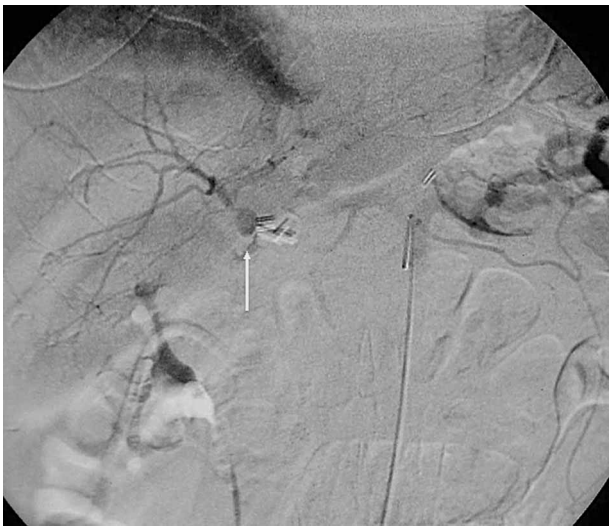

Figure 3. Delayed image following celiac axis contrast injection demonstrates a 1-cm pseudoaneurysm of the right hepatic artery, immediately distal to the surgical clips (arrow).

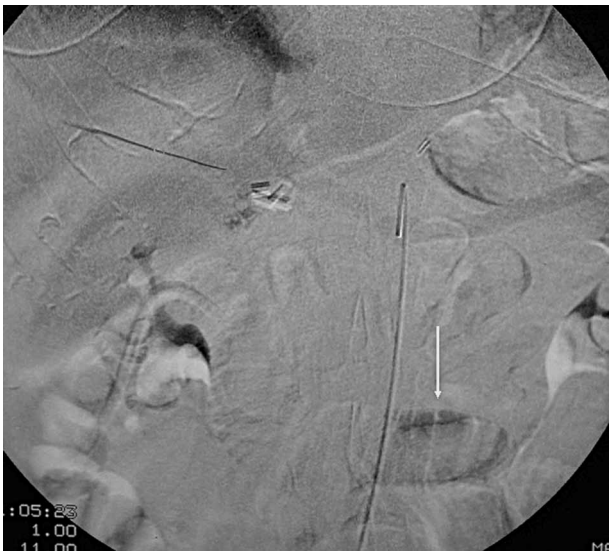

Figure 4. More delayed image from the same injection demonstrates contrast in a loop of small bowel (arrow), indicating that the pseudoaneurysm is the source of the patient's GI bleed.

Management

Of the six patients, four were managed with surgery and two with coil embolization (Table II). Management decisions were largely influenced by presentation and pre-management diagnosis. Patients 2 and 6 were stable at presentation and correctly diagnosed by CT scan and angiography leading to management with coil embolization (Figures 6 and 7). Patient 5 was correctly diagnosed with CT scan and angiography and was managed with excision of the PsA and ligation of the right hepatic artery due to the massive size (11-cm) of the aneurysm leading to bile duct compression and severe jaundice. Patients 1 and 4 underwent emergent surgery for massive bleeding, which included ligation of a ruptured right hepatic

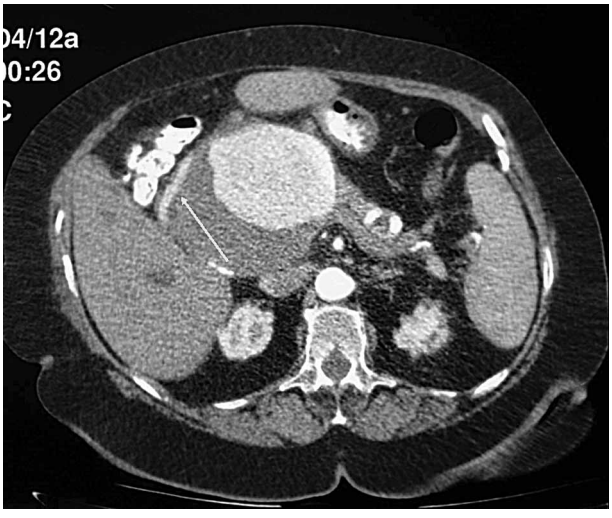

Figure 5. Patient 5: Contrast enhanced CT demonstrates a large, thrombus containing pseudoaneurysm of the right hepatic artery. The feeding artery is coursing along the lateral aspect, entering the pseudoaneurysm anteriorly (arrow).

Table II. Management, Sequelae, and Outcomes.

Patient	Management	Sequelae	Management of Sequelae	Result	Follow-up
1	Ligation RHA; RHL	Liver failure; sepsis	Liver transplant	Alive and well	21 months
2	Coil embolization	None		Alive and well	7 years
3	Ligation RHA; revision of biliary anastomosis	Cholangitis and strictures at 15 months	Right hepatic lobectomy; revision of biliary anastomosis	Alive and well	68 months
4*	1. Ligation RHA; pancreaticoduodenectomy 2. Emboilization IPDA	Biloma at 3 years	Percutaneous drainage	Alive and well	7 years
5	Ligation RHA; excision PsA; removal cystic duct stones	None		Alive and well	38 months
6	Coil embolization	None		Alive and well	21 months

CT – Computerized tomography, PsA – Pseudoaneurysm, HA – Hepatic artery, RHA – Right hepatic artery, IPDA – Inferior pancreaticoduodenal artery, GDA – Gastroduodenal artery, RHL – Right hepatic lobectomy, * – Patient with 2 pseudoaneurysms (IPDA and RHA).

artery PsA and right lobectomy (Patient 1) and pancreaticoduodenectomy (Patient 4). Following a failed attempt at gelfoam embolization, Patient 3 was managed with surgical ligation of a right hepatic artery PsA at the same time as a required revision of a biliary anastomosis.

Results

All six patients are alive and well after a mean follow-up of 53 months (range 21–84 months) (Table II). Three patients, including the two treated with coil embolization, had no significant sequelae. The other three patients required further management for complications of right hepatic artery occlusion. Patient 1 developed liver failure immediately after right hepatic lobectomy and ligation of the right hepatic artery. He underwent successful liver transplantation. Patient 3 developed cholangitis and was diagnosed with biliary strictures at 15 months. A right hepatic lobectomy and revision of a biliary anastomosis were performed. Pathologic evaluation of the right lobe revealed well-established fibrosis. These changes were not present in the left lobe. Patient 4 returned with an infected intrahepatic biloma that was percutaneously drained. Cholangiogram demonstrated no biliary stricture and there has been no recurrence.

Discussion

Presentation

The presentation of gastrointestinal bleeding following an invasive upper abdominal procedure should prompt consideration of the presence of a hepatic artery PsA. The majority of hepatic PsA are iatrogenic, as compared to pancreatic PsA, which are usually caused by pancreatitis [25]. In a literature review of 153 hepatic artery PsA (63% intrahepatic and 37% extrahepatic), Tessier reports the common presenting symptoms as hemobilia (64%), hematemesis (30%), hematochezia (14%), and abdominal pain (20%), with some asymptomatic patients (10%) [7]. There may be other unusual presentations based upon the size and location of the PsA including compressive symptoms of jaundice and pruritis. Our six patients demonstrate the wide spectrum of clinical presentations.

The prior history of hepato-pancreatico-biliary procedures in all of our patients highlights the shifting etiologic trend from traumatic to iatrogenic causation. The six patients had a mean latency time of 142 weeks (range 5 weeks–16 years). Excluding Patient 5, mean latency becomes 58 days, which is more likely to provoke consideration of the correct diagnosis. While the patient with a PsA is more likely to present in the early post-operative period, the outliers demonstrate that late presentation is possible.

Diagnosis

As the majority of patients present with gastrointestinal bleeding, initial evaluation generally includes endoscopic procedures. Endoscopy may show bleeding from the ampulla of Vater and can rule-out other etiologies of bleeding. However, endoscopy often fails to make the correct diagnosis and may even provide misleading information. After failing to make a diagnosis endoscopically, the diagnosis of PsA is most commonly made by angiography [2,3,7,9,26]. Angiography can accurately diagnose PsA and offers the therapeutic options of embolization or stenting. It is useful in identifying additional aneurysms in the vascular tree as 20% of hepatic aneurysms are multiple [23]. Angiography also delineates feeding vessels and fistulas. Both the celiac and superior mesenteric arteries must be filled to avoid a false-negative result due to the presence of a replaced right hepatic artery and to delineate the hepatic arterial blood flow that is important in determining the safety and feasibility of hepatic artery occlusion [2,5,9].

The use of ultrasound has been recommended by some as the initial test for suspected hepatic PsA [27]. Color flow Doppler shows pulsatile flow and confirms

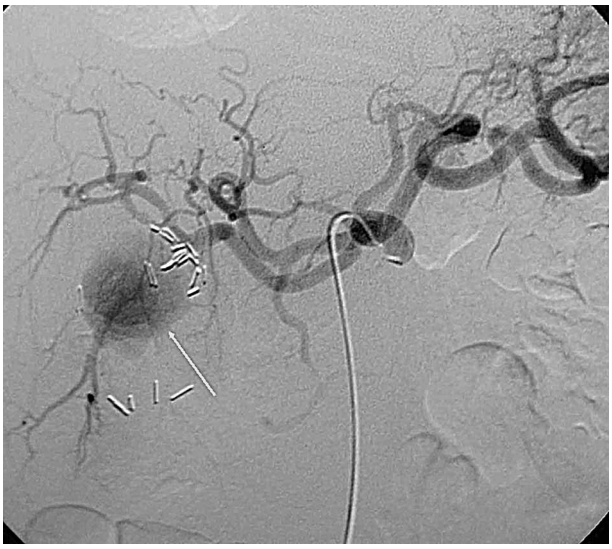

Figure 6. Celiac angiogram demonstrates a 5-cm pseudoaneurysm of the right hepatic artery (arrow) adjacent to multiple surgical clips from a prior laparoscopic cholecystectomy.

vascular patency. Unfortunately, many patients with gastrointestinal bleeding have significant bowel gas that limits visualization with ultrasound. Ultrasound is also highly operator-dependent, and patients' symptoms often demand a more comprehensive evaluation of the abdomen.

Dynamic computerized tomographic angiography (CTA) is an excellent noninvasive examination for the evaluation of visceral aneurysms and vascular anomalies, especially in the stable patient. CT is commonly used for the evaluation of aortic, visceral and renal artery pathology as well as for the diagnosis of splenic artery aneurysms [28]. CT is also useful for evaluation of extravascular structures and should be performed when the presentation is not related to gastrointestinal bleed, such as in the case of Patients 1 and 5. In both of these patients, imaging was performed for indications other than bleeding, and was able to detect the hepatic PsA.

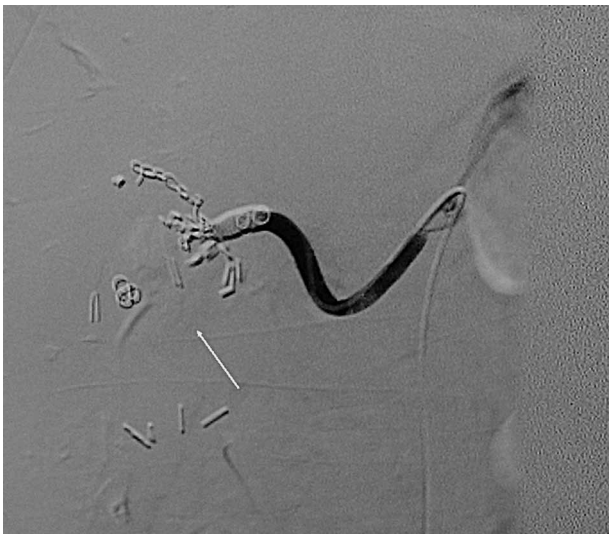

Figure 7. Post-embolization image demonstrates complete occlusion of pseudoaneurysm.

Magnetic resonance angiography (MRA), like CTA, can be performed as a part of a comprehensive examination of the abdomen. Contrast enhanced MRA is a reliable, accurate way to evaluate vascular morphology and has been used for evaluation of mesenteric ischemia, visceral aneurysms, tumor encasement and surgical planning for liver donation [29–33]. There are non-contrast techniques that can be used to evaluate blood flow, especially for the evaluation of stenosis [34,35] MRA is an alternative to CTA, especially in patients with iodinated contrast allergy and renal insufficiency. MRA is also used when reduction of radiation exposure is desired, such as during pregnancy and in the pediatric population. Although not used in the patients presented in this paper, MR imaging would have been an appropriate imaging choice for diagnosis in all but the most unstable.

Management

PsA of the hepatic arterial vasculature have a well-documented propensity for continuous enlargement and eventual rupture with a reported rupture rate of 21–80% [6,9,24,36]. Mortality from rupture is significant at 21–43% [24,27]. Regardless of the presence of symptomatology, diagnosis of a PsA indicates treatment.

Coil embolization is highly effective at controlling bleeding. Embolization is associated with a 25% lower mortality and less morbidity than surgical intervention [7]. When embolizing the right and left hepatic arteries, intrahepatic translobar collaterals usually establish flow immediately and no infarction occurs [37]. The two patients that underwent embolization alone suffered no complications. However, three of the four surgically managed patients had significant sequelae. This cannot be solely attributed to the surgical intervention because those patients undergoing surgery had complicated presentations and co-morbidities, which likely impacted upon the post-surgical course.

Although collateral circulation may prevent hepatic infarction, this is not always the case and in some circumstances, infarction may occur. There have been reports of hepatic infarction with necrosis after embolization due to sacrifice of the involved vessel and risks end-organ thrombosis [38–41]. Patients with underlying liver disease, inadequate collateral flow, or compromised hemodynamic status suffer significant parenchymal infarction and develop liver failure, which is associated with a mortality of 54% [7]. The vessel may also recanalize and require repeat embolization or surgical intervention. Patient 3 had undergone embolization prior to presenting to our institution where the vessel was found to be

recanalized. Other complications include dislodgement or migration of the obstructing material [6,41], coil erosion into the common bile duct [42], hepatic abscess formation and gallbladder fibrosis [4,24].

Although less common, treatment with stent-graft placement has been reported as an alternative to selective arterial embolization when there is concern about hepatic infarction or vascular recanalization. These stents are a non-invasive method of excluding the PsA and retaining hepatic arterial flow. There have been recent case studies documenting the success of stent-graft placement in the treatment of both asymptomatic and ruptured PsA following liver transplantation and other biliary tract surgery [43,44]. Stent placement requires an adequate length of normal caliber artery on both sides of aneurysm to allow for optimal placement. The need for anticoagulation following stent-graft placement may be a contraindication for some patients.

Surgical management includes ligation or excision of the PsA. Like embolization, serious complications can ensue including hepatic necrosis resulting in death, necessitating a liver transplantation, or requiring hepatectomy. Surgery should be considered for patients with PsA who fail embolization or have coexisting complications requiring surgical intervention.

Whether managed by hepatic arterial embolization or surgical ligation and/or resection, these patients require close monitoring during the post-operative period for signs of complications secondary to occlusion of hepatic arterial flow. Post-operative findings that should prompt a search for complications of arterial occlusion include persistently elevated or rising hepatic transaminases, fever, elevated white blood cell count and other signs of intraabdominal sepsis. The possibility of complications may persist well past the immediate post-interventional period, as demonstrated by Patients 3 and 4, who presented with complications 15 months and three years after their respective right hepatic artery ligations.

Conclusions

Our six patients demonstrate the diversity and unpredictability with which a PsA of the extrahepatic arterial vasculature may present. Diagnosis is made with a high index of clinical suspicion in conjunction with radiological imaging. The recommended treatment, if not precluded by underlying patient condition, is selective arterial embolization. This procedure is associated with an excellent prognosis and it enables precise PsA localization and hemorrhage control. The alternative treatment is surgical arterial ligation with or without PsA excision. Following embolization or surgical management, patients must be monitored for complications of impaired arterial flow.

References

[1] Croce MA, Fabian TC, Spiers JP, Kudsk KA. Traumatic hepatic artery pseudoaneurysm with hemobilia. Am J Surg 1994;168:235–8.

[2] Curet P, Baumer R, Roche A, Grellet J, Mercadier M. Hepatic hemobilia of traumatic or iatrogenic origin: recent advances in diagnosis and therapy, review of the literature from 1976–1981. World J Surg 1984;8:2–8.

[3] Czerniak A, Thompson JN, Hemingway AP, Soreide O, Benjamin IS, Allison DJ, et al. Hemobilia: a disease in evolution. Arch Surg 1988;123:718–21.

[4] Yoshida J, Donahue PE, Nyhus LM. Hemobilia: review of recent experience with a worldwide problem. Am J Gastroenterol 1987;82:448–53.

[5] Samek P, Bober J, Vrzgula A, Mach P. Traumatic hemobilia caused by false aneurysm of replaced right hepatic artery: case report and review. J Trauma 2001;51:153–8.

[6] Reber PU, Baer HU, Patel AG, Wildi S, Triller J, Buchler MW. Superselective microcoil embolization: treatment of choice in high-risk patients with extrahepatic pseudoaneurysms of the hepatic arteries. J Am Coll Surg 1998;186:325–30.

[7] Tessier DJ, Fowl RJ, Stone WM, McKusick MA, Abbas MA, Sarr MG, et al. Iatrogenic hepatic artery pseudoaneurysms: an uncommon complication after hepatic, biliary, and pancreatic procedures. Ann of Vasc Surg 2003;17:663–9.

[8] Balsara KP, Dubash C, Shah CR. Pseudoaneurysm of the hepatic artery along with common bile duct injury following laparoscopic cholecystectomy. Surg Endosc 1998;12:276–7.

[9] Genyk YS, Keller FS, Halpern NB. Hepatic artery pseudoaneurysm and hemobilia following laser laparoscopic cholecystectomy. Surg Endosc 1994;8:201–4.

[10] Nicholson T, Travis S, Ettles D, Dyet J, Sedman P, Wedgewood K, et al. Hepatic artery angiography and embolization for hemobilia following laparoscopic cholecystectomy. Cardiovasc Intervent Radiol 1999;22:20–4.

[11] Pistorius GA, Walter P, Hildebrandt U, Defreyne L. Pseudoaneurysm of the hepatic artery. A rare complication after laparoscopic cholecystectomy. Langenbecks Arch Chir 1994; 379:291–3.

[12] Stewart BT, Abraham RJ, Thomson KR, Collier NA. Postcholecystectomy hemobilia: enjoying a renaissance in the laparoscopic era? Aus NZ J Surg 1995;65:185–8.

[13] Yelle JD, Fairfull-Smith R, Rasuli P, Lorimer JW. Hemobilia complicating elective laparoscopic cholecystectomy: a case report. Can J Surg 1996;39:240–2.

[14] Zironi G, Piscaglia F, Gaiani S, Masi L, Bolondi L. Intrahepatic artery pseudoaneurysm: a possible complication of blind thoracocentesis. J Clin Ultrasound 1999;27:151–5.

[15] Ahmen A, Samuels S, Keeffe E, Cheung RC. Delayed fatal hemorrhage from pseudoaneurysm of the hepatic artery after percutaneous liver biopsy. Am J Gastroenterol 2001;96:233–7.

[16] Kowdley KV, Aggarwal AM, Sachs PB. Delayed hemorrhage after percutaneous liver biopsy. Role of therapeutic angiography. J Clin Gastroenterol 1994;19:50–3.

[17] Otah E, Cushin BJ, Rozenblit GN, Neff R, Otah KE, Cooperman AM. Visceral artery pseudoaneurysms following pancreatoduodenectomy. Arch Surg 2002;137:55–9.

[18] Reber PU, Baer HU, Patel AG, Wildi S, Triller J, Buchler MW. Life-threatening upper gastrointestinal tract bleeding caused by ruptured extrahepatic pseudoaneurysm after pancreatoduodenectomy. Surgery 1998;124:114–5.

[19] Aranha GV, O'Neil S, Borge MA. Successful nonoperative management of bleeding hepatic artery pseudoaneurysm following pancreaticoduodenectomy. Dig Surg 1999;16:528–30.

[20] Bussenius-Kammerer M, Ott R, Wutke R, Grunewald M, Hohenberger W, Reck T. Pseudoaneurysms of the hepatic

artery—a rare complication after orthotopic liver transplantation. Chirurg 2001;72:78–81.
[21] Cavallari A, Vivarelli M, Bellusci R, Jovine E, Mazziotti A, Rossi C. Treatment of vascular complications following liver transplantation: multidisciplinary approach. Hepatogastroenterology 2001;48:179–83.
[22] Fichelle JM, Colacchio G, Castaing D, Bismuth H. Infected false hepatic artery aneurysm after orthotopic liver transplantation treated by resection and renohepatic vein graft. Ann Vasc Surg 1997;11:300–3.
[23] O'Driscoll D, Olliff SP, Olliff JFC. Hepatic artery aneurysm. Br J Radiol 1999;72:1018–25.
[24] Messina LM, Shanley CJ. Visceral artery aneurysms. Surg Clin North Am 1997;77:425–42.
[25] Elton E, Howell DA, Amberson S, Dykes TA. Combined angiographic and endoscopic management of bleeding pancreatic pseudoaneurysms. Gastrointest Endos 1997;46:544–9.
[26] Sandblom P. Hemobilia. In: Blumgart LH, editor. Surgery of the liver and biliary tract. Edinburgh: Churchill Livingstone; 1988:1075–89.
[27] Chandramohan, Khan AN, Fitzgerald S, Sherlock D, Tam E. Sonographic diagnosis and follow-up of idiopathic hepatic artery aneurysm, an unusual cause of obstructive jaundice. J Clin Ultrasound 2001;29:466–71.
[28] Heestand G, Sher L, Lightfoote J, Palmer S, Mateo R, Singh G, et al. Characteristics and management of splenic artery aneurysms in liver transplant candidates and recipients. Amer Surg 2003;69:933–40.
[29] Baden JG, Racy DJ, Grist TM. Contrast-enhanced three-dimensional magnetic resonance angiography of the mesenteric vasculature. J. Magn. Reson. Imaging 1999;10:369–75.
[30] Meaney JFM, Prince MR, Nostrant TT, Stanley JC. Gadolinium-enhanced MR angiography of visceral arteries in patients with suspected chronic mesenteric ischemia. J. Magn. Reson. Imaging 1997;7:171–6.
[31] Ernst O, Asnar V, Sergent G, Lederman E, Nicol L, Paris JC, et al. Comparing contrast-enhanced breath-hold MR angiography and conventional angiography in the evaluation of mesenteric circulation. AJR 2000;174:433–9.
[32] Fulcher AS, Szucs RA, Bassignani MJ, Marcos A. Right lobe living donor liver transplantation: Preoperative evaluation of the donor with MR imaging. AJR. 2001;175:1483–91.
[33] Fan Cheng Y, Long Chen C, Liang Huang T, Yi Chen T, Yu Lee T, Sen Chen Y, et al. Single imaging modality evaluation of living donors in liver transplantation: magnetic resonance imaging. Transplantation. 2001;72:1527–33.
[34] Schoenberg SO, Knopp MV, Londy F, Krishnan S, Zuna I, Lang N, et al. Morphologic and functional magnetic resonance imaging of renal artery stenosis: a multireader tricenter study. J Am Soc Nephrol 2002;13:158–69.
[35] de Haan MW, van Engelshoven JMA, Houben AJ, Kaandorp DW, Kessels AG, Kroon AA, et al. Phase-contrast magnetic resonance flow quantification in renal arteries comparison with 133 Xenon washout measurements. Hypertension 2003; 41:114–8.
[36] Abbas MA, Fowl RJ, Stone WM, Panneton JM, Oldenburg WA, Bower TC, et al. Hepatic artery aneurysm: factors that predict complications. J Vasc Surg 2003;38:41–5.
[37] Mays ET, Wheeler CS. Demonstration of collateral arterial flow after interruption of hepatic arteries in man. N Engl J Med 1974;290:993–6.
[38] Hoevels J, Nilsson U. Intrahepatic vascular lesions following nonsurgical percutaneous transhepatic bile duct intubation. Gastrointest Radiol 1980;5:127–35.
[39] Roche A, Doyon D, Harry G, Weingarten A, Edouard A. L'embolisation arterielle hepatique: 35 cas. Nouv Presse Med 1978;7:633–7.
[40] Wagner WH, Lundell CJ, Donovan AJ. Percutaneous angiographic embolization for hepatic arterial hemorrhage. Arch Surg 1985;120:1241–9.
[41] Salam TA, Lumsden AB, Martin LG, Smith III RB. Nonoperative management of visceral aneurysms and pseudoaneurysms. Am J Surg 1992;164:215–9.
[42] Ozkan OS, Walser EM, Akinci D, Nealon W, Goodacre B. Guglielmi detachable coil erosion into common bile duct after embolization of iatrogenic hepatic artery pseudoaneurysm. J Vasc Intervent Radiol 2002;13:935–8.
[43] Paci E, Antico E, Candelari R, Alborino S, Marmorale C, Landi E. Pseudoaneurysm of the common hepatic artery: treatment with a stent-graft. Cardiovasc Intervent Radiol 2000;23:472–4.
[44] Larson RA, Solomon J, Carpenter J. Stent graft repair of visceral artery aneurysms. J Vasc Surg 2002;36:1260–3.