JGH Open Open Access An open access journal of gastroenterology and hepatology

JGHF Journal of Gastroenterology and Hepatology Foundation

doi:10.1002/jgh3.12116

ORIGINAL ARTICLE

Arterial pseudoaneurysms in acute and chronic pancreatitis: Clinical profile and outcome

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Key words
arterial pseudoaneurysms, chronic pancreatitis, endovascular embolisation.

Accepted for publication 3 November 2018.

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Declaration of conflict of interest: The authors declare that they have no conflict of interest.

Abstract

Objective: To evaluate the clinical profile and outcome of arterial pseudoaneurysms (PSA) associated with acute and chronic pancreatitis (CP).

Methods: Records of all patients of pancreatitis from 2010 to 2016 were analyzed retrospectively for the development PSAs; clinical profile and outcome parameters were compared between PSAs associated with acute and CP.

Results: Of the 980 patients, 46 (all males, age 39.70 ± 11.78 years) developed PSAs, including 19 of 600 of acute pancreatitis (AP) and 27 of 380 of CP. The most common clinical presentation was bleeding (37, 80.4%). The majority of patients was managed nonsurgically, with endovascular embolization in 31 (67.4%) and percutaneous thrombin injection in 9 (19.6%) patients. Pseudoaneurysms in patients with AP were associated more often with fluid collections (94.7% vs. 55.6%, $P = 0.004$) with more requirement of surgery compared to patients with CP (15.8% vs. 3.7%, $P = 0.033$). The pattern of arteries involved with PSAs and outcome was similar in AP and CP patients.

Conclusion: Arterial PSAs were more commonly associated with CP compared to AP with similar presentations. Associated fluid collections and requirement of surgical intervention were higher in PSAs in patients with AP compared to patients with CP.

Introduction

Pancreatitis can cause a spectrum of arterial and venous complications.[1] Visceral artery pseudoaneurysm (PSA) is the most common arterial complication and is potentially life threatening.[2] PSAs are more often associated with chronic pancreatitis (CP) compared to acute pancreatitis (AP) and are associated with significant morbidity and mortality because of potential of catastrophic life-threatening bleeding.[3] Arterial hemorrhage, which includes bleeding from PSAs, has been reported to occur in 5% of patients with AP.[4] Vascular complications including gastrointestinal bleeding is reported in 5–10% of patients with CP, and the majority of these were due to bleed from PSAs.[3]

PSAs commonly affect the arteries in close proximity to the pancreas, such as the gastroduodenal artery (GDA), splenic artery (SA), and superior mesenteric artery (SMA).[2,3,5] The rarity of PSAs and heterogeneity of the associated inflammation, lack of uniform guidelines for management, and its potential devastating complications make its optimal management difficult. There are limited data available comparing the differences between the behavior of PSAs associated with AP and CP. The available literature reported that the need of surgical intervention was greater for PSAs associated with CP in comparison to PSAs associated with AP[3] and higher mortality for PSAs associated with AP when compared to CP.[2,3] In the present study, we aimed to assess the clinical profile, presentation, and outcome of PSAs associated with AP and CP, managed in a tertiary referral center from 2010 to 2016.

Patients and methods

Patients. The study was based on retrospective data of all patients of AP and CP admitted in the department of gastroenterology between 2010 and 2016 at a tertiary care referral center. We reviewed each patient's medical records in order to identify patients who developed PSA during the disease course, its etiology, clinical symptoms, and management, and this study was approved by the institutional ethics committee. The diagnosis of AP was made on the basis of revised Atlanta 2012 guidelines,[6] and diagnosis of CP was made as per Marseille criteria.[7]

Ultrasonography (USG) with Doppler study was performed as a screening tool. All the patients had a contrast-enhanced computed tomography (CECT), and wherever possible, the hard copies of computed tomography (CT) scan were retrieved and analyzed. In the absence of hard copies of the CT scan, the reports were used to obtain the finding. Associated AP

episode as suggested by raised serum amylase and/or lipase more than three times the upper limit of normal and imaging evidence of pancreatic/peripancreatic inflammation were noted in CP patients.

All patients who had presented with gastrointestinal or intra-abdominal bleeding were resuscitated and stabilized with intravenous fluid and/or blood transfusions. The management strategy was decided depending on the hemodynamic condition of the patients. Patients who were hemodynamically stable underwent CT angiography. If PSAs were detected on CT angiography, patients were taken for digital subtraction angiography (DSA) with endovascular embolization. In case of failure of endovascular embolization, patients were considered for percutaneous thrombin injection. In hemodynamically stable patients, if PSAs were suitable for direct percutaneous thrombin injection (large PSA with narrow neck), the procedure was carried out. In patients with hemodynamic instability precluding angiography and/or percutaneous thrombin injection, surgical management was performed. The size of PSAs, as measured by the maximum diameter in any plane, and the location of PSAs were noted.

Endovascular technique. DSA and endovascular procedures were performed using the standard technique through the transfemoral route with a 5 F angiography catheter. Following celiac and SMA angiograms, selective angiography was performed using a microcatheter. Endovascular embolization was performed using the platinum coils or N-butyl cyanoacrylate (NBCA). The standard technique was used to embolize the bleeding artery both proximal and distal to the PSA using coils (Fig. 1). The diameter of the coil was selected to be 20% more than the maximum caliber of the involved artery. In case of failure to reach the distal segment, NBCA, which could be propagated distal to the site of vascular abnormality, was chosen. NBCA was mixed with lipiodol, and the dilution depended on the dynamics of blood flow as determined by initial angiogram. Due to limited availability and financial constraints in some patients, the stent grafts were not used in any of our patients.

In case of technical failure, percutaneous ultrasound-guided thrombin injection was considered. Percutaneous thrombin injection was administered with autologous thrombin injection into the neck of the PSA (Fig. 2). The flow in the PSA was checked 5 min after thrombin injection and 24 h later using Doppler USG, and the procedure was considered successful when no flow was observed on both occasions.

Outcome. Endovascular embolization was considered successful when there was nonopacification of PSAs in the final angiogram. Clinical success was assessed at the end of 1 month and was defined as resolution of sign and symptoms for which intervention was initiated. Complications related to the procedure were classified as major and minor according to the Society of Interventional Radiology Clinical Practice 2009.[8] The median follow up of our patients was 35 months (range 6–55 months).

Statistical analysis. The data were analyzed using SPSS software (version 22.0, IBM, Chicago, IL, USA). Data were explored for any outliers, errors, and missing values. Quantitative or numerical variables were represented with measures of central location, such as mean median and measures of dispersion, that is, standard deviation. Comparison of categorical variables between the AP and CP groups was performed using the Chi-square test. Continuous variables and normally distributed data were compared using Student's *t*-test. A P value of less than 0.05 was statistically significant.

Results

Records of 980 patients were reviewed (AP—600, CP—380). Arterial PSAs were found in 46 patients, including 19 (3.2%) patients of AP and in 27 (7.1%) patients of CP. Of the 27 CP patients, 17 (63%) had associated acute inflammation. De novo PSAs were seen in 29 (63%) patients, while 17 (37%) patients had history of prior interventions in the form of percutaneous catheter drain in 9 (19.6%), endoscopic retrograde

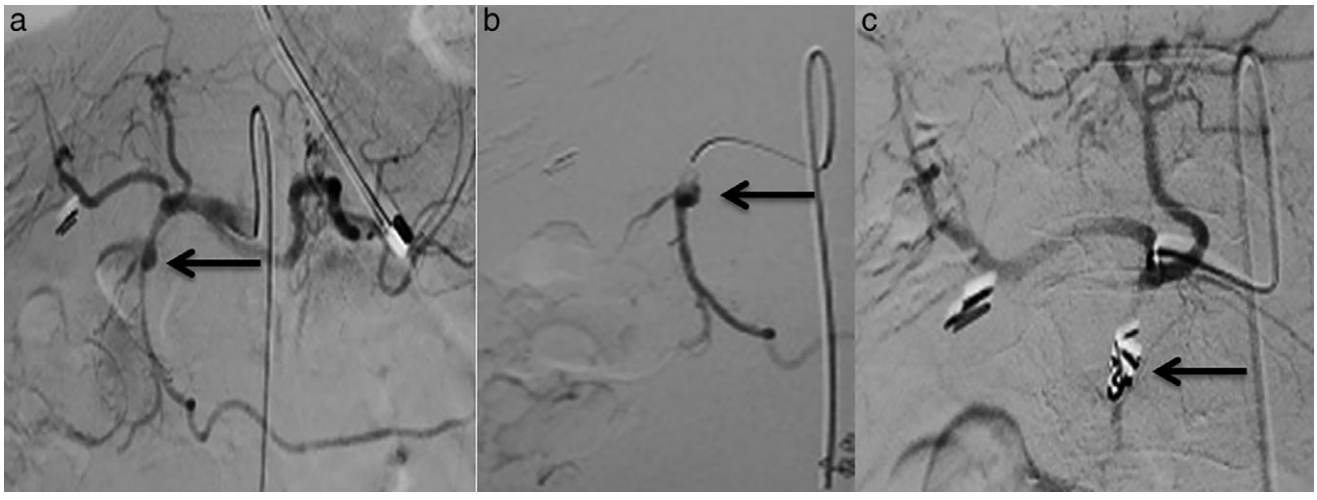

Figure 1 (a) Celiac artery angiography showing pseudoaneurysm (PSA) arising from gastroduodenal artery (GDA); (b) selective angiography of GDA showing PSA; (c) postcoil embolization angiography showing obliteration of PSA.

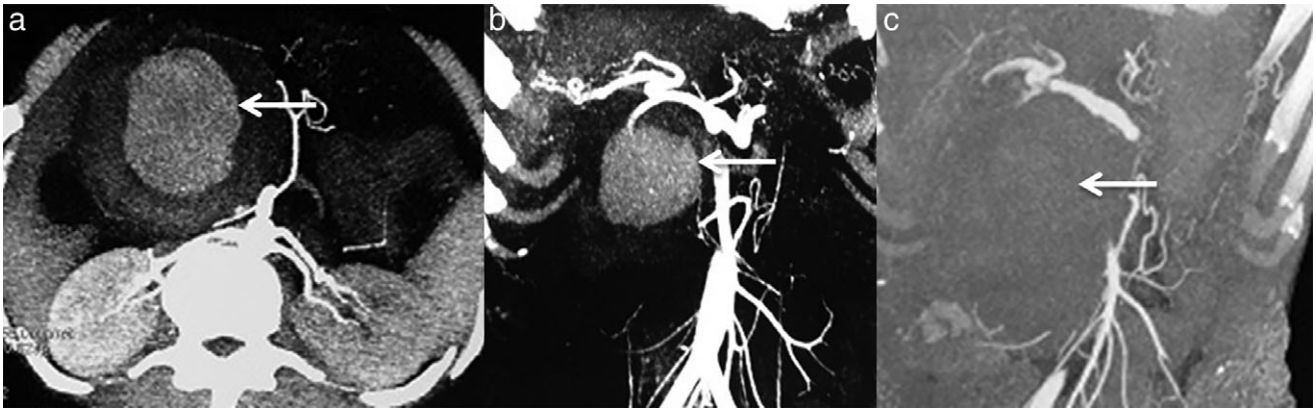

Figure 2 (a) CT angiography showing a large pseudoaneurysm (PSA); (b) axial image showing the PSA in relation to gastroduodenal artery (GDA); (c) postpercutaneous thrombin injection showing resolution of PSA.

Table 1 Patient characteristics and clinical parameters

Characters	AP (*n* = 19)	CP (*n* = 27)	Total (*n* = 46)	Significance (*P*)
Age (year), mean ± SD (range)	34.26 ± 11.24	43.52 ± 10.77	39.70 ± 11.78	0.007
Male:female	19:0	27:0	46:0	
Prior intervention				
De novo	9 (47.4%)	20 (74.1%)	29 (63%)	0.065
Prior events	10 (52.4%)	7 (25.9%)	17 (37%)	
PCD	6 (60%)	3 (42.9%)	9 (19.6%)	0.332
Surgery	1 (10%)	2 (28.5%)	3 (6.5%)	
ERCP	2 (20%)	1 (14.3%)	3 (6.5%)	
Endoscopic transmural drainage	1 (10%)	1 (14.3%)	2 (4.3%)	
Causes of pancreatitis				
Alcohol	13 (68.4%)	25 (92.6%)	38 (82.6%)	0.056
Gallstone	5 (26.3%)	0	5 (10.9%)	
Others	1 (5.3%)	2 (7.4%)	3 (6.7%)	
Clinical presentations				
Gastrointestinal bleeding	14 (73.7%)	23 (85.2%)	37 (80.4%)	0.493
Asymptomatic	3 (15.8%)	2 (7.4%)	5 (10.9%)	
Pain abdomen	1 (5.2%)	2 (7.4%)	3 (6.5%)	
Both bleed and pain	1 (5.2%)	0	1 (2.2%)	
Gastrointestinal bleeding				
Hematemesis	9 (47.4%)	6 (22.2%)	15 (32.6%)	0.002
Melena	1 (5.3%)	17 (63.0%)	18 (39.1%)	
Bleed from PCD site	3 (15.8%)	1 (3.7%)	4 (8.7%)	
Hemoglobin drop	2 (10.5%)	0	2 (4.3%)	
Vital signs				
Hemodynamic instability	5 (26.3%)	5 (18.5%)	10 (21.7%)	0.528
Clinically stable	14 (73.7%)	22 (81.5%)	36 (78.3%)	
Associated peripancreatic collection	18 (94.7%)	15 (55.6%)	33 (71.7%)	0.004

AP, acute pancreatitis; CP, chronic pancreatitis; ERCP, endoscopic retrograde cholangiopancreatography; PCD, percutaneous catheter drain.

cholangiopancreatography (ERCP) in 3 (6.5%), prior surgery in 3 (6.5%), and cystogastrostomy in 2 (4.3%).The development of de novo PSAs was more common in CP than AP (74.1% and 47.4%, respectively, $P = 0.065$) (Table 1).

Clinical parameters. Of the 46 patients with PSAs, alcohol abuse was the most common etiology of pancreatitis in 38 (82.6%). Clinical presentation included gastrointestinal or intra-abdominal bleeding in 37 (80.4%) patients in the form of melena in 18 (39.1%) patients, hematemesis in 15 (32.6%) patients, bleeding from the percutaneous catheter drain site in 4 (8.7%) patients, and sudden drop in hemoglobin without any obvious bleeding source in 2 patients. In five (10.9%) patients, PSAs were detected incidentally during cross-sectional imaging. Three patients presented with acute abdominal pain, and one patient presented with acute abdomen and hematemesis. Hemodynamic instability (tachycardia, systemic hypotension) was observed in 10 patients, while the remaining patients were

Table 2 Artery involved with pseudoaneurysms

	Anatomical location of pseudoaneurysm			
Artery involved	Acute pancreatitis (n = 19)	Chronic pancreatitis (n = 27)	Total (n = 46)	Significance (P)
Gastroduodenal artery	7 (36.8%)†	13 (48.2%)	20† (43.5%)	0.391
Splenic artery	9 (47.4%)	10 (37.1%)†	19† (39.2%)	
Superior mesenteric artery	1 (5.3%)†	2 (7.4%)†	3† (6.5%)	
Left gastric artery	1 (5.3%)	1 (3.7%)	2 (4.3%)	
Inferior pancreaticoduodenal artery	0	2 (7.4%)	2 (4.3%)	
Common hepatic artery	1 (5.3%)	0	1 (2.2%)	
Arteria pancreatica magna	1 (5.3%)	0	1 (2.2%)	

†Two patients have more than one artery involvement.

clinically stable. There was no difference in clinical presentation of PSAs between AP versus CP.

Of the 46 patients, 33 (71.7%) had associated peripancreatic collection. A significantly higher number of patients with AP (n = 18, 94.7%) had associated fluid collection compared to CP (n = 15, 55.6%). Of the 15 CP patients having fluid collection(s), 10 (66.7%) patients had associated acute inflammation.

Size and anatomical location. The most frequent arteries involved were those in close vicinity to pancreas: GDA in 20 (43.5%), SA in 19 (39.2%), and SMA in 3 (6.5%) patients. More than one artery was involved in two (4.3%) patients. The detection and localization of PSAs on CT was accurate in all except one patient, in whom the CT could not localize the site of bleeding, and DSA was performed in view of high suspicion. In this case, a PSA was found to arise from the left gastric artery. The sensitivity of contrast-enhanced CT and/or CT angiography in diagnosing PSA was 97.8% in our study. We did not find any difference in the pattern of arteries involved in AP and CP patients (Table 2). The overall mean size of PSA was 1.88 $\pm$ 1.55 cm, and there was no difference in the size of PSA between AP and CP patients. Four patients had giant a PSA (>5 cm).

Treatment. Endovascular coil embolization was the most frequently performed procedure (n = 24, 52.2%) followed by percutaneous thrombin injection (n = 9, 19.6%). NBCA was used for embolization along with coil in three (6.5%) patients, and NBCA alone was used in four (8.7%) patients. None of the patients had nontarget embolization. Three patients were managed without any intervention as there was evidence of spontaneous thrombosis of PSA and absence of flow on DSA. Surgery was performed as a primary intervention in four (8.7%) patients. Requirement of surgery was higher in AP patients compared to CP patients (Table 3). Of the 33 patients having fluid collections in association with PSA, 8 patients (4 each of AP and CP) subsequently underwent drainage of symptomatic fluid collections with USG-/CT-guided percutaneous catheter drainage (PCD). All of them had successful embolization of PSAs before they were taken for PCD.

Outcome. Angiography and embolization was attempted as primary intervention in 35 (76.1%) patients. Technical success

Table 3 Management of patients with pseudoaneurysms

	Acute pancreatitis (n = 19)	Chronic pancreatitis (n = 27)	Total (n = 46)	Significance (P)
Procedures				
Endovascular coiling	11 (57.9%)	13 (48.2%)	24 (52.2%)	0.033
Percutaneous thrombin injection	1† (5.2%)	8 (29.6%)	9† (19.6%)	
Conservative management	2 (10.5%)	1 (3.7%)	3 (6.5%)	
Surgery	3 (15.8%)	1 (3.7%)	4 (8.7%)	
Endovascular coiling + glue	2 (10.5%)	1 (3.7%)	3 (6.5%)	
Endovascular glue	1† (5.2%)	3 (11.1%)	4† (8.7%)	
Outcomes				
Size of pseudoaneurysm (cm)	1.43 $\pm$ 1.42	2.20 $\pm$ 1.58	1.88 $\pm$ 1.55	0.099
Giant pseudoaneurysm (>5 cm)	1 (5.2%)	3 (11.1%)	4 (8.7%)	0.488
Technical success of embolization	14/14 (100%)	17/21 (80.9%)	31/35 (88.6%)	0.901
Clinical success of embolization	10/14 (71.4%)	16/17 (94.1%)	26/31 (83.9%)	0.371
Major complications of embolization	2 (10.5%)	1 (3.7%)	3 (6.5%)	0.356
Successful hemostasis	17 (89.5%)	23 (85.2%)	40 (87%)	0.671
Rebleed	2 (10.5%)	4 (14.8%)	6 (13%)	0.671
Death	1 (5.3%)	1 (3.7%)	2 (4.3%)	0.798

†One patient having two pseudoaneurysms (PSA) managed with glue and thrombin.

was achieved in 31 (88.6%) patients and clinical success in 26 (83.9%) (Table 3). Successful hemostasis without rebleeding was achieved in 40 patients (87%) after the primary intervention. The remaining six patients (three after coil embolization, two after glue embolization, and one after thrombin injection) underwent repeat interventions for recurrence of bleed. The cause of rebleeding was (i) recanalization of the embolized artery, occurring 5 and 7 days after the embolization in two patients (one after coil embolization and one after thrombin injection); (ii) development of new bleeding sites 2–4 weeks after embolization in three patients; and (iii) persistent retrograde filling of the PSA in one patient. The first two patients were successfully managed, one patient by repeat embolization and another by surgical ligation. Development of new bleeding focus in three patients was successfully managed, one each with coil embolization, glue embolization, and thrombin injection. The patient with retrograde filling of PSA failed to respond to thrombin injection as well as to surgical ligation and finally succumbed. There was no difference in the outcome parameters in PSA associated with AP and CP.

Complications. Nine (25.7%) complications occurred due to endovascular embolization; five patients demonstrated radiological features of focal splenic infarction, three developed splenic abscess, and one patient developed ischemic hepatitis. These five cases of focal splenic infarction and one patient of ischemic hepatitis were classified as minor complications, and patients were asymptomatic for these complications. The patients who developed splenic abscess were classified as major complication, and all of them recovered with antibiotics. Two patients succumbed in this study, one due to multiorgan dysfunction developing as a complication of AP and other due to failure to control bleed even after surgery. There was no difference in complication rate and mortality in PSA associated with AP and CP.

Discussion

We retrospectively analyzed the clinical profile, treatment, and outcome of 46 arterial PSAs in patients with pancreatitis and compared the differences between AP and CP patients. Of the 600 patients of AP, 19 (3.2%) developed PSAs, and of the 380 with CP, 27 (7.1%) developed PSAs. The most common clinical presentation was gastrointestinal or intra-abdominal bleeding in 37 (80.4%) patients. Around two-thirds of patients had associated peripancreatic collection. Most of the patients were managed successfully with interventional radiological procedures consisting of endovascular coil or glue embolization or percutaneous thrombin injection.

In the present study, PSAs were found to be more common in the background of CP than AP. Similar to our study, previous studies have reported underlying CP to be present in 60–84% of patients presenting with PSAs.[3,9,10] Factors incriminated to increase the risk of PSA formation and subsequent bleeding in CP include duration of disease, proximity of vessel near a pseudocyst, communication with biliary or pancreatic duct, to associated splenic vein thrombosis.[11] However, in AP, different factors may be responsible, which include the surrounding inflammation and prior intervention in the form of PCD.[3,11,12]

The most common mode of presentation in our patients was gastrointestinal or intra-abdominal bleed (80%) followed by pain abdomen (6.5%). Patients with AP presented predominantly with hematemesis, and those with CP presented more often with melena. There is heterogeneity in the presentation of PSA in different studies. Previous studies have reported GI bleeding to be present in 24–43% of patients, while pain abdomen was reported in 40–52% of patients.[3,9,10] However, a systematic review reported GI bleed as the most common presentation of PSAs.[2]

We observed that PSAs in AP were more commonly associated with fluid collections (94.7%) in comparison to CP (55.6%). Others have also reported similar experience.[13–15] Hyare et al. noted that 68% of their patients with PSAs had associated fluid collections, and a majority (88%) of them had underlying AP.[14] Hsu et al. reported that five of nine of their patients with CP having PSA had associated fluid collections.[15] Zyromski et al. reported fluid collection in 87% of the patients with PSAs, with 92% of the patients having CP.[13] Of the 27 patients with CP in our study, 63% had associated inflammation with elevated enzymes, suggestive of an episode of AP in recent past. Zyromski et al. had also noted that a majority of patients of CP with PSA had an episode of acute inflammation.[13] In view of the possibility of the risk of bleeding during drainage of fluid collections associated with PSAs, surgery was advocated as the primary modality for management in such cases. However, recent data suggest that pancreatic fluid collections can be safely drained by PCD or through a transmural route after embolization of associated PSAs.[16,17] In the present study, all patients having symptomatic fluid collection along with PSA were successfully managed with PCD after successful prior embolization of PSAs.

The arteries involved in our patients were mainly GDA, SA, and SMA. We did not find a difference in the pattern of involvement in patients of AP versus CP. However, contrary to our previous studies, we found the SA to be the most commonly involved branch of celiac axis in PSA associated with CP,[3,9,10,15] and Hyare et al. noted that GDA involvement is the most common artery in patients with PSA associated with AP.[14]

The incidence of intervention prior to the diagnosis of PSA was more common in patients with AP than in patients with CP (52.4% vs. 25.9% respectively, $P = 0.065$). Development of PSAs after pancreatic surgery is well documented, with iatrogenic injury, long placement of percutaneous drain, and bleed from dehiscence of vascular stump being the possible reasons for development of PSAs after surgery.[13] The reasons for development of PSAs after percutaneous or transmural drainage of pancreatic fluid collection are not well documented in the literature. Iatrogenic vessel injury during transmural drainage or PCD, erosion of vessel by percutaneously placed catheter or indwelling stents placed during transmural drainage, or sudden decompression of collection might be the reasons for development of PSA. It is difficult to postulate how ERCP can be incriminated in the causation of PSA.

With respect to the treatment of arterial PSAs, surgery and endovascular techniques are the two primary options.[3,18–20] The short-term outcome of embolization is well described in the literature, with success rates in the range of 77–100%.[3,10,15,21–23] The technical success rate of our study with endovascular embolization was 87.9%, and clinical success was 82.8%. We did not

find any difference in the technical and clinical success rates in PSA associated with AP versus CP. Hyare et al. reported a technical success rate of 81.5% and clinical success rate of 80% with angioembolization.[14] Failure after endovascular embolization in the form of rebleeding, as described previously, varies between 8 and 20%.[9,10,14]

Nine patients had complications in our study, three major and six minor. Complications were found in patients who underwent embolization of the splenic and hepatic artery. Most of them were minor and were successfully managed with conservative management. There was no difference in the complication rate in PSA associated with AP versus CP. The rates of complication were similar to the other reported studies.[9,10] Four patients were taken for surgery directly in view of hemodynamic instability. The small number of patients requiring surgery in the present and other recent studies is probably due to advancement in interventional radiology and availability of advanced equipment.[3,10,14,18]

In the present study, we did not find any difference in mortality between PSAs associated with AP versus CP. However, surgical intervention was required more often in patients with AP than the patients with CP (15.8% vs. 3.7%). Bergert et al. had reported that the need for surgical intervention was more in PSAs associated with CP (59.3%) than in those in patients with AP (37.5%).[3] They also reported higher mortality in patients with PSAs associated with AP than CP.[3] In a systematic review by Balachandra et al., mortality for PSA associated with AP was 44% compared to 5% in patients with CP.[2] The reason for higher mortality in PSA with AP reported in these studies might be because such patients are seriously ill even before any bleeding complication and have an inherent higher mortality due to underlying necrotizing AP.

Three of our patients had spontaneous thrombosis of PSAs. Past studies have also reported occurrence of this phenomenon in patients with pancreatitis-associated PSAs.[24–26] Incidental PSAs have been documented to occur in 10–21% of patients of CP who underwent angiography for unrelated reasons.[27–30] Despite the presence of this large number of PSAs, clinically overt bleeding occurs in only 1–8% of pancreatitis.[3,4,30] This might be due to spontaneous thrombosis of these PSAs. Trauma during catheterization and decreased splanchnic blood flow with octreotide infusion (received by one patient) are the probable reasons for spontaneous thrombosis in our patients.

Strengths of the present study include description of the largest number of patients with PSAs from a single institution. In addition, the study compares the differences in presentation of PSAs in patients with AP and CP over a long period of time.

To conclude, pancreatitis-related PSAs are more commonly associated with CP than AP. Endovascular management is safe and effective and should be the first modality of treatment. Associated fluid collections and requirement of surgical intervention were greater in PSAs developed in AP in comparison to CP.

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