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Management of pancreatic pseudocysts in pediatric oncology patients☆

Yousef El-Gohary [a], Sara Mansfield [a], Jessica Staszak [b], Abdelhafeez Abdelhafeez [a,b], Lindsay Talbot [a,b], Ching-Hon Pui [c], Robert Gold [d], Andrew J. Murphy [a,b], Andrew M. Davidoff [a,b,*]

[a] Department of Surgery, St. Jude Children's Research Hospital, 262 Danny Thomas Pl, Memphis, TN 38105, USA
[b] Division of Pediatric Surgery, Department of Surgery, University of Tennessee Health Science Center, Memphis, TN 38105, USA
[c] Department of Oncology, St. Jude Children's Research Hospital, 262 Danny Thomas Pl, Memphis, TN 38105, USA
[d] Department of Radiology, University of Tennessee Health Science Center, Memphis, TN, USA

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ABSTRACT

Background: Management of children with pancreatic pseudocysts has historically been adopted from the adult experience where pancreatic pseudocysts greater than 6 cm are unlikely to resolve without intervention. We reviewed the clinical course of pediatric oncology patients with pancreatic pseudocysts.
Methods: A retrospective review of patients treated over a 15-year period was performed. Variables evaluated included cancer type, medications administered, clinical and imaging characteristics of the pancreatic pseudocysts, treatment and outcome.
Results: A total of 132 patients with a median age of 13 (IQR, 9–17) years were identified with pancreatitis. Thirty-one (23.5%) patients developed a pancreatic pseudocyst, of which 84% were associated with PEG-asparaginase treatment. The median pseudocyst size was 7.6 (IQR, 4.4–9.9) cm with 59% being greater than 6 cm. Twenty-two (71%) patients with a pancreatic pseudocyst underwent successful conservative management, while only 9 (29%) required procedural intervention including six percutaneous drainage, one of whom recurred and required surgical cyst-enteric drainage. Two other patients had primary surgical cyst-enteric drainage and one patient underwent endoscopic retrograde cholangiopancreatography with stenting. The indication for intervention was worsening pain rather than pseudocyst imaging characteristics, size or serum amylase/lipase.
Conclusion: Most medication-induced pancreatic pseudocysts in children being treated for cancer, regardless of pseudocyst size, can be managed non-operatively or with transgastric percutaneous drainage. The need for intervention can be safely dictated by patient symptoms.
Level of evidence: III

Acute pancreatitis is a well-recognized complication in pediatric oncology patients that can prolong hospital stay, delay chemotherapy treatment and sometimes require surgical intervention. Acute pancreatitis has been most strongly associated with PEG-asparaginase therapy, which is part of the backbone treatment regimen for acute lymphoblastic leukemia, the most common malignancy in children [1]. Pancreatic pseudocysts are an uncommon complication of acute pancreatitis in children, however, as a result, there is a lack of consensus on the optimal management of these patients. Traditional interventions, mostly adopted from the adult literature, include drainage procedures such as percutaneous external, endoscopic internal and operative internal drainage [2]. Non-operative management has also been proposed including symptomatic pain control, use of somatostatin analogues, and nutrition support either parenterally or enterally via a nasojejunal feeding tube [3]. The general dogma is that pancreatic pseudocysts greater than 6 cm in size will not resolve without procedural intervention [2].

Given that the underlying etiology of pancreatic pseudocysts differs between children with cancer and adult patients for whom consensus guidelines were developed [4], we hypothesized that effective management strategies might be different between these two cohorts of patients. The aim of the study was to characterize the effective management of pancreatic pseudocysts in pediatric oncology patients.

1. Methods

1.1. Study design

Following local Institutional Review Board approval and waiver of informed consent, we performed a retrospective chart review of children who were diagnosed with pancreatitis at St. Jude Children's Research Hospital between 2000 and 2015. The diagnosis of pancreatitis required either a serum amylase or lipase greater than three times normal (0–91 U/L and 0–60 U/L, respectively) or diagnostic imaging

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* Corresponding author at: 262 Danny Thomas Place, Memphis, TN 38105, USA. Tel.: +1 901 595 4157; fax: +1 901 595 2207.
E-mail address: andrew.davidoff@stjude.org (A.M. Davidoff).

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Table 1
Patient demographics. PP: pancreatic pseudocyst, IQR, interquartile range, NA: not applicable.

	All pancreatitis patients n = 132	Pancreatitis without PP n = 101	Pancreatitis with PP n = 31	p-value
Age, median years (IQR)	13 (9–17)	13 (8–17)	13 (9–16)	p = 0.98
Male-to-female (ratio)	79 M: 54 F (1.5:1)	55 M: 46 F (1.2:1)	23 M: 8 F (2.9:1)	p = 0.051
Cumulative Asparaginase dose, median (IQR) U/m^2	NA	5833 (3475–10,288)	7155 (5492–9628)	p = 0.42
Pseudocyst size, median cm (IQR)	NA	NA	7.6 (4.4–9.9)	NA

consistent with pancreatitis. We considered patients to have a pancreatic pseudocyst only if their peripancreatic fluid collection persisted at least 4 weeks beyond the time of initial diagnosis of pancreatitis. Pancreatic pseudocyst size was measured by the largest diameter in centimeters. Variables evaluated included cancer type, medications administered, clinical and imaging characteristics of the pseudocysts, management approach and outcome. Non-operative management primarily consisted of pancreatic rest with total parenteral nutrition, or nasojejunal tube feeding, and/or use of somatostatin analogues.

1.2. Statistical analysis

Continuous variables were compared using the two-tailed Student's *t*-test. Categorical variables were compared using the nonparametric chi square test. A significance threshold of $p < 0.05$ was utilized for all tests. Statistical analysis was performed using GraphPad Prism version 7.0, GraphPad Software (La Jolla, CA, USA) and SAS 9.4 (Cary, NC, USA).

2. Results

A total of 132 patients were diagnosed with pancreatitis over the 15-year period. The median age at diagnosis of pancreatitis was similar between the 31 patients with a pancreatic pseudocyst and the other 101 patients without a pseudocyst (Table 1 and Fig. 1). There was a slight male predilection for developing a pancreatic pseudocyst, but this was not statistically significant ($p = 0.051$). The median amylase and lipase levels at diagnosis for patients with a pancreatic pseudocyst were 277 (IQR, 178–496) units/L and 926 (IQR, 477–1573) units/L, respectively. These levels were comparable to those of patients without a pancreatic pseudocyst: 296 (IQR, 139–494) units/L, $p = 0.70$, and 826 (IQR, 288–1386) units/L, $p = 0.81$, respectively. There was no significant difference in the cumulative dose of asparaginase treatment between patients with or without a pancreatic pseudocyst. The median pancreatic pseudocyst size was 7.6 (IQR, 4.4–9.9) cm. Table 2 lists the underlying diagnoses for patients who developed pancreatitis, with or without pancreatic pseudocysts. Leukemia or lymphoma were the most common diagnoses in both groups, 80 (79.2%) and 27 (87.1%) patients, respectively.

The etiologies of pancreatitis in patients without a pancreatic pseudocyst and the frequencies of each are listed in Table 3, many of which have been previously reported as causative of pancreatitis [1, 5–20]. The two commonest etiologies for the development of pancreatitis in patients without an associated pancreatic pseudocyst were treatment with PEG-asparaginase and gallstone/gallbladder sludge (Table 3). Of the three iatrogenic causes of pancreatitis, one resulted after a traumatic upper esophagogastroduodenoscopy causing duodenal hematoma, one occurred after a laparoscopic subtotal colectomy for colonic adenocarcinoma, and the third developed after embolization of the right hepatic artery for a hepatic mass. The most common cause of pancreatitis in patients with a pancreatic pseudocyst was PEG-asparaginase (83.9%), with other causes listed in Table 4 [1, 7, 21, 22].

The median time to enteral feeding in patients without a pancreatic pseudocyst was 3 (IQR, 0–8) days. Total parenteral nutrition was used to supplement nutrition in 21% of patients without pancreatic pseudocyst for a median duration of 19 (IQR, 12–31) days. Twenty-two (71.0%) patients with a pancreatic pseudocyst were successfully treated non-operatively, which entailed a period of fasting, parenteral or nasojejunal enteral nutrition, and supportive pain control. The median time to enteral feeding in those patients without intervention was 6 (IQR, 4–9) days, with 43% being supported with total parental nutrition for a

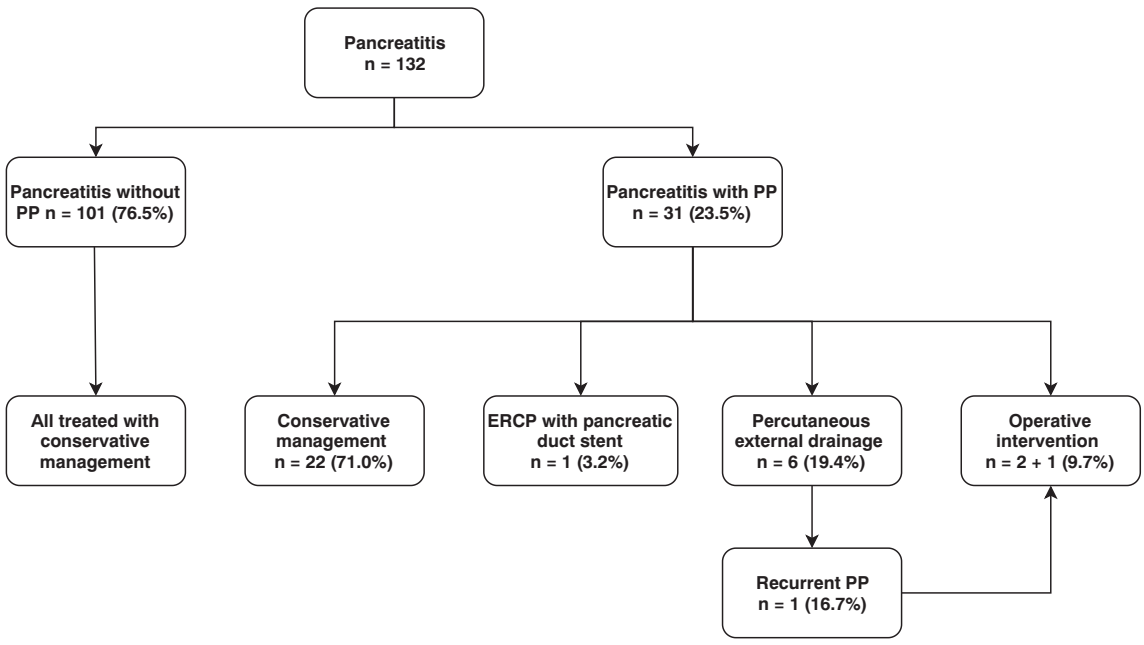

Fig. 1. Flow diagram. ERCP: endoscopic retrograde cholangiopancreatography, PP: pancreatic pseudocyst.

Table 2
Underlying diagnoses of patients with pancreatitis.

Underlying Diagnosis	Pancreatitis without pancreatic pseudocyts n = 101 (%)	Pancreatitis with pancreatic pseudocysts n = 31 (%)
Leukemia or lymphoma	80 (79.2)	27 (87.1)
Solid tumor	14 (6.9)	2 (6.5)
CNS tumor	7 (13.9)	2 (6.5)

Table 3
Underlying etiology of pancreatitis in those patients who did not develop pancreatic pseudocyst.

Etiology	Total number n = 101 (%)
PEG-Asparaginase	33 (32.7)
Gallstone / sludge	10 (9.9)
Steroids	9 (8.9)
6-Mercaptopurine	5 (5.0)
Hypertriglyceridemia	5 (5.0)
Cytarabine	5 (5.0)
Iatrogenic	3 (3.0)
Tacrolimus	2 (2.0)
Pentamidine	2 (2.0)
Methotrexate	2 (2.0)
Sulfamethoxazole/trimethoprim	2 (2.0)
Cisplatin	2 (2.0)
Stavudine	2 (2.0)
Hypercalcemia	2 (2.0)
Mycophenolate mofetil	1 (1.0)
Ribavirin	1 (1.0)
Pancreatic ductal obstruction from mass	1 (1.0)
Crenolanib	1 (1.0)
Amoxicillin/clavulanic acid	1 (1.0)
Fludarabine	1 (1.0)
Cyclophosphamide	1 (1.0)
Pazopinib	1 (1.0)
Retinoid acid	1 (1.0)
Unknown	8 (8.4)

median duration of 17.5 (IQR, 11–42) days. Somatostatin analogues were used in 9% of patients with pancreatic pseudocysts. Out of the 32 pancreatic pseudocyst patients, 8 (31.3%) were treated with antibiotics for febrile neutropenia due to their low absolute neutrophil counts from their chemotherapy regimen. The median duration of antibiotic treatment was 8 (IQR, 7–10) days (range 1–14 days). However, none of these episodes of febrile neutropenia were attributed to an infected pancreatic pseudocyst. None of these patients were found to have necrotizing pancreatitis or infected pancreatic necrosis.

Nine (29.0%) patients required intervention for symptoms. The main symptom that led to intervention was intractable nausea and/or vomiting. The median duration of symptoms prior to intervention was 24 (IQR, 6–46) days. Six patients underwent percutaneous external drainage for a median duration of 9 (IQR, 9–51) days, of which five had a transgastric percutaneous drainage approach and one had a percutaneous extra-gastric approach because the stomach was displaced too far medially, precluding a safe transgastric approach. One of the patients who initially underwent transgastric percutaneous external drainage for a period of 3 months, had a symptomatic recurrence of the pseudocyst 9 months later, and subsequently underwent a laparoscopic transgastric cyst-gastrostomy. Two patients failed conservative, non-operative management secondary to hemorrhagic pancreatitis necessitating an exploratory laparotomy with Roux-en-Y cyst-jejunostomy. They were discharged 10 and 11 days following surgery, respectively. One patient underwent endoscopic retrograde cholangiopancreatography with pancreatic duct stent placement, which was removed 50 days later (96 days after diagnosis) after resolution of the pancreatic pseudocyst (Fig. 1). The two patients with CT-confirmed hemorrhagic pancreatitis had a shorter duration of non-operative management prior to intervention due to significant symptoms of abdominal pain and intractable vomiting. The median delay of resumption of chemotherapy treatment in relation to the development of a pancreatic pseudocyst was 6.5 (IQR, 0–12.3) days. Twenty-nine percent of chemotherapy delays in patients with a pancreatic pseudocyst were related to other complications of cancer treatment including neutropenic enterocolitis, *Clostridium difficile* infection, pulmonary aspergillosis, or awaiting methotrexate levels to normalize.

Table 4
Underlying etiology of pancreatitis in patients with pancreatic pseudocyst. *One patient had advanced pancreatic ductal adenocarcinoma and another patient had advanced transverse colon adenocarcinoma invading the pancreas.

Etiology	Total number n = 31 (%)
PEG-Asparaginase	26 (83.9)
Pancreatic ductal obstruction secondary to mass*	2 (6.5)
Erlotinib	1 (3.2)
Valproic acid	1 (3.2)
Steroids	1 (3.2)

Of the 22 patients with pancreatic pseudocysts who were managed conservatively (Fig. 1), complete radiologic resolution was documented in 17 (73.9%) patients with a median duration to last documented imaging of 99 (IQR 48–253) days. The remaining patients were asymptomatic, and no further imaging or intervention was undertaken from the last documented pancreatic pseudocyst.

3. Discussion

We have shown that a conservative approach was successful in the management of pancreatic pseudocysts in 22 (71.0%) of 31 patients whose median pancreatic pseudocyst size was 7.6 (IQR, 4.4–9.9) cm (Fig. 1). Furthermore, of the six patients who had a minimally invasive percutaneous external drain, only one (16.7%) had a pancreatic pseudocyst recurrence 9 months later, requiring a laparoscopic transgastric cyst-gastrostomy (Fig. 1) [3, 23]. In contrast, in a large adult series of 893 patients with pancreatic pseudocysts, procedural intervention was performed in 810 (91%) patients with a pancreatic pseudocyst diameter greater than 5 cm [2]. Of the 810 adult patients who underwent an intervention, 13 (1.5%) had percutaneous drainage, 46 (5.7%) had pancreatic resection, 341 (42.1%) underwent internal surgical drainage, and 410 (50.6%) underwent endoscopic drainage [2]. Percutaneous drainage is not generally employed in adult patients due to a high complication rate of up to 30% with the frequent need for surgical intervention and a failure rate of 16% [2].

While infrequently required in the pediatric cancer population, the surgical management of pancreatic pseudocysts can be accomplished with minimally invasive laparoscopic techniques, using either a transgastric or intragastric approach [24]. Three patients in our series required surgical intervention because of persistent symptoms despite nonoperative management or percutaneous drainage.

Despite the primarily conservative treatment strategy employed in this series, we did not find significant delays in resumption of chemotherapy associated with pancreatic pseudocysts. The median delay in resumption of chemotherapy related to pancreatic pseudocysts was 6.5 (IQR, 0–12.3) days. Unresolving nausea and vomiting were the main symptoms that prompted interventional or surgical procedures in 9 patients (29%) in this series. Since we did not identify prolonged treatment delays associated with pancreatic pseudocysts, we do not believe that earlier procedural intervention would result in shorter delay of chemotherapy.

We have consistently reserved operative drainage for cases that have failed non-operative management in this series over a 15-year period, and therefore there has not been a bias based on year of diagnosis for choosing percutaneous drainage versus non-operative management or operative enteric drainage. The only two patients in the series that developed hemorrhagic pancreatic pseudocyst occurred in 2011 and

2013. Both necessitated operative drainage since they had significant symptoms. The third patient that required operative intervention had an initial percutaneous drainage procedure in 2015. During the 2-year follow-up, this patient developed a recurrence and underwent laparoscopic cystgastrostomy. Therefore, we recommend that surgical intervention should be based on patient symptoms rather than the size of the pancreatic pseudocyst in the pediatric cancer population.

There were no radiographic signs of infection in any patient and no pancreatic aspirate cultures were obtained in this series to assess for bacterial infection of the pancreatic pseudocyst. Only 8 (31.3%) patients in the pancreatic pseudocyst group were treated with antibiotics for febrile neutropenia secondary to chemotherapy, with a median duration of treatment of 8 (IQR, 7–10) days.

We did not find any difference in the mode of presentation between the pancreatic pseudocyst and non-pseudocyst patient groups. Both groups initially presented with nausea and abdominal pain which led to laboratory confirmation of pancreatitis followed by imaging. The cumulative doses of PEG-asparaginase and the peak amylase and lipase levels between both groups were not significantly different.

Another approach to treating pancreatic pseudocysts is using endoscopic procedures through a transpapillary or cystenteric drainage. These techniques generally employ stents to optimize evacuation of the cyst. However, the failure rate has been reported as high as 23% in a recent adult series [25]. The pediatric literature on these techniques is limited by small case numbers with mixed results [24]. Saluja et al. [26] found no significant difference in the overall complication rate or duration of hospital stay between patients treated with endoscopic cyst-gastrostomy and those with surgical cyst-gastrostomy for the management of pancreatic pseudocysts. However, the surgical approach had a statistically better drainage rate as compared to the endoscopic drainage group. This is in contrast to the large adult pancreatic pseudocyst series of Pan et al. [2] where they found no difference in pancreatic pseudocyst resolution between the surgical and endoscopic groups [2]. However, both groups recommended endoscopic intervention as a first choice in the appropriate clinical setting.

In a Cochrane review of pancreatic pseudocyst management in adults, four randomized controlled trials examining 176 patients who were randomized to endoscopic ultrasound (EUS)-guided drainage, endoscopic drainage, EUS-guided drainage with nasocystic drainage and open surgical drainage were reviewed; only low-quality evidence was found to suggest one intervention over the other with optimal decisions needing to be made on case by case basis [27].

The most common causes of pancreatitis in adults are alcohol and biliary tract disease [2, 28]. In our series, the most common underlying diagnosis of patients with pancreatitis in children with cancer is leukemia or lymphoma, and the commonest instigating factor was PEG-asparaginase treatment, occurring in 59 out of 132 (44.7%) patients with pancreatitis. Asparaginase is a key chemotherapeutic drug that is used for remission induction and consolidation therapy for leukemia and non-Hodgkin lymphoma. Risk factors associated with PEG-asparaginase-induced pancreatitis include Native American ancestry, older age, and higher cumulative dose (>240,000 U/m^2). Furthermore, those who inherit a rare nonsense variant in carboxypeptidase A2 are at markedly increased risk of asparaginase-induced pancreatitis [1]. In our series age, cumulative dose of asparaginase treatment, and the degree of amylase or lipase elevation were not significantly different between patients who did or did not develop a pancreatic pseudocyst (Table 1). There are three forms of asparaginase preparations that are available; the native asparaginase derived from *Escherichia coli* (*E. coli*-asparaginase), a pegylated form of this enzyme (PEG-asparaginase) and a product isolated from *Erwinia chrysanthemi*, i.e. Erwinia asparaginase [29]. The vast majority of patients in this series received PEG-asparaginase. In the early part of the study, three out of 59 patients that received PEG were switched to Erwinia in the thought that it caused less severe pancreatitis.

A limitation of this study is that it is retrospective in nature with a small sample size and was not designed to determine the success rate of any specific procedural interventions. However, we believe that this series provides a reasonably unique perspective on managing pancreatic pseudocysts, especially PEG-asparaginase-induced pancreatic pseudocysts, specifically looking at pediatric oncology patients. Surgeons often face a clinical predicament of when to surgically intervene when consulted on a patient with pancreatic pseudocyst. We believe this series will provide clinicians and surgeons some guidance on their management.

4. Conclusions

Most drug-induced pancreatic pseudocysts in children being treated for cancer, regardless of pseudocyst size, can be safely managed nonoperatively or with minimally invasive procedures, with little morbidity. The need for intervention can be safely dictated by patient symptoms. Thus, serial imaging with computed tomography can be minimized to avoid unnecessary radiation exposure.

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References

[1] Liu C, Yang W, Devidas M, et al. Clinical and genetic risk factors for acute pancreatitis in patients with acute lymphoblastic leukemia. J Clin Oncol 2016;34(18):2133–40.
[2] Pan G, Wan MH, Xie KL, et al. Classification and management of pancreatic pseudocysts. Medicine (Baltimore) 2015;94(24):e960.
[3] Spraker HL, Spyridis GP, Pui CH, et al. Conservative management of pancreatic pseudocysts in children with acute lymphoblastic leukemia. J Pediatr Hematol Oncol 2009;31(12):957–9.
[4] Werlin SL, Kugathasan S, Frautschy BC. Pancreatitis in children. J Pediatr Gastroenterol Nutr 2003;37(5):591–5.
[5] Ko CW, Gooley T, Schoch HG, et al. Acute pancreatitis in marrow transplant patients: prevalence at autopsy and risk factor analysis. Bone Marrow Transplant 1997;20(12):1081–6.
[6] Suzuki M, Sai JK, Shimizu T. Acute pancreatitis in children and adolescents. World J Gastrointest Pathophysiol 2014;5(4):416–26.
[7] Trivedi CD, Pitchumoni CS. Drug-induced pancreatitis: an update. J Clin Gastroenterol 2005;39(8):709–16.
[8] Zerra P, Bergsagel J, Keller FG, et al. Maintenance treatment with low-dose Mercaptopurine in combination with allopurinol in children with acute lymphoblastic leukemia and Mercaptopurine-induced pancreatitis. Pediatr Blood Cancer 2016;63(4):712–5.
[9] Wolfgram PM, Macdonald MJ. Severe hypertriglyceridemia causing acute pancreatitis in a child with new onset type I diabetes mellitus presenting in ketoacidosis. J Pediatr Intensive Care 2013;2(2):77–80.
[10] McGrail LH, Sehn LH, Weiss RB, et al. Pancreatitis during therapy of acute myeloid leukemia: cytarabine related? Ann Oncol 1999;10(11):1373–6.
[11] Nieto Y, Russ P, Everson G, et al. Acute pancreatitis during immunosuppression with tacrolimus following an allogeneic umbilical cord blood transplantation. Bone Marrow Transplant 2000;26(1):109–11.
[12] Hung WY, Abreu Lanfranco O. Contemporary review of drug-induced pancreatitis: a different perspective. World J Gastrointest Pathophysiol 2014;5(4):405–15.
[13] Stefanovic M, Jazbec J, Lindgren F, et al. Acute pancreatitis as a complication of childhood cancer treatment. Cancer Med 2016;5(5):827–36.
[14] Park TY, Oh HC, Do JH. A case of recurrent pancreatitis induced by trimethoprim-sulfamethoxazole re-exposure. Gut Liver 2010;4(2):250–2.
[15] Patel MK, Barvaliya MJ, Patel TK, et al. Stavudine induced acute necrotizing pancreatitis with tetany in a pediatric patient. Indian J Pharmacol 2012;44(4):523–5.
[16] Einollahi B, Dolatimehr F. Acute pancreatitis induced by mycophenolate mofetil in a kidney transplant patient. J Nephropharmacol 2015;4(2):72–4.
[17] da Silva J, Giroldi SB, de Oliveira Basso F, et al. Acute pancreatitis during interferon-alpha and ribavirin treatment for hepatitis C. BMJ Case Rep 2009;2009.
[18] Chams S, El Sayegh S, Hamdon M, et al. Amoxicillin/clavulanic acid-induced pancreatitis: case report. BMC Gastroenterol 2018;18(1):122.
[19] Kawakubo K, Hata H, Kawakami H, et al. Pazopanib-induced severe acute pancreatitis. Case Rep Oncol 2015;8(2):356–8.
[20] Teng HW, Bai LY, Chao TC, et al. Acute pancreatitis during all-trans-retinoic acid treatment for acute promyelocytic leukemia in a patient without overt hypertriglyceridemia. Jpn J Clin Oncol 2005;35(2):94–6.

[21] Ruinemans GM, Balemans C, Mattijssen V, et al. Fatal necrotizing pancreatitis during combined treatment with erlotinib and sunitinib. Lung Cancer 2010;70(3):364–5.
[22] Ray S, Khamrui S, Kataria M, et al. Valproic acid-induced severe acute pancreatitis with pseudocyst formation: report of a case. Cureus 2015;7(8):e297.
[23] Russell KW, Barnhart DC, Madden J, et al. Non-operative treatment versus percutaneous drainage of pancreatic pseudocysts in children. Pediatr Surg Int 2013;29(3):305–10.
[24] Yoder SM, Rothenberg S, Tsao K, et al. Laparoscopic treatment of pancreatic pseudocysts in children. J Laparoendosc Adv Surg Tech A 2009;19(Suppl. 1):S37–40.
[25] Ruckert F, Lietzmann A, Wilhelm TJ, et al. Long-term results after endoscopic drainage of pancreatic pseudocysts: a single-center experience. Pancreatology 2017;17(4):555–60.
[26] Saluja SS, Srivastava S, Govind SH, et al. Endoscopic cystogastrostomy versus surgical cystogastrostomy in the management of acute pancreatic pseudocysts. J Minim Access Surg 2019 (Epub ahead of print).
[27] Gurusamy KS, Pallari E, Hawkins N, et al. Management strategies for pancreatic pseudocysts. Cochrane Database Syst Rev 2016;4:CD011392.
[28] Lankisch PG, Apte M, Banks PA. Acute pancreatitis. Lancet 2015;386(9988):85–96.
[29] Pieters R, Hunger SP, Boos J, et al. L-asparaginase treatment in acute lymphoblastic leukemia: a focus on Erwinia asparaginase. Cancer 2011;117(2):238–49.