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A scoping review on acute gastrointestinal surgical complications in immunocompromised pediatric patients

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Abstract

Background: Gastrointestinal complications are becoming increasingly more common and pose a significant risk on the health of children with compromised immunity caused by various etiologies such as chemotherapy and post-transplantation immunosuppression. We aim to review abdominal complications in immunocompromised children and their respective management.

Main body: This is a scoping review of the literature. PubMed, MEDLINE, Google Scholar, and Scopus libraries were searched for relevant articles. Extracted data included the etiologies of immunocompromised immunity, gastrointestinal and abdominal complications in immunocompromised children, diagnosis, and treatment of these pathologies. Examples of gastrointestinal complications in immunocompromised children include, but not limited to, neutropenic enterocolitis, acute appendicitis, bowel perforation, acalculous cholecystitis, and acute pancreatitis. Our literature review showed that bacterial and fungal infections are the major causes of exacerbation and mortality. The main cause of immunosuppression in children with neutropenic enterocolitis and acute pancreatitis is chemotherapy, and management of these pathologies using intravenous fluids, antibiotic therapy, and granulocyte-stimulating factors is the current standard of care. Surgical intervention is uncommon and reserved for complicated cases. That said, in acute appendicitis and bowel perforation, laparoscopy is the mainstay treatment. However, in systemic infections, nonsurgical interventions such as transfusion and bowel rest are the gold standard. As for acalculous cholecystitis, percutaneous cholecystectomy is superior to laparotomy and other surgical interventions.

Conclusion: Timely diagnosis and management of gastrointestinal complications in the immunocompromised children is key in reducing mortality and morbidity. Both surgical and nonsurgical interventions are needed and should be further studied in order to improve outcomes.

Keywords: Immunocompromised, Neutropenic enterocolitis, Acute appendicitis, Bowel perforation, Acalculous cholecystitis, Acute pancreatitis

Background

Immunocompromised children are at an increased risk of developing life-threatening emergencies and acute gastrointestinal (GI) surgical complications such as pancreatitis, colitis, cholecystitis, viscus perforation, and typhlitis. The immunocompromised condition itself or the treatment regimens and medications that accompany the patient's compromised immunity are the main predisposing factors. Identifying such risks and potential complications as soon as possible is crucial in limiting and preventing escalation in morbidity and mortality rates. In this scoping review, we focus on the published data on the clinical features and management options of the most commonly encountered acute GI surgical pathologies in the immunocompromised pediatric population.

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Methodology

PubMed, MEDLINE, Scopus, and Google Scholar libraries were searched between the years 1989 and 2020 using each of the keywords "Immunocompromised host," "Chemotherapy," "Neoplasm," "Malignancy," "Congenital immunodeficiency," "Transplant," "Post-transplant," "SCID," "Primary Immunodeficiency Diseases" with each of "Neutropenic enterocolitis," "Appendicitis," "Bowel perforation," "Cholecystitis," "Pancreatitis," and "Typhlitis."

The results were independently screened by the authors for relevant articles. Selected articles focused on patients under 18 years of age and included case reports, case series, systematic reviews, and meta-analyses. Extracted data included the etiology behind the patients' immunocompromised condition, the number of cases studied, and their acute surgical complications. Data on the diagnostic methods and treatment regimens for each condition were collected. Articles written only in English were selected.

Table 1 summarizes the published case reports/series of children who developed acute abdominal surgical emergencies in the context of compromised immunity.

Neutropenic enterocolitis or typhlitis

Neutropenic enterocolitis (NE), also known as typhlitis, is an inflammation of the cecum, colon, or small intestine [37]. Exposure to chemotherapy that induces mucositis and mucosal wall interruption and assists in bacterial translocation across the intestinal walls is hypothesized to be the main cause of this condition. Neutropenia exacerbates this bacterial spread following the weakened immune system's response against the infection [38]. Invasive fungal infections also seem to play a role and have been shown to increase mortality and morbidity in patients with NE. Two cases have been reported where patients with simultaneous infection of invasive pulmonary aspergillosis and hepatosplenic candidiasis suffered major exacerbations during chemotherapy, despite optimal treatment with amphotericin B [33].

The most common symptoms seen in patients with NE include high fever and right-sided abdominal pain in the context of neutropenia [39]. Computed tomographic (CT) scan is recommended for documenting a mural wall thickness of more than 5 mm, but a definitive diagnosis of NE is established histologically in the presence of edema with mucosal ulceration and necrosis [40, 41]. In a case of a 13-year-old child with Fanconi anemia having severe neutropenia, abdominal ultrasound (US) showed a 1.18 cm thickening of the ascending colon and cecum. Early management and treatment in this patient resulted in favorable outcomes [35]. Similarly, in a study on 75 children with acute leukemia, 10 episodes of typhlitis (4.5%) were recorded in periods of severe neutropenia, with the cumulative risk being higher in patients with acute myeloid leukemia (AML) than acute lymphoblastic leukemia (ALL). However, despite the relatively low rate of typhlitis, mortality rate was 20% indicating that early detection and intervention are essential in decreasing mortality [42]. Similar results were reported of a group of 18 patients, 12 having ALL and 6 having AML, presenting for 20 episodes of NE. Mortality in this group reached 30% [36]. This demonstrates that chemotherapy and hematological malignancies both predispose to NE.

Management options include supportive, medical, and/or surgical therapy. In the absence of severe complications such as peritonitis, bleeding, or perforation, bowel rest and intravenous fluids along with broad-spectrum antibiotics targeting anaerobes are recommended [40, 43]. Other measures include antifungals targeting *Candida* and medications that help restore neutrophil counts [44]. Granulocyte colony-stimulating factor was shown to expedite neutrophil recovery on some occasions. In cases where the severe aforementioned complications were present, surgery was required [40].

Acute appendicitis

Acute appendicitis is the major causative pathology responsible for abdominal surgery in the pediatric population. It usually occurs as a result of luminal obstruction from a variety of causes that include a fecalith, mucosal inflammation, and lymphoid hyperplasia. The obstruction is then followed by appendiceal inflammation that can progress to transmural inflammation, ischemia, and infarction [45]. Cases of appendicitis are rarely reported in immunocompromised patients due to decreased lymphoid aggregates in this subset of patients [46].

Patients with acute appendicitis usually present with abdominal pain that later migrates to the right lower quadrant. The pain is associated with nausea, vomiting, low-grade fever, and mild leukocytosis. In immunocompromised patients, the absence of leukocytosis is a prominent feature. As for diagnosis, imaging, specifically CT scans or abdominal US scans, is the gold standard [47, 48].

Concerning the treatment of appendicitis, appendectomy remains the mainstay of treatment. Historically, it started in the 1800s with the conventional method of an incision in the right lower quadrant and was later followed in the 1990s with advancements through the introduction of laparoscopy [49]. As a result, laparoscopic appendectomy is currently the gold standard treatment for acute appendicitis. In the case of two pediatric patients with acute leukemia presenting for fungal appendicitis, laparoscopic appendectomy prevented fungal dissemination and led to successful treatment [7].

Table 1 Recorded acute abdominal surgical emergencies in immunocompromised children

Year published	Author	Number of cases	Immunocompromised condition	Acute surgical complication
1995 [1]	Gilad et al	1	Chemotherapy for ALL	Appendiceal and ileal perforation
2003 [2]	Velez et al	1	Chemotherapy for Wilms tumor	Appendiceal perforation
2005 [3]	Kerri et al	1	HIV	Appendicitis
2009 [4]	Bong Hee et al	1	Aspergillosis and chemotherapy for ALL	Appendicitis
2009 [5]	Luer et al	1	Chemotherapy for ALL	Appendicitis
2013 [6]	Larbcharoensub et al	1	Chemotherapy for diffuse large B-cell lymphoma and promyelocytic leukemia	Appendicitis
2016 [7]	Veronica et al	2	Chemotherapy for AML	Appendicitis
2020 [8]	Yada et al	1	Chemotherapy for AML	Appendicitis
1989 [9]	Yoshida et al	1	Down's syndrome	Bowel perforation
1994 [10]	Vadeboncoeur et al	1	Premature infant	Bowel perforation
1999 [11]	Rokhsar et al	2	Non-Hodgkin's lymphoma	Bowel perforation
2006 [12]	Yalcin Polat et al	1	Down's syndrome	Bowel perforation
2007 [13]	Choudhury et al	1	Malnourishment	Bowel perforation
2008 [14]	Cindy et al	3	Posttransplant lymphoproliferative disorder	Bowel perforation
2013 [15]	Kutsch et al	1	Posttransplant lymphoproliferative disorder	Bowel perforation
2013 [16]	Radhakishan et al	12	Chemotherapy for ALL	Bowel perforation
2017 [17]	Maxted et al	1	Post kidney transplant	Bowel perforation
1999 [11]	Rokhsar et al	1	Aplastic anemia	Bowel perforation and neutropenic enterocolitis
2016 [18]	Schaefer et al	10	5 children received BMT for leukemia, 1 child received BMT for SCID, 1 child had lymphoma, 1 child had SCID, 2 children had leukemia	Cholecystitis
2019 [19]	Rose et al	3	2 children with hemoglobinopathy and 1 child with mononucleosis	Cholecystitis
2007 [20]	Lee et al	7	7 children post liver transplantation	Colitis
2017 [21]	Fernandes et al	9	6 children post liver transplantation, 2 post kidney transplantation and 1 post liver-kidney transplantation	Colitis
2019 [22]	Akkelle et al	12	12 children with PIDs	Colitis
1992 [23]	Miller et al	9	6 patients had vertical transmission of infection and 3 had acquired HIV infection through contaminated blood products	Pancreatitis
1992 [24]	Werlin et al	7	7 children post BMT	Pancreatitis
1996 [25]	Koranyi et al	10	3 children with acquired HIV from vertical transmission and 7 from contaminated blood products	Pancreatitis
2002 [26]	Carter et al	1	1 child 12 days post liver transplantation	Pancreatitis
2003 [27]	Eghtesad	26	1 child post liver transplantation	Pancreatitis
2004 [28]	Sastry et al	1	1 child post BMT	Pancreatitis
2006 [29]	Bateman et al	5	5 children post BMT	Pancreatitis
2008 [30]	Cho et al	1	1 child post renal transplantation	Pancreatitis
2016 [31]	Hammer et al	1	1 child with *Candida albicans* infection and cystic fibrosis status post-lung transplantation	Pancreatitis
2019 [32]	Qi et al	1	1 child with acute B lymphoblastic anemia and HSCT	Pancreatitis
2007 [33]	Avci et al	2	Chemotherapy for ALL	Typhlitis
2008 [34]	Haut et al	1	Chemotherapy for AML	Typhlitis
2008 [35]	Tinsa et al	1	Fanconi anemia	Typhlitis
2018 [36]	User et al	18	12 children underwent chemotherapy for ALL 6 children underwent chemotherapy for AML	Typhlitis

ALL, acute lymphocytic leukemia; HIV, human immunodeficiency virus; AML, acute myeloid leukemia; BMT, bone marrow transplant; SCID, severe combined immunodeficiency disorder; PID, primary immunodeficiency disorder; HSCT, hematopoietic stem cell transplantation

However, there is still no consensus on whether the medical or the surgical interventional method should be used as the mainstay treatment in uncomplicated appendicitis in the immunocompromised. In a hemodynamically stable patient, first-line treatment with antibiotics and interventional radiology consultation and/or growth factor support should be considered [50]. The most commonly used antibiotics include meropenem or piperacillin-tazobactam and are administered intravenously for 24 to 48 h. They are followed by approximately 7 days of amoxicillin-clavulanate or ciprofloxacin and metronidazole [51]. In patients with systemic infections, transfusion and bowel rest, along with the use of granulocyte colony-stimulating factors, are common nonoperative treatments [52].

It follows that various bacterial and few fungal infections can present as acute appendicitis in immunocompromised hosts. In a case of a 17-year-old human immunodeficiency virus (HIV)-positive patient, cryptosporidiosis presented as acute appendicitis, causing major life-threatening complications [3]. Three patients undergoing chemotherapy, two having candida and one *Aspergillus* infections, presented for acute appendicitis and underwent appendectomy. Only one of the three patients received curative treatment and survived [6]. Similarly, a 13-year-old leukemic child with aspergillosis developed acute appendicitis. After being admitted for chemotherapy intensification, the child soon developed abdominal pain, intermittent fever, and tenderness in the periumbilical region. Appendectomy for this patient soon followed, and his chemotherapy regime was continued [4]. Human cytomegalovirus (HCMV) viral infections are a very rare cause of acute appendicitis in immunocompromised patients. However, one study on 14 patients showed an association between HCMV and acute appendicitis, whereby cells double positive for early antigens and IL-6/IL-8 were prominent in 9 of 14 (64.4%) of appendicitis patients. HCMV late antigen was also found in 6 of 14 (42.9%) of the patients, and latent HCMV was present in as high as 11 of 14 (78.6%) of the patients as confirmed by in situ hybridization and HCMV IgG antibodies [53].

Another example of acute appendicitis was reported in a 6-year-old child with Wilms' tumor on chemotherapy. At first, the child had low-grade fever, persistent vomiting, and nonspecific intermittent "crampy" sensations at the level of the abdomen with no abdominal tenderness or rebound tenderness at McBurney's point. He was admitted with the suspected diagnosis of sepsis, and soon, his abdominal pain increased and became persistent with right lower quadrant guarding and tenderness at McBurney's point. He also underwent appendectomy followed by nephrectomy for his Wilms' tumor and continued chemotherapy as planned [2].

When dealing with severe cases of acute appendicitis, early intervention and treatment should be initiated to ensure favorable clinical courses. This was demonstrated in the case of an 11-year-old patient with AML who was found to have *Aspergillus* appendicitis and had undergone urgent appendectomy. Prolonged antifungal therapy was given until remission from ALL was established [8]. Similarly, a 4-year-old patient with ALL and having mucormycotic appendicitis was able to recover his neutrophil count after surgery and prolonged antifungal therapy with monitored serum Posaconazole [5].

Bowel perforation

Bowel perforation is defined as the discontinuity of the intestines and is the byproduct of a multitude of disease processes. In premature infants, the major cause is necrotizing enterocolitis, while in children and teenagers, it is usually due to appendicitis. However, in adults, the causes can vary widely [54].

Physical examination can be of importance in diagnosing bowel perforation. Almost all patients with bowel perforation present with abdominal pain, vomiting, nausea, decreased bowel function, and fever. Moreover, a detailed history of previous endoscopic retrograde cholangiopancreatography (ERCP) and colonoscopy should be noted if present [55, 56]. Vital signs assessment is essential for evaluating the presence of a systemic inflammatory response syndrome or sepsis [54]. On abdominal examination, abdominal rigidity and absent or hypoactive bowel sounds indicate bowel perforation [57]. We can distinguish perforation from obstruction through the presence of signs such as palpable peristalsis characteristic of obstruction and collection of a mass, gas, or fluid [58]. Other typical findings suggestive of obstruction include increased tympany and high-pitched bowel sounds [59]. Abdominal examination is also important to rule out acute surgical abdomen, whereby rebound tenderness and involuntary guarding might suggest a diagnosis of appendicitis or cholecystitis and require surgical intervention [60].

Several modalities are available for the evaluation of bowel perforation. A plain abdominal X-ray is cost-efficient and easy to perform and can detect signs of pneumoperitoneum. However, a CT scan of the abdomen and pelvis remains the gold standard in diagnosing the etiology behind the perforation [61]. In a case of a child with ALL having a perforated appendix and ileum, CT scan helped uncover extraluminal air that was otherwise overlooked on plain abdominal film [1]. A similar finding can be seen in Fig. 1.

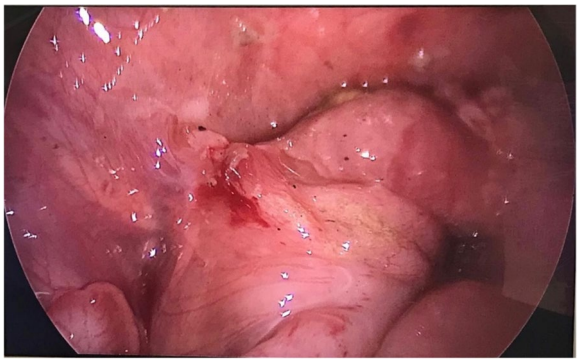

Fig. 1 Showing an example of intraoperative laparoscopic findings of bowel (cecal) inflammation and perforation in a patient with ALL on chemotherapy who presented with severe abdominal pain and findings of free air in the abdomen on CT scan

In the pediatric population, bowel perforation is considered an uncommon entity, due to the vague pathogenesis underlying it, an event that makes its diagnosis misleading and eventually delayed [62]. Fungal infections such as intestinal mucormycosis can be responsible for bowel perforation. Although rare, it is a critical condition that requires intensive care. This is demonstrated in a report of three children presenting with mucormycosis infection post bowel obstruction or perforation, who suffered major exacerbations including death [10]. Two children who presented for ALL were found to have intestinal mucormycosis, and only one survived [16]. Another interesting case was that of a malnourished child who presented with peritonitis and was intraoperatively found to have three perforations that showed characteristic hyphae under histopathological examination of their edges that confirmed the presence of gastrointestinal mucormycosis [13]. Other cases that involve infectious agents and perforation include intestinal tuberculosis. A case of a 17-year-old male with Down syndrome demonstrates how obstructive and perforated ileal tuberculosis caused multiple tuberculosis live abscesses [12]. Transplant pediatric patients are also an important target population when it comes to intestinal perforation. Posttransplant lymphoproliferative disorder (PTLD) is a serious complication and often develops after an Epstein-Barr virus infection. Of 14 patients having PTLD, 8 patients developed abdominal involvement, and 4 of them died of PTLD complications, one of which is bowel perforations. Similarly, this was seen in the case of a 3-year-old child who developed EBV and ulcerations in the sigmoid colon as a result of PTLD [15]. An interesting but rare case of an 11-year old with bowel perforation after peritoneal dialysis catheter removal posttransplantation reflects the complications that arise in the time period between transplantation and catheter removal [17].

Surgical resection of the involved segment is the standard of care in isolated bowel perforations. Platelet and neutrophil counts play a role in the timing of the surgery and the duration of recovery [16]. Historically, laparotomy was the main surgical procedure in the treatment of bowel perforation; however, with the emerging improvements in the field of medicine, laparoscopic intervention is one of the practical options used in identifying and treating the perforation [63]. Creating an intestinal stoma is also a possible surgical method. In a case study on 18 patients suffering from non-Hodgkin's lymphoma and undergoing 19 stomas, 9 children survived. Despite suffering many postoperative complications, stoma creation did not diminish their chances of survival [11].

Acalculous cholecystitis

Acalculous cholecystitis was first described in 1844 [64] and is defined as the acute inflammation of the gallbladder in the absence of gallstones [65]. It occurs due to stasis and an increase in the intraluminal pressure, which also leads to wall ischemia. The stasis is responsible for the colonization of bacteria and thus inflammation. The pressure buildup which causes ischemia can lead to sepsis due to gangrenous changes and perforation [66, 67].

Patients presenting with acalculous cholecystitis usually have right upper quadrant pain, positive murphy sign, food intolerance, nausea, and bloating. Diagnostic evaluation usually includes an US or a cholescintigraphy nuclear scan (HIDA). Another imaging method sometimes used is CT scan [68, 69].

Although acalculous cholecystitis is very uncommon in the pediatric population, it represents the major cause of cholecystitis, reaching up to 50–70% of all cholecystitis patients [65, 70]. This increase in diagnosed cases may be attributed to the improvement in echographic imaging, whereby imaging has shown acute acalculous cholecystitis (AAC) presenting with wall thickening greater than or equal to 3 cm, biliary sludge, gallbladder distension (>5 cm), and wall edema [71]. A retrospective review of immunocompromised children between 2006 and 2013 who underwent percutaneous cholecystostomy (PC) highlights the association between acute acalculous cholecystitis and immunocompromised states in the pediatric population. Ten children aged between 10 months and 15 years and 8 months were included in the study. Six of the immunocompromised children received bone marrow transplant for leukemia (5 children) or severe combined immunodeficiency (SCID) (1 child). The remaining four children had leukemia (2 children), SCID (1 child), and lymphoma (1 child) [18]. Similarly, Rose et al. examined the cholecystectomy specimens from a pediatric

institution over a 24-month period and recorded 3 children (2 children with hemoglobinopathies and 1 with mononucleosis) with eosinophilic cytoplasmic inclusions on the gallbladder [19].

Acalculous cholecystitis in the pediatric population is generally secondary to infection [72], trauma, intra-abdominal inflammatory disorders, and postoperative complication [73]. Immunocompromised children on the other hand can also present with AAC; however, the aforementioned subgroup is deemed as a poor surgical candidate. Thus, PC is more likely to be the preferred procedure in this group of patients. Moreover, in some cases, PC might obviate the need for future cholecystectomy [18]. In a PC, a thin hollow flexible tube is placed in the gallbladder to drain bile outside the body, under imaging guidance. In this process, inflammation and infection are reduced [74]. This procedure is said to be superior to laparoscopic or surgical cholecystectomy, for it decreases the chances of sepsis and shortens hospital stay [18].

Acute pancreatitis

Acute pancreatitis (AP) involves the inflammation of the pancreatic parenchyma. The diagnosis is usually made on clinical grounds. Patients usually present with epigastric pain that is accompanied by nausea and anorexia. The type of pain varies depending on the etiology of the inflammation. When biliary obstruction is the cause, the pain is usually sharp and radiates to the back. On the other hand, when toxic or metabolic exposure is the cause, the pain is usually generalized [75]. When evaluating acute pancreatitis, one should look for physical symptoms that are consistent with acute pancreatitis, lab values showing serum amylase and lipase levels more than three times their upper limits, and US or CT scan to evaluate for any pancreatic damage [76]. Recent reports have shown an increase in the number of patients with pancreatitis in the pediatric population [77]. Several factors have been shown to play a role, as will be discussed below.

To start with, children with cancer receiving certain types of medications have been shown to be at a higher risk of developing pancreatitis. L-Asparaginase, which is used in the treatment of ALL and some non-Hodgkin lymphomas (NHL), is one of these main drugs. Thirty-one (11%) of 275 patients receiving L-asparaginase suffered from abdominal pain, nausea, and vomiting [78]. An example of this can be seen in Fig. 2. No genetic factors have been shown to explain the association between this drug and AP [79]. Corticosteroids, such as dexamethasone and prednisone that are used in induction therapy in ALL pediatric patients, also play a role. Teuffel et al. conducted a meta-analysis on the topic and stated

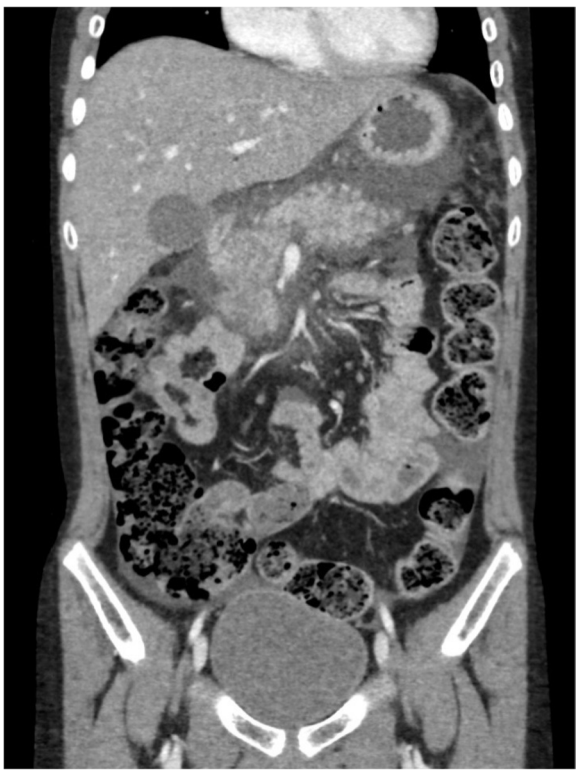

Fig. 2 Showing a CT scan of an 18-year-old patient with ALL who presented with severe epigastric pain

that the risk ratio for AP stimulated by dexamethasone, and prednisone was 3.78% [80]. These findings were further supported by the study conducted by Bai et al. that showed that corticosteroids and valproic acid were the main drugs responsible for the development of pancreatitis in the pediatric population [81]. The trimethoprim/sulfamethoxazole (TMP/SMX) treatment has also been hypothesized to play a role. It is generally used in patients undergoing chemotherapy as prophylaxis against *Pneumocystis jirovecii* infection. However, the TMP/SMX combination has been shown to cause pancreatitis in adults more than in children, with this notion believed to be dose dependent. Other drugs, usually used in stem cell transplantation such as cyclosporine, tacrolimus, cidofovir, foscarnet, and ganciclovir, have also been shown to cause pancreatitis [77]. This was seen in a case report of a 9-year-old boy with chronic granulomatous disease (CGD) who underwent a nonmyeloablative-matched sibling bone marrow transplant and received tacrolimus and mycophenolate mofetil posttransplant. The child developed acute pancreatitis 19 days after the transplant [28].

In addition, hematopoietic stem cell transplantation (HSCT) itself can be a cause of pancreatitis in children. This was shown in a study conducted by Wherlin et al. that stated that 3.5% of 202 children who were treated

with HSCT developed pancreatitis [24]. Similarly, a case of a 16-year-old female with acute B lymphoblastic leukemia (ALL-B) who received HSCT and developed acute pancreatitis 24 days after transplant supports the above notion [32]. Acute pancreatitis is also associated with organ and bone marrow transplantation. Between January 2001 and December 2004, Bateman et al. reported 5 children who received bone marrow transplant and developed acute pancreatitis, with 4 of them also having adenoviral infections [29]. Concerning organ transplantation, acute pancreatitis developed in a child with cystic fibrosis and *Candida albicans* infection post-lung transplantation [31] in a child 12 days after liver transplantation in 2002 [26], in 26 children between January 1986 and December 1999 also after liver transplantation [27], and in a 5-year-old girl after renal transplantation [30].

Other causes that have been suggested to cause pancreatitis include acquired infections in children such as HIV. Korayni et al. reported that in a children's hospital over the course of 6 years, 10 out of 42 children (23.8%) infected with HIV developed pancreatitis [25]. Similar findings were reported in a study by Miller et al., where over a 6-year period, 9 (17%) of 53 pediatric patients infected with acquired immunodeficiency syndrome (AIDS) developed pancreatitis [23].

Regarding the management of AP, it is important to replenish fluids with crystalloids in the first 24 h. It is also essential to monitor the pulmonary, cardiac, and renal functions in the first 48 h. Furthermore, enteral nutrition should be administered as soon as it is tolerated [82]. Endoscopic and surgical approaches in the management of children with AP are uncommon [83]; however, in chronic pancreatitis, endoscopic procedures may be advantageous in the presence of a detectable stricture or pancreatic duct obstruction [84, 85]. On the other hand, surgical procedures that include decompression of dilated ducts or resection of strictures can also be of value in the treatment of chronic pancreatitis [86].

Conclusion

The immunocompromised pediatric population is particularly susceptible to acute abdominal complications that require pertinent diagnosis and treatment. Imaging modalities such as ultrasonography (US) and computed tomography (CT) are useful for the diagnosis of neutropenic enterocolitis, appendicitis, bowel perforation, acute pancreatitis, and acalculous cholecystitis. Histologic evaluation, pancreatic enzymes, and HIDA scan are essential in the cases of neutropenic enterocolitis, acute pancreatitis, and acalculous cholecystitis, respectively. Treatment options vary widely among these conditions and include supportive hydration, broad-spectrum antibiotics, and antifungal medications.

Abbreviations

ALL: Acute lymphocytic leukemia; HIV: Human immunodeficiency virus; AML: Acute myeloid leukemia; BMT: Bone marrow transplant; SCID: Severe combined immunodeficiency disorder; PID: Primary immunodeficiency disorder; HSCT: Hematopoietic stem cell transplantation; CT: Computed tomography; US: Ultrasound; GI: Gastrointestinal; NE: Neutropenic enterocolitis; IBD: Inflammatory bowel disease; PTLD: Posttransplant lymphoproliferative; PC: Percutaneous cholecystostomy; HIDA: Hepatobiliary iminodiacetic acid; AP: Acute pancreatitis; NHL: Non-Hodgkin lymphomas; TMP/SMX: Trimethoprim/sulfamethoxazole; CGD: Chronic granulomatous disease; HCMV: Human cytomegalovirus; ERCP: Endoscopic retrograde cholangiopancreatography; AIDS: Acquired immunodeficiency syndrome.

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Authors' contributions

TBD, MBM, and CA contributed to writing and editing the article. AZ is the surgeon who supervised in writing and finalizing the article. The author(s) read and approved the final manuscript.

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Availability of data and materials

PubMed, MEDLINE, Scopus, and Google Scholar libraries were searched between the years 1989 and 2020 using each of the keywords "Immunocompromised host," "Chemotherapy," "Neoplasm," "Malignancy," "Congenital immunodeficiency," "Transplant," "Post-transplant," "SCID," "Primary Immunodeficiency Diseases" with each of "Neutropenic enterocolitis," "Appendicitis," "Bowel perforation," "Cholecystitis," "Pancreatitis," and "Typhlitis." The selected articles focused on patients under 18 years of age and included case reports, case series, systematic reviews, and meta-analyses. Extracted data included the etiology behind the patients' immunocompromised condition, in addition to their acute surgical complications. Only articles written in the English language were selected.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

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