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MAJOR ARTICLE

Splenic Abscess in the New Millennium: A Descriptive, Retrospective Case Series

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Background. Splenic abscess is a rare infection often resulting from hematogenous spread. Immunocompromised states are commonly comorbid, and the microbiology is heterogeneous.

Methods. We conducted a retrospective review of 33 cases identified by convenience sampling. Cases were treated in our institution's hospital system between May 2012 and February 2021 and classified as proven or probable based on predetermined criteria.

Results. The median age was 57 years, and 58% were men. Common underlying diseases included diabetes mellitus (30%), pancreatic disease (30%), and hematological malignancy (15%). The most common mechanism of pathogenesis was hematogenous spread (n = 13). *Escherichia coli*, enterococcal spp., and anaerobes were frequently implicated. One case was discovered at autopsy and excluded from subsequent analyses. The median duration of antimicrobial therapy (range) was 45 (5–525) days, and the median length of index hospitalization was 20 days. Percutaneous drainage by interventional radiology was common (17 of 32; 53%), and 6 patients underwent splenectomy. Treatment success was achieved in 14 of 32 cases (44%), with clinical stability in 3 of 32 cases (9%). Failures occurred in 13 of 32 (41%) cases, 2 of whom died from splenic abscesses. Two patients (2 of 32) were lost to follow-up.

Conclusions. To our knowledge, this is the largest North American series since the turn of the century and the first to distinguish between proven and probable cases. As reflected in our series, patients with splenic abscess may require prolonged hospitalizations and courses of antimicrobial therapy. Improvements in management are needed.

Keywords. diabetes mellitus; malignancy; pancreas; splenectomy; splenic abscess.

References to the spleen and splenic diseases have appeared in the medical literature since antiquity. Egyptian anatomists were charting the vasculature of the spleen as early as the second millennium BCE, and the sages of traditional Chinese medicine placed it among the 5 *zang* organs [1]. The understanding of splenic function has continued to develop in recent centuries, and numerous diseases are known to affect this solid organ. One rare form of splenic pathology is abscess [2, 3].

Classically, splenic abscess results from hematogenous seeding, preceding trauma, or other mechanisms [2, 4, 5]. Clinical factors associated with splenic abscess include splenic artery embolization [6, 7], endocarditis [8, 9], immunocompromised states [4, 5], and, less commonly, pancreatic disease [10, 11]. Microbiology is contingent upon the host and their respective global region. For example, splenic abscess due to melioidosis is not uncommon in Thailand [12, 13]. Contrarily, gram-positive bacteria, *Enterobacterales*, and anaerobic bacteria account for a sizeable number of culture-positive infections in studies from Taiwan [10, 11], India [14, 15], and the United States [4, 8, 16]. In general, management strategies center on antimicrobial therapy and achievement of source control through percutaneous drainage or splenectomy.

Few large series from North America have focused on splenic abscess [4, 8, 17], and modern evidence is confined to case reports and small case series. In the last 4 decades, the largest data set from a US center was published on 39 cases treated between 1981 and 1996 [4]. We hypothesized that this clinical entity has since undergone transformation due to significant changes in the epidemiology of solid organ and hematological malignancies [18–20], HIV [21], and immunosuppressive therapies [22]. Resultantly, we conducted a retrospective review of 33 cases treated in our institution's hospital system. We aimed to characterize (1) demographics and pathogenesis, (2) microbiology, (3) management strategies, and (4) clinical outcomes.

METHODS

Study Overview

Our study population consisted of all patients receiving care in the Yale New Haven Health System between May 2012 and

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February 2021. Our institution's electronic medical record (EMR) database is searchable as early as 2012, which corresponds to when departments' Epic records were configured in a searchable format by our institution's Joint Data Analytics Team (JDAT). As a result, the present series represents a convenience sample. To identify cases, we manually reviewed the EMRs of all patients with diagnoses associated with 1 or more of the following International Classification of Diseases 10th Revision (ICD-10) codes: splenic abscess (D73.3), splenic cyst (D73.4), splenic infarction (D73.5), splenic lesion (D73.89), and splenic hematoma (S36.029A). For patients who received care before the implementation of the ICD-10, their institution-specific codes corresponding to ≥1 of the above-listed ICD-10 codes of interest were identified and converted to ICD-10 when the EMR database was searched by JDAT. Our institutional pathology database was also queried. The Yale University Institutional Review Board approved our study protocol and waived the need for informed consent. All data were stored in a secure, encrypted fashion over the course of this study's review period.

Case Definition

We defined splenic abscess as a focal area of splenitis due to a proven or probable infectious etiology and contained within the splenic capsule. Perisplenic collections were deemed to be the sequelae of other intra-abdominal processes (eg, peritonitis) and were resultantly excluded. In order to be included, cases had to have demonstrable findings compatible with splenic abscess on imaging studies, intraoperative reports, and/or pathological examination in addition to clinical (eg, fever, leukocytosis, etc.) or microbiological data (eg, aspirate culture) consistent with an infectious process.

Cases with (1) a positive aspirate culture resulting from drainage of a splenic collection and/or (2) pathological confirmation of abscess via tissue examination were defined as proven. Probable cases were defined as having (1) clinical signs and symptoms (eg, rigors) consistent with infection, (2) imaging studies with 1 or more discrete splenic lesions measuring ≥5 mm, (3) supporting microbiological (eg, positive blood culture, antigen test, etc.) or procedural data (eg, purulence noted during percutaneous drainage), and (4) a clinical or radiographic response temporally related to antimicrobial therapy. Probable cases were required to meet all 4 criteria. Cases not satisfying either proven or probable criteria were excluded. During case identification and all subsequent data collection, 2 authors (C.R., M.G.) were required to reach consensus on cases with incomplete or ambiguous information documented in the EMR.

Data Collection

Basic demographic information was recorded from the EMR. Data concerning abdominal trauma, intraperitoneal surgery, injection drug use, bacteremia/fungemia, and other clinical factors preceding the diagnosis of splenic abscess were collected. For each case, the following immunocompromising or gastrointestinal comorbidities were noted: HIV/AIDS, solid organ transplantation, hematopoietic stem cell transplantation, end-stage renal disease (ESRD), active hematological or solid organ malignancy, cirrhosis, diabetes mellitus, inflammatory bowel disease, pancreatic disease (eg, malignancy, pancreatitis, pseudocyst, etc.), and receipt of immunosuppressive therapy for an underlying condition (eg, prednisone, infliximab, etc.). History of prior splenic artery embolization was also recorded. We sorted the presumptive mechanism of abscess pathogenesis into the following categories: hematogenous spread, contiguous spread, superinfection of hematoma or infarcted tissue, multifactorial, and idiopathic.

Length of initial hospital stay was recorded for each case. Readmissions and complications directly related to the splenic abscess were noted. All relevant microbiological and pathological data were extracted. Length of appropriate therapy was recorded, as were the identities of treatment regimens. We defined appropriate therapy as antimicrobial therapy offering adequate coverage for the inciting pathogen(s). Resultantly, length of appropriate therapy includes both empiric and targeted regimens that cover the isolated pathogen(s). For culture-negative cases, the entire length of empiric antimicrobial therapy was recorded as the length of appropriate therapy. The following adverse events related to antimicrobial therapy were recorded if present: dermatological or musculoskeletal complaints, gastrointestinal complaints, liver or kidney abnormalities, creatine kinase (CK) elevation, and neurological complaints. Discontinuation of therapy due to adverse events was recorded. We also recorded surgical or procedural interventions related to management of splenic abscesses. Procedural complications (eg, drain mispositioning, peritonitis, etc.) were recorded when present.

Classification of Outcomes

For summary purposes, outcomes were divided into 4 groups: treatment success, clinical stability, treatment failure, and loss to follow-up. Treatment success was defined as resolution of symptoms and/or radiographic response to therapy without the need for additional antimicrobial therapy or procedural interventions during the patient's follow-up period at our institution. When applicable, the following symptoms and clinical factors were assessed: vital sign derangements, fever curves, leukocytosis trends, vasopressor requirements, and patient-reported symptoms (eg, pain). Radiographic response was defined as reduction in abscess cavity size or complete resolution.

Cases with clinical stability were defined as patients with interval improvement in clinical factors and/or interval decrease in abscess size after initiation of therapy without evidence of complete resolution or further decompensation due to the splenic abscess during their follow-up periods. Treatment failures were defined as cases who lacked response to therapy,

had uncontrolled progression of the infectious process while on therapy, or needed additional interventions after completion of an initial course of therapy. Lack of response to therapy was defined as no improvement in symptoms or clinical factors and/or no decrease in abscess size after treatment initiation. Uncontrolled progression was defined as worsening symptoms or clinical factors and/or increase in abscess size. Deaths attributable to splenic abscess were defined as deaths with direct, plausible links to the splenic abscess and were classified as treatment failure. As such, not all deaths after diagnosis were attributed to splenic abscesses. Cases incidentally uncovered at autopsy are reported separately. The medical records of all cases were reviewed until death, loss to follow-up, or the conclusion of this study's review period in August 2021.

RESULTS

Study Population and Abscess Pathogenesis

In total, we manually reviewed the EMR for 925 patients with ≥1 ICD-10 code(s) of interest seen in our health care system between May 2012 and February 2021. During this time period, we identified 33 cases of splenic abscess treated in our institution's health care system (Table 1). The median age (range) was 57 (29–95) years, and 58% were men. Common underlying diseases were diabetes mellitus (30%), pancreatic disease (30%), and hematological malignancy (15%). Three patients had cirrhosis, 2 patients had HIV, and 1 patient reported injection drug use. Definite endocarditis was present in 9% of cases (3 of 33), with an additional case having possible endocarditis. Four cases had intraperitoneal surgery in the 8 weeks preceding the diagnosis of splenic abscess. Six patients had previously undergone splenic artery embolization. Twelve patients (36%) had bacteremia ≤5 days before or after the diagnosis of splenic abscess was made.

The most common mechanism of pathogenesis was hematogenous spread (n = 13), followed by superinfection of hematoma or infarcted tissue (n = 7). Of patients with presumed hematogenous spread (n = 13), 4 had definite or possible endocarditis, 3 had multiple infectious foci, 2 were hematological malignancy patients with recent candidemia, 1 had an epidural abscess, 1 had an indwelling central line, 1 had typhoid fever, and 1 had no other identified sources. Six cases were multifactorial, 5 cases resulted from contiguous spread, and 2 cases were idiopathic. Four of 5 cases resulting from contiguous spread had intrabdominal pathology. Half of the cases (17 of 33) satisfied criteria to be considered proven.

Thirty-two cases had imaging studies available, and 1 case was found at autopsy. In total, 31 of 32 (97%) cases' initial diagnostic imaging studies were computed tomography (CT) scans, and 1 case underwent a whole-body positron emission tomography CT scan. The median abscess size (range) was 6 (<1–24) cm, and 6 of 33 (18%) had multifocal abscesses. Three of 6 cases with multifocal abscesses had <1-cm lesions. Two cases with <1-cm lesions were caused by *Candida* spp., and the remaining case was due to *Mycobacterium tuberculosis* complex. Figure 1 displays representative examples of CT scans.

Microbiology and Pathology

In total, 32 of 33 cases had microbiological data available. One case was uncovered at autopsy, and no premortem microbiological data were available. One or more positive blood or splenic drainage cultures were available for 30 of 32 cases (94%). The single culture-negative case had received 10 days of cefepime and 9 days of both anidulafungin and vancomycin before percutaneous drainage. It is interesting to note that gram staining of a concurrent perinephric abscess showed 4+ white blood cells and 3+ hyphae. One-third of cases (11 of 32) in the present study had ≥2 species isolated from blood or splenic drainage cultures.

Table 2 details the microbiology data for all cases. *Escherichia coli*, enterococcal spp., and several anaerobes were frequently isolated from blood cultures. A similar list of bacterial species was isolated from splenic drainage cultures. Only 1 splenic abscess case involved the isolation of *Staphylococcus aureus*. Six cases were polymicrobial (≥3 species isolated).

Pathology reports were available for 6 splenectomy cases, and all cases confirmed the presence of abscess. One case of splenic abscess was uncovered at autopsy in an 82-year-old woman with ESRD on hemodialysis. An abscess cavity (5.2 × 2.5 × 1.4 cm) was incidentally identified. The cause of death was deemed to be severe atherosclerotic cardiovascular disease likely leading to cardiac arrhythmia.

Management and Outcome

All cases (n = 32) with premortem diagnosis of splenic abscess received antimicrobial therapy and had an index hospitalization (median length of stay, 20 days). A total of 8 cases had 1 or more readmissions related to the diagnosis of splenic abscess. Readmissions were related to worsening of symptoms, need for repeat drainage procedures, and/or sepsis.

The median duration of antimicrobial therapy (range) was 45 (5–525) days, and 30 of 32 patients received multiple agents. Patients receiving ≥2 antimicrobial agents received a median (range) of 5 (2–8) agents. In total, 28 of 32 (88%) patients received a beta-lactam, and 19 of 32 (59%) received vancomycin. Fluoroquinolones and metronidazole were both used in 17 of 32 (53%) cases. Five patients received a carbapenem, 3 patients an oxazolidinone, and 3 patients a glycylcycline. Antifungal therapy was used in 9 cases. Twelve adverse events related to antimicrobial therapy occurred (4.5 events/1000 days of antimicrobial use), with dermatological complaints (n = 4) and elevated liver enzymes (n = 4; 3 episodes of drug-related transaminitis, 1 episode of drug-induced liver injury) being common. Each of the following adverse events was documented

Table 1. Summary of Splenic Abscess Cases

Age (y), Sex	Comorbidities and Clinical Information	Infection Type (Proven or Probable)	Presumptive Mechanism	Relevant Microbiological Data	Largest Abscess Dimension on Initial Imaging Studies (cm)	Length of Appropriate Therapy, d	Procedural Intervention(s) for Splenic Abscess	Outcome
51, female	Pancreatic cancer on chemotherapy	Probable	Contiguous spread	*Escherichia coli* and *Streptococcus anginosus* bacteremia; concurrent polymicrobial hepatic abscess	4.4	206	None	Failure (uncontrolled progression on initial therapy)
57, female	Recent intraperitoneal surgery	Probable	Contiguous spread	Polymicrobial bacteremia and candidemia preceding splenic abscess diagnosis; concurrent intra-abdominal collections with polymicrobial cultures	2.7	56	None	Success
46, female	Recent intraperitoneal surgery	Proven	Contiguous spread	Splenic drainage culture grew *Bacillus* spp.; polymicrobial peritoneal fluid culture	8.3	17	Percutaneous drainage	Success
80, female	Concurrent perinephric abscess and chronic renal calculi	Probable	Contiguous spread	Splenic drainage culture negative; concurrent perinephric abscess drainage culture negative with gram stain 3+ hyphae	5	28	Percutaneous drainage	Success
29, male	Remote history of necrotizing pancreatitis with multiple complications	Proven	Contiguous spread	Splenic drainage culture grew Group B *Streptococcus*	3	16	Percutaneous drainage	Success
30, male	Endocarditis, DM, IBD with recent prednisone course, remote history of necrotizing pancreatitis with chronic collections	Proven	Hematogenous spread	*Staphylococcus lugdunensis* bacteremia	5.8	43	Percutaneous drainage; splenectomy	Failure (uncontrolled progression on initial therapy)
78, male	DM	Probable	Hematogenous spread	MSSA bacteremia preceding splenic abscess diagnosis	2.1	43	None	Success
57, female	Acute myelogenous leukemia on chemotherapy with recent prednisone course for refractory gingivitis	Probable	Hematogenous spread	*Candida kefyr* fungemia preceding diagnosis of microabscesses	<1	8	None	Stable (death from other causes)
31, female	Postpartum cardiomyopathy with indwelling Hickman line for milrinone infusions	Probable	Hematogenous spread	*Klebsiella pneumoniae* bacteremia	3.5	27	None	Success
61, male	Endocarditis, DM	Probable	Hematogenous spread	*Enterococcus faecalis* bacteremia	3.7	63	None	Lost to follow-up
48, male	Recent intraperitoneal surgery	Probable	Hematogenous spread	*Solobacterium moorei* and *Atopobium rimae* bacteremia	6	49	None	Failure (uncontrolled progression on initial therapy)
89, male	DM	Probable	Hematogenous spread	*Serratia marcescens* bacteremia	5.6	47	None	Success
43, female	Acute myelogenous leukemia on chemotherapy	Probable	Hematogenous spread	CoNS and *Burkholderia cepacia* bacteremia and candidemia preceding splenic microabscess diagnosis; catheter tip culture grew >100 000 CFU CoNS	<1	74	None	Success
40, male	Endocarditis, injection drug use	Proven	Hematogenous spread	*Serratia marcescens* bacteremia; splenic drainage and fluid cultures grew *S. marcescens*	4.5	41	Percutaneous drainage; splenectomy	Failure (uncontrolled progression on initial therapy)

Table 1. Continued

Age (y), Sex	Comorbidities and Clinical Information	Infection Type (Proven or Probable)	Presumptive Mechanism	Relevant Microbiological Data	Largest Abscess Dimension on Initial Imaging Studies (cm)	Length of Appropriate Therapy, d	Procedural Intervention(s) for Splenic Abscess	Outcome
30, male	HIV (CD4 13, VL 140 000) off HAART	Probable	Hematogenous spread	Histoplasma urine antigen >15 ng/mL (reference range <0.2 ng/mL)	1.4	365	None	Success
78, female	Psoriasis on adalimumab	Probable	Hematogenous spread	*Mycobacterium tuberculosis* complex in sputum culture	0.7	525	None	Success
56, male	Recent travel to Bangladesh	Probable	Hematogenous spread	*Salmonella* serotype Typhi bacteremia	3.6	14	None	Lost to follow-up
60, female	DM, possible endocarditis	Probable	Hematogenous spread	*Pseudomonas aureginosa* bacteremia	4	28	None	Stable (death from other causes)
83, female	Pancreatic cancer on chemotherapy, low-grade lymphoma, hypogammaglobulinemia on IVIG	Proven	Idiopathic	Polymicrobial splenic drainage culture; VRE bacteremia	8.5	12	Percutaneous drainage	Failure (death)
82, female	End-stage renal disease on hemodialysis, DM	Proven	Idiopathic	No premortem culture data available	5.2 (on autopsy)	None	None	Discovered on autopsy
66, male	Pancreatic cancer on chemotherapy	Proven	Multifactorial	*Bacteroides fragilis* bacteremia preceding splenic abscess diagnosis; polymicrobial splenic drainage culture	10	42	Percutaneous drainage	Stable (death from other causes)
32, male	Cirrhosis with ascites, DM, recent pancreatitis	Proven	Multifactorial	Splenic drainage culture with *Clostridium difficile*; perisplenic drainage culture also grew *C. difficile*	4	101	Percutaneous drainage	Success
95, male	Uncharacterized pancreatic mass	Proven	Multifactorial	*Bacteroides fragilis* bacteremia preceding splenic abscess diagnosis; multiple polymicrobial splenic drainage cultures	7.5	203	>1 percutaneous drainage	Failure (need for re-intervention; death)
71, male	Cirrhosis without ascites, hepatocellular carcinoma, splenic artery embolization	Proven	Multifactorial	*Cutibacterium avidum* bacteremia preceding splenic abscess diagnosis; splenic drainage culture grew *C. avidum*	16	158	>1 percutaneous drainage	Failure (need for re-intervention)
40, male	Acute promyelocytic leukemia on chemotherapy, DM	Proven	Multifactorial	*Escherichia coli* and *Streptococcus anginosus* bacteremia preceding diagnosis of splenic abscess; splenic drainage culture grew *Escherichia coli*	12	68	Percutaneous drainage; splenectomy	Failure (uncontrolled progression on initial therapy)
53, male	Necrotizing pancreatitis, splenic artery embolization	Proven	Multifactorial	*Escherichia coli* and *Pseudomonas aureginosa* bacteremia; polymicrobial splenic and pancreatic drainage cultures; concurrent hepatic abscess drainage culture grew *E. coli*	12	63	Percutaneous drainage; splenectomy with open drainage of pancreatic fluid collections and partial pancreatectomy	Failure (uncontrolled progression on initial therapy)
51, male	Abdominal trauma with splenic artery embolization for hematoma, HIV (CD4 126, VL 22) on HAART	Proven	Superinfected hematoma	Splenic fluid culture grew *Staphylococcus lugdunensis*	24	5	Splenectomy	Success
70, female	Gastric cancer, splenic artery embolization for spontaneous splenic rupture, DM	Proven	Superinfected hematoma	Splenic drainage culture grew pan-susceptible *Enterococcus* spp.; concurrent intra-abdominal collections grew VRE	16	32	Percutaneous drainage	Success
47, female	Undifferentiated connective tissue disease on hydroxychloroquine	Proven	Superinfected splenic infarct	Splenic drainage culture grew *Clostridium* spp.	6	22	Percutaneous drainage; splenectomy	Failure (uncontrolled progression on initial therapy)

Table 1. Continued

Age (y), Sex	Comorbidities and Clinical Information	Infection Type (Proven or Probable)	Presumptive Mechanism	Relevant Microbiological Data	Largest Abscess Dimension on Initial Imaging Studies (cm)	Length of Appropriate Therapy, d	Procedural Intervention(s) for Splenic Abscess	Outcome
61, male	Pancreatic cancer on chemotherapy	Probable	Superinfected splenic infarct	*Escherichia coli* bacteremia; splenic drainage cultures negative	8	67	>1 percutaneous drainage	Failure (uncontrolled progression on initial therapy)
93, male	Colon cancer and recent intraperitoneal surgery	Probable	Superinfected splenic infarct	*Escherichia coli* and gamma-hemolytic *Streptococcus* spp. bacteremia	8	11	None	Failure (uncontrolled progression on initial therapy)
70, male	DM, splenic artery embolization for bleed related to distal pancreatectomy for neuroendocrine tumor	Proven	Superinfected splenic infarct	*Enterococcus faecalis* and *Lactobacillus* spp. bacteremia; polymicrobial splenic drainage culture	9.5	50	Percutaneous drainage	Success
62, female	Post–essential thrombocytosis myelofibrosis on ruxolitinib, cirrhosis with ascites and TIPS, splenic artery embolization	Proven	Superinfected splenic infarct	Splenic drainage culture grew CoNS	19.7	164	>1 percutaneous drainage	Failure (need for re-intervention)

Abbreviations: CFU, colony-forming unit; CoNS, coagulase-negative *Staphylococcus*; DM, diabetes mellitus; HAART, highly active antiretroviral therapy; IVIG, intravenous immunoglobulin; IBD, inflammatory bowel disease; MSSA, methicillin-sensitive *Staphylococcus aureus*; TIPS, transjugular intrahepatic portosystemic shunt; VL, viral load; VRE, vancomycin-resistant *Enterococcus*.

once: non–*Clostridium difficile* diarrhea, altered mental status, CK elevation, tendon pain. Five patients had antimicrobial agents discontinued due to adverse events.

Percutaneous drainage by interventional radiology was used in the majority of cases (17 of 32; 53%), and 6 cases underwent splenectomy. Five of 6 splenectomies were performed after an inadequate percutaneous drainage procedure. Splenectomy complications included a pleural effusion (n = 1) and pancreatic ductal injury (n = 1), whereas percutaneous drainage procedures were complicated by severe pain due to drain mispositioning (n = 1) and peritonitis (n = 1). Two cases were notable for requiring repeat drainage procedures months (range, 3–8 months) after their initial presentations.

Treatment success was achieved in 14 of 32 cases (44%). Three cases (9%) had stable disease, and all 3 cases had deaths not attributable to the splenic abscess (range, 8–42 days after diagnosis). Failures occurred in 41% (13 of 32) of cases, with 2 of 13 patients having had deaths attributable to the splenic abscess (range, 12–428 days after diagnosis). The median durations of antimicrobial therapy for successes and failures were 45 days and 63 days, respectively. The majority of treatment failures (9 of 13; 69%) resulted from progression of disease while still being treated with an initial antimicrobial regimen, and 5 patients underwent splenectomy due to inadequate source control afforded by percutaneous drainage. For patients with pancreatic pathology, 6 of 10 (60%) had treatment failures. Two patients (2 of 32) were lost to follow-up. Notably, 1 was clinically improving at an office visit 2 weeks after discharge, and 1 had outpatient abdominal imaging showing interval decrease in the size of the splenic lesion.

DISCUSSION

We report individualized information on 33 cases of splenic abscess treated in our institution's health care system. Half of all cases were proven, and this is the first series to apply criteria for distinguishing between probable and proven cases. Diabetes mellitus, pancreatic pathology, and splenic artery embolization were common comorbidities, and 18% of infections were polymicrobial. The median duration of antimicrobial therapy was roughly 6 weeks, and half of the patients underwent procedural interventions. Treatment success was only achieved in 44% of cases, and attributable mortality was 6% (2 of 32). To our knowledge, this is the largest North American series from a single health care system in over 2 decades.

Optimal duration of antimicrobial therapy for splenic abscess remains an open question, and we report a median duration of 45 days. Only 2 other modern series have reported mean durations of 5.6 weeks in patients (n = 27) with endocarditis and splenic abscess [8] and 46 days in a subgroup of patients (n = 33) who did not undergo procedural interventions [10]. These lengths of therapy are consistent with those reported for pyogenic liver abscesses, a more common intra-abdominal

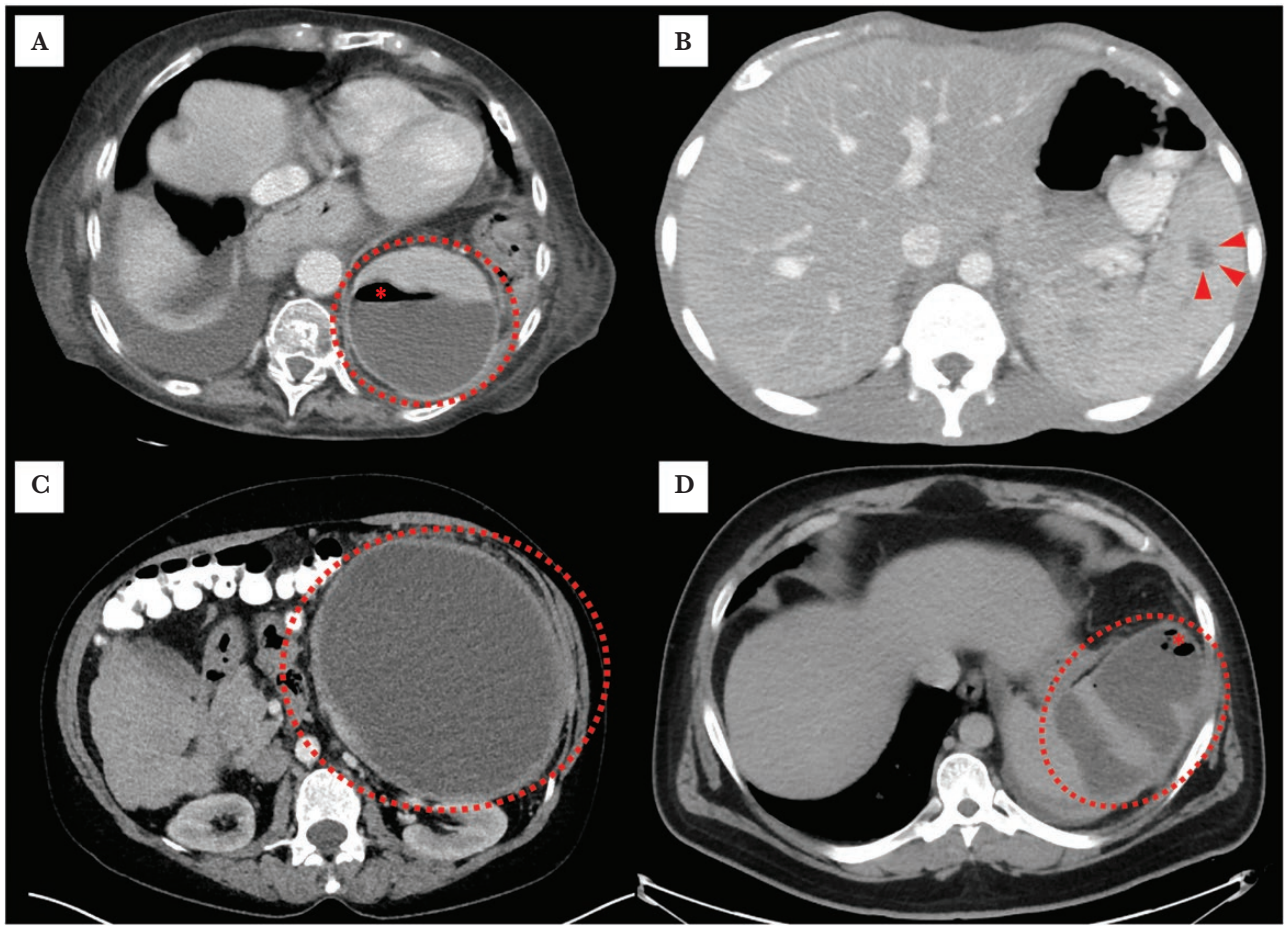

Figure 1. Representative computed tomography scans of splenic abscesses. A, Polymicrobial case in an 83-year-old woman with low-grade follicular lymphoma and pancreatic cancer. An abscess (interrupted circle) with an air–fluid level (asterisk) is visualized. B, Splenic histoplasmosis in a 30-year-old man with HIV. Arrowheads denote a 1.4-cm abscess. C, Coagulase-negative *Staphylococcus* case in a 62-year-old woman with cirrhosis and post–essential thrombocytosis myelofibrosis. An abscess (interrupted circle) is visualized. D, *Escherichia coli* case in a 40-year-old man with acute promyelocytic leukemia. A complex abscess (interrupted circle) with internal foci of gas (asterisk) is present.

solid organ abscess with somewhat comparable pathogenesis and microbiology [23–25]. For example, Yu et al. reported 6-week courses for all liver abscess cases (n = 64) in their prospective study on drainage techniques [23]. Duration of antimicrobial therapy for abscesses is dependent upon multiple factors including site, causative organism(s), and achievement of source control. The Study to Optimize Peritoneal Infection Therapy (STOP-IT) trial found shorter courses to be satisfactory if source control is first achieved [26]. It is important to note that intraperitoneal solid organ infections were underrepresented in the STOP-IT trial, and only 5% and 2% of cases in the control and experimental arms, respectively, arose from the liver. No infections of splenic origin were explicitly reported. As a result, shorter treatment courses may not be appropriate for pyogenic infections of the spleen and liver.

Prompt attainment of source control proved challenging in our study given that 9 cases required multiple procedures, and this contributes to our relatively protracted length of antimicrobial therapy. Additionally, 2 patients had disseminated infections (ie, tuberculosis and histoplasmosis) requiring months of therapy per standard treatment guidelines. Despite the known risk of overwhelming postsplenectomy infections that may require antibiotic prophylaxis or vaccination [27], we do report 1 successful case of just 5 days of antibiotics after splenectomy for a 24-cm abscess due to *Staphylococcus lugdunensis* in a 51-year-old man with well-controlled HIV.

Diabetes mellitus was the most common comorbidity in our series, and others have reported similar findings (prevalence 22%–51%) [10, 11, 14, 15]. By impacting microvasculature and immune function [28], its epidemiology likely affects a number of other pyogenic infections like brain abscess [29], liver abscess [25], and pyomyositis [30]. Another common comorbidity was active malignancy, and both deaths occurred in patients with malignancies. Although HIV has been reported as a common comorbidity accompanying splenic abscesses [4], our present study only identified 2 cases. This shift in epidemiology is likely attributable to the advent of highly active antiretroviral therapy for patients with HIV.

Table 2. Microbiology of Blood and Splenic Drainage Cultures With Speciated Results

Blood Culture (n = 35)	Percutaneous or Intraoperative Splenic Drainage Culture (n = 24)
Atopobium rimae	*Bacillus* spp.
Bacteroides fragilis (n = 2)	*Bacteroides thetaiotaomicron*
Burkholderia cepacia	*Candida albicans*
Candida albicans	*Clostridium difficile*
Candida glabrata	*Clostridium* spp.
Candida kefyr	CoNS (n = 2)
Candida tropicalis	*Cutibacterium avidum*
CoNS	*Eikenella corrodens*
Cutibacterium avidum	*Enterococcus faecalis*
Enterococcus faecalis (n = 2)	*Enterococcus* spp.
Enterococcus faecium (VRE; n = 2)	*Enterococcus* spp. (VRE)
Escherichia coli (n = 6)	*Escherichia coli* (n = 3)
Klebsiella pneumoniae	*Klebsiella* spp.
Klebsiella spp.	*Lactobacillus* spp.
Lactobacillus spp.	*Morganella morganii*
MSSA	*Pseudomonas aureginosa*
Pseudomonas aeruginosa (n = 2)	*Saccharomyces cerevisiae*
Serratia marcescens (n = 2)	*Serratia marcescens*
Solobacterium moorei	*Staphylococcus lugdunensis*
Staphylococcus lugdunensis	*Veillonella parvula*
Streptococcus anginosus (n = 2)	Group B *Streptococcus*
Streptococcus pneumoniae	
Streptococcus spp. (gamma hemolytic)	
Salmonella serotype Typhi	

Unless otherwise stated, each individual entry signifies that the species was isolated from 1 case. It is important to note that several individual splenic abscess cases led to the isolation of multiple species.

Abbreviations: CoNS, coagulase-negative *Staphylococcus*; MSSA, methicillin-susceptible *Staphylococcus aureus*; VRE, vancomycin-resistant *Enterococcus*.

Pancreatic disease (eg, malignancy, acute or chronic pancreatitis, pancreatic pseudocyst, etc.) has less commonly been reported as comorbid with splenic abscess [10, 11], but our study's data corroborate this observation. No unifying mechanism accounted for splenic abscess in patients with pancreatic disease; however, the anatomical proximity of the spleen and pancreas accounts for cases resulting from contiguous spread. In our series, the high treatment failure rate (60%) for patients with pancreatic disease may be skewed by our small sample size, but the observation is likely multifactorial. Possible contributions include nonpancreatic comorbidities and challenges associated with the inflammatory state that often accompanies pancreatic pathology.

The wide spectrum of bacterial and fungal species isolated in the present study is largely consistent with prior reports [4, 10, 11, 14, 15], and polymicrobial splenic abscesses have been reported to range from 1% to 18% [10, 11, 14]. Nonetheless, there were 2 cases in our study with exceptionally rare anaerobic species as the cause of splenic abscess. One case of splenic abscess in a 71-year-old man with cirrhosis was caused by *Cutibacterium avidum*, and only 2 prior cases of *C. avidum* splenic abscess exist in the literature [31, 32]. It is interesting to note that both cases were cardiac patients [31, 32], and 1 had undergone cardiac catheterization before developing splenic abscess [32]. In our case, angiography was also performed as the patient had undergone splenic artery embolization in the weeks preceding diagnosis. We also report a case of splenic abscess due to *Solobacterium moorei* and *Atopobium rimae* bacteremia in a 48-year-old man with recent Nissen fundoplication. To our knowledge, this stands as the first report of either species being identified as the etiology of splenic abscess.

There is a conspicuously low incidence of *S. aureus* in our series when compared with historical data [4, 8]. Two of the largest series from the United States report on cases from the latter 20th century and note a higher incidence of both *S. aureus* and injection drug use in their study populations [4, 8]. By contrast, the present study only identified 1 patient with injection drug use, and it is interesting to note that a recent US series on splenic abscess only reported 1 case [17]. The significance of this finding is unclear given the myriad infectious complications of injection drug use [33], and the relatively modest size of our study may not represent the true incidence of *S. aureus* splenic abscess. It is important to note, however, that one of the abovementioned series only included patients with concurrent endocarditis, and 78% of patients (21 of 27) were reported to inject drugs [8]. This large proportion likely skewed the microbiology of that series. Infectious endocarditis can lead to splenic infarcts, which radiographically mimic splenic abscesses, but all cases in that series would be classified as proven based on our proposed criteria [8].

Although we developed a rigorous case definition and present a large series for this type of infection, the present study has limitations. Principally, the strength of our study's conclusions is limited by its small population size, single geographic locale, and retrospective nature. Use of ICD-10 codes to identify cases also limited our ability to definitively identify all cases during our study period, and we are resultantly unable to comment on changes in incidence or prevalence. The present convenience sample likely underestimates the total number of splenic abscesses treated during our study period. Additionally, the reasoning behind management decisions was the product of individual physicians, and we are unable to fully ascertain how length of therapy or need for intervention was determined.

CONCLUSIONS

Our review of 33 splenic abscess cases treated between 2012 and 2021 confirmed this infection to be a significant source of morbidity and mortality. The median duration of antimicrobial therapy was 45 days, and 6 patients underwent splenectomy despite the availability of modern percutaneous drainage techniques. In contrast to prior reports from North America [4, 8], we identified a large proportion of cases with active

malignancies or pancreatic disease as opposed to injection drug use, infectious endocarditis, or HIV. Further studies on optimal management strategies for select patient populations are indicated.

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Author contributions. C.R. decided study concept and design, collected data, and wrote the manuscript. Z.T. assisted with data collection and revised the manuscript. S.D.G. assisted with data collection and revised the manuscript. M.G. decided study concept and design, revised the manuscript, and conducted study supervision. All authors contributed to the manuscript and its review.

Patient consent. The study protocol was reviewed and approved by Yale University Institutional Review Board (protocol #2000030275). All work performed during the study period was in accordance with the ethical standards of our institution as well as those detailed by the 1964 Helsinki Declaration and its later amendments. Need for informed consent was waived by Yale University Institutional Review Board.

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