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Prevalence and Associated Factors of Abdominal Pain and Disability at One Year Follow-up After an Attack of Acute Pancreatitis

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Abstract

Objectives: To report the prevalence and predictors of abdominal pain and disability one year after an acute pancreatitis (AP) attack.

Methods: Patients were prospectively enrolled between December 2012 and April 2016. Enrolled subjects were contacted at a median of 13 months after enrollment. Multivariable regression models were used to determine factors independently associated with abdominal pain at follow-up.

Results: Response rate was 71% (110/155). Of respondents, median age was 51 years, 58% were female, and 14% had severe AP. At follow-up, 24% of patients reported abdominal pain (65% intermittent, 35% constant), 10% used analgesics regularly, and 6% had regular opioids use. Furthermore, 41% of patients experienced pain-related interference with work or daily activities, and 8% developed disability. On regression analysis, idiopathic etiology [odds ratio (OR), 3.8; 95% confidence interval (CI), 1.1–13.6], organ failure (OR, 3.3; 95% CI, 1.1–7.9), and recurrent AP (OR, 2.9; 95% CI, 1.1–10.6) were independently associated with abdominal pain at follow-up.

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Disability at follow-up was associated with younger age, current smoking, and intensive care unit admission (all $P < 0.05$).

Conclusion: Abdominal pain and disability are potential long-term sequelae of AP. Certain pre-existing factors and pancreatitis features are associated with these outcomes at one-year follow-up of AP.

Keywords

acute pancreatitis; natural history; patient related outcomes; pain; disability

Introduction

The clinical landmark of acute pancreatitis (AP) is severe acute upper abdominal pain. This is secondary to acinar cell injury, cytokine release, leukocyte recruitment, and parenchymal inflammation.[1] The majority of AP patients have a mild clinical course, but 10–20% develop severe AP, characterized by persistent organ failure, local complications, and impaired survival.[2] Several studies have demonstrated that a subset of AP survivors develop long-term sequelae including exocrine pancreatic insufficiency (EPI), diabetes mellitus (DM), micronutrient deficiencies, recurrent acute pancreatitis (RAP), and chronic pancreatitis (CP).[3–7] As the incidence and survival of AP continue rising, more individuals will be at risk for these long-term consequences.[8,9] Therefore, understanding the incidence, prevalence and predictors of late AP complications, is of paramount importance for clinicians and policymakers.

We recently demonstrated in a prospective cohort that patients surviving an attack of AP experience impaired quality of life (QOL) at one-year follow-up compared to no-pancreatitis controls.[10] This effect was mostly due to presence of abdominal pain, analgesic use, and disability at follow-up; however, the prevalence of these factors at long-term follow-up following an AP attack has not been well established. Understanding abdominal pain and disability after recovery of AP is relevant from a clinical, socioeconomic, and patient's perspective. Based on previous data on chronic gastrointestinal disorders such as CP and inflammatory bowel disease, abdominal pain can progressively lead to opioid use, disability, and impaired QOL.[11,12] Survivors of AP may follow a similar pathway if they experience long-term debilitating abdominal pain. Moreover, disability in AP may result in a detrimental socioeconomic impact due to unemployment and work absenteeism, which would overall increase the indirect costs related to AP care. Identifying AP survivors with high risk factors for abdominal pain and disability may offer a therapeutic window for secondary and tertiary prevention of these potential late complications of AP. Therefore, we aim to report the prevalence and associated risk factors of abdominal pain and disability after one year from an AP attack.

MATERIALS AND METHODS

Study Population

This study is a post hoc analysis of a prospective single center cohort study aimed to assess the impact of AP in QOL.[10] Patients admitted to the University of Pittsburgh Medical Center

(UPMC) with a diagnosis of AP were prospectively enrolled during hospitalization. Exclusion criteria were defined as the presence of CP at enrollment, and history of pancreatic cancer. Enrolled participants who survived an attack of AP between September 2011 and May 2015 were contacted at one year after enrollment, with the purpose of obtaining long-term follow-up data on abdominal pain, disability, and QOL. As data on QOL has been previously reported,[10] this study will focus on abdominal pain and disability at long-term follow-up of AP. The study was approved by the University of Pittsburgh Institutional Review Board (IRB # PRO08010374). Informed consent for participation and follow-up was obtained prior to enrollment.

Assessment of Abdominal Pain and Disability

Long-term follow-up data on abdominal pain and disability was obtained using a dedicated questionnaire. Participants were reached through standardized telephone calls conducted by a research coordinator. In the event of a patient not being reached by phone, a voicemail was left with instructions to call back. A total of 5 phone calls per subject were attempted prior to mailing the questionnaire to them.

Patients were asked whether they had experienced abdominal pain within the last 4 weeks and whether it was presumed to be related to pancreatitis. Among subjects who reported abdominal pain, further questions on pattern and severity of pain were asked. Five options were provided to patients to describe their pain pattern: A) mostly pain free with episodes of mild to moderate pain; B) constant mild to moderate pain; C) mostly pain free with episodes of severe pain; D) constant mild to moderate pain with episodes of severe pain; and E) constant severe pain.[13] Use of analgesics and opioids was also assessed by asking patients whether they were taking such medications on a regular basis. Furthermore, subjects were questioned whether they had experienced any type of pain over the last 4 weeks that interfered with their work or usual activities, or if they had developed disability.

Data on Enrollment

Pre-existing factors such as age, sex, race, smoking status, obesity, and comorbidities were obtained at the time of enrollment. Subjects were classified based on body mass index as normal (<25 kg/m^2), overweight (25–29.9 kg/m^2), or obese (≥30 kg/m^2). Comorbidities were adjusted based on the Charlson Comorbidity Index (CCI).[14] Etiology of AP, history of prior AP attacks, clinical course, management, and hospital outcomes were prospectively recorded during hospitalization. Severity assessment followed the revised Atlanta classification in order to stratify patients as mild, moderately severe, and severe.[15] Data on organ failure was collected, and multisystem organ failure referred to involvement of at least two organs (renal, pulmonary, or cardiovascular).[16] Pancreatic interventions included any endoscopic, radiologic, or surgical pancreatic drainage or debridement procedures. Prolonged hospital length of stay was defined as ≥7 days of hospital admission.

Statistical Analysis

Data was presented as proportions for categorical variables, and median with interquartile range (IQR) for continuous variables. To evaluate the association of pre-existent and pancreatitis-related factors with abdominal pain and disability at follow-up, we performed

univariable analyses using Pearson χ^2 test for categorical variables and Wilcoxon rank-sum test for continuous variables. Statistical significance was defined as $P < 0.05$.

Multivariable linear regression models were used to determine the factors independently associated with abdominal pain at long-term follow-up of AP. A backwards selection technique was used to determine significant independent predictors; variables were removed one by one in order of the magnitude of the P for the type III F-test. Models were then re-run until a final model was selected containing only the variables with $P < 0.05$. Given the small sample size of patients with disability, regression models were not performed for this outcome. Data was analyzed using Stata/IC version 12.1 (StataCorp, College Station, Texas).

RESULTS

Baseline Cohort Characteristics

Among 155 eligible AP patients who were contacted for long-term follow-up assessments, 110 subjects provided complete responses (response rate 71%). Patients responded at a median of 13 months from enrollment (IQR, 11–15). There were no significant differences on baseline characteristics between respondents and non-respondents, except from a higher proportion of White subjects among respondents (95% vs. 82%, $P < 0.05$) (Table 1).

Of respondents, median age was 51 years (IQR, 36–67), 58% were female, 95% were white, and 42% were obese. At least one comorbidity was present in 46% of patients, and 45% were transferred from another institution. The majority of patients had a single attack of AP (64%), and biliary was the most common etiology (47%). Based on the revised Atlanta classification, 62% of patients were classified as mild, 24% as moderately severe, and 14% as having severe AP. With regards to management, 26% of patients required admission to the intensive care unit (ICU), and 18% underwent pancreatic interventions.

Prevalence of Abdominal Pain at Follow-up

Prevalence and characteristics of abdominal pain at follow-up are presented in Figure 1. Abdominal pain within the preceding 4 weeks of follow-up was reported by 24% of patients. Half of the patients (50%) reported pain pattern type A, characterized as mostly pain free with episodes of mild to moderate pain. The rest of patients with abdominal pain had pain pattern type B (15%), type C (12%), type D (19%), and type E (4%). Analgesics were used by 10% of patients, and 6% used opioids on a regular basis.

Baseline Factors Associated With Abdominal Pain at Follow-up

Univariable Analysis—Demographics, comorbidities, and clinical profile of AP patients with and without abdominal pain at follow-up are presented in Table 2. Subjects with abdominal pain at follow-up were more likely to be younger (median age 38 vs 56, $P < 0.01$), current smokers (35% vs. 17%, $P < 0.05$), and obese (54% vs 43%, $P < 0.05$) at baseline, when compared with pain-free patients. Furthermore, those with abdominal pain at follow-up were more likely to have idiopathic etiology (27% vs 8%, $P < 0.05$), recurrent pancreatitis (69% vs. 30%, $P < 0.01$), and pancreatic necrosis (50% vs 29%, $P < 0.05$) at

baseline. Biliary pancreatitis was more common in patients free of pain at long-term follow-up than in patients with abdominal pain (55% vs 23%, $P < 0.05$). No significant differences were identified with regards to race, sex, pre-existing comorbidities, or transfer status, between subjects with and without abdominal pain at follow-up.

Multivariable Analysis—Idiopathic etiology (odds ratio [OR], 3.8; 95% confidence interval [CI], 1.1–13.6), RAP (OR, 2.9; 95% CI, 1.1–7.9), and development of organ failure (OR, 3.3; 95% CI, 1.1–10.6) during hospitalization, were independently associated with abdominal pain at follow-up (Table 3). Age, obesity, smoking history, and pancreatic necrosis, were non-significant on regression analysis, and were excluded from the final model.

Prevalence of Disability at Follow-up and Associated Factors—A total of 41% of subjects reported that pain of any origin interfered with their work or daily activities at long-term follow-up. This was classified as severe in 17% of patients, moderate in 14%, and mild in 10%. Furthermore, 8% of AP patients reported to be on disability at the time of follow-up.

Univariable comparisons of demographics, comorbidities, and clinical profile of AP patients with and without disability at follow-up are shown in Table 4. Patients with disability were younger (median age 45 vs 52; $P < 0.05$), and more likely to be smokers (67% vs 17%, $P < 0.001$) at baseline, compared to those without disability. Admission to ICU during the AP hospitalization was also more common in patients with disability (56% vs 24%, $P < 0.05$).

DISCUSSION

Different long-term structural and functional consequences have been attributed to AP. Increasing interest in patient related outcomes (PROs) has led our group to recently report that AP independently impairs the long-term quality of life of subjects surviving an attack of AP. However, other important PROs such as abdominal pain, opioid use, and disability at long-term follow-up after AP have not been previously evaluated. In this study, we found that 24% of AP patients experience abdominal pain at one year after AP. In addition, regular analgesic use was reported by 10% of patients, regular opioid use in 6%, pain-related interference with work or daily activities in 41%, and disability in 8% at one-year follow-up. Factors independently associated with abdominal pain at follow-up included idiopathic etiology, RAP and development of organ failure during the pancreatitis attack.

In this study, we focused on PROs following recovery of AP.[17] Therefore, the precise mechanisms of abdominal pain were not assessed directly; however, there are several potential explanations. First, a possibility is that a subgroup of these patients progressed to CP during the follow-up period. The average time for progression of AP into morphologic changes of definite CP has been reported to be 3–4 years.[18] Since patients with prior diagnosis of CP or imaging changes consistent with CP were excluded from this study at time of enrollment, progression to CP within one-year of follow-up is overall unlikely. Second, it is possible that abdominal pain could be secondary to early CP; however, there is no clear definition or accurate diagnostic modalities for this diagnosis yet.[19] Third, an attack of AP could result in the development of visceral hypersensitivity, which may lower the

threshold for abdominal pain. A previous experimental study in rats demonstrated that acute pancreatic parenchymal inflammation can cause nociceptive neural alteration, which in turn results in visceral hypersensitivity and development of neuropathic chronic pain.[20] Fourth, patients could perceive fat malabsorption related to EPI as painful. A recent meta-analysis demonstrated that 27% of patients develop EPI following an attack of AP.[5] Finally, other causes of abdominal pain unrelated to pancreatitis could have also contributed to our findings.

The independent factors associated with long-term abdominal pain in our cohort included idiopathic etiology, RAP and development of organ failure during the pancreatitis attack. Interestingly, similar factors have been found to predict CP progression in population studies.[7,21] Subjects with such disease phenotypes may benefit from closer outpatient follow-up after hospital discharge. Using a holistic secondary and tertiary prevention approach that includes detecting and removing implicated etiologies, judicious use of opioids, managing AP complications in a multidisciplinary fashion, selecting patients with idiopathic etiology that may benefit of endoscopic therapy, and treating AP sequelae, might be beneficial to reduce the burden of abdominal pain at long-term follow-up.[22,23]

Disability is the consequence of several painful conditions such as acute low back pain[24], migraine headaches,[25] papillary stenosis,[26] functional gastrointestinal disorders,[27] inflammatory bowel disease,[28] and chronic pancreatitis.[12] In AP, data is limited to one single center prospective study in patients with acute necrotizing pancreatitis, which reported that 53% had disability at a median follow-up of 3 years.[29] The lower rate of disability found in our study (8%) is likely explained by a more heterogeneous cohort of patients with different levels of severity of AP, as well as the shorter follow-up period of 1 year. It is unclear whether the rates of disability would have changed in our cohort with longer follow-up data available. Subgroups of AP patients at higher-risk for disability included younger patients, current smokers, or those admitted to the intensive care unit during the attack. Admission to ICU has been previously associated with higher grades of AP severity and probably represents a surrogate marker of severity in our study, suggesting that more severe disease is associated with higher risk of disability.[30] While we did not find disability to be significantly higher in patients with moderately severe or severe AP, this finding may reflect the small number of subjects with disability in our cohort. Physical therapy and regular exercise programs have demonstrated improvement in physical function after ICU discharge, and might be of benefit in AP patients who require ICU care.[31] Similar to management of disability due to chronic pain, implementation of cognitive behavioral interventions and physical therapy programs could reduce the impact of disability following AP and should be considered in future studies.[32]

Our results should be interpreted in the context of several important limitations. Despite our intense follow up protocol, the response rate was only modest, which may have caused selection bias. Since respondents had similar baseline characteristics to non-respondents, except by higher proportion of white subjects among respondents, our study population is likely representative of the source population. In addition, information and recall bias could have been introduced as the questionnaires assessed symptoms occurring in the 4-week period before follow-up. Misclassification bias from incorrectly categorizing patients with

and without abdominal pain and disability is possible due to the follow-up design using telephone interviews. Since there was no control group or data available on pain or disability prior to the AP attack, we were unable to confirm temporality and to assess causality of AP with any of the long-term outcomes described in the cohort. However, our results reflect the prevalence of important patient related outcomes at one-year of an AP attack, and might help planning outpatient follow-up of patients recovering of AP. Finally, referral bias is possible because all subjects were enrolled at a large tertiary-care center. Therefore, our results may not be generalizable to AP patients treated at community settings.

In conclusion, among patients who survive an attack of AP, 24% experience abdominal pain and 8% have disability at one-year follow-up. Idiopathic etiology, RAP, and development of organ failure during the AP attack were independently associated with presence of abdominal pain at one-year follow-up. Moreover, disability at one-year follow-up was associated with younger age, current smoking, and ICU admission during the AP attack. Our findings suggest that subgroups of AP patients at high-risk for experiencing abdominal pain and developing disability at one-year might benefit from close outpatient follow-up by a multidisciplinary team with expertise in pancreatic disorders. Additional studies are required to validate the association of AP with abdominal pain and disability at long-term follow-up.

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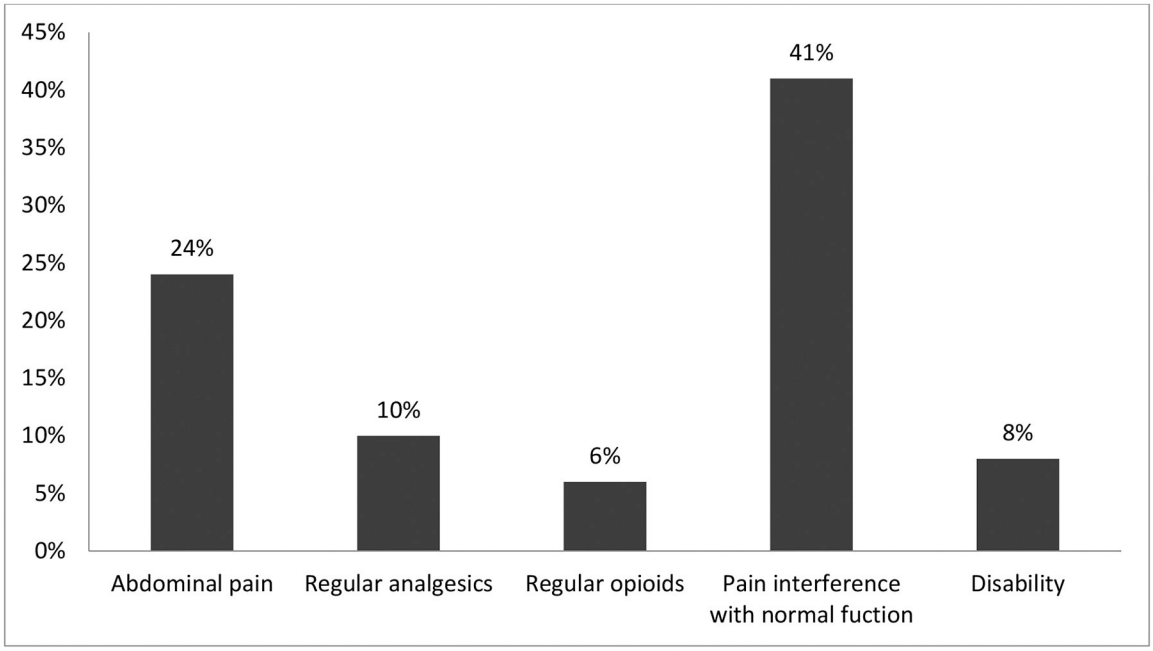

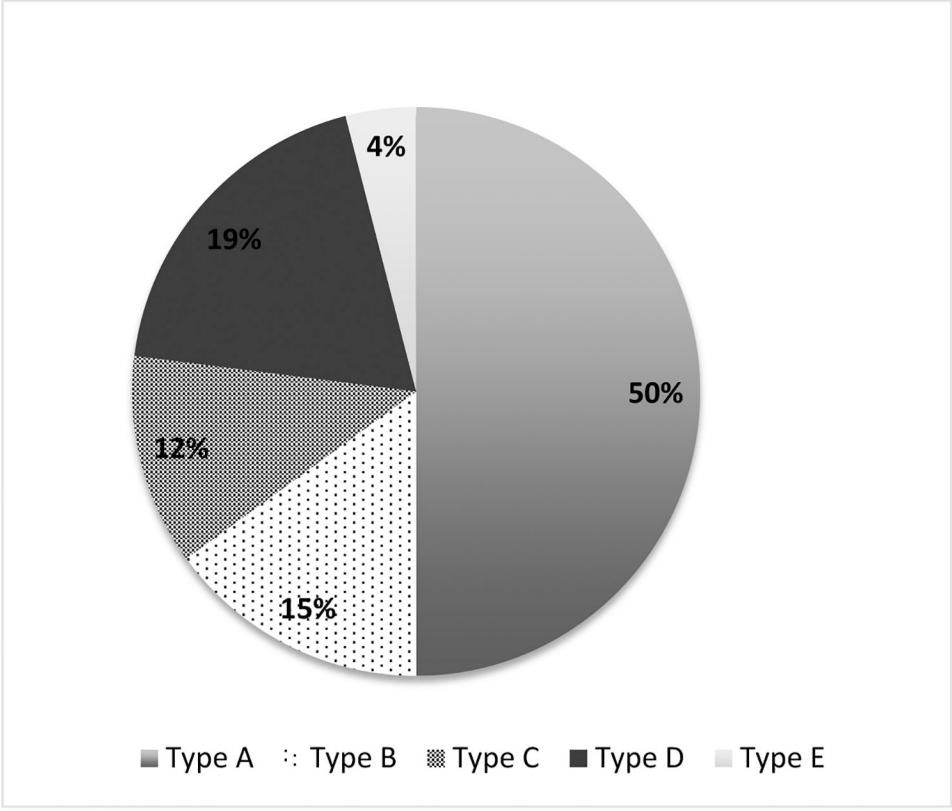

FIGURE 1.

A, Prevalence of abdominal pain, regular analgesic use, pain interfering with normal function, and disability at one-year follow-up of acute pancreatitis. B, Patterns of pain at one-year follow-up of acute pancreatitis. Pain patterns were categorized as type A: mostly

pain free with episodes of mild to moderate pain; type B: constant mild to moderate pain; type C: mostly pain free with episodes of severe pain; type D: constant mild to moderate pain with episodes of severe pain; and type E: constant severe pain.

TABLE 1.

Baseline Demographics, Risk Factors, and Clinical Characteristics Among Respondents and Non-respondents at One-year Follow up

Variables	Respondents (n = 110)	Non-respondents (n = 45)	*P*
Age, median (IQR), y	51 (36–67)	47 (31–60)	0.9
Age groups, y, n (%)			0.36
<45	43 (39.1)	20 (44.4)	
45–60	30 (27.1)	15 (33.3)	
>60	37 (33.6)	10 (22.2)	
Sex, female, n (%)	64 (58.2)	22 (48.9)	0.29
Race, n (%)			0.01
White	104 (94.5)	37 (82.2)	
Black	6 (5.5)	8 (17.8)	
BMI category, n (%)			0.07
Normal/low	29 (26.4)	8 (17.8)	
Overweight	35 (31.8)	9 (20)	
Obese	46 (41.8)	28 (62.2)	
CCI, n (%)			0.8
0	59 (53.6)	24 (53.3)	
1–2	40 (36.4)	15 (33.3)	
≥3	11 (10)	6 (13.3)	
Current smoking, n (%)	23 (20.9)	15 (33.3)	0.13
Transferred, n (%)	50 (45.5)	26 (57.8)	0.16
Etiology, n (%)			0.06
Biliary	52 (47.3)	21 (46.7)	
Alcoholic	5 (4.5)	8 (17.8)	
Idiopathic	14 (12.7)	4 (8.9)	
Other	39 (35.5)	12 (26.7)	
Prior AP attack(s), n (%)	40 (36.4)	20 (44.4)	0.35
Severity by RAC, n (%)			0.4
Mild	68 (61.8)	28 (62.2)	
Moderately severe	27 (24.5)	14 (31.1)	
Severe	15 (13.6)	3 (6.7)	
Multisystem OF, n (%)	14 (12.7)	1 (2.2)	0.45
Pancreatic necrosis, n (%)	33/63 (30)	10/29 (22.2)	0.26
Pancreatic interventions, n (%)	20 (18.2)	6 (13.3)	0.46
ICU admission, n (%)	29 (26.4)	9 (20)	0.4
Length of stay, median (IQR), d	5 (3–8.5)	6 (4–12)	0.12
Prolonged length of stay, n (%)	47 (42.7)	14 (31.1)	0.18

AP indicates acute pancreatitis; IQR, interquartile range; BMI, body mass index; CCI, Charlson comorbidity index; RAC, revised Atlanta classification; OF, organ failure; ICU, intensive care unit.

TABLE 2.

Comparison of Subjects With and Without Abdominal Pain at One-year Follow-up After an Attack of Acute Pancreatitis

Variables	Pain at Follow-up (n = 26)	No Pain at Follow-up (n = 84)	*P*
Age, median (IQR), y	38 (27–52)	56 (39–70)	<0.01
Age groups, y, n (%)			0.07
<45	15 (57.7)	28 (33.3)	
45–60	6 (23.1)	24 (28.6)	
>60	5 (19.2)	32 (38.1)	
Sex, female, n (%)	11 (42.3)	35 (41.7)	0.95
Race, n (%)			
White	25 (96.2)	79 (94.0)	0.68
Black	1 (3.8)	5 (6.0)	
BMI category, n (%)			<0.05
Normal/low	9 (34.6)	16 (19.0)	
Overweight	3 (11.5)	32 (38.1)	
Obese	14 (53.9)	36 (42.9)	
CCI, n (%)			0.74
0	13 (50.0)	46 (54.8)	
1–2	11 (42.3)	29 (34.5)	
≥3	2 (7.7)	9 (10.7)	
Current smoking, n (%)	9 (34.6)	14 (16.7)	<0.05
Transferred, n (%)	13 (50.0)	37 (44.0)	0.59
Etiology, n (%)			<0.05
Biliary	6 (23.1)	46 (54.8)	
Alcoholic	3 (11.5)	2 (2.4)	
Idiopathic	7 (26.9)	7 (8.3)	
Other	10 (38.5)	28 (34.5)	
Prior AP attack(s), n (%)	15 (68.7)	25 (29.8)	<0.01
Severity by RAC, n (%)			0.17
Mild	12 (46.2)	56 (66.7)	
Moderately severe	9 (34.6)	18 (21.4)	
Severe	5 (19.2)	10 (11.9)	
OF, n (%)	7 (26.9)	13 (15.5)	0.18
Multisystem OF, n (%)	5 (19.2)	9 (10.7)	0.25
Pancreatic/peripancreatic necrosis, n (%)	13 (50.0)	24 (28.6)	<0.05
Pancreatic interventions, n (%)	7 (26.9)	14 (16.7)	0.24
ICU admission, n (%)	8 (30.8)	21 (25.0)	0.56
Prolonged length of stay, n (%)	14 (53.8)	33 (39.3)	0.19

AP indicates acute pancreatitis; IQR, interquartile range; BMI, body mass index; CCI, Charlson comorbidity index; RAC, revised Atlanta classification; OF, organ failure; ICU, intensive care unit.

TABLE 3.

Final Regression Model of Factors Associated With Presence of Abdominal Pain at One-year Follow-up After Acute Pancreatitis

Covariate	OR (95% CI)	*P*
Idiopathic etiology	3.8 (1.1–13.6)	0.04
Recurrent episode of AP	2.9 (1.1–7.9)	0.03
Organ failure	3.3 (1.1–10.6)	0.04

OR indicates odds ratio; CI, confidence interval; AP, acute pancreatitis.

TABLE 4.

Comparison of Subjects With and Without Disability at One-year Follow-up After an Attack of Acute Pancreatitis

Variables	Disability at Follow-up (n = 9)	No Disability at Follow-up (n = 101)	*P*
Age, median (IQR), y	45 (36–49)	52 (36–69)	0.15
Age groups, y, n (%)			<0.05
<45	4 (44.4)	39 (38.6)	
45–60	5 (55.6)	25 (24.8)	
>60	0 (0)	37 (36.6)	
Sex, female, n (%)	4 (42.3)	42 (41.7)	0.95
Race, n (%)			0.43
White	8 (88.9)	96 (95.0)	
Black	1 (11.1)	5 (5.0)	
BMI category, n (%)			0.81
Normal/low	3 (33.3)	22 (21.8)	
Overweight	3 (33.3)	32 (31.7)	
Obese	3 (33.3)	47 (46.5)	
CCI, n (%)			0.41
0	3 (33.3)	56 (55.4)	
1–2	5 (55.5)	35 (34.6)	
≥3	1 (11.1)	10 (9.0)	
Current smoking, n (%)	6 (66.7)	17 (16.8)	<0.001
Transferred, n (%)	2 (22.2)	48 (47.5)	0.14
Etiology, n (%)			0.21
Biliary	2 (22.2)	50 (49.5)	
Alcoholic	0 (0)	5 (4.9)	
Idiopathic	1 (11.1)	13 (12.9)	
Other	6 (66.7)	33 (32.7)	
Prior AP attack(s), n (%)	3 (33.3)	37 (36.7)	0.84
Severity by RAC, n (%)			0.51
Mild	4 (44.4)	64 (63.4)	
Moderately severe	3 (33.3)	24 (23.7)	
Severe	2 (22.2)	13 (12.9)	
OF, n (%)	2 (22.2)	18 (17.8)	0.74
Multisystem OF, n (%)	2 (22.2)	12 (11.8)	0.37
Pancreatic/peripancreatic necrosis, n (%)	5 (55.6)	32 (31.7)	0.14
Pancreatic interventions, n (%)	2 (22.2)	19 (18.8)	0.80
ICU admission, n (%)	5 (55.5)	24 (23.7)	<0.05
Prolonged length of stay, n (%)	5 (55.6)	42 (41.6)	0.41

IQR indicates interquartile range; BMI, body mass index; CCI, Charlson comorbidity index; AP, acute pancreatitis; RAC, revised Atlanta classification; OF, organ failure; ICU, intensive care unit.