International Classification of Diseases, Tenth Revision Diagnosis Codes Are Overutilized in the Diagnosis of Chronic Pancreatitis

Michael Ladna, MD, Ishaan Madhok, MD,* Adnan Bhat, MD,* Nicole Ruiz, MD,* Jackson Brown, MD,* Jake Wilson, MD,* Peter Jiang, MD,* Robert Taylor, MD,† Mark Radetic, MD,† John George, MD,† and Christopher Forsmark, MD†*

Background: Retrospective studies and large databases, such as the OneFlorida Clinical Research Consortium, rely on *International Classification of Diseases*, Tenth Revision (ICD-10) diagnosis codes to identify patients with specificity. This study aimed to determine if *ICD-10* codes for CP are overutilized.

Materials and Methods: Retrospective analysis was conducted for patients with *ICD-10* codes K86.0 (alcohol-induced CP) and K86.1 (other CP) from February 2018 to February 2020. Data were extracted from the integrated electronic data repository. This study was approved by the institutional review board. The diagnosis of CP was defined as either being made by a gastroenterologist, proven by biopsy, or having characteristic findings on cross-sectional imaging with appropriate symptoms.

Results: Five hundred four (37%) out of the 1360 patients had no evidence of CP. When broken down by diagnosis code, 41 of 176 charts (23.3%) with K86.0 and 461 of 1184 charts (38.6%) with K86.1 had no evidence of CP. Two hundred ninety-nine of these patients had either a single episode of acute pancreatitis, recurrent acute pancreatitis, or episode of acute necrotizing pancreatitis. Of note, 81 patients had no identifiable abdominal pathology.

Conclusions: Although the OneFlorida database makes multicenter research more accessible, it does not replace labor-intensive chart review given the propensity for overdiagnosis.

Key Words: chronic pancreatitis, *ICD-10* diagnosis code, false positive, misdiagnosis, exocrine pancreatic insufficiency

Abbreviations: AP - acute pancreatitis, CP - chronic pancreatitis, EPI - exocrine pancreatic insufficiency, EUS - endoscopic ultrasonography, GI - gastrointestinal, IBD - inflammatory bowel disease, IBS - irritable bowel syndrome, *ICD-10 - International Classification of Diseases, Tenth Revision*, IRB - institutional review board, PDAC - pancreatic ductal adenocarcinoma, PUD - peptic ulcer disease, RAP - recurrent acute pancreatitis

(*Pancreas* 2025;54: e97–e100)

Large databases, such as the OneFlorida Clinical Research Consortium,[1] rely on *International Classification of Diseases, Tenth Revision* (*ICD-10*) diagnosis codes to identify patients with specificity. Patients enrolled in retrospective research studies are often collected via the use of *ICD-10* diagnosis codes. These codes can be subject to error due to overdiagnosing, especially in diseases that are difficult to diagnose, such as chronic pancreatitis (CP).

Symptoms of CP can be nonspecific and mimic many other gastrointestinal (GI) disorders such as inflammatory bowel disease (IBD), irritable bowel syndrome (IBS), peptic ulcer disease (PUD), and biliary colic. Abdominal pain is an exceedingly common symptom among the general population with 10,300 (41.3%) out of 24,929 study participants reporting abdominal pain via the GI Patient-Reported Outcomes Measurement Information System questionnaire.[2] CP, on the other hand, is not a common diagnosis, with a prevalence of 25–98 per 100,000[3] and an incidence of 4–5 new cases per 100,000 per year.[4] The most common symptom of CP is recurrent or chronic abdominal pain, which is seen in approximately 80% of patients.[5] This still leaves approximately 20% of CP patients who can present with atypical symptoms.

Radiographic findings are typically subtle or not present in early CP. There is the additional difficulty of interpreting radiographic findings in the healthy elderly pancreas, which has been shown to mimic anatomic changes of CP. Autopsies of the healthy elderly pancreas have revealed the presence of ductal proliferation, lobular degeneration, cavitation, fatty replacement, and calcium carbonate calculi.[6,7] Further autopsies of the healthy pancreas also revealed main pancreatic duct dilatation, cystic lesions, and calcifications in 0.49%, 0.21%, and 0.05% of patients, respectively. All these abnormalities had age-dependent increases in incidence in both sexes, except for calcifications, which had age-dependent increases in men only. Eighty-four percent of main pancreatic duct dilatation, 87% of cystic lesions, and 50% of calcifications could not be explained by pathology and were attributed as being age-related.[8] When postmortem pancreatography was compared with histology in pancreatic autopsy specimens, 81% of the pancreatograms had characteristics consistent with CP despite no histologic evidence of CP in any of the patients. This high false-positive rate was likely due to ductular changes in advancing age, which included perilobular and intralobular fibrosis and hyperplasia of ductal epithelium.[9]

Considering the difficulties in establishing a diagnosis of CP and the propensity for false positives, it is likely that CP is being overdiagnosed, and *ICD-10* diagnosis codes for CP are being overutilized. This study aimed to determine if *ICD-10* diagnosis codes for CP were overutilized and to recognize alternative pathologies that likely contributed to this occurrence.

METHODS AND MATERIALS

Study Design

This study was approved by the institutional review board. A retrospective analysis was conducted of all patients with *ICD-10* codes K86.0 (alcohol-induced CP) and K86.1 (other CP) who were seen in either an outpatient or inpatient setting (whether the encounter was associated with the primary diagnosis of CP

From the *Department of Internal Medicine and †Division of Gastroenterology, Hepatology, and Nutrition, Department of Medicine, University of Florida, Gainesville, FL.

Received for publication March 14, 2024; accepted July 3, 2024.

Address correspondence to: Michael Ladna, MD, Division of Hospital Medicine, University of Florida, 1600 SW Archer Rd, Gainesville, FL 32608 (e-mail: Michael.ladna@gmail.com).

There was no funding involvement in this research

C.F. is a consultant for Abbvie. The other authors declare no conflict of interest.

Author Contributions: M.L.: data collection, project formulation, statistical analysis, writing manuscript, final approval. I.M., N.R., A.B., J.B., J.S., J.W., M.R.: data collection. R.T.: data collection, project formulation, writing manuscript. J.G.: editing of manuscript. C.F.: principal investigator, project formulation, editing of the manuscript.

Supplemental digital contents are available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.pancreasjournal.com).

DOI: 10.1097/MPA.0000000000002399

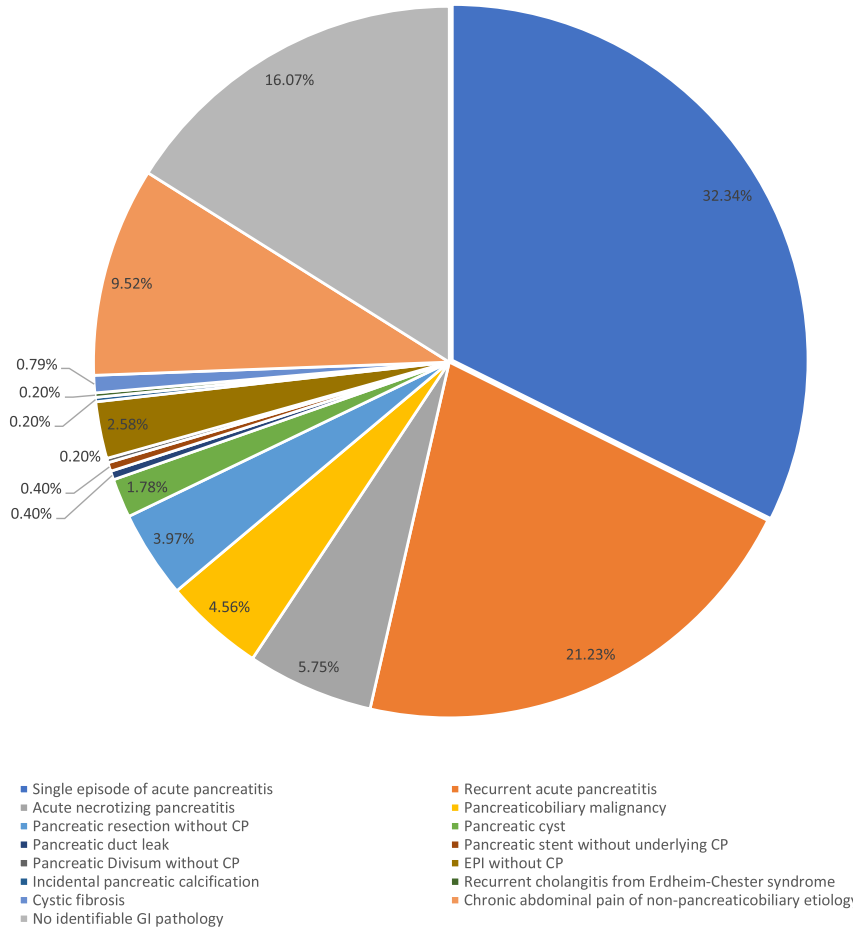

FIGURE 1. Pie graph showing alternative diagnoses, which may have led to *ICD-10* diagnosis code for CP being documented.

or an associated secondary diagnosis of CP) at the University of Florida Health Shands Hospital, a tertiary care center and a pancreatic center of excellence from February 2018 to February 2020. The data were extracted from the institution's integrated electronic data repository.

Study Outcome Measures

The primary aim of this study was to evaluate the mislabeling of patients without any evidence of CP with an *ICD-10* diagnosis code for CP. The secondary aim was to evaluate for pathologies other than CP that likely contributed to the misdiagnosis and mislabeling as CP.

Definitions

The diagnosis of CP was defined as either being made by a gastroenterologist at our institution, proven on histopathology via a biopsy, or having characteristic findings of CP on cross-sectional imaging with appropriate symptoms. For cross-sectional imaging computed tomography, magnetic resonance cholangiopancreatography, and endoscopic ultrasound (EUS) were all deemed appropriate. For computed tomography, the characteristic imaging findings included pancreatic calcifications and pancreatic ductal dilatation. For magnetic resonance cholangiopancreatography, the characteristic imaging findings included reduced T1 signal intensity, main pancreatic duct dilatation or irregularity, dilation of side branches, and the presence of at least 1 stricture. There are 4 parenchymal and 5 ductal criteria for CP diagnosis using EUS (refer to Table 3 in the Appendix, http://links.lww.com/MPA/B241). Those patients who did not meet the above definitions for the diagnosis of CP were further investigated via chart review of the electronic medical record to determine the likely pathology that may have resulted in a mistaken *ICD-10* label for CP.

Statistical Analysis

All variable and outcome distributions are summarized as percentages for categorical variables, and means with SDs for all continuous variables. SPSS (IBM Corp, Armonk, NY) was used for the calculation of descriptive statistics.

RESULTS

A total of 1360 unique patient charts were reviewed, of which 176 carried *ICD-10* code K86.0 (alcohol-induced CP) and 1184

TABLE 1. Baseline Characteristics of All Patients With *ICD-10* Diagnosis Code for CP Divided Into Those With and Without CP

Characteristics	*ICD-10* Code for CP and CP (n = 856)	*ICD-10* Code for CP and No CP (n = 504)
Age, mean ± SD, y	57.49 ± 13.99	54.66 ± 15.68
Sex (male), n (%)	474 (55.4)	251 (49.8)

TABLE 2. Alternative Diagnoses That Likely Resulted in *ICD-10* Diagnosis Code for CP Being Linked to the Patient's Chart

Alternative Diagnosis	K86.0 Alcohol-Induced CP (n = 41)	K86.1 Other CP (n = 463)	Total (n = 504)
Single episode of AP	24 (58.54)	139 (30.02)	163 (32.34)
RAP	5 (12.19)	102 (22.03)	107 (21.23)
Acute necrotizing pancreatitis	2 (4.88)	27 (5.83)	29 (5.75)
Pancreaticobiliary malignancy*	1 (2.44)	22 (4.75)	23 (4.56)
Pancreatic resection without CP	0	20 (4.32)	20 (3.97)
Pancreatic cyst	0	9 (1.94)	9 (1.78)
Pancreatic duct leak	0	2 (0.43)	2 (0.4)
Pancreatic stent without underlying CP	0	2 (0.43)	2 (0.4)
Pancreatic divisum without CP	0	1 (0.22)	1 (0.2)
EPI without CP	0	13 (2.81)	13 (2.58)
Incidental pancreatic calcification on cross sectional imaging	0	1 (0.22)	1 (0.2)
Recurrent cholangitis from Erdheim-Chester syndrome	0	1 (0.22)	1 (0.2)
Cystic fibrosis	1 (2.44)	3 (0.65)	4 (0.79)
Chronic abdominal pain of nonpancreaticobiliary etiology†	0	48 (10.37)	48 (9.52)
No identifiable GI pathology	8 (19.51)	73 (15.77)	81 (16.07)
Total	41	463	504

Values are presented as n (%).

*Pancreatic adenocarcinoma or cholangiocarcinoma.

†IBS, IBD, gastroparesis, PUD, functional abdominal pain.

carried *ICD-10* code K86.1 (other CP). A total of 504 (37%) out of the 1360 patient charts did not have any evidence of CP on chart review. The mean age of patients with an appropriate *ICD-10* code for CP was 57.49 ± 13.99 years, whereas for the group with an inappropriate *ICD-10* code for CP, the mean age was 54.66 ± 15.68 (Table 1). When broken down by diagnosis code, 41 of 176 charts (23.3%) with K86.0 and 461 of 1184 charts (38.6%) with K86.1 did not have any evidence of CP (Table 2). Most of the patients with inappropriate *ICD-10* codes for CP had another pancreatic pathology of some form (Table 2). Of these patients, 163 had a single episode of acute pancreatitis (AP) of any etiology, 107 had recurrent AP (RAP), 29 had at least one episode of acute necrotizing pancreatitis, 22 had pancreaticobiliary cancer (pancreatic or cholangiocarcinoma), 19 had pancreatic resection without underlying CP, 48 had chronic abdominal pain of nonpancreaticobiliary etiology, and 81 had no identifiable abdominal pathology per chart review (Fig. 1).

DISCUSSION

We found that even at a tertiary care center and pancreatic center of excellence, many patients with no evidence of CP were labeled at some point during their care with an *ICD-10* diagnosis code for CP. This difficulty in pinpointing a diagnosis of CP reflects the difficulty of diagnosing a disease with nonspecific symptoms and little pathognomonic radiographic evidence especially early in the disease course. Mislabeling these patients with CP may lead to unnecessary testing and treatment, which raises health care costs and exposes patients to a higher risk of harm that comes with additional testing. It could also lead to a delay in the appropriate diagnosis, which could result in significant harm, such as if a malignancy is missed. The majority of these patients had an alternative pancreatic pathology with a total of 299 (59.3%) having either a single episode, recurrent episodes, or at least 1 episode of acute necrotizing pancreatitis. Although a non-insignificant proportion of patients with AP (whether single episode, recurrent, or necrotizing) go on to develop CP, the majority do not. One study that looked at 669 patients from December 2003 until March 2007 found that 17% of patients go on to develop recurrent pancreatitis after the first episode of AP, whereas only 7.6% of patients go on to develop CP after the first episode of AP.[10] When 532 patients were followed up for an average of 7.8 years after the initial AP episode, CP developed only in patients with continued alcohol use with a cumulative incidence of 13% at 10 years and 16% at 20 years.[11] The rate of developing CP after RAP is much higher than a single episode with 38% and 42% of RAP patients going on to develop CP at 2- and 10-year follow-ups, respectively.[11] This risk is not evenly distributed and is strongly associated with the etiology of AP and lifestyle habits afterward. One study found that the progression to CP from alcoholic pancreatitis, biliary pancreatitis, idiopathic pancreatitis, and pancreatitis of other etiologies was 26% ($P < 0.01$), 1.7%, 13%, and 6.7%, respectively. This study also showed that patients who continued to drink at the same level and those who stopped had significant disparity in progression to CP of 41% versus 13%, respectively.[12] Thus, the majority of patients with AP and RAP do not develop CP, and this risk is greatly diminished if the etiology of AP was not alcoholic and if patients quit alcohol use.

A significant number of patients had no evidence of a pancreaticobiliary pathology, with 48 (9.5%) having chronic abdominal pain of nonpancreaticobiliary pathologies such as IBS or IBD and 81 (16%) having no evidence of any GI pathology whatsoever. Functional abdominal pain can be a distressing entity for both patients and physicians. Patients are left with a sense of frustration and often exert significant pressure on their health care providers to provide a diagnosis of some sort to move on from the diagnostic to the therapeutic course. In addition, patients with functional abdominal pain and IBS have higher rates of depression and anxiety, which could be exacerbated via a delay in diagnosis or misdiagnosis.[13]

Another reason for the overdiagnosis of CP is that many health care providers associate exocrine pancreatic insufficiency (EPI) with CP, especially if cystic fibrosis is not present. Providers may be tempted to label the patient as having CP if there is evidence of EPI. The presence of EPI does not always indicate an

underlying diagnosis of CP. For instance, damage from necrotizing pancreatitis can be severe enough to result in EPI without other features characteristic of CP.[14] Development of diabetes mellitus (DM) has also been closely associated with CP; however, recent data show that even a single episode of AP is closely associated with new DM. In patients with moderate and severe AP, diabetes developed in 10.7% and 16.1% of these patients, respectively.[12] The rate of DM development differed depending on the etiology of AP, with 20.6% of alcoholic AP ($P < 0.01$), 9.1% of biliary AP, 9.9% of idiopathic AP, and 5.3% of all other AP going on to develop DM. [12] The severity of AP and the presence of necrosis were also associated with a statistically significant increase in development in DM. A meta-analysis reported that 39% versus 14% of patients with severe versus mild AP went on to develop DM, respectively, and 28% and 12% of patients with necrosis and without necrosis developed DM, respectively.[15] Thus, the development of EPI or DM after even a single episode of AP is not uncommon and does not necessarily indicate a progression to CP.

Even gastroenterologists can find diagnosing CP challenging, which is exemplified in that the total number of EUS criteria required for the diagnosis of CP is not well established due to high variability among observers.[16] EUS can also yield high false-positive rates, especially in patients with abdominal pain from dyspepsia,[17,18] elderly patients,[19,20] patients with a history of smoking or alcohol abuse,[21,22] obese patients,[23] and patients with DM.[24] A study of EUS done on 120 patients aged 40–61 years without clinical evidence of CP found that at least 1 parenchymal and/or ductular abnormality was identified in 28% of patients with a trend of increasing abnormalities with age: <41 years (23%), 40–60 years (25%), and >60 years (39%).[20]

Data extracted from the OneFlorida Clinical Research Consortium database[1] for CP could be especially vulnerable to this overdiagnosis. Although the OneFlorida Clinical Research Consortium[1] makes multicenter research more accessible, it does not replace labor-intensive chart review when conducting studies on diseases that are difficult to diagnose such as CP. Limitations of this study are those inherent to any retrospective studies including incomplete medical documentation and data. This study did not assess the proportion of patients who had the *ICD-10* diagnosis code for CP removed on follow-up encounters. There will also always be confounding variables even if matching and regression analyses are used. Future research initiatives should focus on the education of health care providers and a more streamlined diagnosis algorithm for CP such as a standardized screening tool that can be applied to patients at high risk for CP such as those with a single or recurrent alcoholic AP who continue to consume alcohol.

REFERENCES

1. OneFlorida+ – Clinical Research Network. Accessed May 23, 2024. https://onefloridaconsortium.org/
2. Lakhoo K, Almario CV, Khalil C, et al. Prevalence and characteristics of abdominal pain in the United States. *Clin Gastroenterol Hepatol*. 2021;19:1864–1872.e5.
3. Machicado JD, Dudekula A, Tang G, et al. Period prevalence of chronic pancreatitis diagnosis from 2001–2013 in the commercially insured population of the United States. *Pancreatology*. 2019;19:813–818.
4. Yadav D, Timmons L, Benson JT, et al. Incidence, prevalence, and survival of chronic pancreatitis: a population-based study. *Am J Gastroenterol*. 2011;106:2192–2199.
5. Singh VK, Yadav D, Garg PK. Diagnosis and management of chronic pancreatitis: a review. *JAMA*. 2019;322:2422–2434.
6. Andrew W. Senile changes in the pancreas of Wistar Institute rats and of man with special regard to the similarity of locule and cavity formation. *Am J Anat*. 1944;74:97–127.
7. Pancreatic lithiasis in the aged. Its clinicopathology and pathogenesis. Accessed January 27, 2023. https://pubmed.ncbi.nlm.nih.gov/6690361/
8. Ikeda M, Sato T, Morozumi A, et al. Morphologic changes in the pancreas detected by screening ultrasonography in a mass survey, with special reference to main duct dilatation, cyst formation, and calcification. *Pancreas*. 1994;9:508–512.
9. Schmitz-Moormann P, Himmelmann GW, Brandes JW, et al. Comparative radiological and morphological study of human pancreas. Pancreatitis like changes in postmortem ductograms and their morphological pattern. Possible implication for ERCP. *Gut*. 1985;26:406–414.
10. Ahmed Ali U, Issa Y, Hagenaars JC, et al. Risk of recurrent pancreatitis and progression to chronic pancreatitis after a first episode of acute pancreatitis. *Clin Gastroenterol Hepatol*. 2016;14:738–746.
11. Lankisch PG, Breuer N, Bruns A, et al. Natural history of acute pancreatitis: a long-term population-based study. *Am J Gastroenterol*. 2009;104:2797–2805.
12. Takeyama Y. Long-term prognosis of acute pancreatitis in Japan. *Clin Gastroenterol Hepatol*. 2009;7:S15–S17.
13. Fond G, Loundou A, Hamdani N, et al. Anxiety and depression comorbidities in irritable bowel syndrome (IBS): a systematic review and meta-analysis. *Eur Arch Psychiatry Clin Neurosci*. 2014;264:651–660.
14. Beyer G, Habtezion A, Werner J, et al. Chronic pancreatitis. *Lancet*. 2020;396:499–512.
15. Zhi M, Zhu X, Lugea A, et al. Incidence of new onset diabetes mellitus secondary to acute pancreatitis: a systematic review and meta-analysis. *Front Physiol*. 2019;10:637.
16. Gardner TB, Gordon SR. Interobserver agreement for pancreatic endoscopic ultrasonography determined by same day back-to-back examinations. *J Clin Gastroenterol*. 2011;45:542–545.
17. Sahai AV, Mishra G, Penman ID, et al. EUS to detect evidence of pancreatic disease in patients with persistent or nonspecific dyspepsia. *Gastrointest Endosc*. 2000;52:153–159.
18. Lariño-Noia J, de la Iglesia D, Iglesias-García J, et al. Morphological and functional changes of chronic pancreatitis in patients with dyspepsia: a prospective, observational, cross-sectional study. *Pancreatology*. 2018;18:280–285.
19. Bhutani MS, Arantes VN, Verma D, et al. Histopathologic correlation of endoscopic ultrasound findings of chronic pancreatitis in human autopsies. *Pancreas*. 2009;38:820–824.
20. Rajan E, Clain JE, Levy MJ, et al. Age-related changes in the pancreas identified by EUS: a prospective evaluation. *Gastrointest Endosc*. 2005;61:401–406.
21. Yusoff IF, Sahai AV. A prospective, quantitative assessment of the effect of ethanol and other variables on the endosonographic appearance of the pancreas. *Clin Gastroenterol Hepatol*. 2004;2:405–409.
22. Petrone MC, Arcidiacono PG, Perri F, et al. Chronic pancreatitis–like changes detected by endoscopic ultrasound in subjects without signs of pancreatic disease: do these indicate age-related changes, effects of xenobiotics, or early chronic pancreatitis? *Pancreatology*. 2010;10:597–602.
23. Al-Haddad M, Khashab M, Zyromski N, et al. Risk factors for hyperechogenic pancreas on endoscopic ultrasound: a case-control study. *Pancreas*. 2009;38:672–675.
24. Bolado F, Prieto C, Vila JJ, et al. Chronic pancreatitis–like changes detected by endoscopic ultrasound in type 1 diabetics are not associated with gastrointestinal symptoms or nutritional deficiencies. *Pancreas*. 2017;46:102–105.