AAVBC

AMERICAN ACADEMY OF VALUE BASED CARE

Chronic Pancreatitis

Quick Reference Guide

2026

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1 CLINICAL SNAPSHOT

Definition: Chronic pancreatitis (**CP**) is a progressive inflammatory disorder characterized by permanent structural damage, functional loss, and fibrosis of the pancreas, leading to exocrine insufficiency and/or endocrine dysfunction (diabetes mellitus).[1,2] In adults, alcohol-induced CP (**K86.0**) and idiopathic CP (**K86.1**) together account for **>80% of cases** in developed countries.[1,2] Diagnosis requires imaging evidence (CT, MRI/MRCP, endoscopic ultrasound) of **pancreatic atrophy**, **ductal dilation**, or **calcifications** in the clinical context of chronic abdominal pain and/or pancreatic insufficiency.[1,3] Unlike acute pancreatitis, CP is **irreversible** and requires multifaceted management: pancreatic enzyme replacement therapy (**PERT**), diabetes management, pain control, and nutritional support; particular diagnostic vigilance is required in older adults for silent disease and sarcopenia.

ICD-10 Codes

K86.0 (alcohol-induced chronic pancreatitis) and **K86.1** (other chronic pancreatitis, including idiopathic) are the primary codes: both **map to HCC 79** under CMS-HCC V28.[4,5] **K86.81** (exocrine pancreatic insufficiency; **EPI**) must be coded when documented, as it is a key comorbidity affecting **nutritional status**.[2] **F10.xx** (alcohol use disorder) codes must be sequenced alongside K86.0 **at every encounter** when alcoholic etiology applies: AUD is the root cause and must be documented and managed.[6] **E13.xx** (type 3c diabetes mellitus, pancreatogenic) should replace or supplement **E11.xx** when diabetes is clearly secondary to pancreatic destruction; **E13.xx** maps to **HCC 37** or **38** depending on complexity.[5] **K86.2** (pancreatic cyst), **K86.3** (pancreatic pseudocyst), **K86.89** (other specified pancreatic disorders) are coded when documented clinically or radiologically. **Do NOT use Z87.19** ('history of pancreatitis') when disease is active: this suppresses HCC mapping and **flattens risk documentation**.

Prevalence and Burden: The annual incidence of chronic pancreatitis (CP) in developed countries is approximately 4–10 per 100,000 population, with prevalence estimates ranging from 10–15 per 100,000 and higher rates observed in regions with elevated alcohol consumption.[7] Alcohol-induced CP typically develops **10–20 years after** sustained heavy alcohol use (>80 g/day for men, >60 g/day for women) while idiopathic CP often presents later in life with similar disease progression.[2] In Medicare populations ≥65 years, disease burden and complications increase substantially: ~40–60% develop EPI;[2] ~70–80% develop **endocrine dysfunction** with brittle diabetes and increased **hypoglycemia risk**,[2,8] and 25–50% demonstrate **osteoporosis** by bone density testing.[9] Vitamin malabsorption, weight loss, malnutrition, and sarcopenia are common and contribute to frailty, falls, and **functional decline** in older adult patients.[7,9] **Only ~50–60%** of patients receive **optimized PERT dosing**,[10] while inadequate pain control, undertreatment of diabetes, and delayed hepatocellular carcinoma screening remain important drivers of preventable complications, hospital utilization, and high-cost readmissions.[9]

HCC V28/RAF Mapping

ICD-10 CODES[4]	HCC CATEGORY (V28)[5]	RAF WEIGHT (CNA)[5]	DOCUMENTATION REQUIREMENT
K86.0 (alcohol-induced chronic pancreatitis)	HCC 79	0.357	Document pancreatitis as chronic with imaging (CT/MRI/EUS) evidence of atrophy, ductal changes, or calcifications; specify **alcohol use as etiology** with frequency and quantity documented, coding F10.xx alongside K86.0 at every visit;[1] distinguish from acute pancreatitis; confirm >6 weeks symptom duration
K86.1 (Other chronic pancreatitis, including idiopathic)	HCC 79	0.357	Imaging findings (atrophy, ductal dilation, calcifications, fat replacement). Document >[6] weeks duration and excluded etiologies (alcohol, CF, hyperparathyroidism)
K86.81 (Exocrine pancreatic insufficiency/EPI)	—	—	No HCC mapping but critical for justifying the complexity of care; Fecal elastase **<200 mcg/g** or **clinical PERT response**;[1] note steatorrhea, weight loss, or nutritional deficiency; reassess annually
K86.89 (Other specified disease of pancreas)	Varies	Varies	Clearly state specific pancreatic condition (**pseudocysts**, **steatorrhea**, **bile duct stenosis**) and link it to supporting diagnostic test results, clinical observations, and the treatment plan
F10.xx (Alcohol use; uncomplicated) with K86.0	Sequenced with K86.0; HCC 139	0.329	Code F10.20/F10.21 (**moderate AUD**) or F10.10/F10.11 (**mild AUD**) based on severity; alcohol use must be documented at every encounter alongside K86.0, not just at initial diagnosis; **document AUDIT-C** or quantity/frequency; active use, remission status, and any AUD pharmacotherapy (naltrexone, acamprosate, baclofen)[1]
F17.210 (nicotine dependence, cigarettes, uncomplicated)	None	—	Document nicotine product is **cigarettes** (not chewing tobacco, cigars, vapes), patient **usage patterns** (frequency and duration), and confirm **absence of complications** (not in withdrawal, remission, nicotine-induced disorder)
Z87.19 (History of pancreatitis) [AVOID during active disease]	Non-HCC — flat risk	No HCC capture	DO NOT use when patient has active disease (chronic abdominal pain, elevated pancreatic enzymes, ongoing PERT, recent imaging findings); **this is the single most common CP coding error**;[2] acceptable only after sustained remission with **documented resolution of CP symptoms**

ICD-10 CODES[4]	HCC CATEGORY (V28)[5]	RAF WEIGHT (CNA)[5]	DOCUMENTATION REQUIREMENT

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; CP = chronic pancreatitis; CMS = Centers for Medicare & Medicaid Services; CNA = community non-dual aged; EPI = exocrine pancreatic insufficiency; EUS = endoscopic ultrasound; F10.xx = alcohol use disorder codes; HCC = hierarchical condition category; ICD-10 = International Classification of Diseases, 10th Edition; K86.x = pancreatic disease codes; MEAT = monitor/evaluate/assess/treat; PERT = pancreatic enzyme replacement therapy; RAF = risk adjustment factor; T3cDM = type 3c diabetes mellitus (pancreatogenic); V28 = CMS-HCC model version 28

RAF values represent the Community Non-Dual Eligible Aged (CNA) coefficient from the 2024 CMS-HCC model; values vary across patient populations based on eligibility and care setting

Risk-Adjusted Care Resources per Patient/Year[4,11]

Risk-adjusted care resource allocation — MA base rate ($10,402.34) × RAF coefficient

Non-alcohol induced pancreatitis	Alcohol-induced pancreatitis
~$3.7K	**~$6.2K**
HCC 79 · RAF 0.357	HCC 79 + HCC36

RAF values represent the Community Non-Dual Eligible Aged (CNA) coefficient from the 2024 CMS-HCC model; values vary across patient populations based on eligibility and care setting

AAVBC PERSPECTIVE

***From the AAVBC's perspective**, value-based care in pancreatitis should focus on preserving nutritional status, functional independence, and quality of life through **clinically individualized care** that reflects the needs of older and medically complex patients. In chronic pancreatitis, this includes **early investigation** of subtle signs of disease progression such as weight loss, steatorrhea, brittle glycemic control, recurrent abdominal symptoms, and functional decline to support earlier diagnosis and intervention. **AAVBC emphasizes** that care delivery in older adults should incorporate frailty, biological age, comorbidities, and caregiver support alongside disease severity when guiding nutrition and longitudinal management. Accurate documentation should capture the full clinical complexity: imaging abnormalities such as pancreatic calcifications or ductal changes must be **explicitly linked** to documented chronic pancreatitis diagnoses, as radiographic findings alone **do not** constitute a coded diagnosis. In geriatric populations, restrictive low-fat dietary recommendations may **worsen malnutrition and sarcopenia**; care pathways should instead prioritize high-calorie, high-protein nutrition with pancreatic enzyme replacement therapy when indicated. Integrated, **closed-loop care pathways** across gastroenterology, nutrition, endocrinology, and primary care can support earlier intervention, reduce downstream hospital utilization, and improve long-term outcomes.*

2 RECOGNITION AND DIAGNOSIS

Screenings For Suspected CP Covered Under Medicare Part B

TEST	FREQUENCY	CPT CODE	CLINICAL INDICATION
Serum lipase/ amylase	**At diagnosis**; then per clinical context (**yearly** in stable CP)	82656 (lipase), 82150 (amylase)	Lipase is more sensitive/specific than amylase for pancreatic disease;[2] lipase may be mildly elevated or normal in CP; rising lipase in a CP patient may signal **acute exacerbation** or complications[1]
Fecal elastase-1	**At diagnosis**; repeat **annually** or if malnutrition worsens	82656 (or specialty lab: fecal elastase)	**Gold standard for EPI screening**; elastase **<200 mcg/g confirms EPI** and justifies PERT initiation;[1,3] Values 200–500 suggest mild insufficiency (>500 normal); levels guide PERT dose and nutritional intervention[1,2]
Pancreatic imaging: CT	**At diagnosis**; every **2–3 years** if stable; sooner if complications	71260 (CT abdomen/ pelvis with IV contrast)	Detects ductal dilation, atrophy, parenchymal calcifications, cysts, pseudocysts; CT is widely available and sensitive for advanced CP; MRI/EUS provide **superior ductal detail and tissue characterization**[1–3]
MRI/MRCP (preferred for detailed ductal assessment)	**At diagnosis** or when **CT findings unclear**; assess for ductal obstruction	74181 (MRCP)	Excellent soft-tissue contrast + no radiation; detects ductal strictures, sludge, stones, biliary involvement, and early fibrosis; may reveal **ductal stones** or **protein plugs** suitable for **ERCP intervention**[2,3]
AUDIT-C	**Annually** with AUD history or at every E/M with CP diagnosis	96160 (standardized screening instrument)	Alcoholic screening if **cause of CP unclear**; AUDIT-C **≥4** (men)/**≥3** (women) indicates harmful use; positive screen triggers brief intervention and AUD pharmacotherapy offer (**naltrexone preferred**; avoid disulfiram in older adults)

TEST	FREQUENCY	CPT CODE	CLINICAL INDICATION
Vitamin D/ bone density screening (DEXA)	**At diagnosis**; repeat every **2 years** if on PERT	76070 (DEXA), 82306 (Vitamin D, 25-OH)	High prevalence of **osteoporosis** (25–50%)[9] and **Vitamin D deficiency** in CP due to fat malabsorption;[10] early identification guides calcium/Vitamin D supplementation and fracture prevention

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; CP = chronic pancreatitis; CMS = Centers for Medicare & Medicaid Services; CPT = Current Procedural Terminology; CT = computed tomography; DEXA = dual-energy x-ray absorptiometry; E/M = evaluation and management; EPI = exocrine pancreatic insufficiency; ERCP = endoscopic retrograde cholangiopancreatography; EUS = endoscopic ultrasound; HEDIS = Healthcare Effectiveness Data and Information Set; IV = intravenous; MRCP = magnetic resonance cholangiopancreatography; MRI = magnetic resonance imaging; PERT = pancreatic enzyme replacement therapy

Subtle Early Signs in Older Adults (>65)

SIGN/SYMPTOM	CLINICAL SIGNIFICANCE
Abdominal pain (epigastric to left upper quadrant, often radiating to back)	Hallmark symptom but **absent in ~30%** of older patients = silent CP;[1,2] pain is typically continuous or recurring, worse with fatty foods: severity ranges mild to severe; **opioid-sparing strategies** (pregabalin, duloxetine, celiac plexus block) preferred[10]
Steatorrhea (fatty, foul-smelling stools); weight loss; malnutrition	Indicates exocrine insufficiency (**EPI**); weight loss is a powerful predictor of frailty in older adults: monitor BMI, prealbumin, albumin → nutritional assessment and high-protein supplementation[2]
New-onset diabetes mellitus or brittle/labile glucose control	Pancreatogenic (T3cDM) diabetes requires **insulin more often** and earlier than T2DM; **hypoglycemia risk** elevated; distinguish from pre-existing T2DM by imaging and elastase findings; HbA1c may not reflect **true glycemic control** if glucose swings are rapid[1,8]
Painless jaundice; pale stools; dark urine	Suggests **ductal obstruction** or extrahepatic **cholestasis** from pancreatic head enlargement/stricture; may indicate biliary stricture, cyst, or pseudocyst compressing common bile duct;[1,2] imaging (**MRCP**) indicated to assess for ERCP candidacy
Nausea, vomiting, early satiety	May signal pancreatic exocrine secretion insufficiency, gastric outlet obstruction (from duodenal encroachment), or comorbid gastroparesis; assess medication list for agents that worsen motility[1,3]
Unexplained anemia; fatigue; bone pain	Vitamin B12/fat-soluble vitamin **malabsorption in EPI**; anemia elevates cardiac stress in older adults; bone pain signals osteoporosis/ osteopenia from Vitamin D malabsorption and chronic inflammation[10]

ABBREVIATIONS: CP = chronic pancreatitis; DEXA = dual-energy x-ray absorptiometry; EPI = exocrine pancreatic insufficiency; ERCP = endoscopic retrograde cholangiopancreatography; HbA1c = hemoglobin A1c; MRCP = magnetic resonance cholangiopancreatography; T2DM = type 2 diabetes mellitus; T3cDM = type 3c diabetes mellitus (pancreatogenic); VBC = value-based care

Risk Factors

FACTOR	RISK SIGNAL	NOTES
Advanced age (≥60 years)	**~20% of all CP**;[12] more idiopathic/autoimmune etiology; higher rates of painless disease and endocrine insufficiency[12]	Age >60 is independent risk factor for AP→CP progression (**HR 5.29**);[13] fastest diabetes onset among CP subtypes (~5;8 yrs)
Genetic predisposition (PRSS1, SPINK1, CFTR, CTRC)	<10% of all CP;[2] **PRSS1** causes hereditary CP in **80% of carriers**; pathogenic variants in ~48% of idiopathic CP	PRSS1 confers **~60×** pancreatic cancer risk;[14] begin cancer surveillance at **age 40** or 20 yrs after diagnosis
Male sex; racial disparity	Two-thirds of CP patients are male;[2] Black patients have higher CP prevalence; non-White patients have 3.3× odds of alcoholic etiology[2]	Genetic testing underutilized in non-White populations (1% vs 11%);[15] most variant data derived from White cohorts
Prior acute pancreatitis (RAP→CP continuum)	**10%** develop CP after first AP; **36%** after recurrent AP;[16] ≥3 episodes is strong risk factor (HR 4.18)	Alcohol-related AP has shortest time to CP (**~4 months**; HR 9.16);[13] older adults more likely to have had cumulative subclinical episodes
Atypical/silent presentation	Pain **absent or minimal**; relies on imaging + labs; weight loss/ malnutrition first clue	Older CP patients may have **fibrosis-induced** nerve loss, altered pain thresholds, or coexisting neuropathy masking pain; requires **proactive screening**[12]
Frailty; sarcopenia; low BMI	**Protein catabolism** from EPI + inflammation; accelerates functional decline and falls	Use Clinical Frailty Scale (CFS) at baseline and annually; early **nutrition intervention** (high-protein, high-calorie)[12,17]
Polypharmacy; opioid burden; medication interactions	Opioid use increases **constipation**, **cognitive impairment**, **fall risk**; NSAIDs + CKD → AKI risk	Pain management should favor pregabalin, duloxetine, PPI-assisted PERT; **deprescribe opioids** if possible; celiac plexus block for refractory pain[1,2]
Metabolic bone disease; high fall risk	Osteoporosis in **25–50%** of CP patients;[15] Vitamin D malabsorption + chronic inflammation[14,15]	**DEXA** at baseline + every 2 years; supplement Vitamin D (2,000–4,000 IU/day), calcium (1,200 mg/day); consider monitoring for vertebral fractures[17]
Cognitive decline; depression; isolation	Chronic pain + malnutrition + social isolation fuel **cognitive decline** and **depression** in older CP	Screen PHQ-9 and MoCA **annually**; link to geriatric psychiatry, social work, and palliative care early if frailty advancing[12]

FACTOR	RISK SIGNAL	NOTES

ABBREVIATIONS: AKI = acute kidney injury; CFS = Clinical Frailty Scale; CP = chronic pancreatitis; CKD = chronic kidney disease; DEXA = dual-energy x-ray absorptiometry; E/M = evaluation and management; EPI = exocrine pancreatic insufficiency; MoCA = Montreal Cognitive Assessment; NSAID = nonsteroidal anti-inflammatory drug; PHQ-9 = Patient Health Questionnaire—9; PPI = proton pump inhibitor; VBC = value-based care

Underlying Conditions that Exacerbate or Progress to CP

UNDERLYING CONDITION	RISK SIGNAL	CLINICAL NOTES
Biliary disease and biliary sludge	**Most common etiology in the elderly**;[9] biliary lithiasis risk increases with age, especially in older women[9]	Recurrent gallstone/sludge passage causes **subclinical micro-attacks** healing via fibrosis;[14] 10% develop CP after first AP, 36% after recurrent AP;[15] sludge-induced AP **equivalent in severity** to gallstone AP[18,19]
High-risk polypharmacy (drug-induced pancreatitis)	Higher rates in older adults;[9] ACEi (**OR 6.12**) and statins (OR **4.97**) are highest-risk common geriatric agents[20]	Recurrent drug-induced AP can **transition to chronic state**;[21] key agents: statins, ACEi, thiazides, azathioprine, DPP-4 inhibitors;[9,20] review medication list in any AP of unclear etiology[20]
Late-onset idiopathic CP	**~25%** of CP is idiopathic;[22] >90% of Type 1 AIP patients are >60[9]	Must differentiate from malignancy (**both cause painless jaundice** in older adults);[9] IgG4 elevated in AIP but also in 4–7% of pancreatic cancer;[23] ~10% present with malabsorption alone[10]
Autoimmune pancreatitis (Type 1 IgG^4)	Prevalence **<1%**; predominantly older males (>60);[9] mimics pancreatic cancer; responds dramatically to corticosteroids[23]	Ischemia from narrowed mesenteric arteries triggers localized necrosis and fibrotic remodeling; ductal hypertension causes acinar cell hypoperfusion[14]
Synergy of moderate alcohol + tobacco	Both present in **>50%** of CP;[22] absolute risk **~18%** for either alone vs **~30%** combined;[14] moderate intake (25–50 g/day) increases risk[14]	Effects **potentiated when combined**;[14] cessation of either substantially reduces progression risk;[14] alcohol-related AP has **shortest time to CP**[14,22]
Prior acute pancreatitis (RAP → CP)	**10%** develop CP after first AP; **36%** after recurrent AP;[15] age >60 is independent progression risk factor[3]	Alcohol-related AP has shortest time to CP;[14] **≥3 episodes** may represent early CP without morphological changes;[15] biliary AP without cholecystectomy among greatest recurrence risk factors[1]

ABBREVIATIONS: CP = chronic pancreatitis; AP = acute pancreatitis; ACEi = angiotensin-converting enzyme inhibitor; OR = odds ratio; DPP-4 = dipeptidyl peptidase-4; AIP = autoimmune pancreatitis; IgG^4 = immunoglobulin G4; RAP = recurrent acute pancreatitis

Diagnostic Thresholds

TEST/MARKER	DIAGNOSIS CRITERION	NOTES
Serum lipase	**>90 U/L** at presentation; chronic CP may show normal or mildly elevated lipase	**More sensitive/specific** than amylase; in advanced CP, lipase may be normal despite ongoing EPI[2]
Fecal elastase-1 (gold standard for EPI)	**<200 mcg/g** = confirms EPI; **≥200** rules out EPI; **200–500** = mild insufficiency	Requires stool collection; elastase <200 justifies PERT initiation and guides dosing[2]
Pancreatic imaging (CT/MRI/EUS)	Ductal dilation, atrophy, parenchymal calcifications, cysts, fibrosis = **CP diagnosis**	ACG 2020 CP guidelines: imaging + clinical findings define CP; **EUS** has highest sensitivity for **early changes**[1,15]
Fecal chymotrypsin; 72-hour fecal fat	**Chymotrypsin absent** (qualitative) or 72-hour fat >7 g/day confirms steatorrhea	Less commonly ordered now; elastase preferred; **used historically** to quantify EPI severity
Vitamin B12, fat-soluble vitamins (A, D, E, K)	Low B12 (**<200 pg/mL**), low 25-OH Vitamin D (**<20 ng/mL**) in EPI; PT prolonged if K deficiency	Malabsorption in EPI requires supplementation (**Vitamin D** 2,000–4,000 IU/day, **B12** monthly IM if needed)[2]

ABBREVIATIONS: ACG = American College of Gastroenterology; CP = chronic pancreatitis; CT = computed tomography; E/M = evaluation and management; EPI = exocrine pancreatic insufficiency; EUS = endoscopic ultrasound; IM = intramuscular; IU = international units; MRCP = magnetic resonance cholangiopancreatography; MRI = magnetic resonance imaging; PERT = pancreatic enzyme replacement therapy; PT = prothrombin time

CLINICAL PEARL

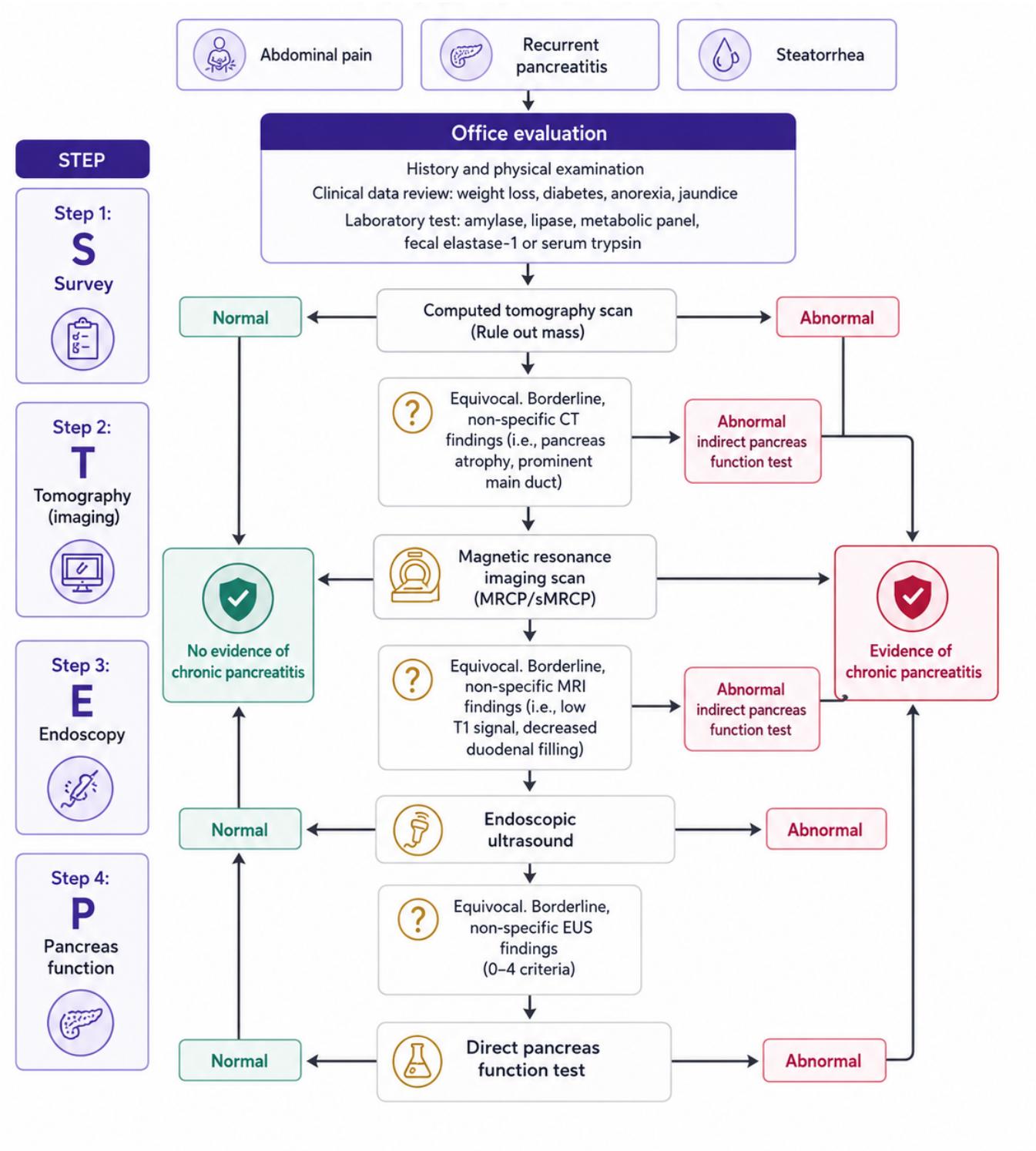
In older adults with CP, painless or mild presentation with weight loss, abnormal imaging, and low fecal elastase **is the norm**; ~30% of CP patients have no pain, with 3.27× higher odds of painless disease after age 55.[24,25] Maintain high suspicion for CP in any patient >65 with unexplained malnutrition, osteoporosis, brittle diabetes, or steatorrhea: lipase is **often normal** in advanced disease.[10,26] **Early PERT** prevents maldigestion-driven frailty; untreated EPI leads to nutritional deficiency, poor quality of life, and increased mortality.[17] For T3cDM, the ADA recommends annual screening and early insulin; **avoid GLP-1 agonists and sulfonylureas**.[8,14]Alcohol etiology must be documented; cessation improves pain and **reduces hospitalizations**.[26] Pancreatic cancer risk is **~8× baseline**; new diabetes in CP **warrants imaging** (pancreatic CT).[27]

Common Oversights

OVERSIGHT/SHORTCUT	WHY IT MATTERS — WHAT TO DO INSTEAD
Assuming normal lipase rules out CP	In advanced CP, lipase may be normal **despite severe EPI**; diagnosis rests on imaging + clinical context + elastase, not enzyme alone[2]
Not screening for EPI when CP is diagnosed	EPI develops in 30–50% of CP patients and is the primary driver of malnutrition and frailty; obtain fecal elastase at diagnosis and periodically thereafter, and initiate PERT when confirmed[2]
Not assessing alcohol use or referring for AUD treatment	**Fewer than half** of CP patients with alcohol etiology achieve sustained cessation;[28] document alcohol use frequency/quantity at every visit and refer for formal AUD treatment, not counseling alone
Treating CP pain exclusively with opioids	Opioids increase constipation, cognitive decline, falls in older adults; prefer pregabalin, duloxetine, PPI-assisted PERT, and consider celiac plexus block[26]
Dismissing weight loss as 'normal aging'	Weight loss in CP signals malnutrition + sarcopenia + frailty risk; requires **proactive nutrition intervention** and PERT optimization[29]

ABBREVIATIONS: AUD = alcohol use disorder; CP = chronic pancreatitis; EPI = exocrine pancreatic insufficiency; HCC = hierarchical condition category; PERT = pancreatic enzyme replacement therapy; T2DM = type 2 diabetes mellitus; T3cDM = type 3c diabetes mellitus (pancreatogenic)

Diagnostic Pathway for Suspected Chronic Pancreatitis



Structured evaluation from symptoms and office assessment by imaging, endoscopy, and pancreatic function testing

Key Differentials in Elderly

PRESENTATION	DIFFERENTIAL	KEY TESTS
Chronic epigastric pain + steatorrhea	**Celiac disease**; pancreatic cancer; IBS; cystic fibrosis	Tissue transglutaminase IgA (**celiac**);[17] fecal elastase **<200** (EPI);[1] CT imaging; if age >55 + new-onset pain, exclude malignancy with EUS[1]
Painless jaundice + weight loss	Pancreatic cancer; biliary stricture; hepatic disease; cyst/pseudocyst compression	**MRCP** to visualize duct;[1] if duct obstruction confirmed, consider **ERCP**; malignancy screening (imaging, CA 19–9) if age >60 + new presentation[27]
Brittle diabetes + weight loss	T2DM with complications;[8] pancreatogenic (T3c) diabetes; cystic fibrosis–related diabetes	Fecal elastase **<200** (EPI);[1] pancreatic imaging; document diabetes onset timing relative to GI symptoms[8]
Unexplained osteoporosis + B12 deficiency	Chronic malabsorption (celiac, Crohn's); atrophic gastritis; pernicious anemia	Tissue transglutaminase IgA (celiac); fecal elastase **<200** (EPI); **Vitamin D level**;[17] consider endoscopy if B12 + **intrinsic factor** antibody positive[17]
Acute abdominal pain + lipase elevation	Acute pancreatitis (often in same patient with CP history); pseudocyst rupture; biliary colic	CT imaging to assess for acute inflammation, pseudocyst, or obstruction;[14] consider ERCP if **ductal obstruction** identified

ABBREVIATIONS: CP = chronic pancreatitis; CT = computed tomography; EPI = exocrine pancreatic insufficiency; ERCP = endoscopic retrograde cholangiopancreatography; EUS = endoscopic ultrasound; GI = gastrointestinal; IBS = irritable bowel syndrome; IgA = immunoglobulin A; MRCP = magnetic resonance cholangiopancreatography; T2DM = type 2 diabetes mellitus; T3cDM = type 3c diabetes mellitus (pancreatogenic)

Comorbidity Screening

CONDITION	PREVALENCE/ASSOCIATION	SCREENING APPROACH
Exocrine pancreatic insufficiency (EPI)/ steatorrhea	40–60% of CP develop clinical EPI;[17] risk increases with disease duration and severity	Fecal elastase-1; 72-hour fecal fat if elastase unavailable; once confirmed, **PERT dosing** 1,000–4,000 lipase units per kilogram per meal[17]
Pancreatogenic (Type 3c) diabetes mellitus	70–80% of CP patients with advanced fibrosis develop endocrine insufficiency;[17] brittle glucose control common[14]	HbA1c + fasting glucose; often requires insulin **despite normal BMI**; titrate insulin cautiously to avoid hypoglycemia in older adults; consider CGM

CONDITION	PREVALENCE/ASSOCIATION	SCREENING APPROACH
Osteoporosis/ Osteopenia; pathologic fractures	25–50% of CP develop metabolic bone disease;[30] Vitamin D malabsorption + chronic inflammation + malnutrition[30,31]	**DEXA** at baseline + every 2 years; Vitamin D level (target >30 ng/mL); supplement Vitamin D 2,000–4,000 IU/day + calcium 1,200 mg/day
Alcohol use disorder (when K86.0)	Alcohol-induced CP implies active or recent heavy use; high relapse and psychosocial complexity[17]	**AUDIT-C**; referral to addiction medicine/ psychiatry; naltrexone preferred in older adults (avoid disulfiram); monitor abstinence or reduction
Pancreatic cancer risk (long-term surveillance)	Relative risk increased **5–20 fold** in CP;[32] risk increases with smoking, chronic inflammation, family history[11]	No evidence-based screening protocol for unselected CP; case-by-case: if age >55, smoking, or atypical imaging (new stricture, cyst growth), discuss **EUS/CA 19–9** with gastroenterology

ABBREVIATIONS: AUDIT-C = Alcohol Use Disorders Identification Test — Consumption; CA 19–9 = carbohydrate antigen 19–9; CGM = continuous glucose monitoring; CP = chronic pancreatitis; DEXA = dual-energy x-ray absorptiometry; E/M = evaluation and management; EPI = exocrine pancreatic insufficiency; EUS = endoscopic ultrasound; HbA1c = hemoglobin A1c; IU = international units; PERT = pancreatic enzyme replacement therapy; T3cDM = type 3c diabetes mellitus (pancreatogenic)

Staging/Severity Matrix

No single classification system captures all dimensions of CP. The **M-ANNHEIM** classification is the most comprehensive, integrating etiology, clinical stage (0–IV), and a severity index;[33] it is applicable across age groups, though **elderly-onset CP** (≥60 years) shows a distinct profile with more idiopathic etiology, painless disease, and higher endocrine insufficiency.[9,12,33] **COPPS** is the only prospectively validated hospitalization predictor, and its accuracy holds irrespective of age at onset, etiology, and smoking status, making it the most universally applicable severity tool across all adult CP populations.[34] **Frailty assessment** should be layered onto any staging system: 41% of hospitalized US CP patients meet frailty criteria, frailty independently doubles mortality (**aHR 2.05**), and nearly two-thirds of frail CP patients are younger than 65.[35,36]

M-ANNHEIM Classification — Primary CP Staging System

M-ANNHEIM COMPONENT[33]	CRITERIA/VALUES[33]	CLINICAL USE	DOCUMENTATION CUE
Etiology (Risk Factor Categories)	**M** = Multiple risk factors; **A** = Alcohol; **N** = Nicotine; **N** = Nutritional; **H** = Hereditary; **E** = Efferent duct; **I** = Immunological; **M** = Miscellaneous/ metabolic; each scored as **definite**, **probable**, or **questionable**	Captures all contributing etiologies simultaneously; reflects **multifactorial nature** of CP; enables risk-factor interaction analysis across visits	Document each identified risk factor with **confidence level** (e.g., "A-definite, N-definite, H-questionable"); update at each visit
Clinical Stage (0–IV)	**Stage 0** = subclinical (risk factors present, no symptoms); **Stage I** = no pain, with (Ia) or without (Ib) complications; **Stage II** = pain ± complications; **Stage III** = persistent pain ± severe complications (EPI, diabetes, pseudocyst, biliary obstruction); **Stage IV** = end-stage (pancreatic burnout ± organ failure)	Stage 0–I → primary care monitoring; Stage II → pain optimization + PERT evaluation; Stage III–IV → GI/surgery comanagement + palliative care consideration	Record stage number (0–IV) with substage; note pain status, EPI (elastase value), diabetes status, structural complications
Severity Index (Point-Based Score)	Points assigned for: pain intensity, pain control method (analgesics → surgery), surgical/interventional history, EPI, diabetes, morphologic complications; summed into Minor (≤5), Increased (6–9), Advanced (10–14), Marked (15–19), Severe (≥20)	Higher scores predict need for endoscopic/ surgical intervention; serial scoring tracks disease trajectory; only system alongside COPPS that includes a severity scoring component	Document total severity index score at baseline and annually; note individual component scores to identify worsening domains
Fecal Elastase-1 Integration	FE-1 <200 µg/g upgrades M-ANNHEIM stage in ~14% of patients vs steatocrit alone; FE-1 detects EPI in 68% vs 53% by steatocrit	FE-1 is the preferred exocrine function test for accurate M-ANNHEIM staging; more sensitive than steatocrit for subclinical EPI detection	Record elastase value with date; note whether it altered M-ANNHEIM stage assignment

ABBREVIATIONS: M-ANNHEIM = Multiple risk factors–Alcohol, Nicotine, Nutritional, Hereditary, Efferent duct, Immunological, Miscellaneous/metabolic; CP = chronic pancreatitis; EPI = exocrine pancreatic insufficiency; COPPS = Chronic Pancreatitis Prognosis Score; GI = gastroenterology; PERT = pancreatic enzyme replacement therapy; FE-1 = fecal elastase-1; µg/g = micrograms per gram; EPF = exocrine pancreatic function

Alternative Staging and Assessment Tools (Summary)

SYSTEM	WHAT IT DOES	KEY VALUES	WHEN TO USE
ACG 2020 Diagnostic Criteria[26]	Separates definite vs probable/suspicious CP based on imaging + ≥1 clinical criterion (EPI, diabetes, recurrent AP)	Definite → aggressive intervention; suspicious → watchful waiting	**Initial diagnostic workup**; establishing diagnostic certainty for documentation
APA Composite Nomenclature (2014)[3]	Combines TIGAR-O etiology + M-ANNHEIM/ Cambridge Imaging Grade (0–IV) + Physiology Stage (A–E, X) into a single string	e.g., "Chronic Toxic (Alcohol, smoking)-induced Pancreatitis, Imaging Grade III, Physiology Stage C"	Standardized **clinical documentation** and research reporting in US practice
COPPS (Chronic Pancreatitis Prognosis Score)[34]	5 parameters (BMI + HbA1c + CRP + platelet count + pain intensity) → Stages A (5–6), B (7–9), C (10–15)	Only prospectively validated score predicting 12-month hospitalization risk; analogous to Child-Pugh for cirrhosis; does not correlate with Cambridge classification	Baseline and serial reassessment; Stage C triggers proactive readmission prevention
Fecal Elastase-1[3]	<200 µg/g = severe EPI; 200–500 = mild EPI; >500 = preserved function	Guides PERT dosing intensity; severe EPI triggers nutrition referral	At diagnosis and when malabsorption symptoms persist despite PERT
DEXA T-score	T-score >−1.0 = normal; −1.0 to −2.5 = osteopenia; <−2.5 = osteoporosis	Fracture risk stratification; osteoporosis warrants calcium/ Vitamin D + bisphosphonate	Baseline + every 2 years in all CP patients
Clinical Frailty Scale (CFS)	1–3 = robust; 4–5 = vulnerable/mildly frail; 6–7 = moderately–severely frail; 8–9 = very severely/terminally ill	CFS ≥5 signals need for palliative/geriatric comanagement	Baseline and annually; advancing score triggers escalation

ABBREVIATIONS: ACG = American College of Gastroenterology; CP = chronic pancreatitis; EPI = exocrine pancreatic insufficiency; AP = acute pancreatitis; APA = American Pancreatic Association; TIGAR-O = Toxic-metabolic, Idiopathic, Genetic, Autoimmune, Recurrent and severe acute pancreatitis, Obstructive; M-ANNHEIM = Multiple risk factors–Alcohol, Nicotine, Nutritional, Hereditary, Efferent duct, Immunological, Miscellaneous/metabolic; COPPS = Chronic Pancreatitis Prognosis Score; BMI = body mass index; HbA1c = glycated hemoglobin; CRP = C-reactive protein; µg/g = micrograms per gram; PERT = pancreatic enzyme replacement therapy; DEXA = dual-energy x-ray absorptiometry; CFS = Clinical Frailty Scale

⚠ RED FLAG - URGENT (SAME-DAY/ED EVALUATION)

Any of the following clinical scenarios in a patient with known or suspected chronic pancreatitis warrants same-day emergency department evaluation:

- Sudden severe epigastric pain + elevated lipase (acute pancreatitis or pseudocyst rupture)
- Persistent vomiting + dehydration
- Signs of sepsis (fever + elevated WBC + hypotension) suggesting infected pseudocyst
- Signs of acute hepatic decompensation (jaundice + coagulopathy) from biliary obstruction
- Acute mental status change (hepatic encephalopathy, hypoglycemia) in patient with CP + diabetes

⚠ RED FLAG - RAPID (24–48 HOURS)

Any of the following clinical scenarios in a patient with known or suspected chronic pancreatitis warrants rapid evaluation or action within 24-48 hours:

- New or worsening diabetes with brittle glucose control
- Significant weight loss over 2–4 weeks + steatorrhea suggesting acute decompensation of EPI
- Persistent elevated lipase without acute symptoms (possible malignancy surveillance)
- New jaundice without fever (possible cyst/pseudocyst obstruction or malignancy)
- Recurrent acute pancreatitis episodes indicating ductal obstruction candidate for ERCP

3 MEAT DOCUMENTATION ESSENTIALS

Accurate diagnosis and management of adult chronic pancreatitis (CP) requires emphasis on the VBC imperative: **early recognition** of exocrine insufficiency, **aggressive nutritional intervention**, **pain management** without opioid escalation, and T3cDM optimization. The most common clinical oversights in CP are **dismissing weight loss as normal aging**, assuming normal lipase rules out disease, failing to code EPI (K86.81) alongside CP, and treating pain exclusively with opioids. For older adults, atypical presentation and comorbidity burden require a geriatric-centered approach that prioritizes functional independence and quality of life over aggressive intervention. The MEAT framework below ties each element into **appropriate clinical action**:

*A **68-year-old male** with 18-month history of mild epigastric discomfort after fatty meals, progressive weight loss (**12 lbs over 6 months**), and recent brittle fasting glucose despite long-standing metformin. Examination reveals mild epigastric tenderness; otherwise unremarkable. **Labs**: fasting glucose 187, HbA1c 8.2%, lipase 74 U/L (normal), albumin 3.1 g/dL. CT abdomen*

shows pancreatic atrophy with subtle ductal dilation. ***Fecal elastase****: 156 mcg/g. History of one prior acute pancreatitis episode (presumed idiopathic, 15 years ago). PCPs frequently miss idiopathic CP in older patients when pain is mild and lipase is normal; weight loss is attributed to aging or comorbid disease, delaying diagnosis and PERT initiation.*

MONITOR: "Weight: 185 → 173 lbs over 6 months (−6.5%). HbA1c: 7.1 → 8.2 over 8 months. Fecal elastase: **156 mcg/g (severe EPI)**. Lipase: 74 U/L (normal — **does not exclude CP**). Albumin: 3.1 g/dL. Stool quality: patient reports greasy, **foul-smelling stools** ×3 months. Baseline DEXA, vitamin D, B12 ordered. AUDIT-C: 0 (no alcohol use). CFS: 4 (vulnerable)."

EVALUATE: "CT abdomen (4/15): pancreatic atrophy, subtle ductal dilation, no calcifications, no mass. Fecal elastase (4/18): 156 mcg/g → confirms EPI. No EUS performed yet; consider if CT findings equivocal or malignancy concern arises. **M-ANNHEIM staging**: moderate (EPI confirmed + early endocrine dysfunction + imaging changes). MoCA: 26/30 (baseline). PHQ-9: 8 (mild depression)."

ASSESS: "Late-onset idiopathic chronic pancreatitis with confirmed EPI (elastase 156) and probable pancreatogenic (T3c) diabetes — **K86.1** + **K86.81** + **E13.9**. Associated: malnutrition with weight loss (**E44.1**); mild depression (**F32.0**). Prior acute pancreatitis episode 15 years ago supports necrosis-fibrosis progression. Normal lipase does not exclude CP in advanced fibrosis. **Frailty risk**: CFS 4 (vulnerable), albumin 3.1, progressive weight loss → high-cost VBC risk."

TREAT: "PERT: pancrelipase 50,000 lipase units with meals, 25,000 with snacks; **add omeprazole 20 mg daily** to optimize PERT efficacy. Nutrition: high-protein, high-calorie diet; dietitian referral placed. Supplements: vitamin D 4,000 IU/day + calcium 1,200 mg/day; B12 level pending. Diabetes: transition **from metformin to basal insulin** (glargine 10 units nightly, titrate to fasting glucose <140); avoid glinides (hypoglycemia risk). Pain: **pregabalin 75 mg BID**, titrate to 150 mg BID if tolerated; celiac plexus block if refractory. Referrals: gastroenterology (EUS consideration + PERT optimization), dietitian, geriatrics (CFS 4 + progressive decline). DEXA ordered; if T-score <−2.5, initiate alendronate 70 mg weekly. Follow-up: 4 weeks for weight check, glucose log review, PERT response."

Clinical Documentation Elements

- **Etiology:** If K86.0 (alcohol-induced), sequence F10.xx at every encounter (F10.20 moderate active; F10.21 in remission; F10.10 mild). Alcohol quantity/frequency must be documented **at every visit**
- **Avoid K86.9** (unspecified) when K86.0 or K86.1 can be supported by imaging. Avoid F10.99 when severity/remission status are determinable. AUD is the clinical root cause, not a secondary note. Only 50% of CP patients with alcohol etiology receive any AUD treatment → **F10.xx at every visit**
- **Severity Markers:** Imaging findings (M-ANNHEIM grade); fecal elastase value; diabetes status (none/controlled/brittle); DEXA T-score if available
- **EPI Diagnosis and PERT:** Elastase value; documented initiation of PERT with starting dose; response assessment (stool quality, weight trend)
- **Comorbidities:** T3cDM with insulin initiation details; pain assessment + management plan (drug, dose, response); **AUDIT-C if AUD**

- **VBC Metrics:** Weight + BMI; albumin/prealbumin; **CFS score**; AUDIT-C; vitamin D level; DEXA date; HbA1c/glucose; annual visit summary for quality metrics and complete documentation

Reframing Common Documentation Shortcuts

INSTEAD OF…	DOCUMENT…
'Chronic pancreatitis' (no etiology specified)	**K86.0** 'Alcohol-induced chronic pancreatitis' **+ F10.20/F10.21**, OR **K86.1** 'Idiopathic chronic pancreatitis'; specify imaging findings (atrophy, ductal changes, calcifications)
'Diabetes management' (no type distinction)	**E13.xx** 'Type 3c diabetes mellitus, pancreatogenic' with date of onset relative to CP diagnosis; document insulin initiation + dose; note **higher hypoglycemia risk** vs. T2DM
'Pancreatic insufficiency' or 'malabsorption' (no PERT dosing)	**K86.81** 'Exocrine pancreatic insufficiency'; fecal elastase <200; 'PERT initiated at 40,000 units lipase per meal; response: stool frequency decreased, weight stable.'
'Pain controlled' (unspecified agents)	'Pain 4/10 on pregabalin 300 mg BID + **duloxetine** 60 mg daily; avoided opioids per VBC guideline; patient reports improved function.'; include medication, dose, response
'History of pancreatitis' (after active disease phase)	**Do NOT use Z87.19** during active disease; acceptable only after documented sustained remission: 'Clinical remission confirmed: asymptomatic x 12 months, normal recent elastase, imaging stable, alcohol abstinent.'

ABBREVIATIONS: K86.0 = alcohol-induced chronic pancreatitis (ICD-10); F10.20 = alcohol use disorder, moderate, active (ICD-10); F10.21 = alcohol use disorder, moderate, in remission (ICD-10); K86.1 = idiopathic chronic pancreatitis (ICD-10); E13.xx = type 3c diabetes mellitus codes (ICD-10); CP = chronic pancreatitis; T2DM = type 2 diabetes mellitus; T3cDM = type 3c diabetes mellitus (pancreatogenic); K86.81 = exocrine pancreatic insufficiency (ICD-10); PERT = pancreatic enzyme replacement therapy; BID = twice daily; VBC = value-based care; Z87.19 = personal history of diseases of the digestive system (ICD-10); EPI = exocrine pancreatic insufficiency; HCC = hierarchical condition category; E/M = evaluation and management

DOCUMENTATION IS THE BEDROCK OF CP MANAGEMENT IN VBC. EVERY NOTE MUST INCLUDE

(**1**) Etiology and sequenced codes (K86.0 + F10.xx OR K86.1); (**2**) Current PERT regimen + response; (**3**) Diabetes type & control (T3cDM vs. T2DM); (**4**) Weight & nutrition status; (**5**) Pain severity & current non-opioid strategy; (**6**) Annual assessments (elastase, Vitamin D, bone density, frailty). This documentation directly supports **HCC mapping**, **specialist comanagement**, and **quality metric reporting**.

4 TREATMENT AND REFERRAL QUICK GUIDE

CP treatment begins with the least invasive **but most impactful interventions:** alcohol and smoking **cessation**, stepwise non-opioid **analgesia** (NSAIDs → pregabalin/duloxetine → weak opioids, reserving strong opioids as last resort), and **PERT** at ≥40,000 lipase units/meal for **confirmed EPI**. Notably, all FDA-approved PERT products (Creon, Zenpep, Pancreaze, Pertzye, Viokace) are **brand-name only**, equipotent at equivalent lipase doses, and often require formulary-driven switching to manage **substantial** out-of-pocket costs.[10,15,17,26] Co-analgesics such as pregabalin and generic antidepressants offer value-based alternatives to opioids, and interdisciplinary treatment combining behavioral pain management with medical therapy **lowers overall healthcare costs** while reducing opioid dependence risk: a critical consideration given that ongoing smoking reduces the efficacy of both endoscopic and surgical interventions.[10,15,22]

Therapy Escalation Criteria

TRIGGER	ACTION
Elastase <200 mcg/g unresponsive to standard PERT dosing (40,000 units/meal)	Refer to gastroenterology for dose optimization ± pancreatic enzyme trial (Creon vs. Pertzye);[1,2] **consider PPI trial** if not already on board
Recurrent acute pancreatitis episodes or suspected ductal obstruction (on MRCP)	Refer to pancreatic endoscopery (ERCP) for possible sphincterotomy, stone extraction, stricture dilation, or stent placement[10]
Uncontrolled pain despite pregabalin/duloxetine ± PPI-enhanced PERT	Refer to pain management and/or gastroenterology for celiac plexus block, EUS-guided block, or other interventional options[10]
New-onset jaundice or imaging evidence of biliary/pancreatic cyst >3 cm	Refer to gastroenterology + hepatology for assessment; possible EUS-guided cyst drainage if symptomatic or malignancy concern[9]
CFS ≥5 (moderate–severe frailty) + ongoing malnutrition despite PERT	Refer to geriatrics + nutrition + palliative medicine; consider home health, PT/OT, feeding support, and advance care planning[36]

ABBREVIATIONS: PERT = pancreatic enzyme replacement therapy; PPI = proton pump inhibitor; MRCP = magnetic resonance cholangiopancreatography; ERCP = endoscopic retrograde cholangiopancreatography; EUS = endoscopic ultrasound; CFS = Clinical Frailty Scale; PT/OT = physical therapy/occupational therapy; CP = chronic pancreatitis

ACG 2020/AGA 2023 Aligned Recommendations

CATEGORY	RECOMMENDED AGENT/ APPROACH[17,26]	NOTES[17,26]
Exocrine Enzyme Replacement (PERT)	Pancrelipase (generic), Creon, Pancreaze, Zenpep, or Pertzye; start at least **40,000–50,000 USP** units of lipase per meal with half-dose for snacks; titrate to symptom response; all porcine-derived formulations are **equally effective at equivalent doses**	ACG 2020 recommends PERT as **first-line for EPI**; start dosing during meals and increase if steatorrhea persists; AGA 2023 confirms minimum **40,000 USP units/meal** with dose adjustment based on meal size and fat content; monitor weight, stool pattern, and fat-soluble vitamin levels; doses >120,000 units/meal **seldom required**
PPI/H2-Blocker (Acid Suppression with PERT)	**Omeprazole 20 mg daily or famotidine 20 mg BID**; improves PERT efficacy by protecting enzymes from gastric acid degradation	AGA 2023 states H2 or PPI therapy is needed with **non–enteric-coated preparations** and should be added if PERT response is suboptimal; ACG 2020 supports acid suppression to increase PERT efficacy; monitor long-term PPI use for **B12 deficiency** and **bone density**
Pain Management (Opioid-Sparing First-Line)	**Pregabalin 150–600 mg/day** divided BID–TID, or duloxetine 60 mg daily; celiac plexus block for refractory pain; avoid NSAIDs if CKD present	ACG 2020 recommends **non-opioid analgesics** and co-analgesics before opioids due to risks of dependence, constipation, and cognitive impairment; pregabalin reduces pain by 36% after 3 weeks even in patients on strong opioids;[3] celiac plexus block reduces pain in ~50% of patients short-term but effect is temporary
Diabetes Management (T3cDM-Specific)	Insulin often required **earlier than T2DM**; metformin may be used in mild hyperglycemia (HbA1c <8%); avoid sulfonylureas (hypoglycemia risk) and **GLP-1 RAs** (pancreatitis risk); monitor for hypoglycemia closely in older adults	T3cDM carries **increased hypoglycemia risk** due to loss of counter-regulation;[8] ADA 2026 recommends HbA1c <8% in older adults with complex health to minimize hypoglycemia; metformin associated with survival benefit and reduced pancreatic cancer risk; optimize PERT concurrently to stabilize glucose

CATEGORY	RECOMMENDED AGENT/ APPROACH[17,26]	NOTES[17,26]
Vitamin D/Bone Health	Vitamin D supplementation if deficient (**target >30 ng/mL**); calcium 1,200 mg/day; bisphosphonate (alendronate 70 mg weekly) if DEXA T-score <−2.5 or prior fracture; weight-bearing exercise recommended	AGA 2023 recommends baseline DEXA **repeated every 1–2 years**; screen fat-soluble vitamins at diagnosis then annually; ACG 2020 notes increased osteoporosis and fracture risk in CP; overall prevalence of bone disease ~two-thirds of CP patients; deficiencies of vitamins D and K are associated with osteopathy and fractures; treatment reduces fracture rates

ABBREVIATIONS: PERT = pancreatic enzyme replacement therapy; USP = United States Pharmacopeia; ACG = American College of Gastroenterology; EPI = exocrine pancreatic insufficiency; AGA = American Gastroenterological Association; PPI = proton pump inhibitor; BID = twice daily; H2 = histamine-2 receptor; TID = three times daily; NSAIDs = nonsteroidal anti-inflammatory drugs; CKD = chronic kidney disease; T3cDM = type 3c diabetes mellitus (pancreatogenic); T2DM = type 2 diabetes mellitus; HbA1c = glycated hemoglobin; GLP-1 RA = glucagon-like peptide-1 receptor agonist; ADA = American Diabetes Association; DEXA = dual-energy x-ray absorptiometry; CP = chronic pancreatitis

Non-Pharmacologic Treatment and Lifestyle Modification

INTERVENTION	TARGET/RECOMMENDATION	NOTES
Nutritional Counseling + Dietary Modification	Fat-reduced diet (**40–50 g/day**); high-protein (**1.2 g/kg/day** minimum, higher if sarcopenic); small frequent meals; avoid alcohol/high-fat / high-fiber triggers	Registered Dietitian Nutritionist (RDN) is **essential**; monitor BMI, albumin, prealbumin quarterly if malnourished; **high-protein supplemental drinks** if unable to meet oral intake[26,29]
Physical Activity/ Rehabilitation	Encourage walking, strength training as tolerated; physical therapy if frail (**CFS ≥4**) to prevent sarcopenia and falls; gentle yoga/tai chi may improve pain perception	Exercise preserves muscle mass, bone density, mood, and cognition in older CP patients; **PT/OT referral** for frail patients (**CFS ≥5**) with fall risk[26]
Alcohol Cessation/AUD Treatment	Motivational interviewing; alcohol support groups; naltrexone 50 mg daily (preferred in older adults) or acamprosate 666 mg TID (renally dosed); avoid disulfiram	Only evidence-based intervention to slow alcohol-induced CP progression; **naltrexone is safer** in older adults than disulfiram; addiction medicine or psychiatry comanagement strongly recommended[37]

INTERVENTION	TARGET/RECOMMENDATION	NOTES
Social Support/ Palliative Care Integration	Link to case management, mental health, social work if depression/ isolation present; early palliative care discussion (**CFS ≥5**, **CKD Stage 4–5**, or **recurrent hospitalizations**)	CP + frailty + polypharmacy + chronic pain fuel depression, isolation, and functional decline; **early palliative input** improves symptom control, quality of life, and reduces inappropriate ED use[26]

ABBREVIATIONS: RDN = Registered Dietitian Nutritionist; BMI = body mass index; CFS = Clinical Frailty Scale; PT = physical therapy; OT = occupational therapy; CP = chronic pancreatitis; AUD = alcohol use disorder; TID = three times daily; CKD = chronic kidney disease; ED = emergency department

Medication Safety and Dose Adjustments

AGENT/CLASS	RISK	ADJUSTMENT[17,26]	MONITORING[17,26]
Naltrexone (AUD) + Opioids	HIGH	**Do not co-prescribe**; stop naltrexone 7–10 days before any opioid use; counsel on precipitated withdrawal and post-discontinuation overdose risk	Coordinate with addiction medicine before any opioid initiation; reassess pain control **≥2 weeks** post-naltrexone start
NSAIDs + CKD (eGFR <60)	HIGH	Avoid if **eGFR <**30; if eGFR 30–60, use lowest dose/shortest duration; prefer acetaminophen or topical NSAID	Baseline eGFR + potassium; **recheck at 2 weeks** and **2 months** if NSAID unavoidable
Pregabalin (Pain Management)	HIGH	Minimize opioid + pregabalin doses **if insulin-dependent**; target HbA1c **7–8%** to avoid hypoglycemia; use CGM if available	Glucose **2–4×/day**; educate on masked hypoglycemia symptoms from CNS depression; monthly insulin dose review
Pregabalin + CKD or + Opioids	MODERATE -HIGH	Reduce dose if **CrCl <60** (e.g., 75 mg BID for CrCl 30–60); avoid co-prescribing with opioids if possible; start low, titrate slowly	Fall risk, cognition, serum creatinine **quarterly**; if renal decline, taper or discontinue
PERT + Opioid /Anticholinergics	MODERATE	Take PERT with meals; minimize opioids; review anticholinergic burden (may delay enzyme transit)	Stool pattern **monthly**; adjust PERT dose if steatorrhea re-emerges despite adherence
Long-term PPI (with PERT)	MODERATE	Continue PPI if PERT response requires it; use lowest effective dose; reassess need annually	**Annual** B12, magnesium, DEXA; supplement B12 (1,000 mcg IM monthly) if <200 pg/ mL

AGENT/CLASS	RISK	ADJUSTMENT[17,26]	MONITORING[17,26]
Duloxetine (NSAID Alternative)	LOW-MODERATE	Preferred over NSAIDs in CP + CKD; start 30 mg daily, increase to 60 mg; monitor sodium (SIADH risk, especially age ≥70)	Baseline sodium + creatinine; recheck sodium at **2 weeks**, then every **3–6 months**

ABBREVIATIONS: AUD = alcohol use disorder; NSAIDs = nonsteroidal anti-inflammatory drugs; CKD = chronic kidney disease; eGFR = estimated glomerular filtration rate; HbA1c = glycated hemoglobin; CGM = continuous glucose monitoring; CNS = central nervous system; CrCl = creatinine clearance; BID = twice daily; PERT = pancreatic enzyme replacement therapy; PPI = proton pump inhibitor; DEXA = dual-energy x-ray absorptiometry; B12 = vitamin B12; IM = intramuscular; CP = chronic pancreatitis; SIADH = syndrome of inappropriate antidiuretic hormone secretion

When to Refer

CRITERION	SPECIALIST	URGENCY
Diagnosis confirmation (imaging atypical, elastase borderline) or complex CP (recurrent pancreatitis, ductal involvement)	**Gastroenterology** (Pancreatic/Biliary specialist if available)	**ROUTINE** (2–4 weeks) for uncomplicated CP; URGENT (1 week) if suspected acute exacerbation or malignancy concern
Malnutrition/sarcopenia/ unresponsive EPI despite PERT optimization	**Registered Dietitian Nutritionist** (RDN); consider home health for in-home assessment if homebound.	**ROUTINE** (1–2 weeks) for mild malnutrition; URGENT (same week) if albumin <2.5 or rapidly declining weight.
Uncontrolled pain despite pregabalin/duloxetine/ PPI-enhanced PERT	**Pain Management Specialist**; **Gastroenterology** (for celiac plexus block/EUS-guided intervention)	**ROUTINE** (2–4 weeks) for stable pain; EXPEDITED (1 week) if pain impairing function/ADLs
Alcohol-induced CP with active AUD or substance use disorder	**Addiction Medicine**/ **Psychiatry**; Outpatient Alcohol Support Programs (AA, SMART Recovery)	**URGENT** (same week) if active heavy use/withdrawal risk; **ROUTINE** (1–2 weeks) if stable in early remission
Frailty (CFS ≥5) + CP comorbidities; functional decline/falls/depression	**Geriatric Medicine**; **Palliative Care**; Case Management; Mental Health (if depression PHQ-9 >10)	**URGENT** (1 week) for CFS 6–9 or recent ED visit; **ROUTINE** (2–4 weeks) for CFS 4–5 with stable function.
Pancreatic cancer surveillance (age >55, smoking, family history, atypical imaging findings, CA 19–9 elevation)	**Gastroenterology** (EUS/ biopsy specialist); **Oncology** if malignancy suspected	**EXPEDITED** (1–2 weeks) if imaging findings concerning or CA 19–9 >37 U/ mL;[27] **ROUTINE** (routine surveillance) if no red flags

CRITERION	SPECIALIST	URGENCY

ABBREVIATIONS: CP = chronic pancreatitis; EPI = exocrine pancreatic insufficiency; PERT = pancreatic enzyme replacement therapy; RDN = Registered Dietitian Nutritionist; PPI = proton pump inhibitor; EUS = endoscopic ultrasound; ADLs = activities of daily living; AUD = alcohol use disorder; AA = Alcoholics Anonymous; CFS = Clinical Frailty Scale; PHQ-9 = Patient Health Questionnaire—9; ED = emergency department; CA 19–9 = carbohydrate antigen 19–9

Follow-Up Timing

STAGE/ CATEGORY[2,26]	FREQUENCY[2,26]	LABS/ASSESSMENTS TO MONITOR[2,26]
Newly Diagnosed CP (First 3 Months)	Every **2–4 weeks** until PERT dose optimized; then every **4–8 weeks** until stable	Stool pattern, weight, pain severity (11-point scale), **PERT adherence**; fecal elastase at diagnosis; repeat at 4–6 weeks if dose adjusted; fasting glucose if diabetic
Stable CP on PERT (EPI Controlled)	Every **3–4 month**s (if CFS <4); every **4–6 weeks** if CFS ≥4 or comorbidities unstable	Weight/BMI, albumin quarterly, pain control, stool frequency, medication adherence; **annual**: fecal elastase, Vitamin D, CBC, B12, LFTs, HbA1c (if diabetic), AUDIT-C (if alcohol-related)[17]
Complicated CP (Malnutrition, CFS ≥5, Recurrent Acute Exacerbations)	Every **2–4 weeks** until malnutrition reverses or acute event resolved; then every **4–6 weeks**	Weight (goal: stabilization); albumin bi-weekly **if <2.5**; glucose if T3cDM; PHQ-9; Timed Up & Go if fall risk; AUDIT-C if applicable[15]
T3cDM + CP (Brittle Diabetes)	Every **2–4 weeks** until glucose stabilized; then every **4–12 weeks**	Fasting/pre-meal/bedtime glucose; HbA1c every 3 months (target 7–8%); insulin dose log; hypoglycemia events; **eGFR if on SGLT2i**; annual lipid panel[38]
Alcohol-Induced CP (F10.xx + K86.0)	Every **1–4 weeks** initially (AUD medication titration + PERT); then every **4–8 weeks** if stable abstinence	**AUDIT-C each visit**; UDS if relapse risk; LFTs, lipase; thiamine/folate/B12; psychiatric screening; naltrexone/ acamprosate adherence; social support assessment[26]
Annual Comprehensive Reassessment	Every **12 months** for all CP patients; sooner if clinical change	Fecal elastase; Vitamin D + DEXA (if prior osteopenia/ osteoporosis); CFS; MoCA (if ≥75 or CFS ≥4); pain med review + taper plan; HCC re-documentation (weight, albumin, EPI severity, T3cDM, pain); cancer surveillance (imaging ± CA 19–9) if age >55 + smoking + family Hx or new-onset diabetes with weight loss

STAGE/ CATEGORY[2,26]	FREQUENCY[2,26]	LABS/ASSESSMENTS TO MONITOR[2,26]

ABBREVIATIONS: CP = chronic pancreatitis; PERT = pancreatic enzyme replacement therapy; CFS = Clinical Frailty Scale; EPI = exocrine pancreatic insufficiency; BMI = body mass index; CBC = complete blood count; B12 = vitamin B12; LFTs = liver function tests; HbA1c = glycated hemoglobin; AUDIT-C = Alcohol Use Disorders Identification Test—Consumption; T3cDM = type 3c diabetes mellitus (pancreatogenic); PHQ-9 = Patient Health Questionnaire—9; eGFR = estimated glomerular filtration rate; SGLT2i = sodium-glucose cotransporter-2 inhibitor; F10.xx = alcohol use disorder codes; AUD = alcohol use disorder; UDS = urine drug screen; DEXA = dual-energy x-ray absorptiometry; MoCA = Montreal Cognitive Assessment; HCC = hierarchical condition category; CA 19–9 = carbohydrate antigen 19–9; Hx = history

Comorbidity Management

COMORBIDITY	APPROACH	CAUTION
Type 3c Diabetes Mellitus (Pancreatogenic)	**Insulin-first approach** due to islet loss; metformin acceptable if HbA1c <8% and not malnourished; target **HbA1c 7–8%** in older adults; refer endocrinology if uncontrolled[39]	**Avoid sulfonylureas** (hypoglycemia); **avoid GLP-1 RAs and DPP-4i** (pancreatitis risk); TZDs contraindicated if CKD/CHF (fluid retention, fracture risk)
CKD Stage 3–5 + CP	Coordinate with nephrology if eGFR <30; **avoid NSAIDs**; reduce ACEi/ARB if K >5.5 mEq/L; monitor phosphate in CKD Stage 4–5	**SGLT2i**: DKA risk if impaired insulin production or vomiting; bisphosphonates contraindicated if eGFR <30 (consider denosumab); PPI may worsen bone density in advanced CKD
COPD/ Pulmonary Disease + CP	Heightened **respiratory depression risk with opioids**; prefer pregabalin/duloxetine; if opioids necessary, use lowest dose + respiratory monitoring[17,26]	Avoid concurrent opioids + benzodiazepines + gabapentinoids (additive CNS/respiratory depression); **calcium supplementation may increase secretions in some COPD patients**
Osteoporosis/ Bone Loss + CP	DEXA baseline + every 1–2 years; calcium **1,200 mg/day** + Vitamin D **2,000–4,000 IU/day**; alendronate **70 mg weekly** if T-score <−2.5 and eGFR ≥30[40]	**Bisphosphonate**: take upright 30 min on empty stomach; contraindicated if eGFR <30; monitor for **ONJ** if >5 years; denosumab alternative for severe osteoporosis or CKD Stage 4–5
Depression/ Anxiety + CP + Chronic Pain	Screen PHQ-9 at baseline + annually; **duloxetine 60 mg** daily treats pain + depression simultaneously; refer mental health if PHQ-9 >10; **CBT** effective for CP pain[41]	**SSRIs**: monitor sodium (hyponatremia risk); benzodiazepines **NOT first-line** (addiction risk, especially if AUD history); coordinate with psychiatry if on multiple psychotropics

COMORBIDITY	APPROACH	CAUTION
Alcohol Use Disorder (if K86.0 alcohol-induced CP)	Naltrexone **50 mg daily** (preferred in older adults) or acamprosate **666 mg TID**; motivational interviewing; link to support groups + addiction medicine[26]	Naltrexone + opioids = **overdose risk** (see MEDSAFE table); monitor LFTs (rare hepatotoxicity); **avoid disulfiram** (hepatotoxicity + delirium risk in older adults); UDS if relapse suspected

ABBREVIATIONS: T3cDM = type 3c diabetes mellitus (pancreatogenic); HbA1c = glycated hemoglobin; GLP-1 RAs = glucagon-like peptide-1 receptor agonists; DPP-4i = dipeptidyl peptidase-4 inhibitors; TZDs = thiazolidinediones; CKD = chronic kidney disease; CHF = congestive heart failure; CP = chronic pancreatitis; eGFR = estimated glomerular filtration rate; NSAIDs = nonsteroidal anti-inflammatory drugs; ACEi = angiotensin-converting enzyme inhibitor; ARB = angiotensin receptor blocker; K = potassium; SGLT2i = sodium-glucose cotransporter-2 inhibitor; DKA = diabetic ketoacidosis; PPI = proton pump inhibitor; COPD = chronic obstructive pulmonary disease; CNS = central nervous system; DEXA = dual-energy x-ray absorptiometry; ONJ = osteonecrosis of jaw; PHQ-9 = Patient Health Questionnaire—9; CBT = cognitive behavioral therapy; SSRIs = selective serotonin reuptake inhibitors; AUD = alcohol use disorder; TID = three times daily; LFTs = liver function tests; UDS = urine drug screen

Cost-Smart Options

BRAND (EST. COST)	GENERIC/ALTERNATIVE (EST. COST)	EST. SAVINGS	COST-SMART TIP
Creon 6 (20K units lipase) ~$800/month	Pancrelipase generic (same strength) **~$150/month**; Pancreaze/ Zenpep/ Pertzye **~$200–$350/month**	**~$450–$650/month**	**Generic pancrelipase is bioequivalent** to Creon; switch to generic first when available; Creon preferred if prior therapeutic response; support with PA if needed
Pregabalin (brand Lyrica) ~$400/month (300 mg/day)	Pregabalin generic (same dose) **~$40/month**; duloxetine generic **~$30/month**; capsaicin cream OTC **~$10/month**	**~$360/month**	**Generic pregabalin/duloxetine are cost-effective** first-line for CP pain; capsaicin topical OTC for **localized abdominal pain** (low cost, modest efficacy); NSAIDs avoided in CKD, so non-drug or opioid-sparing is imperative
Naltrexone (brand ReVia) ~$200/month	Naltrexone generic 50 mg daily **~$30/month**; long-acting Vivitrol (monthly IM) **~$1,200/month** for specialized AUD treatment	**~$170/month generic**	**Generic naltrexone is highly cost-effective** for AUD in alcohol-induced CP; Vivitrol IM monthly reserved for compliance barriers or specialist settings. Acamprosate (Campral) generic ~$50/month as an alternative if naltrexone is contraindicated.

BRAND (EST. COST)	GENERIC/ALTERNATIVE (EST. COST)	EST. SAVINGS	COST-SMART TIP
Omeprazole/ Pantoprazole brand PPI ~$150/month	Omeprazole generic 20 mg daily **~$10/month**; famotidine generic **~$5/ month** (if H2 blocker sufficient)	**~$140/ month**	**Generic omeprazole is highly cost-effective**; use with PERT; monitor B12 annually (supplementation cost ~$10/month IM if needed); Deprescribe PPI after 12 months if PERT + dietary modification adequate
Bisphosphonate (Fosamx brand) ~$100/month	Alendronate generic 70 mg weekly **~$10/month**; denosumab (Prolia) **~$300/dose** every 6 months if DEXA severe	**~$90/month**	**Generic alendronate 70 mg weekly is gold-standard**, cost-effective osteoporosis therapy; combine with calcium + Vitamin D (OTC, ~$20/month); Denosumab reserved for **severe osteoporosis** (T-score <−3.5) or bisphosphonate intolerance.
Vitamin D3 + Calcium Supplements (brand multivitamin formulations) ~$50/month	Vitamin D3 2000 IU + Calcium Citrate 500 mg OTC bulk bottles ~**$10/ month**; or Vitamin D 50,000 IU weekly Rx ~**$15/month** for deficiency correction	**~$40/month**	OTC Vitamin D3 2000–4000 IU + calcium citrate 1000–1200 mg daily is first-line; **inexpensive**; Rx high-dose Vitamin D (50,000 IU weekly x 6–8 weeks) for severe deficiency, then maintain with OTC; Recheck Vitamin D annually; supplement cost-effective **preventive measure for CP osteoporosis**

ABBREVIATIONS: PERT = pancreatic enzyme replacement therapy; PA = prior authorization; CP = chronic pancreatitis; OTC = over the counter; CKD = chronic kidney disease; AUD = alcohol use disorder; IM = intramuscular; PPI = proton pump inhibitor; B12 = vitamin B12; DEXA = dual-energy x-ray absorptiometry; IU = international units; Rx = prescription; T-score = standardized bone density score

Patient Education and Adherence

Patient education in CP should reinforce **daily self-management** anchored to five priorities:

(**1**) PERT adherence with every meal and snack to prevent malabsorption and weight loss[14,26]

(**2**) Dietary modification with small frequent meals, fat restriction (40–50 g/day), and alcohol avoidance[14]

(**3**) Opioid-sparing pain management using pregabalin or duloxetine first-line, with celiac plexus block for refractory cases

(**4**) T3cDM-specific glucose management, with insulin titrated cautiously to avoid hypoglycemia[8]

(**5**) Annual screening for bone loss (DEXA) and fat-soluble vitamin deficiencies.[30,40]

For alcohol-induced CP, naltrexone and counseling should be offered at every visit as the only disease-modifying intervention; alcohol cessation slows fibrotic progression and reduces pancreatic cancer risk.[32]

Patients should be **explicitly coached to report new or changed pain**, unexplained weight loss, worsening diabetes, jaundice, or persistent diarrhea unresponsive to enzymes: these overlap with **pancreatic cancer presentation** and require prompt imaging and gastroenterology referral.

Quality Metrics Tie-In

Chronic pancreatitis intersects **multiple quality measurement domains**: chronic disease management (diabetes care, osteoporosis screening), substance use disorder treatment (alcohol screening, AUD engagement), opioid safety, care transitions (medication reconciliation, readmissions), and geriatric-focused measures (functional status, advance care planning). **No CP-specific HEDIS or MIPS measures currently exist**, but the disease's high comorbidity burden: EPI-driven malnutrition, T3cDM, metabolic bone disease, chronic pain, and AUD means that cross-cutting national measures apply simultaneously across the care trajectory and can anchor internal quality dashboards for value-based CP programs.

MEASURE	STANDARD	NOTES
HEDIS: Care for Older Adults (COA)- Medication Review	**Denominator**: MA enrollees ≥66, continuously enrolled; **Numerator**: 4 sub-measures documented annually: (1) functional status assessment, (2) medication review, (3) pain assessment, (4) advance care planning; **Exclusions**: hospice, ESRD	Most directly applicable cross-cutting measure for **elderly CP patients**; aligns with frailty screening (CFS), PERT adherence review, and pain reassessment at each visit
HEDIS: Care for Older Adults (COA)- Pain Assessment	**Denominator**: enrollees ≥66 with SNP enrollment; **Numerator**: pain assessment documented during the year; **Exclusions**: hospice	Pain affects **>80%** of CP patients and is the primary driver of disability and psychiatric comorbidity; structured pain assessment should distinguish pancreatic nociceptive pain from centrally mediated/ neuropathic pain = **determines therapy**: endoscopic/ surgical vs. regabalin/behavioral therapy
HEDIS: Osteoporosis Management in Women Who Had a Fracture	**Denominator**: women 67–85 with fracture; **Numerator**: osteoporosis testing/treatment within 6 months **Exclusions**: hospice, ESRD	CP confers **~2.7× fracture risk** due to EPI-driven vitamin D/K malabsorption; DEXA and PERT optimization are first steps
HEDIS: Diabetes Care - Eye Exam	**Denominator**: enrollees 18–75 with diabetes (includes E13.xx); **Numerator**: retinal exam performed; **Exclusions**: hospice, ESRD	T3cDM carries **retinopathy risk**; coding as E13.xx ensures mapping in the denominator for annual eye exams

MEASURE	STANDARD	NOTES
HEDIS: Diabetes Care - Blood Sugar Controlled	**Denominator**: enrollees 18–75 with diabetes; **Numerator**: HbA1c tested + ≤8%; **Exclusions**: hospice, ESRD	T3cDM is insulin-first; target HbA1c 7–8% in older adults; avoid incretins, sulfonylureas, SGLT2 inhibitors
HEDIS: Medication Reconciliation Post-Discharge (MRP)	**Denominator**: enrollees ≥18 discharged from inpatient; **Numerator**: medication reconciliation within 30 days; **Exclusions**: hospice	Critical for CP patients with polypharmacy (PERT + PPI + insulin + pain agents); **identifies drug interactions** and PERT adherence gaps post-hospitalization
MIPS #418: Osteoporosis Management in Women Who Had a Fracture	**Denominator**: women ≥50 with fracture; **Numerator**: DEXA ordered or pharmacotherapy prescribed within 6 months	CP-associated osteoporosis prevalence **25–50%**; DEXA baseline + every 2 years; Vitamin D 2,000–4,000 IU/day + calcium 1,200 mg/day; bisphosphonate if T-score ≤−2.5
MIPS #134: Depression Screening (PHQ-9)	**Denominator**: patients ≥12; **Numerator**: screened with PHQ-9 AND follow-up plan if positive; **Exclusions**: active depression with current treatment	Chronic pain + malnutrition + social isolation **fuel depression in CP**; PHQ-9 ≥10 triggers mental health referral; duloxetine treats pain + depression simultaneously
MIPS #047: Advance Care Planning	**Denominator**: patients ≥65; **Numerator**: ACP or surrogate documented, OR discussion documented but patient declined	Billable as CPT **99497/99498 during AWV**; prioritize if CFS ≥5, CKD Stage 4–5, or recurrent hospitalizations
Annual Wellness Visit (AWV)	CPT **G0438** (initial)/**G0439** (subsequent); documents functional/cognitive screen, fall risk, ACP, depression screen	Platform for annual CP reassessment: CFS, MoCA (if ≥75), AUDIT-C, pain review, PERT adherence, Vitamin D/DEXA status; no patient copay

ABBREVIATIONS: HEDIS = Healthcare Effectiveness Data and Information Set; COA = Care for Older Adults; MA = Medicare Advantage; SNP = Special Needs Plan; ESRD = end-stage renal disease; CP = chronic pancreatitis; CFS = Clinical Frailty Scale; PERT = pancreatic enzyme replacement therapy; EPI = exocrine pancreatic insufficiency; DEXA = dual-energy x-ray absorptiometry; E13.xx = pancreatogenic diabetes ICD-10 codes; T3cDM = type 3c diabetes mellitus (pancreatogenic); HbA1c = glycated hemoglobin; SGLT2 = sodium-glucose cotransporter 2; MRP = Medication Reconciliation Post-Discharge; PPI = proton pump inhibitor; IU = international units; PHQ-9 = Patient Health Questionnaire—9; ACP = advance care planning; CPT = Current Procedural Terminology; AWV = annual wellness visit; CKD = chronic kidney disease; MoCA = Montreal Cognitive Assessment; AUDIT-C = Alcohol Use Disorders Identification Test—Consumption; MIPS = Merit-based Incentive Payment System

QUALITY OUTCOME TARGETS FOR VBC SUCCESS

(1) Hospital readmission for acute pancreatitis or decompensation: **<5% at 30 days**, **<10% at 90 days**
(2) Emergency department utilization: **<2 visits/patient/year** for CP-related complaints
(3) Malnutrition/sarcopenia reversal: **≥70%** of malnourished CP patients achieve **albumin ≥3.5 g/dL within 6 months** of PERT optimization + RDN intervention
(4) Opioid deprescribing: **≥30% reduction** in opioid use among CP patients over 12 months via pregabalin/duloxetine substitution.
(5) HbA1c control in T3cDM: **≥60%** of CP patients with **E13.xx** achieve **HbA1c 7–8%** (balanced to avoid hypoglycemia)
(6) Alcohol abstinence in alcohol-induced CP (K86.0 + F10.xx): **≥50% verified abstinence** or **≥40% reduction in consumption** at 6 and 12 months with naltrexone/addiction medicine involvement.
(7) Specialist coordination/no-show rate: **<15%** of recommended gastroenterology/RDN referrals are no-shows; **<10% delay** >30 days beyond referral date.

5 CODING REMINDERS AND CASE EXAMPLES

Documentation Specificity

- **Stage/Severity:** Specify etiology (**K86.0** alcohol-induced vs **K86.1** idiopathic) **AND M-ANNHEIM grade** (mild/moderate/severe) based on imaging + clinical findings;[33] example: "Idiopathic CP (K86.1), moderate severity per M-ANNHEIM (pancreatic atrophy + ductal dilation on MRI; fecal elastase 156 mcg/g confirming EPI)"; In older adults, **add CFS** (1–9) to flag frailty-driven escalation; EPI severity (elastase value) determines PERT dosing and nutritional risk;[17] etiology + elastase + diabetes type + weight trend + pain control are the **five pillars of CP documentation**
- **Etiology:** If **K86.0**, sequence **F10.xx at every encounter** (F10.20 moderate active; F10.21 in remission; F10.10 mild); alcohol quantity/frequency must be documented at every visit, **not just at diagnosis**;[26] avoid **K86.9** when **K86.0** or **K86.1** can be supported by imaging; avoid F10.99 when severity/remission status are determinable; **AUD is the clinical root cause**, not a secondary note; alcohol cessation counseling is recommended for all patients with CP per ACG[26]
- **Current status:** Most recent labs: lipase, fecal elastase (**<200 mcg/g confirms EPI**), glucose/HbA1c (E13.xx if pancreatogenic, **E11.xx if pre-existing T2DM**),[8] weight/BMI, albumin/prealbumin, Vitamin D level; **always code K86.81 when EPI present**; current PERT dose (units/meal), response (stool frequency, weight, adherence), PPI use; **if T3cDM**:

insulin type/dose, glucose logs, HbA1c target 7–8%; pain severity (0–10), current analgesics (pregabalin/duloxetine/opioid doses), functional impact

- **Associated conditions:** K86.81 (EPI, always code when elastase **<200 mcg/g**), E13.xx (T3cDM), K86.2/K86.3 (cysts/pseudocysts), M81.x (osteoporosis), E55.9 (Vitamin D deficiency), D51.x (B12 deficiency), E44.x/E46 (malnutrition), F10.xx (AUD if K86.0), N18.x (CKD); each must be explicitly coded when present: **compound HCC eligibility** separate from CP base
- **Chronicity:** Date of CP diagnosis, imaging summary (CT/MRI/EUS), prior acute episodes, duration of symptoms; functional impact: weight loss (amount/timeframe), falls/mobility, PHQ-9, cognitive status if ≥75 or frail; PERT optimization timeline (start date, dose escalation, elastase rechecks); **AUD progression if K86.0**: AUDIT-C, AUD meds, sobriety duration; CP is **NOT "history of pancreatitis" (Z87.19)** during active disease or ongoing PERT: use Z87.19 only after **documented sustained remission** (>12 months asymptomatic, stable imaging, off PERT)

Annual Clinical Review and Confirmation

- **Annual review:** K86.0 and K86.1 both map to **HCC 79** and must be reassessed in-person or via synchronous video each reporting year while disease remains active and PERT is being used. Update disease severity (**M-ANNHEIM grade**), EPI status (fecal elastase value), diabetes type and control (E13.xx vs. E11.xx; HbA1c; insulin dose), weight/BMI/albumin trend (malnutrition progression?), PERT dose and response, Vitamin D level + DEXA (if prior osteopenia/osteoporosis), AUD treatment status and AUDIT-C (if K86.0), pain control (scale 0–10; analgesic list), frailty score (CFS in ≥65 years), and SDOH
- **Visit modality:** In-person or video telehealth with meaningful evaluation qualifies. Document elastase result (or recent documentation) at annual visit= justifies **HCC 79 + K86.81 coding**. Current PERT dose/response, recent labs (lipase, glucose/HbA1c if diabetic, albumin, Vitamin D, Cr/eGFR if on pain meds), imaging if repeated (stable vs. new findings), medication adherence (PERT, insulin, pain meds, AUD meds if applicable), functional capacity (weight stable? able to work? falls? sarcopenia?), and depression screening (PHQ-9). **If K86.0**: **AUDIT-C**, **F10.xx status** (active vs. remission), naltrexone/AUD med adherence
- **Clinical context:** Under CMS-HCC V28, K86.0/K86.1 both map to **HCC 79 at RAF ~0.357**. Z87.19 (history of pancreatitis) is **NOT acceptable** while PERT is active, elastase remains abnormal, weight is declining, or imaging shows stable/progressive findings. **Continuity of the clinical record** and proactive escalation (RDN, gastroenterology, geriatrics) matter more than any individual code
- **Avoid rollover:** Do not copy last year's CP note forward **without updating M-ANNHEIM** severity grade, elastase value, PERT dose/response, weight/albumin (malnutrition tracking), diabetes type and HbA1c (brittle control?), DEXA T-score trend, AUDIT-C (**if K86.0**), pain level and analgesic response, frailty score, and goals of care. Elastase, weight, glucose control, and function all shift over time: **reassess at each annual visit**. Never default to Z87.19

(history code) simply because imaging is stable; if a patient is on PERT, has abnormal elastase, or has experienced weight loss, use active-disease codes (**K86.0/K86.1 + K86.81**). After documented **>12-month remission OFF PERT** with stable elastase, imaging, and weight, **Z87.19 becomes acceptable**

Good Documentation is Comprehensive Coding

EHR SHORTCUT/RISK	PREFERRED DOCUMENTATION LANGUAGE
Vague: 'Chronic pancreatitis'	Better: 'Idiopathic chronic pancreatitis (**K86.1**) with exocrine pancreatic insufficiency (**K86.81**); fecal elastase 156 mcg/g; on PERT 40,000 lipase units TID with meals; stool frequency improved since initiation.'
Vague: 'Diabetes management' (no distinction of type)	Better: 'Type 3c diabetes mellitus, pancreatogenic (**E13.35**), secondary to chronic pancreatitis; recent imaging shows severe pancreatic atrophy; initiated insulin glargine 20 units at bedtime; fasting glucose improved to 120–150 mg/dL; no hypoglycemic episodes to date.'
Vague: 'Abdominal pain, controlled' (no drug listed)	Better: 'Chronic epigastric pain attributed to chronic pancreatitis; currently **managed with pregabalin** 300 mg BID (improved pain from 7/10 to 4/10); avoided opioids per VBC guideline; patient reports improved ability to walk and engage in activities. No adverse effects from pregabalin noted.'
Vague: 'History of pancreatitis' (during active disease phase)	**AVOID during active disease**; use only after documented sustained remission. Better example of misuse: Patient with active weight loss, elevated lipase, on PERT: **coding Z87.19 'history' is WRONG**. Use '**K86.1** Idiopathic chronic pancreatitis' instead.
Vague: 'Malnutrition' (no intervention)	Better: 'Moderate malnutrition (BMI 18.2, albumin 2.8 g/dL, prealbumin 19 mg/dL) attributed to EPI from chronic pancreatitis; referred to RDN for **high-protein**, **high-calorie nutritional plan**; PERT dose increased to 50,000 units lipase per meal (from 40,000); counseled on frequent small meals; weight trend to follow at next visit.'
Vague: 'Alcohol use' (no frequency/quantity/F10 code listed)	Better: 'Alcohol-induced chronic pancreatitis (**K86.0**); patient reports **2 standard drinks daily**, down from **prior 6–8/day**; moderate alcohol use disorder (**F10.20**) active. AUDIT-C 6; enrolled in outpatient addiction medicine and naltrexone 50 mg daily initiated today; will monitor abstinence progress and reinforce AAVBC CP education at next visit.'

ABBREVIATIONS: K86.1 = other chronic pancreatitis (idiopathic); K86.81 = exocrine pancreatic insufficiency; PERT = pancreatic enzyme replacement therapy; TID = three times daily; E13.35 = type 3c diabetes mellitus with background retinopathy; BID = twice daily; VBC = value-based care; Z87.19 = personal history of diseases of the digestive system (avoid during active disease); BMI = body mass index; EPI = exocrine pancreatic insufficiency; RDN = Registered Dietitian Nutritionist; K86.0 = alcohol-induced chronic pancreatitis; F10.20 = alcohol use disorder, moderate, active; AUDIT-C = Alcohol Use Disorders Identification Test—Consumption

CLINICAL PEARL: AVOIDING COMMON CP CODING ERRORS

The single most common CP documentation error is failing to code **K86.81** (exocrine pancreatic insufficiency) when EPI is present: this is the hallmark complication driving **malnutrition** and **frailty** in older adults,[2,15,26] and its absence from the chart signals to specialists and auditors that PERT management is **not being tracked**. The second most common error is miscoding alcohol-induced CP (**K86.0**) without sequencing F10.xx (alcohol use disorder) as the root cause at every visit: **fewer than half** of CP patients with alcohol etiology achieve sustained cessation, and compliance is lowest among those followed by primary care alone.[28] Documentation that names etiology (alcohol vs. idiopathic), EPI severity (elastase value), diabetes type (E13.xx pancreatogenic vs. E11.xx type 2), and functional status (weight trend, pain control) captures the real clinical picture and drives accurate coding.

EHR Workflow Tips

[EHR TIP — Smartphrase] Build a .cp dot phrase that auto-populates **etiology** (K86.0/K86.1), **M-ANNHEIM severity grade**, **fecal elastase value**, **PERT dose/response**, **diabetes type** (E13.xx vs E11.xx), **weight trend**, **pain score**, and **AUD status** (F10.xx if applicable): sourced from labs, imaging, and encounter notes; reduces unspecified K86.9 coding and supports MEAT documentation in a single paste

[EHR TIP — Smartphrase] Build **modular dot phrases** for common CP scenarios: .cp.epi (elastase value + PERT dose + response), .cp.t3cdm (insulin regimen + HbA1c + hypoglycemia events), .cp.aud (AUDIT-C + alcohol quantity + naltrexone status), .cp.bone (DEXA T-score + Vitamin D level + supplementation), .cppain (severity + nonopioid regimen + functional status); each phrase **pulls structured data** and reduces free-text variability

[EHR TIP — Alert] No fecal elastase on file: configure a gap alert for **any active K86.0/K86.1 without a documented fecal elastase result**; surfaces patients who may have undiagnosed EPI and are not yet on PERT

[EHR TIP — Alert] No AUDIT-C documented: fire a secondary alert for **active K86.0 without AUDIT-C screening** at the current encounter; supports HEDIS ASF compliance and ensures AUD is addressed as root cause at every visit

[EHR TIP — Alert] No advance care planning documented: fire alert for active **K86.x + CFS ≥5 without ACP (CPT 99497)** on file; supports **MIPS #047** compliance and early goals-of-care documentation in frail CP patients

[EHR TIP — Best Practice] Problem list hygiene: maintain active K86.0 or K86.1 with K86.81 (EPI) **coded separately**, each associated condition coded individually (E13.xx T3cDM, F10.xx AUD, M81.x osteoporosis, E55.9 Vitamin D deficiency, E46 malnutrition); mark resolved conditions with resolution date to **prevent carry-forward coding errors**

[EHR TIP — Workflow] Annual diagnosis confirmation: flag any **active K86.x without a face-to-face encounter** and MEAT documentation in the past 12 months; fires at year-end to support HCC mapping and RAF continuity before the sweep deadline; annual reassessment should include updated elastase, DEXA (if prior osteopenia), CFS, and M-ANNHEIM grade

[EHR TIP — Order Set] CP initial workup bundle: serum lipase, fecal elastase, CT abdomen with contrast (or MRCP), CBC, CMP, Vitamin D 25-OH, HbA1c; new-diagnosis add-on bundle includes

gastroenterology referral, RDN referral, addiction medicine referral (if K86.0), and DEXA order: a**ll triggered at K86.x problem list entry**

[EHR TIP — Auto-prompt] Triggered **referral cascade at new K86.x diagnosis**: gastroenterology, registered dietitian, addiction medicine (if F10.xx); during ongoing management: **CFS ≥5** fires geriatric medicine + palliative care referral prompt; **pain ≥6/10** without documented nonopioid trial fires pain management referral prompt

Brief Case Examples

SUCCESS: COMPREHENSIVE DOCUMENTATION

SCENARIO

68-year-old male with 18-month history of mild epigastric discomfort after fatty meals, progressive weight loss (**12 lbs over 6 months**), and recent brittle fasting glucose (120–180 mg/dL) despite metformin monotherapy; **exam**: mild epigastric tenderness; labs: fasting glucose 187, HbA1c 8.2%, lipase 74 U/L (normal), albumin 3.1 g/dL; **CT abdomen**: pancreatic atrophy with subtle ductal dilation; **fecal elastase 156 mcg/g**; prior acute pancreatitis episode 15 years ago (presumed idiopathic); PCP recognized the weight loss + brittle glucose + low elastase pattern and initiated CP workup **rather than attributing symptoms to aging or T2DM progression**

Documentation: "Idiopathic chronic pancreatitis (**K86.1**), moderate severity per **M-ANNHEIM** (pancreatic atrophy + ductal dilation on CT; fecal elastase 156 mcg/g **confirming EPI**); exocrine pancreatic insufficiency (**K86.81**) = initiated PERT pancrelipase 40,000 units lipase TID with meals + omeprazole 20 mg daily; type 3c diabetes mellitus (E13.35) secondary to CP — transitioned from metformin to insulin glargine 20 units at bedtime given islet function loss; pain 2/10 managed with pregabalin 300 mg BID, opioids avoided; malnutrition (E46): albumin 3.1, referred to RDN for high-protein diet (1.2 g/kg/day); DEXA ordered, Vitamin D3 2,000 IU daily + calcium citrate 500 mg TID initiated; follow-up labs at 4 weeks (weight, stool pattern, glucose logs); elastase recheck at 8 weeks"

Outcome: K86.1 + K86.81 (EPI) + E13.35 (T3cDM) + E46 (malnutrition) all captured with clinical anchors (elastase value, PERT dose, insulin rationale, albumin level, weight trend); documentation supports HCC mapping for CP and T3cDM as separate conditions; at 3 months: stool frequency normalized, weight gained 4 lbs (prealbumin 19→23 mg/dL), fasting glucose 120–150 mg/dL with zero hypoglycemic episodes, pain 2/10 without opioids; patient returned to light activity; **averted ED visits through proactive PERT** dosing, **nutrition support**, and **T3cDM-specific insulin management**

PITFALL: INSUFFICIENT DOCUMENTATION

Documentation as written: "Chronic pancreatitis, on enzymes; diabetes on metformin; weight loss" — coded as K86.9 (unspecified); diabetes coded as E11.9 (T2DM); **no elastase value**, **no PERT dose**, **no EPI code**, **no M-ANNHEIM grade**, **no pain assessment**, **no nutrition status** documented

Consequence: Etiology unspecified (**K86.9 instead of K86.1**); EPI (**K86.81**) not coded despite active PERT use; T3cDM miscoded as T2DM (E11.9 instead of E13.35)= **misses insulin-first management** imperative and hypoglycemia risk; malnutrition (albumin 3.1) not captured; weight loss attributed to "aging" without investigation; no documentation of PERT dose, response, or adherence; chart **understates clinical complexity and impairs specialist coordination**

RAF Impact: Unspecified disease of pancreas (**K86.9; no HCC mapping**); missed comorbidity documentation underrepresents the patient's clinical reality and is ineligible for RAF. With proper documentation (C25.0 + C78.7 + EPI + secondary DM + biliary stent history), the record reflects the substantive multi-specialty work the team is doing

Fix: Update problem list and assessment: "Idiopathic chronic pancreatitis (**K86.1**), moderate per M-ANNHEIM; exocrine pancreatic insufficiency (**K86.81**), elastase 156 mcg/g, on PERT 40,000 units TID with response [describe]; type 3c diabetes (**E13.35**), on insulin glargine [dose], HbA1c [%], target 7–8%; malnutrition (E46), albumin 3.1, RDN referral placed; pain [score/10] on pregabalin [dose], opioid-free" — document MEAT for each condition at the next encounter; order DEXA and Vitamin D if not yet completed

REFERENCES

1.Tenner S, Vege SS, Sheth SG, et al. American College of Gastroenterology Guidelines: Management of Acute Pancreatitis. *Off J Am Coll Gastroenterol ACG*. 2024;119(3). https://journals.lww.com/ajg/fulltext/2024/03000/american_college_of_gastroenterology_guidelines_..aspx

2.Vege SS, Chari ST. Chronic Pancreatitis. *N Engl J Med*. 2022;386(9):869-878. doi:10.1056/NEJMcp1809396

3.Conwell DL, Lee LS, Yadav D, et al. American Pancreatic Association Practice Guidelines in Chronic Pancreatitis: evidence-based report on diagnostic guidelines. *Pancreas*. 2014;43(8):1143-1162. doi:10.1097/MPA.0000000000000237

4.Centers for Medicare & Medicaid Services. *2026 Final ICD-10-CM Mappings (CMS-HCC V28)*. U.S. Department of Health and Human Services; 2025. https://www.cms.gov/medicare/health-plans/medicareadvtgspecratestats/risk-adjustors/2026-model-software

5.Centers for Medicare & Medicaid Services. *CMS-HCC Model Relative Factor Tables (Community, Non-Dual, Aged)*. 2024. https://www.cms.gov/files/document/revised-cms-hcc-model-relative-factor-tablespdf

6.US Preventive Services Task Force. Unhealthy Alcohol Use in Adolescents and Adults: Screening and Behavioral Counseling Interventions. *JAMA*. 2018;320(18):1899-1909. doi:10.1001/jama.2018.16789

7.Yadav D, Lowenfels AB. The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*. 2013;144(6):1252-1261. doi:10.1053/j.gastro.2013.01.068

8.American Diabetes Association Professional Practice Committee. 2. Diagnosis and Classification of Diabetes: Standards of Care in Diabetes—2024. *Diabetes Care*. 2023;47(Supplement_1):S20-S42. doi:10.2337/dc24-S002

9.Lee LS, Gardner TB. Pancreatic Cysts and Pancreatitis in the Older Adult. *Am J Gastroenterol*. 2025;120(Suppl 10):S27-S33. doi:10.14309/ajg.0000000000003641.04

10.Cohen SM, Kent TS. Etiology, Diagnosis, and Modern Management of Chronic Pancreatitis: A Systematic Review. *JAMA Surg*. 2023;158(6):652-661. doi:10.1001/jamasurg.2023.0367

11.Centers for Medicare & Medicaid Services (CMS), National Center for Health Statistics (NCHS), American Hospital Association (AHA), American Health Information Management Association (AHIMA). *ICD-10-CM Official Guidelines for Coding and Reporting, FY 2026*. U.S. Department of Health and Human Services; 2025. https://www.cms.gov/medicare/coding-billing/icd-10-codes

12.Hirth M, Härtel N, Weiss C, et al. Clinical Course of Chronic Pancreatitis in Elderly Patients. *Digestion*. 2019;100(3):152-159. doi:10.1159/000494349

13.Park JY, Bang S, Jeon TJ, Cho JH, Lee KJ. Risk of and factors influencing the progression from acute to recurrent acute to chronic pancreatitis. *Pancreatol Off J Int Assoc Pancreatol IAP Al*. 2025;25(5): 624-630. doi:10.1016/j.pan.2025.04.004

14.Beyer G, Habtezion A, Werner J, Lerch MM, Mayerle J. Chronic pancreatitis. *Lancet Lond Engl*. 2020;396(10249):499-512. doi:10.1016/S0140-6736(20)31318-0

15.Singh VK, Yadav D, Garg PK. Diagnosis and Management of Chronic Pancreatitis: A Review. *JAMA*. 2019;322(24):2422-2434. doi:10.1001/jama.2019.19411

16.Sankaran SJ, Xiao AY, Wu LM, Windsor JA, Forsmark CE, Petrov MS. Frequency of progression from acute to chronic pancreatitis and risk factors: a meta-analysis. *Gastroenterology*. 2015;149(6): 1490-1500.e. doi:10.1053/j.gastro.2015.07.066

17.Whitcomb DC, Buchner AM, Forsmark CE. AGA Clinical Practice Update on the Epidemiology, Evaluation, and Management of Exocrine Pancreatic Insufficiency: Expert Review. *Gastroenterology*. 2023;165(5):1292-1301. doi:10.1053/j.gastro.2023.07.007

18. Sirtl S, Bretthauer K, Ahmad M, et al. Severity of Gallstone-, Sludge-, or Microlithiasis-Induced Pancreatitis-All of the Same? *Pancreas*. 2024;53(8):e633-e640. doi:10.1097/MPA.0000000000002349

19. Żorniak M, Sirtl S, Beyer G, et al. Consensus definition of sludge and microlithiasis as a possible cause of pancreatitis. *Gut*. 2023;72(10):1919-1926. doi:10.1136/gutjnl-2022-327955

20.Twohig PA, de- Madaria E, Thakkar S, et al. Quantifying the Risk of Drug-Induced Pancreatitis With Angiotensin-Converting Enzyme Inhibitors and Statins Using a Large Electronic Medical Record Database. *Pancreas*. 2021;50(8):1212-1217. doi:10.1097/MPA.0000000000001895

21.Chadalavada P, Simons-Linares CR, Chahal P. Drug-induced acute pancreatitis: Prevalence, Causative agents, and Outcomes. *Pancreatol Off J Int Assoc Pancreatol IAP Al*. 2020;20(7):1281-1286. doi: 10.1016/j.pan.2020.07.401

22. Thierens ND, Verdonk RC, Löhr JM, van Santvoort HC, Bouwense SA, van Hooft JE. Chronic pancreatitis. *Lancet Lond Engl*. 2025;404(10471):2605-2618. doi:10.1016/S0140-6736(24)02187-1

23.Hart PA, Zen Y, Chari ST. Recent Advances in Autoimmune Pancreatitis. *Gastroenterology*. 2015;149(1):39-51. doi:10.1053/j.gastro.2015.03.010

24. Vujasinovic M, Asplund E, Kourie M, et al. Painless chronic pancreatitis: experiences from a high-volume center. *Scand J Gastroenterol*. 2023;58(4):417-421. doi:10.1080/00365521.2022.2137692

25. Yousefli Z, Kassir Z, Faghih M, et al. Clinical Characteristics of Chronic Pancreatitis Patients Presenting With Acute Pancreatitis, Abdominal Pain, or Primary Painless Disease. *Pancreas*. 2026;55(1):e63-e69. doi:10.1097/MPA.0000000000002552

26.Gardner TB, Adler DG, Forsmark CE, Sauer BG, Taylor JR, Whitcomb DC. ACG Clinical Guideline: Chronic Pancreatitis. *Off J Am Coll Gastroenterol ACG*. 2020;115(3). https://journals.lww.com/ajg/fulltext/2020/03000/acg_clinical_guideline__chronic_pancreatitis..aspx

27.National Comprehensive Cancer Network. NCCN Clinical Practice Guidelines in Oncology (NCCN Guidelines®): Pancreatic Adenocarcinoma. April 22, 2026. https://www.nccn.org/professionals/physician_gls/pdf/pancreatic.pdf

28. Srivoleti P, Yang AL, Jin DX, Banks PA, McNabb-Baltar J. Provider type influences adherence to lifestyle changes in chronic pancreatitis. *Pancreatol Off J Int Assoc Pancreatol IAP Al*. 2021;21(1):42-45. doi: 10.1016/j.pan.2020.11.021

29. Parhiala M, Ukkonen M, Sand J, Laukkarinen J. Osteoporosis and sarcopenia are common and insufficiently diagnosed among chronic pancreatitis patients. *BMC Gastroenterol*. 2023;23(1):124. doi: 10.1186/s12876-023-02756-w

30.Koh A, Oyende O, Humes DJ, Lobo DN. Risk of osteopaenia, osteoporosis and osteoporotic fractures in patients with chronic pancreatitis: A systematic review and meta-analysis. *Clin Nutr Edinb Scotl*. 2023;42(7):1086-1094. doi:10.1016/j.clnu.2023.05.019

31.Duggan SN, Purcell C, Kilbane M, et al. An association between abnormal bone turnover, systemic inflammation, and osteoporosis in patients with chronic pancreatitis: a case-matched study. *Am J Gastroenterol*. 2015;110(2):336-345. doi:10.1038/ajg.2014.430

32.Kirkegård J, Mortensen FV, Cronin-Fenton D. Chronic Pancreatitis and Pancreatic Cancer Risk: A Systematic Review and Meta-analysis. *Am J Gastroenterol*. 2017;112(9):1366-1372. doi:10.1038/ajg.2017.218

33.Schneider A, Löhr JM, Singer MV. The M-ANNHEIM classification of chronic pancreatitis: introduction of a unifying classification system based on a review of previous classifications of the disease. *J Gastroenterol*. 2007;42(2):101-119. doi:10.1007/s00535-006-1945-4

34.Beyer G, Mahajan UM, Budde C, et al. Development and Validation of a Chronic Pancreatitis Prognosis Score in 2 Independent Cohorts. *Gastroenterology*. 2017;153(6):1544-1554.e. doi:10.1053/j.gastro.2017.08.073

35.Kumar V, Barkoudah E, Jin DX, Banks P, McNabb-Baltar J. Hospital Frailty Risk Score (HFRS) Predicts Adverse Outcomes Among Hospitalized Patients with Chronic Pancreatitis. *Dig Dis Sci*. 2023;68(7):2890-2898. doi:10.1007/s10620-023-07946-w

36.Kim Dae Hyun, Rockwood Kenneth. Frailty in Older Adults. *N Engl J Med*. 2024;391(6):538-548. doi: 10.1056/NEJMra2301292

37.Arvanitakis M, Ockenga J, Bezmarevic M, et al. ESPEN guideline on clinical nutrition in acute and chronic pancreatitis. *Clin Nutr Edinb Scotl*. 2020;39(3):612-631. doi:10.1016/j.clnu.2020.01.004

38.Aslanian HR, Lee JH, Canto MI. AGA Clinical Practice Update on Pancreas Cancer Screening in High-Risk Individuals: Expert Review. *Gastroenterology*. 2020;159(1):358-362. doi:10.1053/j.gastro.2020.03.088

39.Hart PA, Bellin MD, Andersen DK, et al. Type 3c (pancreatogenic) diabetes mellitus secondary to chronic pancreatitis and pancreatic cancer. *Lancet Gastroenterol Hepatol*. 2016;1(3):226-237. doi: 10.1016/S2468-1253(16)30106-6

40.Barkin JA, Barkin JS. Chronic Pancreatitis and Bone Disease. *J Clin Densitom Off J Int Soc Clin Densitom*. 2020;23(2):237-243. doi:10.1016/j.jocd.2019.08.004

41.Petrik ML, Freeman ML, Trikudanathan G. Multidisciplinary Care for Adults With Chronic Pancreatitis: Incorporating Psychological Therapies to Optimize Outcomes. *Pancreas*. 2022;51(1):. doi:10.1097/MPA.0000000000001953

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