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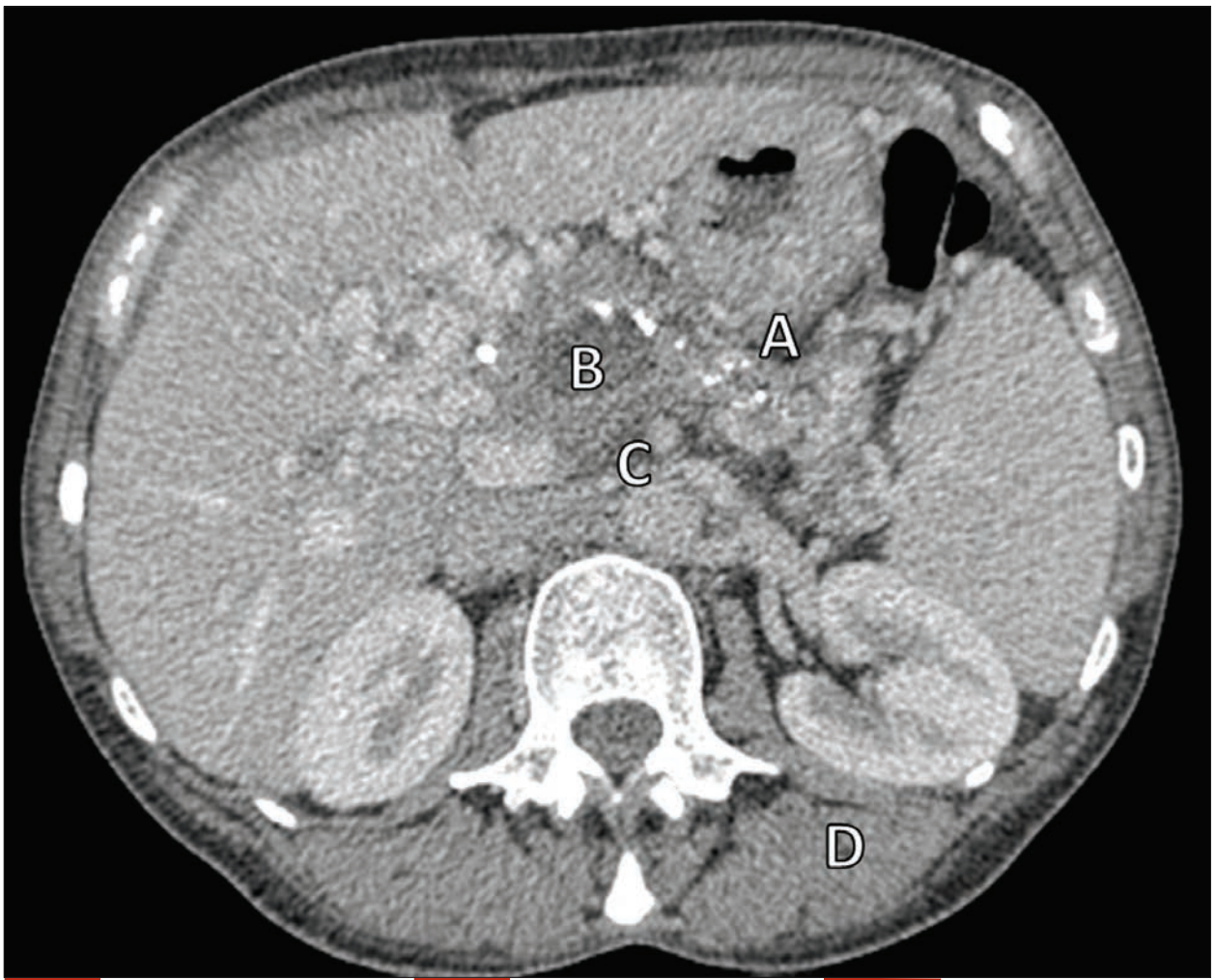

FIGURE 1
Computed tomography image of a patient with chronic pancreatitis: chronic inflammation leads to calcification (A) and fibrosis of the adjacent coeliac plexus (B) causing pain. Fibrosis with ductal strictures and pseudocysts of the pancreatic head (C) lead to exocrine insufficiency which combined with chronic pain leads to malnutrition and low muscle mass (D)

How do patients present in primary care?

CHRONIC PANCREATITIS IS AN IRREVERSIBLE, PROGRESSIVE CONDITION WHERE SCARRING OF the pancreas occurs in response to inflammation from many aetiologies including excessive alcohol consumption, smoking, autoimmune disease and post acute severe pancreatitis (see table 1, p14).

It presents with relapsing, remitting upper abdominal pain accompanied by features of malabsorption and malnutrition due to pancreatic exocrine insufficiency and endocrine deficiency with diabetes.

How should patients be investigated?

Recognising chronic pancreatitis can be challenging especially in its early stages and patients require multidisciplinary management. GPs, in conjunction with secondary care colleagues, have a crucial role in managing the often complex needs of these individuals.

What are the management strategies?

CAUSES AND RISK FACTORS

Chronic pancreatitis is a progressive condition characterised by irreversible fibrosis of the pancreatic parenchyma, accompanied by calcification and dilatation of the main pancreatic duct and its branches.[1] The prevalence of chronic pancreatitis is variable, with estimates between 4 per 100,000 population (in a UK study) and 52.4 per 100,000.[2-5] A mismatch exists between reported incidence and prevalence in many secondary care studies »

Table 1

Classification of chronic pancreatitis[1]

Toxic-metabolic
- Alcohol
- Tobacco smoking
- Hypercalcaemia
- Chronic renal failure
- Medication (azathioprine, tetracycline, valproate, oestrogens)

Idiopathic
- Early onset
- Late onset
- Tropical

Genetic
- Autosomal dominant
- Autosomal recessive (cystic fibrosis)

Autoimmune
- Isolated autoimmune chronic pancreatitis
- Syndromic associations (Sjögren's syndrome, inflammatory bowel disease, primary biliary cirrhosis)

Recurrent and severe acute pancreatitis
- Post necrotic and severe acute pancreatitis
- Post radiotherapy
- Ischaemia

Obstructive
- Pancreatic divisum
- Duct obstruction (tumour or stones)

suggesting chronic pancreatitis is under recognised, especially with historical post-mortem series reporting prevalence rates up to 13%.

One cause for this mismatch is that once diagnosed many patients are lost to secondary care follow-up. Therefore, although a GP may only see two new cases during their career they are likely to encounter patients requiring recurrent consultations.[3,5,6]

Fibrotic changes in the pancreas develop following multiple inflammatory insults and can be due to a variety of causes, see table 1, above. However, excessive alcohol consumption is the most common cause and studies have estimated it to account for 45-84% of cases in the Western world.[3,7] Smoking has also been shown to be a major independent, dose-related risk factor for chronic pancreatitis.[5,7]

Table 2

Differential diagnosis of chronic pancreatitis[15]

- Acute cholecystitis
- Biliary colic
- Acute pancreatitis
- Irritable bowel syndrome
- Peptic ulcer disease
- Pancreatic cancer
- Post-herpetic neuralgia
- Gastroparesis
- Intestinal obstruction, ischaemia, or infarction
- Abdominal aortic aneurysm
- Thoracic radiculopathy
- Myocardial infarction

PRESENTATION

Patients with chronic pancreatitis can present insidiously and are challenging to diagnose especially in the early phases. Symptoms including upper abdominal pain, weight loss and loose stools are non specific and can be attributed to other conditions, see table 2, above.[8]

Classically the pain is localised to the epigastrium, radiates to the back or flanks and is described as dull in nature. Duration and frequency of pain episodes vary but as the disease progresses attacks can intensify and lengthen.[9]

The timing of the development of symptoms from pancreatic exocrine insufficiency (diarrhoea and malnutrition) and endocrine failure (diabetes) can vary significantly, either preceding the onset of pain or taking many years to manifest.[10] Diabetes is more likely to develop as the duration of chronic pancreatitis increases, where pancreatic exocrine insufficiency has developed after pancreatic resection and in those with early onset calcification.[11]

Rarely, chronic pancreatitis can present with jaundice from biliary obstruction or cholangitis due to inflammation of the pancreas around the bile duct. Clinical distinction between chronic pancreatitis and pancreatic cancer can be difficult. Urgent referral for investigation should be made where the possibility of underlying pancreatic cancer exists.[12]

ASSESSMENT AND SCREENING FOR COMPLICATIONS

When first assessing patients with symptoms of chronic pancreatitis it is helpful to start by excluding other differential diagnoses, see table 2, above. Clinical suspicion of chronic pancreatitis alone is insufficient for diagnosis and a composite assessment is necessary. Symptoms and clinical history, cross-sectional imaging, computed tomography (CT), and assessment of nutritional status is recommended, see table 3, opposite[13] with referral to secondary care for confirmation of diagnosis, see table 4, opposite.

A histological diagnosis is possible for chronic pancreatitis but a biopsy is rarely performed because of its invasive nature, risk of complications and poor intra-observer agreement of chronic pancreatitis features.[14]

NICE guidance on the management of chronic pancreatitis in primary care advocates blood tests and supports abdominal ultrasound to exclude gallstones as a cause.

CT is the preferred first-line diagnostic investigation especially where there is a history of alcohol misuse.[15] The sensitivity of CT exceeds that of ultrasound, (75-90% vs 60-70%) although the specificity is similar 85% vs 80-90%.[16] Magnetic resonance imaging (MRI) is used for detailed ductal assessment in secondary care.[17]

Full blood count (FBC) and biochemistry may be normal, but liver function can be affected if there is concomitant liver disease, or in the instance of common bile duct compression by strictures or pseudocysts. HbA_{1c} or fasting glucose should be checked to screen for diabetes.[11,18] Serum tumour markers and amylase testing are not helpful in the diagnosis of chronic pancreatitis and should not be performed, although amylase remains useful in diagnosing acute pancreatitis.[15]

Malnutrition is a major complication resulting from fat and protein malabsorption in addition to deficiencies of vitamins A, D, E and K.[19] Up to 63% of chronic pancreatitis patients have fat soluble vitamin deficiencies.[19,20] Vitamin D deficiency occurs in many patients, and also in the general population, and although its presence is not diagnostic, deficiencies should be corrected.[21] Several serum markers have been associated with pancreatic exocrine insufficiency including magnesium, haemoglobin, albumin, pre albumin and retinol binding protein A.[22] Vitamin E has been shown to be the single most useful parameter to check as a marker of malnutrition,[22] see table 3, oppsite.

Osteoporosis and a high prevalence of low trauma fracture are seen in patients with chronic pancreatitis with one meta-analysis reporting 65% of chronic

pancreatitis patients affected by osteoporosis or osteopenia.[23] In addition to the reduction in bone mass, patients with chronic pancreatitis have been shown to have significant reductions in lean body mass and fat mass despite normal body mass index scores,[20] see figure 1, p13.

Exocrine function can be assessed with faecal elastase-1 (FEL-1). This is an indirect test of pancreatic function using FEL-1 as a surrogate. Other methods to assess pancreatic function directly have been described but are highly invasive and not widely available. FEL-1 measurement is easily performed; patients collect a pea-sized amount of faeces for analysis. It is highly specific and sensitive in severe disease but less sensitive and specific in mild to moderate disease.[24] It is felt to be the most appropriate first-line investigation for screening for pancreatic exocrine insufficiency in at-risk patients.[25]

Patients with chronic pancreatitis are at high risk of pancreatic cancer[26] and have increased mortality largely due to cardiovascular complications.[27] A reduction of 20-30% in ten-year survival is seen in chronic pancreatitis compared with the general population,[28] so continued follow-up is required.

MANAGEMENT

NICE recommendations for management of patients with alcohol-related chronic pancreatitis as well as international guidelines advocate a multidisciplinary approach.[13,17,29] The treatment of chronic pancreatitis is the same regardless of aetiology.

Aims of therapy include pain control, management of exocrine and endocrine insufficiency and management of complications.[30] Input from chronic pain teams, dietetics and diabetologists can be beneficial for complex pain and management of exocrine and endocrine insufficiency respectively. Surgery may be indicated for selected patients.

Complete abstinence from alcohol and smoking cessation is essential for all patients to slow disease progression[31] and improve pain.[7,32]

Chronic pain and the burden of incurable disease may affect relationships and social interactions and patients with chronic pancreatitis are prone to social isolation. Support groups may be of benefit where knowledge and experiences can be shared to boost coping strategies.[8] Patients with psychosocial problems may struggle to attend clinics and comply with treatment; often presenting as emergencies which adds to the management challenges.[29]

Pain can initially be managed with simple analgesia with the addition of a weak opioid if necessary. It is not uncommon for patients with chronic pancreatitis to require rapidly increasing doses of strong opioids risking the development of tolerance and hyperalgesic side effects. Adjuncts such as gabapentin tricyclic antidepressants have been shown to be beneficial.[33] Care should be taken when prescribing to avoid polypharmacy and opioid dependence. Strong opioid prescribing should be avoided in primary care if possible but, if considered, it should prompt a referral to secondary care to review alternatives. Many patients with chronic pancreatitis with a history of alcohol misuse could be at high risk for »

Table 3

Summary of recommended investigations at diagnosis and follow-up[13,15,43,44]

Investigation	At diagnosis	Follow-up
Abdominal ultrasound	Helpful to exclude gallstones	Not routinely
Computed tomography	Recommended	Not routinely Only if concern regarding development of complications or suspicion of malignancy
Liver function tests	Exclude biliary obstruction or coexistent liver disease	Annual
Fasting glucose/HbA_{1c}	Recommended	Annual screening for endocrine insufficiency
Other blood tests		Annual full blood count, prothrombin time, albumin, vitamin D, vitamin A, selenium, zinc, vitamin E
Quantitative faecal fat (faecal elastase-1)	Recommended	Annual screening for exocrine insufficiency
Bone mineral density assessment	Recommended	Every 1-2 years if osteopenia at baseline

Table 4

Referral of chronic pancreatitis to secondary care[15]

Routine referral Gastroenterology or specialist pancreatic centre	Confirmation of diagnosis or recurrent attacks of acute pancreatitis
Urgent referral	Complications of chronic pancreatitis Pancreatic cancer, pseudocyst, biliary obstruction, duodenal or gastric outlet obstruction, fistulae, splenic or portal vein thrombosis
Urgent admission	Episode of acute pancreatitis
Dietitian	Nutritional assessment and supporting treatment of exocrine insufficiency
Diabetologist	Management of diabetes mellitus secondary to chronic pancreatitis

key points

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Chronic pancreatitis is a progressive inflammatory condition characterised by irreversible fibrosis of the pancreatic parenchyma, accompanied by calcification and dilatation of the main pancreatic duct and its branches. There are multiple aetiologies including excessive alcohol consumption, smoking, autoimmune disease and post acute severe pancreatitis. However, excessive alcohol consumption is the most common cause.

The prevalence of chronic pancreatitis is variable, with estimates between 4 and 52.4 per 100,000. A mismatch exists between reported incidence and prevalence in many studies suggesting chronic pancreatitis is under recognised. One cause for this mismatch is that once diagnosed many patients are lost to secondary care follow-up. Therefore, although a GP may only see two new cases during their career they are likely to encounter patients requiring recurrent consultations.

Chronic pancreatitis presents with relapsing, remitting upper abdominal pain accompanied by features of malabsorption and malnutrition due to pancreatic exocrine insufficiency and diabetes due to endocrine deficiency. Diabetes is more likely to occur as the duration of chronic pancreatitis increases, where pancreatic exocrine insufficiency has developed after pancreatic resection and in those with early onset calcification.

Patients with suspected chronic pancreatitis should be referred to gastroenterology or a specialist pancreatic centre for confirmation of the diagnosis. Providing there is no need for urgent admission or referral, NICE recommends that GPs should arrange blood tests and abdominal ultrasound to exclude other conditions such as gallstones. In secondary care, CT is the preferred diagnostic investigation especially if there is a history of alcohol misuse. Faecal elastase-1 is the first-line investigation for screening for pancreatic exocrine insufficiency.

Complete abstinence from alcohol and smoking cessation is essential for all patients to slow disease progression and improve pain. Patients with evidence of malnutrition, malabsorption or pancreatic exocrine insufficiency should be treated with pancreatic enzyme replacement therapy to improve symptoms, nutritional status and quality of life.

Initially pain should be managed by prescribing simple analgesia with the addition of a weak opioid if necessary. Adjuncts such as gabapentin and tricyclic antidepressants have been shown to be beneficial. Care should be taken when prescribing to avoid polypharmacy and opioid dependence. Strong opioid prescribing should be avoided in primary care if possible but, if considered, it should prompt a referral to secondary care to review alternatives. Uncontrolled pain should trigger referral to secondary care for multidisciplinary assessment by physicians, surgeons and specialist pain clinics.

Table 5

Guidance for pancreatic enzyme replacement therapy (PERT)[33,34]

- PERT should be started in secondary care and prescribing transferred to primary care with agreement from the GP
- Starting dose of 50,000 units with meals and 25,000 units with snacks recommended but may need increasing
- Gastric acid suppression with proton pump inhibitors or H_2-receptor antagonists may be necessary to improve absorption
- Most enzyme supplements are porcine but have been approved for use by both Jewish and Islamic communities
- PERT should not be prescribed for pain alone

rapid development of dependence symptoms.

A trend towards significant pain relief from improved nutrition and pancreatic enzyme replacement therapy (PERT) has been shown in one systematic review,[34] although PERT is not recommended for pain alone in chronic pancreatitis.[15] In selected cases, jejunal feeding has been shown to improve pain, although oral nutrition remains the preferred route.[35]

Uncontrolled pain should trigger referral to secondary care for multidisciplinary assessment by physicians, surgeons and specialist pain clinics.[13] Endoscopic and surgical treatments should be considered as an adjunct to medical therapy in all cases initially or if pain symptoms increase, especially before opioid escalation occurs. Endoscopic therapy to dilate strictures, remove stones or drain pseudocysts can improve pain.[17] Endoscopic ultrasound-guided coeliac plexus block has been shown to reduce pain in up to 55% of patients with chronic pancreatitis so can be considered in the tertiary care setting.[36]

Surgery can be considered in cases of main pancreatic duct dilatation to remove obstruction (pancreatic jejunostomy) providing short-term benefit in up to 80% of patients, with 60% reporting benefit for more than two years.[37] Pancreatic resection is indicated in the presence of pain and small duct disease or enlargement of the pancreatic head, or if previous drainage procedures have failed.[8]

Surgery for chronic pancreatitis is a significant consideration with morbidity rates of 30-60% although mortality is less than 5%.[38] Recovery includes readjustment to opioid dosing and adjusting to life with insulin. Patients should be counselled extensively prior to undertaking surgery so that they are fully aware of the risks and benefits as well as the possibility that improvement is not guaranteed.[8]

Patients with evidence of malnutrition, malabsorption or pancreatic exocrine insufficiency should be treated with PERT to improve symptoms, nutritional status and quality of life,[33,39] see table 5, above.

The majority (80%) of chronic pancreatitis patients can be managed with a normal diet and PERT, with 10-15% requiring nutritional supplementation and 5% requiring enteral tube feeding.[40] Interventions providing balanced dietary advice have been shown to be as effective as providing supplements to optimise malnutrition in patients with chronic pancreatitis so early input from a dietitian is vital.[41]

The majority of patients can be managed with a normal diet (30% fat content) and PERT. However, if calorie intake is low, protein supplementation can be considered.[40]

It has been suggested that baseline bone density assessment is performed in all individuals with chronic pancreatitis given the high risk of osteoporosis, fractures and the associated morbidity and cost. In addition, advice regarding weight-bearing exercise, smoking and alcohol cessation and dietary advice to maximise calcium and vitamin D intake should be provided as standard.[42]

FOLLOW-UP

Patients should remain on at least yearly review which should include: routine blood tests, liver function tests (cholestasis or biliary obstruction), and markers of malnutrition (albumin,

coagulation, FBC),[43] see table 3, p15. If the patient is not already known to have pancreatic exocrine insufficiency, stool testing for steatorrhoea with quantitative measures such as FEL-1 should be used.[43]

Annual screening for diabetes mellitus with HbA_{1c} should be offered.[18] Additionally, osteoporosis screening, every one to two years, should be considered for those with malnutrition or high-risk indicators (previous low trauma fracture).[43] There is no role for routine pancreatic cancer screening, however, an urgent suspected cancer pathway referral to gastroenterology or hepatobiliary surgery should be made for patients with unexplained progressive weight loss.[44] American guidelines advocate starting cancer screening only in those with hereditary pancreatitis and in chronic pancreatitis patients with a family history of pancreatic cancer. [45]

Ongoing support in primary care is vital to help with smoking cessation, abstinence from alcohol and treatment compliance. The management of this patient group is challenging especially in preventing opiate dependence.

CONCLUSION

Chronic pancreatitis has multiple causes, the most common of which is excessive alcohol consumption. Patients present with severe pain among other features including malnutrition and weight loss.

Early identification of chronic pancreatitis can be challenging and patients are at risk of complications such as osteoporosis, diabetes and pancreatic cancer.

Diagnosis should be confirmed in secondary care, but input from community and primary care is vital for ongoing care with a multidisciplinary team approach early on to help guide treatment and manage complications.

Competing interests: None

REFERENCES

1 Etemad B, Whitcomb DC. Chronic pancreatitis: Diagnosis, classification, and new genetic developments. *Gastroenterology* 2001;120(3):682-707
2 Wang LW, Li ZS, Li SD et al. Prevalence and clinical features of chronic pancreatitis in China: a retrospective multicenter analysis over 10 years. *Pancreas* 2009;38(3):248-54
3 Levy P, Barthet M, Mollard BR et al. Estimation of the prevalence and incidence of chronic pancreatitis and its complications - A prospective survey in adults attending gastroenterologists in France. *Gastroenterol Clin Biol* 2006;30(6-7):838-44
4 Hirota M, Shimosegawa T, Masamune A et al. The sixth nationwide epidemiological survey of chronic pancreatitis in Japan. *Pancreatology* 2012;12(2):79-84
5 Lévy P, Domínguez-Muñoz E, Imrie C et al. Epidemiology of chronic pancreatitis: burden of the disease and consequences. *United European Gastroenterol J* 2014;2(5):345-54
5 Dominguez-Munoz JE, Lucendo A, Carballo LF et al. A Spanish multicenter study to estimate the prevalence and incidence of chronic pancreatitis and its complications. *Rev Esp Enferm Dig* 2014;106(4):239-45
6 Olsen TS. Incidence and clinical relevance of chronic inflammation in pancreas in autopsy material. *Acta Pathol Microbiol Scand A* 1978;86(5):361-65
7 Cote GA, Yadav D, Slivka A et al. Alcohol and smoking as risk factors in an epidemiology study of patients with chronic pancreatitis. *Clin Gastroenterol Hepatol* 2011;9(3):266-73
8 Duggan SN, Chonchubhair HMN, Lawal O et al. Chronic pancreatitis: A diagnostic dilemma. *World J Gastroenterol* 2016;22(7):2304-13
9 Goulden MR. The pain of chronic pancreatitis: a persistent clinical challenge. *Br J Pain* 2013; 7(1):8-22
10 Rebours V, Boutron-Ruault MC, Schnee M et al. The natural history of hereditary pancreatitis: a national series. *Gut* 2009;58(1):97-103
11 Rickels MR, Bellin M, Toledo FGS et al. Detection, evaluation and treatment of diabetes mellitus in chronic pancreatitis: Recommendations from PancreasFest 2012. *Pancreatology* 2013;13(4):336-42
12 Adsay NV, Bandyopadhyay S, Basturk O et al. Chronic pancreatitis or pancreatic ductal adenocarcinoma? *Sem Diagnost Pathol* 2004;21(4):268-76
13 National Institute for Health and Care Excellence. CG100. Alcohol-use disorders: diagnosis and management of physical complications. NICE. London. 2010 (Updated April 2017)
14 Ammann RW, Muellhaupt B. The natural history of pain in alcoholic chronic pancreatitis. *Gastroenterology* 1999;116(5):1132-40
15 National Institute for Health and Care Excellence. Clinical Knowledge Summaries: Pancreatitis- chronic, management of suspected chronic pancreatitis. NICE. London. 2016
16 Bolondi L, Li Bassi S, Gaiani S et al. Sonography of chronic pancreatitis. *Radiol Clin North Am* 1989;27(4):815-33
17 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):1143-62
18 Ewald N, Hardt PD. Diagnosis and treatment of diabetes mellitus in chronic pancreatitis. *World J Gastroenterol* 2013;19(42):7276-81
19 Duggan SN, Smyth ND, O'Sullivan M et al. The prevalence of malnutrition and fat-soluble vitamin deficiencies in chronic pancreatitis. *Nutr Clin Pract* 2014;29(3):348-54
20 Haaber AB, Rosenfalck AM, Hansen B et al. Bone mineral metabolism, bone mineral density, and body composition in patients with chronic pancreatitis and pancreatic exocrine insufficiency. *Int J Pancreatol* 2000;27(1):21-7
21 Lindkvist B, Phillips ME, Dominguez-Munoz J. Clinical, anthropometric and laboratory nutritional markers of pancreatic exocrine insufficiency: Prevalence and diagnostic use. *Pancreatology* 2015;15:589-97
22 Lindkvist B, Domínguez-Munoz JE, Luaces-Regueira M et al. Serum nutritional markers for prediction of pancreatic exocrine insufficiency in chronic pancreatitis. *Pancreatology* 2012;12(4):305-10
23 Duggan SN, Smyth ND, Murphy A et al. High prevalence of osteoporosis in patients with chronic pancreatitis: a systematic review and meta-analysis. *Clin Gastroenterol Hepatol* 2014;12(2):219-28
24 Loser C, Mollgaard A, Folsch UR. Faecal elastase 1: a novel, highly sensitive, and specific tubeless pancreatic function test. *Gut* 1996;39(4):580-6
25 Dominguez-Munoz JE, Hardt PD, Lerch MM et al. Potential for screening for pancreatic exocrine insufficiency using the fecal elastase-1 test. *Dig Dis Sci* 2017;62(5):1119-30
26 Raimondi S, Lowenfels AB, Morselli-Labate AM et al. Pancreatic cancer in chronic pancreatitis; aetiology, incidence, and early detection. *Best Prac Res Clin Gastroenterol* 2010;24(3):349-58
27 Bang UC, Benfield T, Hyldstrup L et al. Mortality, cancer, and comorbidities associated with chronic pancreatitis: A Danish nationwide matched-cohort study. *Gastroenterology* 2014;146(4):989-94
28 Seicean A, Tantau M, Grigorescu M et al. Mortality risk factors in chronic pancreatitis. *J Gastrointest Liver Dis* 2006;15:21-6
29 British Society of Gastroenterology. Commissioning evidence-based care for patients with gastrointestinal and liver disease. www.bsg.org.uk/clinical/commissioning-report/chronic-pancreatitis.html. Last accessed 1st May 2017
30 Callery MP, Freedman SD. A 21-year-old man with chronic pancreatitis. *JAMA* 2008; 299(13):1588-94
31 Talamini G, Bassi C, Falconi M et al. Smoking cessation at the clinical onset of chronic pancreatitis and risk of pancreatic calcifications. *Pancreas* 2007;35(4):320-6
32 Olesen SS, Juel J, Graversen C et al. Pharmacological pain management in chronic pancreatitis. *World J Gastroenterol* 2013;19(42):7292-301
33 D'Haese JG, Ceyhan GO, Demir IE et al. Treatment options in painful chronic pancreatitis: a systematic review. *HPB* (Oxford). 2014;16(6):512-21
34 Taylor JR, Gardner TB, Waljee AK et al. Systematic review: efficacy and safety of pancreatic enzyme supplements for exocrine pancreatic insufficiency. *Alim Pharmacol Therap* 2009;31(1):57-72
35 Stanga Z, Giger U, Marx A et al. Effect of jejunal long-term feeding in chronic pancreatitis. *J Parenteral Enter Nutr* 2005;29(1):12-20
36 Gress F, Schmitt C, Sherman S et al. Endoscopic ultrasound-guided celiac plexus block for managing abdominal pain associated with chronic pancreatitis: A prospective single center experience. *Am J Gastroenterol* 2001;96(2):409-16
37 Warshaw AL, Banks PA, Castillo CFD. AGA technical review: Treatment of pain in chronic pancreatitis. *Gastroenterology* 1998;115(3):765-76
38 Ho CK, Kleeff J, Friess H et al. Complications of pancreatic surgery. *HPB* (Oxford) 2005;77:99-108
39 Sandhu B, Sistrun S, Naniwadekar A et al. Good nutrition, as measured by Mynutritionindex, in chronic pancreatitis patients improves clinical outcome. *Gastroenterology* 2010;138([S392 (M1377)]):S392
40 Meier R, Ockenga J, Pertkiewicz M et al. ESPEN guidelines on enteral nutrition. *Pancreas Clin Nutr* 2006;25(2):275-84
41 Singh S, Midha S, Singh N et al. Dietary counseling versus dietary supplements for malnutrition in chronic pancreatitis: A randomized controlled trial. *Clin Gastroenterol Hepatol* 2008;6(3):353-59
42 Duggan SN, Conlon KC. Bone health guidelines for patients with chronic pancreatitis. *Gastroenterology* 2013 Oct;145(4):911
43 BMJ. Best Practice - Chronic Pancreatitis. 2016. http://bestpractice.bmj.com/best-practice/monograph/67.html. Last accessed July 25th 2017
44 National Institute for Health and Care Excellence. Clinical Knowledge Summaries- Gastrointestinal tract (upper) cancers- recognition and referral. NICE. London. 2015 www.nice.org.uk
45 DiMagno EP, Reber HA, Tempero MA. AGA technical review on the epidemiology, diagnosis, and treatment of pancreatic ductal adenocarcinoma. *Gastroenterology* 1999;117(6):1464-84

Useful information

Core
Patient information on chronic pancreatitis
www.corecharity.org.uk/gut-liver-and-pancreas/pancreas/chronic-pancreatitis/

NICE Clinical Knowledge Summaries
Pancreatitis - Chronic: Management of suspected chronic pancreatitis, revised 2016
www.cks.nice.org.uk/pancreatitis-chronic

British Society of Gastroenterology
Commissioning evidence-based care for patients with gastrointestinal and liver disease
www.bsg.org.uk/clinical/commissioning-report/chronic-pancreatitis.html

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