Editorial

# A practical approach for the diagnosis and management of pancreatic cystic lesions—the Hong Kong consensus recommendations

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## Introduction

The incidental detection of pancreatic cystic lesions (PCLs) has increased in recent years, primarily due to improved imaging resolution and the broader use of cross-sectional imaging (1-3). International studies report a prevalence ranging from 2.2% to 44.7%, influenced by factors such as population, imaging modality, and the indication for imaging, with prevalence rising with age (1-4). While most PCLs do not progress to cancer, their high prevalence and uncertain malignant potential remain a cause for concern (3). The Hong Kong consensus recommendations aim to provide a comprehensive approach to diagnosing and managing patients with PCLs tailored to the clinical context of Hong Kong and East Asia (1).

## Classification of PCLs

PCLs are classified as either benign or neoplastic. Benign PCLs include simple cysts, lymphoepithelial cysts, and retention cysts, while neoplastic cysts can be further divided into serous PCLs, mucinous PCLs, and cystic degeneration of solid pancreatic tumors (3). Mucinous cysts have a mucin-producing epithelial lining, including intraductal papillary mucinous neoplasms (IPMNs) and mucinous cystic neoplasms (MCNs) (3). The key difference between IPMNs and MCNs is their connection to the pancreatic ductal system. MCNs do not communicate with the ducts, while IPMNs are connected to the main pancreatic duct (MD-IPMN) or its branches (BD-IPMN) (2-4). Common mixed solid and cystic lesions include cystic degeneration of neuroendocrine tumors, solid pseudopapillary tumors (SPTs), adenocarcinoma, and mucinous cystic lesions with a solid component (3). Accurate classification of PCLs is crucial as the risk of malignant transformation varies among the subtypes. MCNs are malignant in up to 34% of cases. Serous PCLs have a minimal risk of malignancy, while SPTs are considered malignant tumors with both local and metastatic potential (1,3,5). For IPMN, the Kyoto guidelines mention the mean rates of high-grade dysplasia (HGD) or invasive carcinoma (IC) in resected BD-IPMN and MD-IPMN are 31% (range, 15–48%) and 62% (range, 36–100%), respectively. Within these, the rates of IC are 19% (range, 6–38%) and 43% (range, 11–81%), respectively (4).

## Detection and diagnosis of PCLs

Several international organizations provide diagnostic guidelines for PCL (1,4-8). Mass screening for PCL is not

**Table 1** "High risk" and "Worrisome" features in intraductal papillary mucinous neoplasms

| |
|---|
| "High risk" features |
| Main pancreatic duct ≥10 mm |
| Obstructive jaundice |
| Solid mass |
| Cancer or high-grade dysplasia on cytology |
| Mural nodule ≥5 mm |
| "Worrisome" features |
| Main pancreatic duct 5–9 mm |
| Cyst ≥3 cm |
| Lymphadenopathy |
| ↑ Carbohydrate antigen 19-9 |
| Mural nodule <5 mm |
| Cyst growth ≥5 mm/2 years |
| Change in the caliber of pancreatic duct with distal pancreatic atrophy |
| Thickened/enhancing cyst walls |
| New-onset diabetes mellitus |

recommended (1). Genetic mutations like BRCA1 and BRCA2 increase the risk of pancreatic cancer (PC) around two- to six-fold. Patients with chronic pancreatitis have a 16-fold higher risk of PC than those without chronic pancreatitis, and patients with new-onset diabetes (NOD) have a 6- to 8-fold higher risk of PC (1). Other risk factors include smoking, obesity, and metabolic syndrome (1,3).

Contrast-enhanced computed tomography (CT) and magnetic resonance imaging (MRI) are preferred for diagnosing and monitoring PCLs, with MRI being the preferred choice due to the absence of ionizing radiation, which poses cancer risks, especially in younger patients requiring long-term surveillance and repeated imaging (1,3). The American College of Radiology (ACR) guidelines recommend including five elements in a radiology report: lesion size, presence of 'worrisome features' or 'high-risk stigmata', growth on serial imaging, and the multiplicity of PCLs (6). Endoscopic ultrasound (EUS) with fine needle aspiration (FNA) enables cyst cytology and fluid analysis, which includes testing for carcinoembryonic antigen (CEA), glucose, amylase, and next-generation markers. This can help differentiate mucinous from non-mucinous PCLs and aid in diagnosing PCLs with malignancy (1,4-8). EUS is usually recommended for suspected MCNs or IPMNs with worrisome features (*Table 1*). Intra-cystic glucose can differentiate mucinous from non-mucinous PCNs with 91% sensitivity and 86% specificity (1). A CEA level >192 ng/mL has a sensitivity of 50–75% and specificity of 84–92% for distinguishing mucinous from non-mucinous cysts (9). Emerging EUS-guided needle-based confocal laser endomicroscopy (nCLE) shows promise for improved risk stratification and diagnosis of HGD, although nCLE is not widely available (1).

## Recommendations for surveillance

"High-risk" clinical features include obstructive jaundice, recurrent pancreatitis, an elevated serum carbohydrate antigen 19-9 (CA19-9) level, or, if cytology is obtained, the presence of cells demonstrating HGD or neoplasia, and new-onset or worsening diabetes (1,3,4). "Worrisome" characteristics on imaging include main pancreatic duct dilation ≥5 mm, cyst size ≥3 cm, and presence of solid component or mural nodule in the PCL (1,4-8) (*Table 1*). PCLs that have the potential to become malignant are managed by active surveillance or surgical excision as long as the patient is fit for surgery (1,3). It is also recognized that surveillance-detected PC in patients with IPMNs is more commonly observed in the early stages (10). Recommendations for monitoring duration and intervals vary among expert groups, but generally, patients with larger cysts and higher-risk features should undergo more intensive follow-up. The Hong Kong consensus recommendations align with most other societal guidelines, noting that cyst size ≥3 cm is associated with a higher risk of HGD or IC. Additionally, NOD, history of pancreatitis, main pancreatic duct dilation ≥5 mm, presence of a mural nodule <5 mm, increasing cyst size (>5 mm/2 years), and elevated CA19-9 are considered worrisome features (1) (*Figure 1*).

The American Gastroenterological Association (AGA) recommends MRI surveillance every 1 year, then every 2 years, with discontinuation at 5 years or if surgery is no longer an option (8). The International Association of Pancreatology (IAP) advises lifelong CT/magnetic resonance cholangiopancreatography (MRCP) surveillance every 2 years, with more frequent follow-up for cysts >3 cm (4). The European Study Group on Cystic Tumors of the Pancreas (ESGCTP) suggests 6-monthly surveillance for 1 year, followed by annual monitoring if no risk factors exist (7). The Hong Kong consensus recommends monitoring for patients with IPMN or MCN who have no

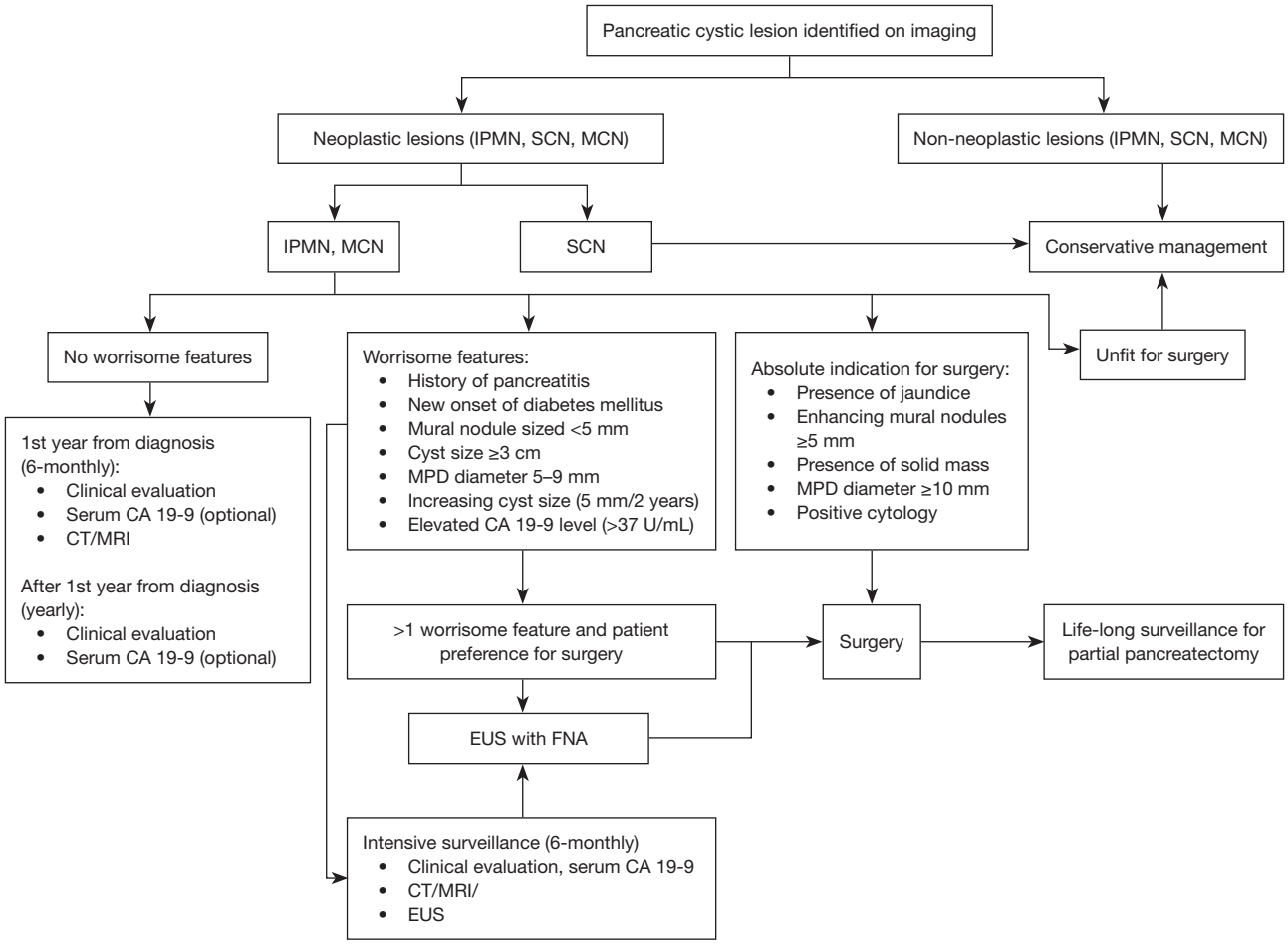


**Figure 1** Approach to surveillance and management of patients with PCL. Adapted from (1). CA19-9, carbohydrate antigen 19-9; CT, computed tomography; EUS, endoscopic ultrasound; FNA, fine needle aspiration; IPMN, intraductal papillary mucinous neoplasm; MCN, mucinous cystic neoplasm; MPD, main pancreatic duct; MRI, magnetic resonance imaging; PCL, pancreatic cystic lesions; SCN, serous cystic neoplasm.

indications for surgery or worrisome features. This includes clinical evaluation, MRI/CT imaging, and CA 19-9 tests every 6–12 months, followed by annual monitoring. Patients with worrisome features but no absolute surgical indication should have surveillance every 6 months (1). The potential risk of undetected malignant transformation must be weighed against surveillance's cost, inconvenience, and invasiveness. Experts recommend continuing surveillance until age 75 years, and individualizing follow-up for patients aged 76–85 years (4,5,7). Physicians should be aware that PC can arise either from the IPMN itself or from unaffected pancreatic tissue, a phenomenon known as a "field defect; therefore, long-term postoperative surveillance is essential" (1,11).

## Management and prognosis of PCL

Surgery may be considered for fit patients with symptomatic neoplastic PCLs. PCLs with high-risk features, such as main duct or mixed-type IPMN and SPTs, should be referred for surgical excision due to the significant risk of malignancy (1). Absolute indications for surgery include jaundice, mural nodules ≥5 mm, solid masses, main pancreatic duct ≥10 mm, and positive cytology in patients with IPMN (1,4-8). Also, patients with symptoms like pancreatitis, nausea, vomiting from intestinal obstruction, or abdominal discomfort should undergo surgical evaluation regardless of cancer risk (3). The Hong Kong consensus offers recommendations similar

 

to those of other societies. It also suggests that patients with multiple worrisome features may be candidates for surgery, depending on EUS findings and patient preferences (1). Surgery is recommended for fit patients when a PCL undergoes malignant transformation, and adjuvant chemotherapy is beneficial for most patients with resectable pancreatic ductal cancer following such transformation (1) (*Figure 1*).

NOD occurs in up to a quarter of patients after partial pancreatectomy (1). Following the procedure, low levels of insulin, glucagon, and other pancreatic polypeptides contribute to rapid fluctuations in glucose, sometimes referred to as 'brittle diabetes' or 'type 3c diabetes'. Management strategies are primarily based on recommendations for type 1 diabetes (12). Patients should be screened for diabetes using fasting plasma glucose (FPG) ≥126 mg/dL, hemoglobin A1c (HbA1c) ≥6.5%, or a 2-hour plasma glucose ≥200 mg/dL after a 75-g oral glucose tolerance test (13). Insulin replacement should be initiated when necessary, keeping in mind that patients with type 3c diabetes typically require lower insulin doses and are at a higher risk of hypoglycemia compared to those with type 1 diabetes (12).

Additionally, the incidence of pancreatic exocrine insufficiency (PEI) is higher following pancreaticoduodenectomy compared to distal pancreatectomy (1). For patients with PCLs, the diagnosis of post-pancreatectomy PEI is based on clinical and laboratory findings, typically including patient-reported changes in bowel function, weight loss, and other symptoms (1,14). While direct tests like the secretin-cholecystokinin stimulation or endoscopic function tests are accurate but costly and invasive, indirect tests (blood, fecal, or breath samples) are more affordable but less specific. In Hong Kong, PEI is usually diagnosed based on symptoms, and pancreatic enzyme replacement therapy (PERT) is initiated (1,14). In Hong Kong, the typical approach is to start patients on low doses of PERT (e.g., 20,000–30,000 PhU per meal) and gradually increase the dosage until symptoms resolve. Symptom assessment using tools like the Pancreatic Exocrine Insufficiency Questionnaire (PEI-Q) is recommended at diagnosis and during follow-up to monitor treatment response (1,15). For patients with poor response, increasing the PERT dose or adding proton pump inhibitors (PPIs) to reduce gastric acid and minimize enzyme degradation should be considered. Additionally, clinical evaluation for malnutrition should include screening for deficiencies in calcium, zinc, and liposoluble vitamins (1).

## Future directions and challenges

While the Hong Kong consensus provides a robust framework, challenges remain. The limited availability of advanced diagnostic tools like nCLE and the cost of long-term surveillance present significant barriers, particularly in resource-constrained settings. Further research into the genetic and molecular makeup of PCLs will pave the way for more precise, individualized management strategies.

Another area of opportunity lies in patient education. Empowering patients with knowledge about their condition and the rationale behind surveillance and treatment plans can enhance adherence and reduce anxiety associated with a PCL diagnosis.

## Conclusions

The Hong Kong consensus recommendations represent a comprehensive guide in management of PCLs. By integrating global best practices with regional insights, these guidelines provide a robust, patient-centered approach to a complex and increasingly prevalent condition. With continued advancement in scientific knowledge about the genetic and molecular makeup of PCLs, and innovation in developing precise diagnostic tests, we are inching closer to a future where the burden of PCLs is effectively mitigated.

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