

Pathology

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More than 85% of pancreatic cancers are ductal adenocarcinomas. The remaining tumours constitute a variety of pathologies with individual characteristics. Endocrine tumours of the pancreas are rare. These are covered in Chapter 57. Ductal adenocarcinomas arise most commonly in the head of the gland. They are solid, scirrhous tumours, characterised by neoplastic tubular glands within a markedly desmoplastic fibrous stroma. Fibrosis is also a characteristic of chronic pancreatitis, and histological differentiation between tumour and pancreatitis can be difficult. Ductal adenocarcinomas infiltrate locally, typically along nerve sheaths, along lymphatics and into blood vessels. Liver and peritoneal metastases are common. Proliferative lesions in the pancreatic ducts can precede invasive ductal adenocarcinoma. These are termed pancreatic intraepithelial neoplasia or PanIN, and can demonstrate a range of structural complexity and cellular atypia. Cystic tumours of the pancreas may be serous or mucinous. Serous cystadenomas are typically found in older women and are large aggregations of multiple small cysts, almost like bubble wrap. They are benign. Mucinous tumours, on the other hand, have the potential for malignant transformation. They include mucinous cystic neoplasms (MCNs) and intraductal papillary mucinous neoplasms (IPMNs). MCNs are seen in perimenopausal women, show up as multilocular thick-walled cysts in the pancreatic body or tail and, histologically, contain an ovarian-type stroma. IPMNs are more common in the pancreatic head and in older men, but an IPMN arising from a branch duct can be difficult to distinguish from an MCN. IPMNs arising within the main duct are often multifocal and have a greater tendency to prove malignant. Thick mucus seen extruding from the ampulla at ERCP is diagnostic of a main duct IPMN. Mucinous tumours can be confused with pseudo-

pancreatic cancer. Demographic factors Age (peak incidence 65–75 years) Male gender Black ethnicity Environment/lifestyle Cigarette smoking Genetic factors and medical conditions Family history Two first-degree relatives with pancreatic cancer: relative risk increases 18- to 57-fold Germline BRCA2 mutations in some rare high-risk families Hereditary pancreatitis (50- to 70-fold increased risk) Chronic pancreatitis (5- to 15-fold increased risk) Lynch syndrome (HNPCC) Ataxia telangiectasia Peutz-Jeghers syndrome Familial breast-ovarian cancer syndrome Familial atypical multiple mole melanoma Familial adenomatous polyposis – risk of ampullary/duodenal carcinoma Diabetes mellitus Obesity HNPCC, hereditary non-polyposis colorectal cancer.

cysts (Summary box 72.8 and Figure 72.32). Occasionally, lymphoepithelial cysts, lymphangiomas, dermoid cysts and intestinal duplication cysts can show up in the pancreas. Solid pseudopapillary neoplasms are rare, slowly progressive but malignant lesions seen in women of

childbearing age, and manifest as large, part-solid, part-cystic tumours. Tumours arising from the ampulla or from the distal common bile duct can present as a mass in the head of the pancreas and constitute around a third of all tumours in that area. Adenomas of the ampulla of Vater are diagnosed at endoscopy as polypoid submucosal masses covered by a smooth epithelium. They can harbour foci of invasive carcinoma; the larger the adenoma, the greater the risk. Biopsies taken at endoscopy may not always include the malignant focus. Endoscopic surveillance, endoscopic resection or even surgical transduodenal ampullary excision should be considered (Figure 72.33). Patients with familial adenomatous polyposis (FAP) can present with multiple duodenal polyps. Malignant transformation in a duodenal polyp is a significant cause of mortality in these patients, mandating endoscopic follow-up and pancreaticoduodenectomy in selected patients with high-grade dysplasia within the polyp. Ampullary adenocarcinomas often present early with biliary obstruction. Their natural history is distinctly more favourable than that of pancreatic ductal adenocarcinoma. Ampullary carcinomas are relatively small when diagnosed, which may account for their better prognosis. Occasionally, other malignant neoplasms can arise at the ampulla, such as carcinoid tumours and high-grade neuroendocrine carcinomas. -

Investigate with MRCP and
EUS+FNA Send fluid for CEA,
cytology (CEA ≥ 192 ng/mL
indicates mucinous neoplasm)
IPMN or MCN Relative indications
for surgery: Unfit for surgery No
indication for surgery Absolute
indications for surgery: • Growth
rate ≥ 5 mm/year • Carcinoma or
high-grade dysplasia • Serum

CA19-9 >37 U/L on cytology •
Main pancreatic duct dilated •
Solid mass 5–9.9 mm • Jaundice
(tumour related) • Cyst diameter
>40 mm • Enhancing mural
nodules ≥ 5 mm • New-onset
diabetes mellitus • Main
pancreatic duct dilated ≥ 10 mm •
Acute pancreatitis • Enhancing
mural nodules <5 mm Surgery
Acceptable surgical risk with 1 or 2
indications or high surgical risk but
>2 indications Surgery Figure
72.32 Management algorithm for
cystic neoplasms of the pancreas.
CA19-9, carbohydrate antigen 19-

9; CEA, carcinoembryonic anti gen;
EUS, endoscopic ultrasonography;
FNA, /f_i ne-needle aspiration;
IPMN, intraductal papillary
mucinous neoplasm; MCN,
mucinous cystic neoplasm; MRCP ,
magnetic resonance
cholangiography and
pancreatography; NET,
neuroendocrine tumour. (Adapted
fr Study Group on Cystic Tumours
of the Pancreas. European
evidence-based guidelines on
pancreatic cystic neoplasms. Solid
pseudopapillary Cystic NET Serous
cystic neoplasm neoplasm Other

benign cyst Consider surgery
Consider surgery Manage
conservatively High surgical risk
with 1 indication Monitor at 6 and
12 months Monitor 6-monthly and
then annually with: with: • Clinical
evaluation • Clinical evaluation •
Serum CA19-9 • Serum CA19-9 •
MRCP/EUS • MRCP/EUS

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