Intraductal papillary mucinous neoplasm of the pancreas: a key to diagnosis

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Learning objectives

Conduct an overall review of IPMN considering epidemiological data, concepts of anatomopathology and molecular biology, aspects of its natural history and manifestations in term of imaging. We placed particular emphasis on the key factors for correct diagnosis and classification, especially on findings that are suspicious for malignancy or invasion. The role of other complementary diagnostic techniques and management are also discussed.

Key factors for radiological diagnosis, classification and management are shown.
Background

Introduction

The role of diagnostic imaging techniques is crucial in focal pancreatic lesions, not only because radiologists often discover the injury as an incidental finding, but also because imaging is decisive in differential diagnosis.

This is particularly important in the wide range of pancreatic cystic lesions (PCL) that vary from benign congenital lesions to neoplasms that include Intraductal Papillary Mucinous Neoplasm (IPMN), a known precursor of pancreatic cancer.

IPMN was first described by Ohhashi in 1982 in endoscopic retrograde cholangiopancreatography (ERCP). The increase in the number of reported cases of IPMN in recent years may be due to a greater knowledge of the entity and technical improvements in imaging rather than to a real increase of its incidence.

IPMN, along with mucinous cystic neoplasms (MCN) and pancreatic intraepithelial lesions (PanIN), is a well-defined precursor for pancreatic cancer, which is a macroscopic radiographically detectable entity, unlike PanIN which is more frequent but not visible radiographically.

Description

The term adopted by the World Health Organization (WHO) in 2004 to describe this entity is Intraductal Papillary Mucinous Neoplasm (IPMN).

Classification

Imaging techniques allow classification of IPMNs and definitive diagnosis is based on pathological findings. This classification has implications for prognosis and clinical management and depends on localisation:

- Main-duct IPMN (MD-IPMN): Macroscopically, dilatation of the main pancreatic duct (MPD) may be diffuse or segmental, associated with intraluminal mucin accumulation. It may present papillary projections, calcifications and varying degrees of glandular atrophy. Associated to malignancy (in situ or invasive carcinoma) in 57-92%.

Fig. 1
• Branch duct IPMN (BD-IPMN): Dilatation of the lateral branches of MPD, which usually adopt the shape of a cyst. Communication between the cyst and the MPD. Lesions are multifocal in slightly over 60% of cases. Associated with malignancy in 6-46%. The rate of invasive carcinoma is 15%.

Fig. 2

• Mixed IPMN (M-IPMN): Presents dilatation of the main duct and the branches.

Fig. 3

**Epidemiology**

IPMN is increasingly recognised but its real incidence remains unknown. It accounts for 0.5% of all pancreatic neoplasms found in autopsies and 7.5% of all diagnosed pancreatic lesions. No difference in the incidence of IPMN is found between men and women, with peak incidence in the 6th decade of life.

**Pathology, molecular genetics, natural history**

Histologically, a proliferation of neoplastic mucinous cells is observed. They form papillae and lead to the dilatation of the main duct or its branches due to endoluminal mucin accumulation.

Fig. 4

Depending on the epithelium, different histological subtypes with different prognoses have been characterised:

• Gastric foveolar type: Almost always low-grade lesions. Mucin secretion: positive for MUC5AC and MUC6, and negative for MUC1 and MUC2.
• Intestinal type: Usually intermediate or high-grade dysplasia. MUC2 and MUC5AC expression.
• Pancreatobiliary type: Often high-grade lesions. MUC 1 and MUC5AC expression.
• Intraductal papillary oncocytic neoplasm: Usually present high-grade dysplasia. MUC 1 and MUC6 expression. The majority have no KRAS2 gene mutation.
• Intraductal tubulopapillary neoplasm: Controversial entity, more recently recognised. Also absence of KRAS2 gene mutation, no visible mucin. MUC6 expression.
Genetic studies have determined KRAS2 gene mutation in over 90% of cases of pancreatic cancer. The same mutations are found in 38.5-100% of IPMNs, with a frequency directly proportional to the degree of dysplasia. This mutation is an early event in the genesis of IPMN.

Other genes involved in pancreatic cancer that have also been reported in IPMNs at different degrees are p16/CDKN2A, SMAD4 and TP53. Aberrant hypermethylation of DNA contributes to the inactivation of tumour suppressor genes in IPMNs. It is increasingly detected and proportional to the degree of dysplasia.

Only a small percentage of IPMNs do not present mutations characteristic of pancreatic cancer and this is the case of the IPMNs of patients with Peutz-Jeghers syndrome.

IPMNs are multifocal in up to 65% of cases and 5-10% affects the whole pancreatic gland.

IPMNs exhibit varying degrees of malignancy from adenoma to invasive neoplasia, including dysplasia, borderline tumours and in situ carcinoma. This adenoma-carcinoma sequence suggests that the epithelium is pre-malignant and may progress to malignancy.

Although most invasive carcinomas are associated with in situ carcinoma, they sometimes coexist with adenoma and borderline subtype. The global incidence of invasive carcinoma associated with IPMN is from 20 to 40%.

Scientific studies coincide in reporting that IPMN progresses with time, at least in MD-IPMN. Ongoing studies aim to illustrate this mechanism and determine the time necessary for such transformation.

Accumulated evidence shows that IPMN is a marker for increased risk of developing pancreatic cancer. Some studies report an increased risk of up to 22.5 times that of the general population. Pancreatic cancer may arise in IPMN, but also in other areas of the pancreas distant from the IPMN. It is believed to evolve from malignant PanIN lesions.

Pancreatic cancer in IPMN has a better prognosis than ductal adenocarcinoma when located in dilated ducts, but prognosis does not differ when it invades pancreatic parenchyma. Survival at 5 years in patients with no pancreatic parenchymal invasion
is superior to 95%, but drops to 40% in patients with invasion; detection of invasion is therefore crucial.

The association between IPMNs and extrapancreatic lesions is controversial as while some authors report a major prevalence of other neoplasms (colon, stomach, etc), other recent studies point out that IPMN is not associated with systemic carcinogenesis.

Fig. 7

**Clinical presentation**

IPMN is usually an incidental finding in asymptomatic patients.

The most common clinical manifestations are pain, weight loss, steatorrhea, diabetes and pancreatitis.
**Fig. 1:** MRCP, MIP reconstruction. Diffuse dilation of whole MPD and extrahepatic biliary tract.

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Fig. 2: MRCP, MIP reconstruction. Cystic dilation of lateral branches in the whole pancreas, the largest in size are located in the uncinate process and in the tail.

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Fig. 3: A. Normal pancreas. B. BD-IPMN. Isolated or multifocal dilatations of lateral branches, often in head or uncinate process. May be single locules (arrowhead), or, more typically, grape-like bunches of polymorphic cysts (short arrow). They contain mucin (orange), papillary proliferation of the epithelium with occasional formation of mural nodules (long arrow) that can be visualised with imaging techniques. C. MD-IPMN. Shows a dilated main pancreatic duct containing mucin (orange). Associates varying degrees of atrophy and fibrosis (purple). A specific, though not very sensitive sign is the bulging of the papilla into the duodenal lumen (thick arrow). Papillary proliferation of the epithelium in the MPD walls form nodules (short arrow). Some scattered calcifications in walls or inside MPD (stars). D. M-IPMN. Combination of findings described in MD-IPMN and BD-IPMN. May evolve to malignancy, accompanied by parenchymal invasion (long arrow).

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**Fig. 4:** H-E stain, 40x (A) that shows normal pancreatic duct. 20x image (B) shows a dilated duct with epithelial mucinous hyperplasia and micropapillary pattern.

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**Fig. 5:** H-E stain, 200x. Detail of intestinal-type IPMN with moderate dysplasia.
**Fig. 6:** H-E stain, 400x. Biliary subtype IPMN with areas of dysplasia from mild to moderate.
Fig. 7: H-E stain, 20x. Ductal epithelium (ellipse) with micropapillary structure (arrow). We observe a tree-like structure (arrowhead), which would correspond to a bigger papilla.

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Radiological findings

Ultrasound has a limited role in the diagnosis of IPMN.

Multiphasic CT scans should be performed with intravenous contrast as it provides better spatial resolution. Dilatation observed in MD-IPMN (often >1cm) and diffuse or segmental tortuosity of MPD, sometimes associated with atrophy, mural nodules or calcifications.

Fig. 8

BD-IPMN show single or multiple cystic lesions, often polymorphic and located in the head of the pancreas.

Fig. 9

CT is used not only to diagnose and classify IPMNs, but also to detect signs of malignancy or invasion such as:

- diameter of MPD ≥ or equal to 10 mm (for other authors, the cut-off is 6 mm or 8 mm, respectively),
- wide communication (>10mm) between BD-IPMN and MPD,
- altered pancreatic attenuation adjacent to MPD or to cystic lesion (accuracy for the diagnosis of invasion of 89%),
- mural nodules in the Wirsung duct or in a cystic lesion,
- cystic lesion ≥ 3 cm.

Accuracy of radiographic assessment for the diagnosis of invasion is 89% in the presence of alteration in the density of the pancreas next to MPD/cyst, which is more visible in the arterial phase images.

Some findings are indicative, but have not been demonstrated to be statistically significant: presence of adenopathies, protrusion into MPD in ampulla of Vater (association with malignancy), and common bile-duct dilatation or calcification of lesion (association with invasion).

Fig. 10
Magnetic resonance (MRI) findings are similar to those of CT.

Magnetic resonance cholangiopancreatography (MRCP) is better than CT to show the communication of BD-IPMNs with MPD.

MRCP also shows internal defects that correspond to mural nodules or mucin plugs. A substantial improvement of the sensitivity of MRCP has been reported after the administration of secretin.

Most authors agree on the potential usefulness of techniques of diffusion-weighted imaging (DWI) in pancreatic cystic lesions. Further studies should be performed to determine their usefulness.

Limitations to progress are numerous: no unified terminology, controversies in the interpretation of laboratory results, extreme variability and complexity of molecular environment that determines the image, multiple designs of the diffusion-weighted sequences, etc. This accounts for the great disparity in ADC values published in literature (due to different sequences, different b values applied, and different methods of calculation of ADC…). It is thus difficult to draw conclusions that can really be of use to the radiologist in routine clinical practice. ADC values of the CSF are $3.5 \times 10^{-3}$ mm²/s for Irie, 5.9 for Yamashita and 4.1 for Boraschi. Yamashita and Boraschi report opposite results regarding ADC values to differentiate mucinous tumours and serous cystadenomas (SCA), while Irie states that ADC is not useful in this case due to the wide range of ADC shown by SCA, which overlap. However, Inan reports low values of ADC for mucinous lesions, but obtains similar results for SCA.

Most authors point out that mucinous lesions (IPMN, NQM) and all lesions containing thicker fluid (abscesses, hydatid cysts, some pseudocysts) show lower ADC values than those of CSF or simple cysts.

Greater consistency is therefore needed in working techniques in order to obtain more homogeneous data to establish reference values as real tools for differential diagnosis.
In our centre, diffusion-weighted sequences are routinely included in explorations of the pancreas (b 0, 50, 600). A visual assessment of the diffusion-weighted sequences is made, as well as ADC mapping and morphological sequences, but quantitative analysis and measurement of ADC value is not carried out. In practice, it is useful in some cases of pseudocysts to demonstrate the solid or thick component (in which case surgical drainage would be preferable to endoscopic drainage) or to visualise solid areas of cystic lesions. We have not observed a restricted diffusion (according to visual criteria applied in CNS) in mucinous lesions, but a behaviour that suggests severely impaired diffusivity but with considerable overlap among the different pancreatic cystic lesions.

Fig. 13

Other diagnostic techniques

- **Endoscopic retrograde cholangiopancreatography (ERCP).** Shows dilatation of MPD and/or its branches. Prominent ampulla of Vater with mucus secretion is a very specific finding, with low sensitivity. ERCP is a method to obtain cytological sample or biopsy. It is a complementary technique in selected cases, but is not used routinely in the assessment of IPMN.
- **Endoscopic ultrasonography (EUS).** A complementary imaging technique when CT/MRI findings are inconclusive. Can be very useful to detect signs of invasion. Can be considered in lesions >3cm (if they present mucin, surgery is performed) and in lesions 2-3cm in order to rule out signs of alarm (see Management and Prognosis).
- **Ultrasound-guided fine-needle aspiration biopsy (FNA).** Allows collecting samples of intracystic fluid whose analysis is helpful for differential diagnosis. High levels of CEA, mucin and amylase are observed in IPMN. Malignant IPMNs show higher values of Ca 19.9 and CEA than benign IPMNs.

Differential diagnosis

- **Chronic pancreatitis.** When MD-IPMN involves the whole MPD, it may be very difficult to differentiate it from chronic pancreatitis (CP), as both present dilatation of the MPD and glandular atrophy. Calcifications are typical of CP, but can also be seen in MD-IPMN. Both entities are related and often coexist, as it believed that MD-IPMN may cause CP due to chronic obstruction of the MPD. In practice, when MPD is dilated and presents glandular atrophy with or without calcifications and the patient has no clinical history of CP, MD-IPMN should be suspected.

Fig. 14
Fig. 15

- *Serous cystadenoma* (SCA). Usually polymicrocystic, honeycomb patterned and often with a central scar, sometimes calcified. Yet 10-25% of SCAs are oligocystic and do require differential diagnosis with BD-IPMN: the main difference is that SCA does not communicate with the MPD.

Fig. 16

- *Mucinous cystic neoplasm* (MCN). Cystadenoma, cystadenocarcinoma. 95% of these lesions affect women. They are potentially malignant and are characterised by the presence of ovarian-type stroma. They can be uni- or multilocular and show no communication with MPD.

Fig. 17

- *Non-neoplastic mucinous cyst* (NNMC). A controversial entity described by Kosmahl in 2002 and not recognised by all pathologists (some classify it as adenoma-type IPMN). Typically, they are homogeneous, uni- or multilocular cysts, with no solid component, and small-sized. Differential diagnosis with BD-IPMN should be made.

- *Pseudocyst*. Usually a unilocular lesion without communication with MPD that does not present diagnostic doubts. But sometimes, communication between the pseudocyst and the MPD is present. In this case, clinical context and the presence of interior debris are useful for diagnosis. It should always be considered that pancreatitis may be caused by an IPMN. An antecedent of acute pancreatitis is therefore not *necessarily* indicative of a pseudocyst.

Fig. 18

**Management and prognosis**

It must be borne in mind that all symptomatic cystic neoplasms in operable patients should be surgical.

When the finding is incidental, management will depend on subtype of IPMN, as they have different prognoses.

The aim of surgery is to cure the symptoms and prevent dissemination of the disease or transition to invasion.
Although time of progression has not been established, some studies have determined that the risk of malignancy increases with advanced age, the presence of symptoms and mural nodules, affectation of MPD, dilatation of MPD > 10mm, and in BD-IPMN subtypes of >3cm.

Nevertheless, one should consider that the absence of symptoms does not rule out malignancy, and that the risk of recurrence after resection in case of invasive carcinoma is much greater (46%) than after resection for non-invasive disease (1.3 %). Survival at 5 years is almost 100% after resection of non-invasive IPMN and 46% when invasive.

Surgical resection should be performed in all patients with MD-IPMN and M-IPMN that have a reasonable life expectancy.

Controversy exists regarding BD-IPMN, although the most adequate management is a combination of observation and resection, considering: age, symptoms, concomitant disease of the patient, size, mural nodules, atypical cytology.

Pancreatectomy may be indicated for the resection of MD-IPMN (with intraoperative cytology of margins), and focal resection of lesion is recommendable in BD-IPMN (when multifocal, the dominant lesion is removed with surveillance of other lesions).

Recurrence can be observed in BD-IPMN and in MD-IPMN that present false negative margins.

Patients with colloid and oncocytic invasive subtypes have a better prognosis than those with tubular adenocarcinoma.

The current recommendations for follow-up are:

- Operated patients with benign IPMN: yearly CT/MRI, then space out follow-ups after some years
- Operated patients with invasive IPMNs: CT/MRI every 6 months
- Non-operated patients:
  - Cysts < 1cm: yearly CT/MRI
  - Cysts of 1-3 cm: First, perform EUS+CRM or ERCP to rule out risk factors (mural nodules, atypias, and dilatation of MPD). If ruled out, follow-up with CT/MRI every 6-12 months for cysts 1-2 cm and every 3-6 months for cysts 2-3 cm.
During follow-up, appearance of symptoms, mural nodules, size > 3cm or dilatation of MPD >6 mm are indications for surgery.
Fig. 8: Thoracic CT performed for aphonia shows atrophy of pancreatic parenchyma and stricturing, dilatation and beading of MPD, with no evidence of nodules inside. Radiological diagnosis was probable MD-IPMN with no signs of invasion. Ultrasonography was compatible with mucinous tumour. Distal pancreatectomy was performed with final diagnosis of severe chronic pancreatitis and borderline MD-IPMN, with no evidence of dysplasia in resection margins.

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Fig. 9: Patient with repeated attacks of acute pancreatitis (AP) in context of chronic pancreatitis (CP) of enolic aetiology. CT in 2006 (A, B) shows signs of AP and a small parenchymal collection. CT performed in 2007 (C, D) reveals progression of signs of CP with greater dilation of MPD and increase of calcifications. MRCP performed in 2010 for jaundice (E, F, G) shows dilatation of the biliary tract and of the MPD from the head of the pancreas. Low T1 signal (G) of pancreas, compatible with CP. CT performed during AP attack (H): evidence of size increase of pancreatic head, with several collections inside. Last control CT (I, J, K): interestingly, pseudotumoural aspect of pancreatic head. EUS-FNA showed mucin and severe cytological atypia in relation to IPMN. Given the patient's history or untreatable pain for chronic pancreatitis, total pancreatectomy was performed. Pathology results: MD-IPMN with moderate dysplasia.

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**Fig. 10:** Patient with no antecedents of interest visits for toxic syndrome. CT (A, B, C) shows global dilatation of main pancreatic duct, with glandular atrophy and no calcifications. Radiological diagnosis: MD-IPMN with no signs of invasion. EUS-FNA reveals IPMN with no atypias. Cephalic pancreaticoduodenectomy was performed; pathology results: adenoma-type MD-IPMN.

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Fig. 11: Cystic lesion detected by CT (not shown) performed for acute pancreatitis. Subsequent MRI: HASTE axial sequence (A) and coronal (B) show beaded dilatation of MPD and dilatation of some branch ducts. MIP reconstruction (C) of MRCP reveals important fusiform dilatation of MPD and Todani type I choledochal cyst. Dynamic study (D-G) at same level as image A shows enhancement of mural nodule inside MPD at level of pancreatic head. Diffusion sequences (b 0 in H, b 50 in I, b 600 in J, ADC map in K) clearly show mural nodule, with intensity more similar to parenchymas than to CSF. Behaviour of intracystic fluid shows intermediate signal intensity in relation to CSF and spinal cord, which, in this context, may be related to the presence of mucin. Cephalic pancreaticoduodenectomy was performed: borderline M-IPMN. L. H-E stain, 200x: shows prominent nucleoli, with multiple layers of nuclei and disorganisation and moderate atypia.
Fig. 12: CT performed for sigmoiditis (not shown) shows lobulated cystic lesion (A, B) in body of the pancreas. EUS-FNA results: cystadenoma with no evidence of mucin nor a mucous secretory epithelium. MIP reconstruction of MRCP (C) shows communication between cyst and MPD. Diffusion-weighted sequences (b 0 in E, b 50 in F, b 600 in G and ADC map in H) reveal insointensity between cyst and spinal cord in b600 and hypointensity of the cyst in ADC map, which suggests that the contents is not serous fluid. Radiological diagnosis: BD-IPMN. The patient had increases level of serous CEA and an increase of size was observed in control MRCPs. Distal pancreatectomy was performed; pathology results: intestinal-type borderline M-IPMN.

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**Fig. 13:** Each row includes a different patient with probable BD-IPMN (with no histological confirmation) to illustrate the different behaviours observed in diffusion sequences. b0 (B, G), b50 (C, H) and b600 (D, I) were used. Both are grape-like cystic lesions communicating with the MPD (A, F). In case 1, the cyst is isointense with the spinal cord in b600 (D) and hypointense in ADC map relative to CSF (E). In case 2, the cyst is hypointense in b600 relative to the spinal cord (I) and isointense with CSF (J) on ADC map.

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**Fig. 14:** CT performed in May 2006 (A-D) for abdominal pain that shows pancreatic atrophy with dilatation of MPD and some small calcifications (long arrow) inside. Diagnosis was chronic pancreatitis. Note the intramural nodule in the head of the pancreas (short arrow). MRCP (E-G) performed in June 2006: chronic pancreatitis, with dilatation and beading of MPD. Also dilatation of some branch ducts and intraductal nodule of the pancreatic head. CT (H-L) repeated in June 2007 for abdominal pain shows a mass in the head of the pancreas (thick arrow in K and L). Cephalic pancreaticoduodenectomy, pathology results: adenocarcinoma in IPMN.

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Fig. 15: Chronic cystic pancreatitis. Abdominal CT performed for altered hepatic tests shows cystic lesion (A) in head of the pancreas formed by several locules. Coronal reconstruction (B) of helical CT data obtained during portal phase shows cystic lesion at the level of the main locule and increase of density of adjacent peripancreatic fat. Dilatation of the main pancreatic duct (C). With a presumptive diagnosis of cystic tumour, EUS was performed: probable cystic neoplasm. FNA detected inflammatory cells. Cephalic pancreaticoduodenectomy AP results: chronic pancreatitis with no evidence of malignancy.

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**Fig. 16:** Serous cystadenoma (SCA). Incidental finding in abdominal CT (A, B) performed for sigmoiditis (not shown) of a lobulated cystic lesion in the body-tail of the pancreas. C. HASTE sequence shows cystic lesion formed by several locules. D. T1-weighted sequence with fat saturation shows hyperintensity of cyst content. Normal pancreas signal. Diffusion-weighted sequences (b 0 in E, b 50 in F, b 600 in G, ADC map in H) show that the lesion has intermediate signal intensity between serous fluids (e.g. CSF) and parenchymas (e.g. spinal cord) that behave similarly to biliary fluid. Pathology results confirmed SCA and reported presence of blood inside, consistent with T1-weighted image signal and may explain the diffusion behaviour different from that expected from serous fluid in SCA.

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**Fig. 17:** Study performed for altered hepatic tests show a cystic lesion in the head of the pancreas. (A). CT (B) detects discrete dilatation of the MPD. Study was completed with MRCP that revealed the same findings. HASTE sequence (C) shows a sedimented level inside the lesion. No communication with the MPD is seen. Diffusion-weighted sequences (b 0 en D, b 50 in E, b 600 in F, ADC map in G) show a behaviour of cystic lesion more similar to that of spinal cord than to CSF, and a signal in ADC map more hypointense than that of CSF. EUS-FNA diagnosed probable mucinous cystadenoma. As the patient also suffered multifocal hepatocellular carcinoma, surgery was not performed on pancreatic lesion.

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**Fig. 18:** Patient referred for study for antecedent of pancreatitis. CT (A) findings of intra- and extrapancreatic fluid collections. With suspicion of pseudocyst, ultrasound control was performed (B): stability of lesion with internal echoes. Subsequent MRCP reveals communication between the lesion and the MPD (C and D). Diffusion-weighted sequences (b 0 in E, b 50 in F, b 600 in G, ADC map in H) show lesion with intermediate signal between serous fluids (e.g. CSF) and parenchymas (e.g. spinal cord). EUS-FNA (not shown) compatible with pseudocyst. Cultures yielded Proteus mirabilis and Streptococcus viridans. Endosonography-guided cystoduodenostomy was performed.

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Conclusion

Pancreatic IPMN is a premalignant condition that can develop into cancer.

The role of imaging techniques is:

• Establish diagnosis,
• Classify subtype (main duct, branch duct, mixed),
• Determine localisation,
• Detect signs of malignant transformation and/or invasions.
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