Intraductal papillary mucinous neoplasms, a radiologic-pathologic correlation.

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Learning objectives

1. To describe the radiological findings of the three types of typical intraductal papillary mucinous neoplasm (IPMN); 
   Main Duct, Side Branch, Mixed.

2. Highlight the features, which distinguish IPMN from other cystic pancreatic lesions using radiologic-pathologic correlation.

3. Provide a framework for work-up, diagnosis, follow-up and treatment of IPMNs.

Background

IPMNs are potentially malignant intraductal epithelial neoplasms that are composed of mucin-producing columnar cells. These lesions show cyst formation, papillary proliferation and varying degrees of atypia.

IPMNs can be classified into three types, Main duct (MD-IPMN), branch duct (BD-IPMN), and mixed type, based on imaging studies and histology.

They were first described in the 1980’s [1], the incidence rate and natural history of IPMN are not fully known. IPMN is being recognized with increasing frequency around the world and the number of pancreatic resections for IPMN is rising [2].

Imaging findings OR Procedure details

Definition:

MD-IPMN is characterized by segmental or diffuse dilation of the main pancreatic duct (MPD) of>5mm without other causes of obstruction [3]. Fig. 1 on page 5

Pancreatic cysts of >5mm in diameter that communicate with the MPD should be considered as BD-IPMN (pseudocyst is another consideration). Fig. 2 on page 6, Fig. 4 on page 7.
IPMN usually present in older patients, with a greater male predominance.

**Imaging:**

**Ultrasonographic Findings:** Ultrasound can show a septate cystic lesion and dilated MPD. Not sufficient for diagnosis.

**CT Findings:**

**BPD type:** Lobulated multicystic with thin, irregular, peripheral ring-enhancing walls. Fig. 4 on page 7, Fig. 5 on page 8

**MPD type:** Markedly dilated tortuous main duct. Fig. 1 on page 5 May see nodular lesions lining the duct. Fig. 6 on page 9 Calcification may also be seen.

**Combined type:** Multicystic lesion in the uncinate process communicating with a grossly dilated main duct. Atrophy of the gland may be seen upstream from tumour.

**MR Findings:**

Lobulated, clustered cysts and dilated main ducts. Can identify communication between cystic lesions & ducts.

May detect nodular filling defects. Can also assess for biliary obstruction.

**EUS:**

Endoscopic ultrasonography allows superior visualization; can guide biopsy & aspiration.

Elevated CEA is a marker that distinguishes mucinous from non-mucinous cysts, but not benign from malignant cysts.

**Histological subtypes:**

There are 4 subtypes of cell origin of the papillary component of IPMNs; Gastric, intestinal, hepatobiliary and oncocytic. Gastric is the most common and oncocytic is the least common. These are histopathological diagnoses and they cannot be distinguished on radiology.

It is felt that gastric and intestinal sub-types have less malignant potential and that carcinomas from intestinal types are less aggressive [4].
Differential Diagnosis:

**Serous cystic neoplasms** are the most common of the cystic neoplasms. They have a four to one female to male predominance. They are characteristically microcystic, although they may be macrocystic. Since they are only very rarely malignant, symptomatic and enlarging lesions only should be surgically resected.

**Mucinous cystic neoplasms** mostly affect women between the 4th and 6th decades of life; they are mostly localized in the tail of the pancreas. They do not communicate with the duct and contain an ovarian-type stromal component. Surgery is indicated in any case in a diagnosis of MCN as these tumors have the potential for malignancy.

**Solid papillary neoplasms** are benign in more than 90% of cases and affect mainly young women. Since they are not easily distinguishable from pancreatic cancer in terms of the differential diagnosis, surgery is indicated.

**Management:** [3]

The management of IPMN is dictated by its subtype.

**BD-IPMN Table 1, Table 2**

The incidence of malignancy in BD-IPMN is lower than MD-IPMN, but remains significant, up to 25%. They occur mostly in elderly patients and surgical resection is not always possible. "High risk stigmata" have been validated, such as jaundice, enhancing mural nodularity and main duct >10 mm, and their presence should prompt surgical resection.

If these are not present, a set of "worrisome features" contained in table 1 should also prompt consideration for surgery. If these are not identified, further evaluation with EUS +FNA is advised, and if cytology is negative and no more "worrisome features" are identified, follow-up surveillance can be performed dictated by lesion size.

**MD-IPMN**

According to published literature, MD-IPMNs have >50% presence of malignancy. Due to the high incidence of malignancy, surgical resection should be strongly considered in all surgically fit patients. MD dilatation of 5-9 mm should be considered a "worrisome feature" as in BD types and should warrant evaluation (EUS+FNA).
In MD-IPMN, unlike BD, no consistent features of malignancy have been established (such as duct size, presence of mural nodules or symptoms).

**Fig. 6 on page 9**

**Mixed type IPMNs** are treated as MD types.

**Multiple BD-IPMNs** are considered a "field change", but do not have an increased rate of malignancy when compared with solitary BD-IPMNs and should be treated according to the largest cyst as per single BD types.

**Images for this section:**

**Fig. 1:** Axial T2 MRI, axial contrast enhanced CT and MIP HASTE MRI with pathological correlation. There is gross dilation of the main pancreatic duct (curved arrow) with cystic dilation at the tail. Gross pathology shows cross-sectional cuts through the pancreatic
gland with gross ductal dilatation and cystic lesion distally. Histopathology showed intestinal type IPMN with moderate dysplasia without evidence of carcinoma.

**Fig. 2:** Coronal contrast enhanced CT, HASTE T2 MRI and pathological correlation. Fig 2 and 3 Multiple cystic dilatations within the pancreatic head. Communication with the main duct, without dilatation of the duct. Gross pathology shows a small cystic lesion in the pancreatic head. Probing is used by the pathologist to identify communication with the main duct to confirm the diagnosis of IPMN. Histopathology showed gastric type IPMN with low-grade dysplasia.
**Fig. 3:** Axial MRI, Axial contrast enhanced CT and MIP HASTE MRI with pathological correlation. Fig 2 and 3 Multiple cystic dilatations within the pancreatic head. Communication with the main duct, without dilatation of the duct. Gross pathology shows a small cystic lesion in the pancreatic head. Probing is used by the pathologist to identify communication with the main duct to confirm the diagnosis of IPMN. Histopathology showed gastric type IPMN with low-grade dysplasia.
Fig. 4: Axial contrast enhanced CT. Fig 4 and 5. The pancreas is markedly atrophic and there is an area of higher attenuation within the uncinate process. The presence of this within an atrophic pancreas (curved arrow) should raise suspicion of a lesion. The multi-cystic lesion appears more consistent with a serous cystic neoplasm, but in a male patient, this is still more likely to be an IPMN. Histopathology showed branch type IPMN with low grade dysplasia.
Fig. 5: T2 HASTE axial and MIP and T2 axial MRI with pathological correlation. Fig 4 and 5. The multi-cystic lesion appears more consistent with a serous cystic neoplasm, but in a male patient, this is still more likely to be an IPMN. Gross pathology shows fatty replacement of the pancreas (curved arrow) and a mulit-cystic tumor. Histopathology showed branch type IPMN with low grade dysplasia.
Fig. 6: Axial contrast enhanced CT and pathological correlation. Cystic lesion with enhancing nodularity within the main pancreatic duct. Gross pathology shows a large cystic lesion with several soft tissue nodules. Histopathology showed IPMN with prominent oncocytic morphology and high grade dysplasia.
Fig. 7: Axial contrast enhanced CT and pathology correlation. There is a cystic abnormality in the body of the pancreas with soft tissue nodularity (arrow). There is communication with and dilatation of the main duct (curved arrow). Gross pathology shows a cystic lesion containing soft tissue tumor. Histopathology showed invasive moderately differentiated mucinous adenocarcinoma arising in an IPMN with intestinal mucosa.
Fig. 8: Axial and Coronal CT and T2 MRI with pathological correlation. Multi-loculated fluid collection continuous with, but without dilation of the main duct. Gross pathology shows a normal appearing pancreas with a multi-cystic lesion in the body. Histopathology revealed intestinal type IPMN with minimal atypic.
**Fig. 9:** T2 Axial and MIP HASTE T2 MRI with pathological correlation. Cystic lesion at the tail of the pancreas without dilation of the main duct. MRCP shows communication with the main duct. No soft tissue component is identified. Gross pathology shows a multicystic lesion in the tail of the pancreas. Cross-sections through this shows multiple cysts communicating with the main duct. Histopathology showed IPMN of mixed type involving both the main and branch ducts. Gastric type mucosa with low grade dysplasia.
Table 1: Management of BD-IPMN (1)
Table 2: Management of BD-IPMN (2)

Adapted from International consensus guidelines 2012 for the management of IPMN and MCN of the pancreas
Conclusion

IPMN is a relatively new diagnosis, and the natural history of the disease is not fully understood.

Despite the increase in familiarity with this lesion, the incidence rate and natural history of IPMN remain unknown. Therefore, an aggressive approach to the treatment of IPMN is currently practiced.

This educational exhibit describes the different types of IPMN, illustrated with radiological and pathological examples, and provides information on the current guidelines for surveillance and treatment of these lesions.

References

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International consensus guidelines 2012 for the management of IPMN and MCN of the pancreas.


Intraductal Papillary-Mucinous Neoplasms of the Gastric and Intestinal Types May Have Less Malignant Potential Than the Pancreatobiliary Type

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