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Intraductal oncocytic papillary neoplasm of the pancreas: A systematic review

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ABSTRACT

Background: Intraductal oncocytic papillary neoplasm of the pancreas (IOPN–P) is a rare subtype of intraductal papillary mucinous neoplasm (IPMN). This study was performed to summarize the clinicopathological features and management of IOPN-P.
Methods: English-language articles were searched from MEDLINE and EMBASE from the first report of IOPN-P in 1996 until 1 May 2019 following the methodology in the PRISMA guidelines.
Results: In total, 66 patients from 24 full articles were included in the final data analysis. The patients' average age was 61 years, and the male/female ratio was 1. Most lesions were large (average size, 5.50 cm), located in the pancreatic head, and found either incidentally or by uncharacteristic abdominal symptoms. IOPN-P was usually a cystic and solid lesion with or without mural nodules on radiological examination. A definitive diagnosis was often acquired from fine needle aspiration biopsy or postoperative pathology. All tumors were diagnosed as carcinoma *in situ* or minimally invasive carcinoma, necessitating surgical resection. The prognosis of IOPN-P was better than that of other IPMN subtypes, even when metastasis occurred. Recurrence after surgical resection of IOPN-P was rare.
Conclusions: IOPN-P is rare among IPMN subtypes with unique pathological characteristics. Because of the nontypical symptoms and radiological findings, a definitive preoperative diagnosis usually depends on multimodal examinations. Management and surveillance of IOPN-P after surgical resection should be differentiated from those of other pancreatic benign cystic lesions because of its relative malignancy, but IOPN-P should also be differentiated from other IPMN subtypes and malignant cystic tumors because of its favorable prognosis.

Introduction

Intraductal oncocytic papillary neoplasm of the pancreas (IOPN–P), also called oncocytic type intraductal papillary mucinous neoplasm (O-IPMN), is a rare cystic neoplasm of the pancreas. IOPN-P is characterized by complex, arborizing papillary structures that are lined by eosinophilic cells and was first described as an independent entity by Adsay et al. [1] in 1996. IOPN-P was later classified as one of four subtypes of intraductal papillary mucinous neoplasm (IPMN) in the World Health Organization guideline, namely gastric type, intestinal type, pancreatobiliary type, and oncocytic type [2]. IOPN-P is now considered a variant of IPMN; however, given the histological and genetic molecular differences among IOPN-P, typical IPMN, and pancreatic adenocarcinoma (PDAC), the unique pathogenetic mechanism can be deduced from an in-depth study of IOPN-P [3].

Most previous studies have focused on the unique pathological and histological features of IOPN-P, but few have summarized the clinicopathologic features and the diagnostic and treatment modalities of IOPN-P because of the limited number of cases containing complete data [4–6]. Since Adsay et al. [1] reported the first case series of IOPN-P, other research groups have also summarized small numbers of IOPN-P cases published as single case reports or case series [7,8]. A conclusive guideline and consensus regarding the management of IOPN-P are lacking because of the rarity of this neoplasm [9]. Therefore, it is necessary to summarize the effective diagnostic and treatment modalities to reduce the possibility of misdiagnosis and malpractice. This systematic review was

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performed using evidence-based methods to elucidate the clinicopathologic features, diagnostic modalities, and therapies of IOPN-P for better clinical practice.

Methods

Literature search

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) methodology was followed during the performance and reporting of this systematic review [10]. Two researches (Y.Z.W. and J.L.) independently conducted an online literature search using MEDLINE (via PubMed) and EMBASE (via Embase.com) until 1 May 2019. No filters regarding study type, language, or publication time were applied. The "related articles" function was used to include more articles. The search terms are shown below:

MEDLINE (via PubMed):
#1"intraductal oncocytic papillary neoplasm"; #2"oncocytic-type intraductal papillary mucinous neoplasm"; #3 pancreas* OR pancrea*; #4 #1 AND #2AND#3
EMBASE (via Embase.com):
#1'intraductal oncocytic papillary neoplasm'; #2'oncocytic-type intraductal papillary mucinous neoplasm'; #3 #1 AND #2

Study selection

All studies of IOPN-P were searched without distinguishing study types. Only English-language articles that partially or completely reported demographic and clinicopathologic data (age, sex, symptoms, serological and imaging examination findings, treatment, pathologic characteristics, and prognosis) were included. Other articles such as conference reports, letters, reviews, retrospective studies, and expert consensus without original data were excluded. Additionally, repetitive studies and studies from the same institution/authors were combined. Moreover, articles reporting IOPN in organs other than the pancreas, such as the liver and extrahepatic biliary tract, were excluded. Two reviewers (Y.Z.W. and J.L.) screened the titles and abstracts. The full text of all eligible articles was read by both reviewers. The references of the eligible articles were browsed to identify additional studies, as recommended by the Cochrane Handbook for Systematic Reviews [11]. Discrepancies between the two reviewers were settled through discussion with a third reviewer (J.C.G.).

Data extraction and study quality assessment

Two reviewers (Y.Z.W. and J.L.) independently extracted the data using a standardized form. When available, the following information was extracted for each patient with IOPN-P: title, study type, author(s), publication year, age, sex, lesion location, lesion size, symptoms, serological tumor biomarkers, imaging examination findings (e.g., ultrasonography, contrast-enhanced computed tomography [CT], magnetic resonance imaging [MRI], endoscopic retrograde cholangiopancreatography, magnetic resonance cholangiopancreatography, endoscopic ultrasonography, and fluoro-2-deoxy-D-glucose positron emission tomography [FDG-PET]), fine needle aspiration (FNA) pathology, preoperative diagnosis, treatment, postoperative pathology, postoperative diagnosis, and prognosis. All related text, tables, and figures were also reviewed for data extraction. Differences were settled by a third author (J.C.G.) to reach a consensus. Primary data are summarized in *Supplementary File 1*. The United States Preventive Services Task Forces rating criteria [12] were used to evaluate the study quality.

Statistical analysis

Categorical variables are presented as frequency and percentage. Continuous variables are presented as median and corresponding range.

Results

In total, 24 full articles including 66 patients were extracted for full review (Fig. 1). Abdominal pain and nonspecific abdominal discomfort were the most common symptoms in patients with IOPN-P because of the relatively large median size of the lesions (5.50 cm). Serologic examinations showed no abnormal levels of tumor biomarkers, such as carbohydrate antigen 19–9 (CA19-9) and carcinoembryonic antigen (CEA). Preoperative imaging examination revealed multilocular cystic and/or solid lesions and mural nodules with a dilated main pancreatic duct (MPD). Many of the mural nodules exhibited elevated FDG uptake on FDG-PET. FNA pathology was usually the most accurate preoperative examination for a diagnosis of IOPN-P because of the acquisition of oncocytic cells. Preoperative and postoperative immunohistochemical analyses were also applied to confirm the diagnosis based on the expression of several specific markers. Most of the postoperative diagnoses of IOPN-P were malignant, necessitating radical resection. However, the prognosis was better than that for other subtypes of IPMN, even when metastasis was present, because of the slow progression of IOPN-P. The preoperative data, cyst features, and preoperative examination findings are shown in Tables 1 and 2. The preoperative diagnosis and diagnostic modality and treatment methods are summarized in Table 3.

Discussion

Since Adsay et al. [1] first reported a series of 11 cases of IOPN-P, IOPN-P has gradually become recognized and explored. Several previous studies have focused on the unique pathologic characteristics of IOPN-P, including complex, thick, arborizing papillae with delicate stroma lined by multiple layers of cuboidal and columnar epithelium with abundant granular, eosinophilic cytoplasm scattered with goblet cells [9,13,14]. However, given the limited number of complete cases of IOPN-P, the pathogenesis, clinical behavior, and management of the disease remain unclear [14].

Preoperative demographic data show that the age of patients with IOPN-P ranges from 36 to 84 years, with men and women equally affected (male:female ratio = 1). The symptoms of IOPN-P are similar to those of IPMN, the main one of which is often nonspecific abdominal discomfort [8]. IOPN-P mainly arises from the pancreatic head, but it can also arise in other areas such as the body and tail, as Liszka et al. [8] reported. The average size of an IOPN-P is 5.50 cm (range, 1–15 cm).

The conventional serological tumor biomarkers CA19-9 and CEA are often used to diagnose and predict the prognosis of pancreatic lesions [15]. In the present study, however, most of the patients did not present with elevated tumor biomarkers. This implies that these conventional tumor biomarkers might not be suitable for the diagnosis of IOPN-P.

In preoperative imaging examinations, IOPN-P is usually a multilocular cystic or cystic and solid lesion with possible mural nodules on enhanced CT and MRI. Meanwhile, a dilated MPD and filling defects can be detected using endoscopic retrograde cholangiopancreatography and magnetic resonance cholangiopancreatography. Mirko et al. [14] analyzed the imaging

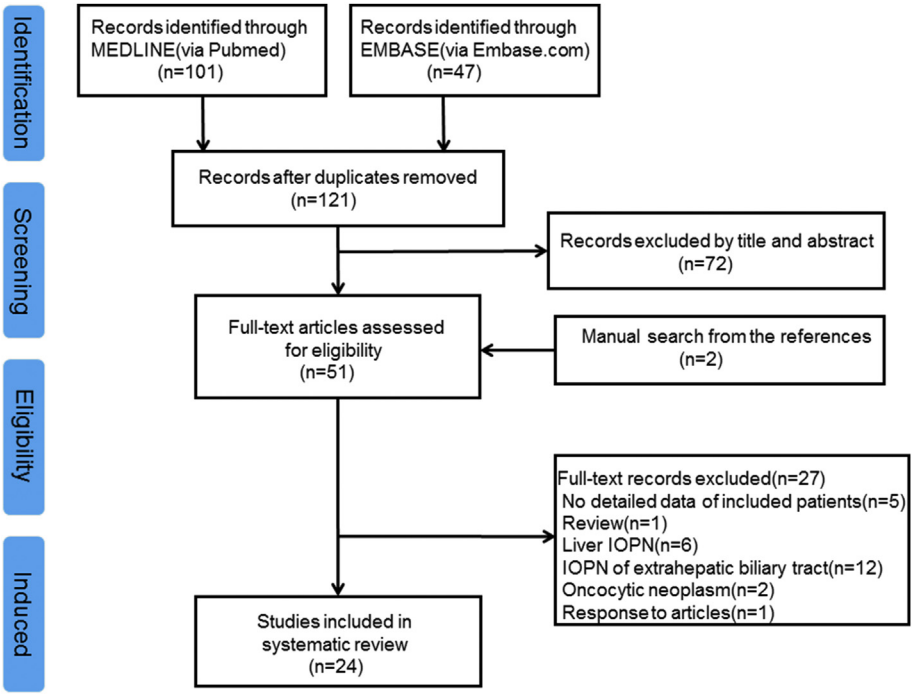

Fig. 1. PRISMA flow chart of the literature search.

characteristics of 18 patients with IOPN-P by contrast-enhanced CT and MRI and found that MPD dilation (83.3%) and enhanced mural nodules (62.5%) often existed in IOPN-P. However, invasion and compression symptoms were less common [14]. In the present study, IOPN-P mainly presented with hyperintensity on T2-weighted MRI and hypointensity on T1-weighted MRI, and the mural nodules often showed hypointensity on T1 and T2-WI; these findings are also in accordance with the study by Mirko et al. [14,16,17]. Interestingly, the use of FDG-PET has been reported in the diagnosis of IOPN-P [16,18,19]. FDG-PET is generally applied to evaluate the degree of malignancy, prognosis, degree of invasion, and metastasis of pancreatic cancer and to distinguish pancreatic cancer from other pancreatic cystic lesions [20,21]. However, the experience in using FDG-PET for diagnosis of IPMN and other pancreatic cystic diseases is limited. A systematic review by Srinivasan et al. [22] showed that the positive predictive value, negative predictive value, sensitivity, specificity, and accuracy of FDG-PET in identifying malignancy of IPMN and other cystic lesions were better than those of conventional diagnostic techniques, such as CT or MRI. Therefore, malignant IPMN may be accurately diagnosed by FDG-PET. However, limitations still exist. The first limitation is the lack of large cohort studies to further determine the status of FDG-PET in the diagnosis of IPMN. The second is that FDG-PET cannot differentiate IOPN-P from other subtypes of IPMN. Therefore, whether FDG-PET can be used as a specific method to diagnose IOPN-P remains unclear. Preoperative cytologic analysis or histological examination may be the gold standard diagnostic technique. The use of FNA biopsy, pancreatic juice cytologic analysis, or endoscopic biopsy preoperatively may reveal cells or tissues with oncocytic characteristics [23–25]. Immunohistochemical analysis of oncocytic cells or cystic tissue often show positive MUC1 and focal MUC5AC and MUC6 expression but negative CA19-9, neuron-specific enolase, chromogranin, synaptophysin, and CDX2 expression; in contrast, these are often overexpressed in other cystic and solid pancreatic lesions, such as pancreatic ductal adenocarcinoma (PDAC), pancreatic neuroendocrine tumors, and pancreatic acinar cell carcinoma [1]. However, Klausen et al. [26] found that MUC1 was strongly expressed in the gastric and intestinal subtypes, which exhibited high-grade dysplasia, rather than the oncocytic subtype. Moreover, the accuracy of FNA biopsy is sometimes doubtful when a negative result is obtained [27].

In this study, almost all patients with IOPN-P underwent radical resection such as pancreatoduodenectomy or distal pancreatectomy because of the malignant potential of IOPN-P. Additionally, all postoperative pathology results of patients who underwent operations were minimally invasive carcinoma or carcinoma *in situ*, and the number of invasive carinoma was nearly a half, which is in contrast to the study by Mirko et al., in which only two lesions presented foci of invasion [14]. Regional lymph node metastasis and distant metastasis only appeared in a small number of cases. Although IOPN-P can be a malignant lesion, the invasive parts are usually inconspicuous and are often confined to the pancreas [28]. This may be why the prognosis of IOPN-P is better than that of other subtypes of IPMN, even when metastasis has occurred or in patients who require a reoperation for recurrence [29,30]. In our study, most patients were alive with disease during the follow-up period (37/50). One patient with IOPN-P who developed brain metastasis was alive throughout the 20-month postoperative follow-up period [31]. Moreover, one patient who underwent a second operation for recurrence was also alive during the 16-month follow-up period [28]. However, a network meta-analysis by Qi et al. [32] showed that IOPN-P was not associated with better overall survival than other subtypes of IPMN. A single-center retrospective study indicated that the lymph node status rather than the histological subtype was the main prognostic factor, indicating that the histological subtype may be not so closely related to the patients' prognosis [33]. Therefore, large cohort studies will be required to confirm the prognosis of IOPN-P. Given the malignant potential and possibility of lymph node and distant metastasis, radical resection should be conducted as for other IPMN subtypes according to the 2012 international consensus guidelines for pancreatic IPMN and mucinous cystic neoplasm management [34]; these radical

Table 1
Patient demographic data, symptoms, cyst features, tumor characteristics, and prognosis.

Items		Median(range),n/d(%)
Age(Years)		61(36–84)
	<50	5/42
	50≤≤70	28/42
	>70	9/42
Gender	Female	33/66(50)
	Male	33/66(50)
	Male/Female	33/33
Location	Head	40/62(64)
	Body/Tail	15/62(25)
	Diffuse	6/62(9)
	Remnant body	1/62(2)
Tumor size(cm)		5.50(1–15)
	<6 cm	21/39
	≥6 cm	18/39
Symptoms	Abdominal pain	9/36
	Non-specific abdominal symptoms (such as abdominal discomfort and nausea)	8/36
	Pancreatitis	4/36
	Painless jaundice	2/36
	Diabetes Mellitus	1/36
	Right-sided weakness and tripping	1/36
	Incidental	11/36
Cyst features	Cystic	40/42
	Monolocular	3/40
	Multilocular	21/40
	Monolocular or Multilocular	16/40
	Solid with focal cyst	1/42
	Solid	1/42
	CA19-9(−)	9/13
	CA19-9(+)	4/13
	CEA(−)	7/16
	CEA(+)	9/16
	Chromogranin(−)	14/18
	Chromogranin(+)	4/18
	Synaptophysin(−)	12/16
	Synaptophysin(+)	4/16
	NSE(−)	8/12
	NSE(+)	4/12
	MUC1(−)	14/31
	MUC1(+)	17/31
	MUC2(−)	22/32
	MUC2(+)	10/32
	MUC5-AC(−)	1/30
	MUC5-AC(+)	29/30
	MUC6(−)	4/29
	MUC6(+)	25/29
	CDX2(−)	25/27
	CDX2(+)	2/27
Metastasis	None	45/55
	Lymphogenous metastasis	6/55
	Liver metastasis	3/55
	Brain metastasis	1/55
Postoperative pathology diagnosis	Invasive carcinoma	27/66(41)
	High-grade dysplasia(carcinoma *in situ*)	39/66(59)
Follow-up(months)		17.0(1–103)
	Alive without disease	37/50
	Dead of disease	4/50
	Dead of perioperative complications and other causes	9/50

CA19-9: carbohydrate antigen 19–9; CEA: carcino-embroyonic antigen; NSE: neuron specific enolase; MUC: mucin; CDX2: caudal-related homeobox transcription factor 2.

techniques include pancreatoduodenectomy for pancreatic head lesions and distal pancreatectomy for pancreatic body and tail lesions. However, partial or segmental resection can also be accepted under the premise of a negative margin. For distant metastasis and recurrence, a favorable prognosis can also be acquired after reoperation.

An appropriate surveillance strategy after surgery for IPMN has not been established because of the lack of large-scale retrospective studies and inadequate follow-up of patients with IPMN who have undergone surgical resection. Therefore, as an independent entity distinct from other IPMN subtypes with a relatively favorable prognosis, the management and follow-up strategy for IOPN-P should be different from that of IPMN. In a study of IPMN in Japan that included 257 consecutive patients with IPMN who underwent surgical treatment, the authors reported that extrapancreatic recurrence rather than remnant pancreatic recurrence influenced the overall survival [35]. Furthermore, a positive resection margin was the only independent factor for remnant pancreatic recurrence, and invasive IPMN, mixed-type neoplasia, an elevated serum CA19-9, and intraoperative transfusion were the

Table 2
Preoperative examinations.

Examinations	Presentations	n/d
Serum CA19-9	Normal	15/17
	Elevated CA19-9	2/17
Serum CEA	Normal	16/17
	Elevated CEA	1/17
Abdominal US	Cystic lesion	6/6
Enhanced CT	Cystic lesion with solid component or mural nodule	7/19
	Only cystic lesion	11/19
	Solid lesion	1/19
MRI	CR with hypointensity on T1-WI	2/2
	CR with hyperintensity on T2-WI	2/2
	MN with hypointensity on T1 and T2-WI	1/1
ERCP/Endoscopy	Dilated PD and filling defect	8/8
	Prominent papillary	2/3
	Normal papillary	1/3
EUS	Cystic lesion	2/6
	Cystic lesion with mural nodule	4/6
FDG-PET	Elevated FDG uptake in the mural nodule	4/6
	Tumor diffused FDG uptake	2/6
Cytologic analysis	No malignant cell	4/12
	Adenocarcinoma	3/12
	Non-diagnostic carcinoma cell	1/12
	IOPN	4/12

CA 19–9: carbohydrate antigen 19–9; CEA: carcinoembryonic antigen; US: ultrasound; CT: computed tomography; MRI: magnetic resonance imaging; CR: cystic region; T1-WI: T1-weighted images; T2-WI: T2-weighted images; MN: mural nodule; ERCP: endoscopic retrograde cholangiopancreatography; PD: pancreatic duct; EUS: endoscopic ultrasonography; FDG-PET: fluoro-2-deoxy-D-glucose-positron emission tomography; FNA pathology: fine needle aspiration pathology.

Table 3
Preoperative diagnosis, diagnostic methods, and treatment.

Items		n/d(%)
Preoperative diagnosis	IPMN	14/47
	MCN/MCC	10/47
	PDAC	13/47
	IOPN	8/47
	Neuroendocrine tumor	1/47
	Undefined cystic mass	1/47
Diagnostic methods	Postoperative pathology	61/66(92)
	FNA pathology	3/66(5)
	Pancreatic juice pathology	2/66(3)
Treatment	PD	20/64(31)
	PPPD	4/64(6)
	DP or DP + splenectomy	9/64(14)
	Total pancreatectomy	2/64(3)
	Partial pancreatectomy	4/64(6)
	Abdominal transplant surgery	1/64(2)
	Surgical resection(unclear detailed)	24/64(38)

IPMN: intraductal papillary mucinous neoplasm; MCN: mucinous cystic neoplasm; MCC: mucinous cystadenocarcinoma; PDAC: pancreatic ductal adenocarcinoma; IOPN: intraductal oncocytic papillary neoplasm; FNA: fine needle aspiration; PD: pancreaticoduodenectomy; PPPD: pylorous-preserving pancreaticoduodenectomy; DP: distal pancreatectomy.

independent factors for extrapancreatic recurrence [35]. Therefore, they suggested that patients with IPMN who are at risk for remnant pancreatic recurrence undergo continual surveillance every 6 months for >5 years and that patients at risk for extrapancreatic recurrence undergo surveillance every 3 months for 1 year after the operation and every 6 months thereafter [35]. Another study also showed that recurrence of noninvasive branch duct IPMN is infrequent and could be managed conservatively [29]. The risk factors for IOPN-P recurrence are similar to those for other IPMN subtypes, but noninvasive or minimally invasive neoplasia, a normal CA19-9 level, and remnant pancreatic recurrence of IOPN-P may contribute to the favorable prognosis of IOPN-P. The 5- and 10-year recurrence rates for IOPN-P are higher than those for other IPMN subtypes (31% vs. 17% and 46% vs. 19%, respectively), and there is a high rate of recurrence for IOPN-P after more than 5 years, and this risk is increased after than 10 years [30]. However, even in patients who undergo reoperation, the overall survival is favorable and death is rare [36]. Therefore, long-term surveillance for IOPN-P is needed. We recommend a distinct long-term surveillance and management strategy for recurrent IOPN-P as shown in Fig. 2.

In recent years, research of the pathogenesis of IPMN has extended deep into the molecular and genetic fields of study. *KRAS* mutations play an essential role in the pathogenesis of IPMN and PDAC [37]. Point mutations at codon 12 in exon 2 have been reported in 31%–68% of patients with IPMN [38,39]. Interestingly, although *KRAS* mutations play an important role in the

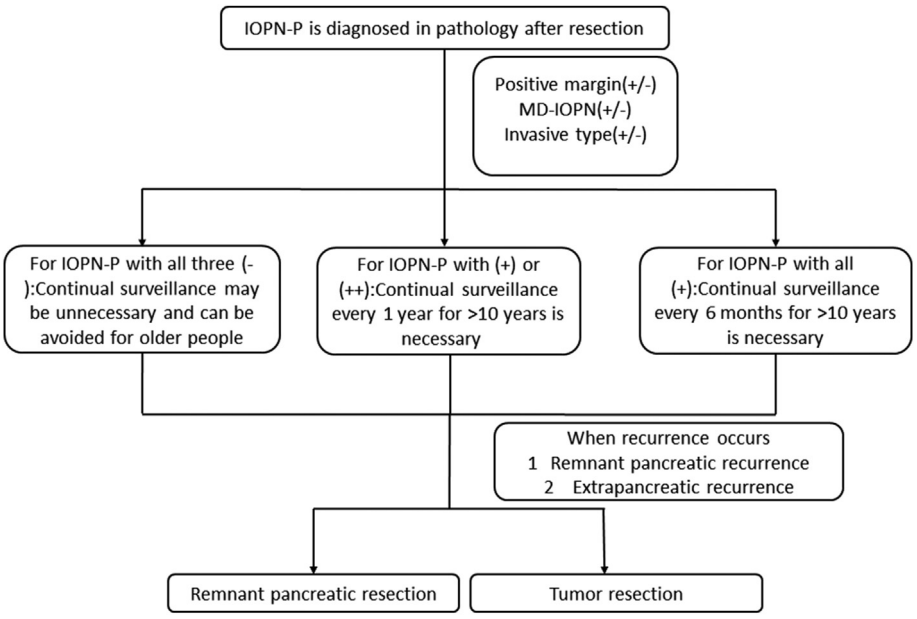

Fig. 2. Surveillance and management strategy for recurrent IOPN-P

pathogenesis of IPMN, these mutations might not be necessary in the pathogenesis of IOPN-P. A study by Basturk et al. [4] showed that the mutations of *KRAS*, *GNAS*, and *RNF3* that are common in patients with IPMN were rarely discovered in patients with oncocytic-type IPMN. *ARHGAP26*, *ASXL1*, *EPHA8*, and *ERBB4* mutations were found in more patients with IOPN-P than in patients with other subtypes [4]. Chang et al. [40] also confirmed this result, finding no *KRAS* mutations in patients with oncocytic-type IPMN [40]. Therefore, given that IOPN-P is genetically distinct from other subtypes of IPMN, IOPN-P can be distinguished by its typical gene profile, such as *ARHGAP26*, *ASXL1*, *EPHA8*, and *ERBB4* mutations. However, the specificity of this method requires further study.

IOPN-P can be easily misdiagnosed as other IPMN subtypes or pancreatic cystic lesions until preoperative FNA biopsy reveals oncocytic cells or postoperative pathologic examination shows oncocytic characteristics. A short review of IOPN-P in 2016 summarized the possible differential diagnoses, including other intraductal growth neoplasms and oncocytic lesions of the pancreas [13]. IOPN-P with a solid component should also be distinguished from typical PDAC because the solid component may show a pseudoinvasive appearance, which mimics the behaviors of PDAC; however, unlike IPMNs, IOPN-P seldom give rise to PDAC [41]. Using highly specific preoperative cytology and immunohistochemical analysis, most other diagnoses can be excluded, and the correct diagnosis can even be made preoperatively [42]. The typical and key characteristics of IOPN-P in the clinicopathologic preoperative examination and clinical management are summarized in Table 4.

Conclusion

IOPN-P accounts for an extremely small fraction of IPMN, and very few cases have been reported. The morbidity of oncocytic type IPMN is low; however, because of the possibly different

Table 4
Typical and key characteristics of IOPN-P

Items	Typical and key characteristics
Age	*50–70*
Gender	*M = F*
Symptom	*Abdominal pain/Non-specific abdominal symptoms*
Location	*Mainly head*
Median size	*5.*50 cm
Enhanced CT	*Cystic lesion or cystic-solid lesion with mural nodules*
MRI	*Cystic lesion with hyperintensity on T2-WI*
EUS	*Dilated pancreatic duct with filling defect*
FDG-PET	*Diffuse elevated FDG uptake with high FDG uptake of mural nodule*
FNA pathology	*Oncocytic cell/Adenocarcinoma*
Operation	*PD/DP*
Postoperative pathology	*Foci invasive carcinoma/CIS*
Immunohischemical features	*MUC1(+), MUC5AC(+),MUC6(+)*
Metastasis	*Few*
Prognosis	*Favorable even when metastasis and recurrence*
Molecular findings	*KRAS mutations are lacking*

CT: computed tomography; MRI: magnetic resonance imaging; T2-WI: T2-weighted images; EUS: endoscopic ultrasonography; FDG-PET: fluoro-2-deoxy-D-glucose-positron emission tomography; FNA: fine needle aspiration; PD: pancreatoduodenectomy; DP: distal pancreatectomy; CIS: carcinoma *in situ*; MUC: mucin.

pathogenesis and the better prognosis of IOPN-P, it is necessary to summarize the characteristics of IOPN-P and use multimodal examinations to make a definitive preoperative diagnosis. Previous studies paid more attention to the pathological characteristics than the clinical management of IOPN-P. As research has deepened, the focus has shifted to the prognosis, the molecular and genetic pathogenesis, and the differences between IOPN-P and other subtypes of IPMN. In the present study, we used an evidence-based systematic review to summarize the demographic and clinicopathologic characteristics and the management of IOPN-P from the first report in 1996. We also briefly introduced recent studies of the molecular pathogenesis of IOPN-P. In conclusion, IOPN-P may be an independent entity that should be distinguished from typical IPMN, and large-scale cohort studies are needed to further explore the peculiarity of IOPN-P.

Disclosure statement

The authors declare that they have no conflict of interest.

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Authors' contributions

Study design: Yi-Zhi Wang and Jun Lu. Data search, analysis, and interpretation: Yi-Zhi Wang and Jun Lu. Initial draft: Yi-Zhi Wang. Critical revision for important intellectual content: Bo-Lun Jiang and Jun-Chao Guo. All authors approved the final version of the manuscript and are accountable for all aspects of the work. Jun-Chao Guo has full responsibility for the conduct of the study, has access to the data, and controlled the decision to publish.

Conflicts of interest

None declared.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.pan.2019.07.040.

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