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Cytology and KRAS/GNAS Molecular Testing of Pancreatic Cyst Fluid for Risk Stratification of Intraductal Papillary Mucinous Neoplasms: a Single-Center Study with Histological Correlation

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Abstract

Background Accurate preoperative risk stratification of intraductal papillary mucinous neoplasms (IPMNs) remains a major challenge in pancreatic surgery. Cytology obtained through endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) demonstrates high specificity but limited sensitivity, whereas molecular analysis of cyst fluid—particularly KRAS and GNAS mutations—has emerged as a promising complementary diagnostic tool. **Methods** We conducted a narrative review combined with a retrospective single-center observational study of patients evaluated for suspected IPMN between 2018 and 2025 who underwent EUS-FNA with cytology and KRAS/GNAS testing followed by surgical resection. Histology was used as the reference standard. **Results** A total of 105 patients were included, of whom 70 underwent EUS-FNA and 25 surgical resection. Final histology showed low-grade dysplasia in 12 cases (48%) and high-grade dysplasia in 13 cases (52%), with no invasive carcinoma detected. Cytology demonstrated a sensitivity of 38.5% and specificity of 75% for advanced neoplasia. Molecular testing achieved 100% sensitivity but low specificity. A combined diagnostic strategy increased sensitivity to 92.3% compared with 38.5% for cytology alone, although with reduced specificity. **Conclusions** A multimodal diagnostic approach integrating morphology, cytology, and molecular testing improves risk stratification of IPMNs and supports surgical decision-making within multidisciplinary pancreatic teams.

Keywords: intraductal papillary mucinous neoplasm; pancreatic cyst fluid; endoscopic ultrasound-guided fine needle aspiration; KRAS mutation; GNAS mutation; molecular profiling

Introduction

The widespread use of high-resolution cross-sectional imaging has led to a rapidly increasing detection of pancreatic cystic lesions, posing a growing diagnostic and management challenge for pancreatic surgeons and multidisciplinary teams. Among these lesions, intraductal papillary mucinous neoplasms (IPMNs) represent one of the most clinically relevant entities because of their recognized potential to progress to pancreatic ductal adenocarcinoma (PDAC).[1,2]

IPMNs arise from the pancreatic ductal epithelium and are characterized by mucin-producing epithelial proliferation with varying degrees of dysplasia. According to ductal involvement, they are classified as main duct IPMN, branch duct IPMN, or mixed-type IPMN. Main duct involvement is associated with a significantly higher risk of malignant transformation compared with branch duct lesions.[3,4]

Over the past two decades, several international consensus guidelines have been developed to guide the management of IPMNs. The Fukuoka consensus guidelines and subsequent updates

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integrate cyst morphology, main pancreatic duct dilation, mural nodules, and clinical symptoms to determine indications for surgical resection or surveillance.[35]

Despite these advances, preoperative risk stratification remains challenging, particularly for branch-duct IPMNs lacking clear high-risk stigmata. Imaging alone often fails to reliably distinguish low-grade from high-grade dysplasia, leading to uncertainty in clinical decision-making.[6]

Endoscopic ultrasound-guided fine needle aspiration (EUS-FNA) represents a cornerstone of pancreatic cyst evaluation. Cytological examination of cyst fluid remains highly specific when positive, yet its sensitivity is limited by low cellularity, sampling variability, and operator dependence.[7]

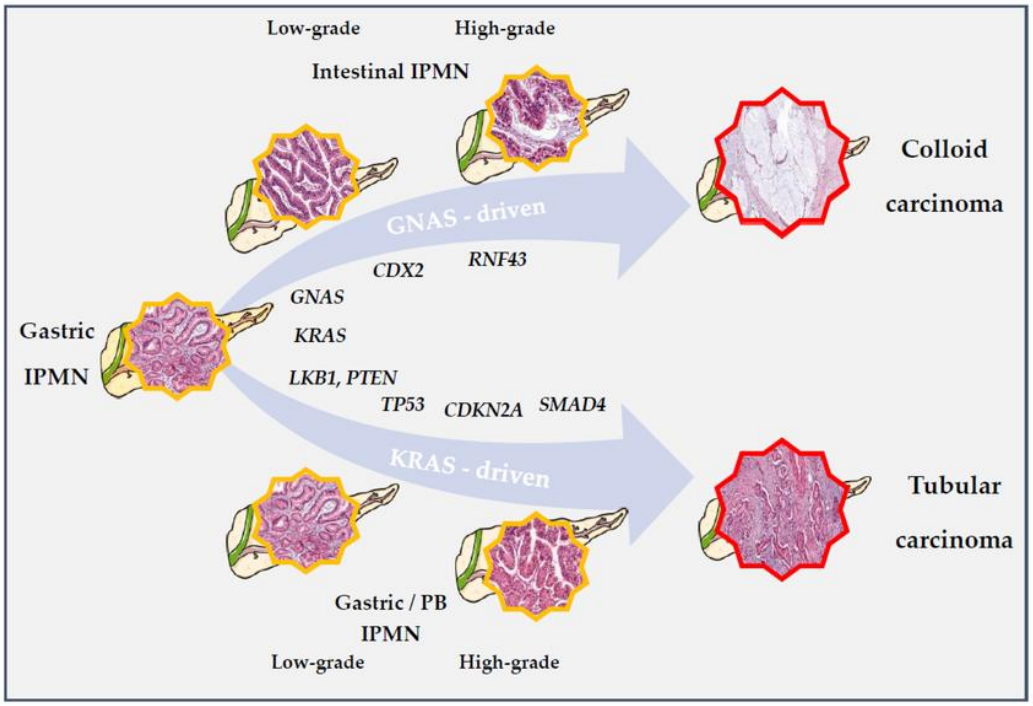
Molecular analysis of pancreatic cyst fluid has emerged as an important adjunct diagnostic tool. Mutations in the KRAS oncogene represent early events in pancreatic tumorigenesis and are frequently detected in mucinous pancreatic cysts.[8]

In addition, activating mutations in the GNAS gene have been identified as highly characteristic of IPMNs and may help distinguish them from other cystic lesions.[9]

The integration of cytological findings with molecular analysis may therefore provide a more comprehensive diagnostic framework for the management of pancreatic cysts. Several studies have demonstrated that the combination of cytology, molecular markers, and imaging features improves the diagnostic classification of pancreatic cystic lesions.[10]

The aim of the present study was to evaluate the complementary diagnostic roles of cytology and KRAS/GNAS molecular testing in the preoperative evaluation of intraductal papillary mucinous neoplasms and to explore the potential value of a multimodal diagnostic strategy supported by histologically confirmed surgical specimens.

The molecular progression of IPMNs and the role of KRAS and GNAS alterations are illustrated in Figure 1.



(**a**)

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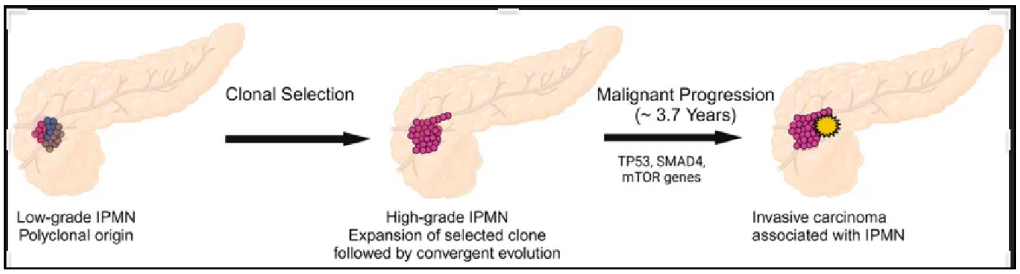

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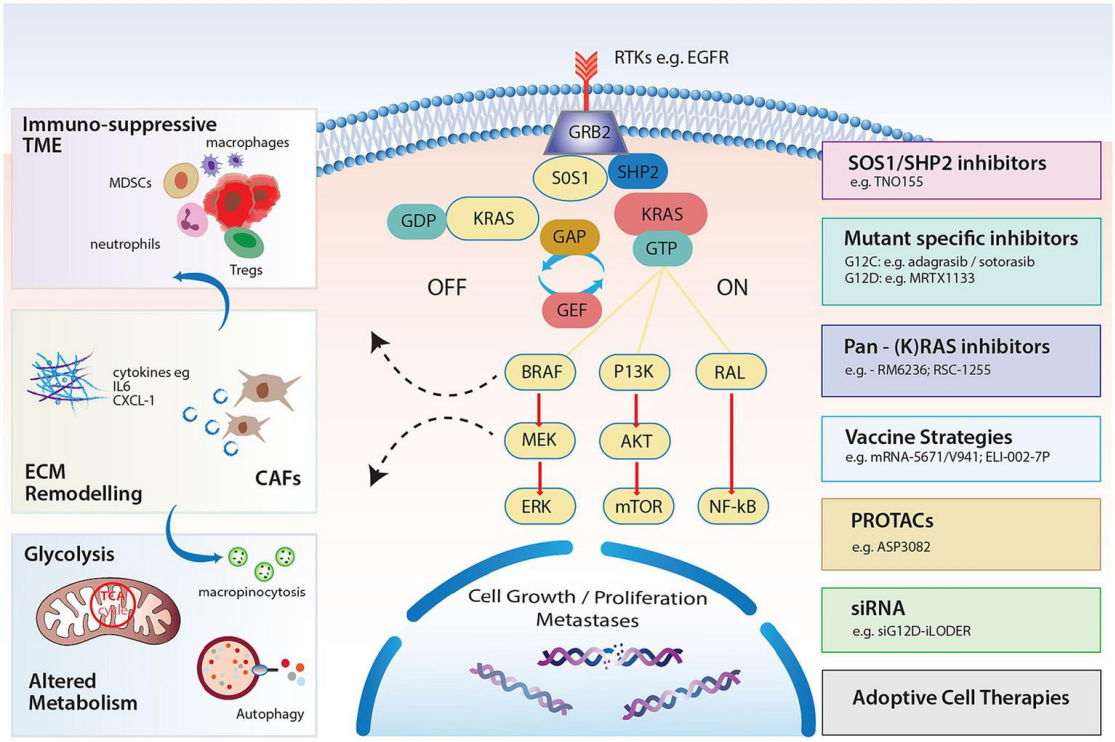

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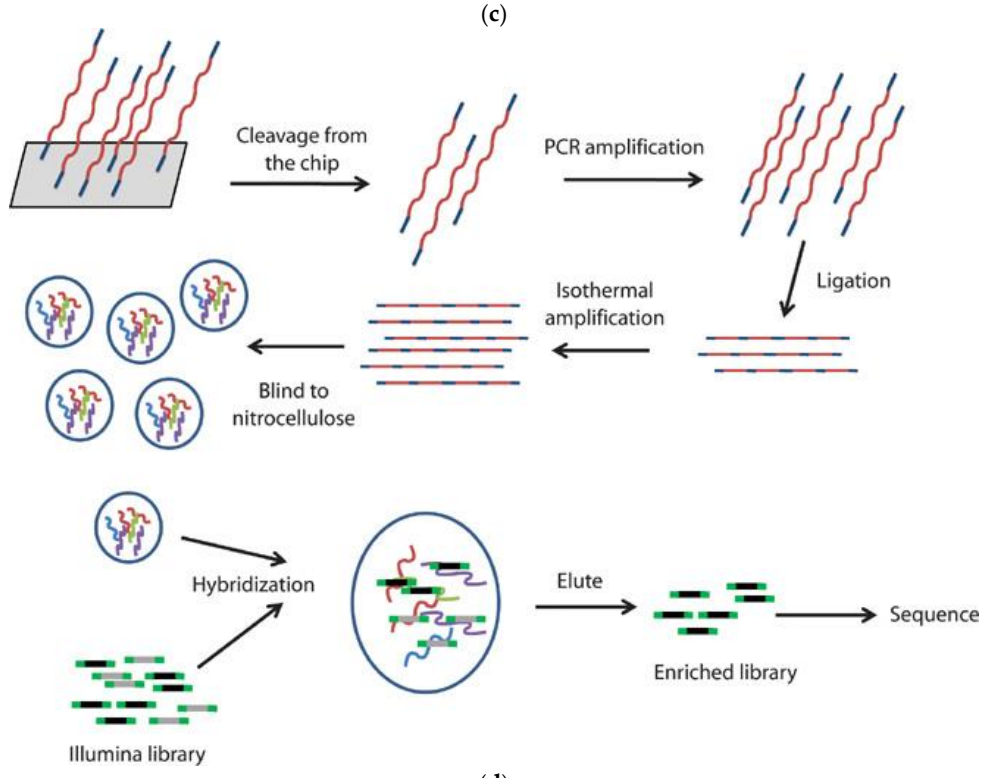

(**d**)

Figure 1. (**a–d**). Molecular progression model of intraductal papillary mucinous neoplasms.

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Methods

Study Design

We performed a retrospective single-center observational study including consecutive patients evaluated for suspected IPMN between 2018 and 2025 at a tertiary pancreatic surgery center. Patients were included if they met the following criteria: suspected IPMN based on cross-sectional imaging, EUS-FNA with cyst fluid sampling, cytological analysis performed, KRAS and/or GNAS molecular testing available. Clinical, radiological, cytological, molecular, and histopathological data were retrieved from institutional electronic records.

The study protocol was approved by the local Institutional Review Board, and all procedures were conducted in accordance with the Declaration of Helsinki.

Endoscopic Ultrasound and Cyst Fluid Analysis

EUS examinations were performed using standard equipment. Cyst size, location, communication with the pancreatic duct, mural nodules, and ductal dilation were recorded. Cyst fluid samples were obtained through EUS-guided fine needle aspiration and divided for cytological and molecular analysis.

Cytology

Cytological specimens were classified according to the **Papanicolaou Society of Cytopathology (PSC) classification system**. Diagnostic categories included:

- Non-diagnostic
- Negative
- Atypical
- Neoplastic benign
- Neoplastic other
- Suspicious for malignancy
- Positive/malignant

For diagnostic accuracy analyses, the threshold for malignancy was defined as **suspicious for malignancy or positive/malignant**.

Molecular Analysis

Molecular testing evaluated the presence of **KRAS and GNAS mutations** using PCR-based assays. Molecular testing was considered positive when either KRAS or GNAS mutations were detected.

Reference Standard

Histological diagnosis on surgical specimens was used as the reference standard. IPMN dysplasia was classified as low-grade dysplasia (LGD), high-grade dysplasia (HGD), invasive carcinoma, according to WHO criteria. For the purpose of diagnostic performance analysis, advanced neoplasia was defined as high-grade dysplasia or invasive carcinoma on final surgical histology**.**

Statistical Analysis

Continuous variables were summarized as median with interquartile range. Categorical variables were expressed as frequencies and percentages.

Diagnostic performance was evaluated by calculating sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV).

Statistical analyses were performed using standard statistical software (e.g., R statistical software, R Foundation for Statistical Computing, Vienna, Austria)

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Results

Cohort Characteristics

A total of **105 patients** with suspected IPMN were included in the study. Baseline characteristics are summarized in Table 1.

The median patient age was **72 years**, and the cohort showed a slight male predominance. EUS-FNA was performed in **70 patients (66.7%)**, while **25 patients (23.8%)** underwent surgical resection.

Table 1.—Baseline characteristics.

Variable	Value
Patients	105
Median age	72 years
Female	49 (46.7%)
Male	56 (53.3%)
Median cyst size	8 mm (IQR 6–32)
EUS-FNA performed	70 (66.7%)
Surgical resection	25 (23.8%)
Worrisome features	39 (37.1%)
High-risk stigmata	10 (9.5%)

Histological Findings

Among the **25 patients undergoing surgical resection**, final histology demonstrated:

- 12 cases of low-grade dysplasia (48%)
- 13 cases of high-grade dysplasia (52%)

No cases of invasive carcinoma were observed.

Cytological Findings

The distribution of PSC cytology categories compared with histology is shown in Table 2.

Table 2.—Cytology versus histology.

Cytology category	HGD	LGD
Atypical	3	4
Negative	3	3
Neoplastic benign	2	2
Suspicious for malignancy	3	3
Positive/malignant	2	0

Cytology demonstrated relatively high specificity but limited sensitivity for the detection of advanced neoplasia.

For diagnostic accuracy analysis, cytology results classified as “suspicious for malignancy” or “positive/malignant” were considered positive for advanced neoplasia, whereas all other categories were considered negative.

Molecular Analysis

KRAS/GNAS molecular results were available in **20 of 25 resected patients (80%)**. Molecular positivity demonstrated very high sensitivity for advanced neoplasia but low specificity.

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Diagnostic Performance

The diagnostic performance of cytology, molecular testing, and the combined approach is summarized in Table 3.

Table 3.—Diagnostic performance.

Test	Sensitivity	Specificity	PPV	NPV
PSC cytology	38.5%	75.0%	62.5%	52.9%
KRAS/GNAS testing	100%	12.5%	63.2%	100%
Combined strategy	92.3%	33.3%	60.0%	80.0%

The combined diagnostic strategy significantly improved sensitivity compared with cytology alone.

Discussion

Our findings highlight the complementary diagnostic roles of cytology and molecular analysis in the preoperative evaluation of IPMNs. Cytology demonstrated relatively high specificity but limited sensitivity for detecting advanced neoplasia, consistent with previous reports describing the diagnostic limitations of cyst fluid cytology.[711]

Conversely, KRAS and GNAS testing showed very high sensitivity but markedly reduced specificity for high-grade dysplasia. This observation is biologically plausible because these mutations primarily represent markers of mucinous lineage rather than predictors of dysplastic grade.[89]

KRAS mutations occur early during pancreatic tumorigenesis and are detected in a large proportion of mucinous pancreatic cysts.[8] GNAS mutations, in contrast, are strongly associated with IPMNs and rarely observed in other pancreatic cystic neoplasms.[9]

Our findings confirm previous studies demonstrating that molecular analysis improves the diagnostic classification of pancreatic cysts, particularly when integrated with cytological and clinical information.[1012]

The present findings are broadly consistent with the growing body of evidence supporting molecular analysis of pancreatic cyst fluid as a complementary diagnostic tool in IPMN evaluation. Early sequencing studies showed that cyst fluid mutational profiling could improve the diagnostic and prognostic stratification of pancreatic cystic lesions, particularly in cases not fully characterized by imaging or conventional biomarkers. Subsequent studies demonstrated that the identification of KRAS and GNAS alterations increases the detection of mucinous/IPMN lineage, while additional alterations such as TP53, SMAD4, and CDKN2A may better support recognition of biologically advanced lesions. Notably, Singhi et al. reported high sensitivity of preoperative cyst fluid NGS for IPMN, whereas pooled evidence from a later meta-analysis confirmed overall diagnostic utility but also highlighted relevant heterogeneity across studies. More recent evidence and the Kyoto 2024 guidelines support the use of molecular markers as adjuncts within a multimodal framework rather than as isolated decision tools. At the same time, recent cohort data suggest that the incremental value of KRAS/GNAS testing may be limited when conventional diagnostic work-up is already sufficiently informative, reinforcing the importance of selective use in diagnostically equivocal cases. These considerations are summarized in Table 4.

Table 4. Selected studies evaluating KRAS/GNAS molecular analysis in pancreatic cyst fluid for IPMN risk stratification.

Study	Year	Study design	Main finding	Clinical relevance
Amato et al.	2014	Targeted NGS analysis	Molecular profiling of cyst fluid improved diagnostic and prognostic	Early evidence supporting molecular testing as adjunct to

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			classification of pancreatic cystic neoplasms	conventional assessment
Jones et al.	2016	Clinical impact study	KRAS/GNAS mutations improved identification of mucinous cysts, particularly when conventional markers were indeterminate	Suggested that molecular testing may influence preoperative classification
Rosenbaum et al.	2017	Cytology + molecular analysis	KRAS/GNAS mutations improved recognition of mucinous cysts; TP53/SMAD4/CDKN2A mutations associated with high-risk lesions	Demonstrated complementary role of molecular testing with cytology
Singhi et al.	2018	Preoperative NGS study	KRAS/GNAS mutations showed high sensitivity for IPMN and specificity for mucinous cystic lesions	Landmark study supporting integration of molecular testing in cyst evaluation
Volckmar et al.	2019	Prospective biomarker study	NGS analysis distinguished IPMN from pseudocysts and identified multiclonal driver alterations	Highlighted biological heterogeneity of pancreatic cystic neoplasms
McCarty et al.	2021	Systematic review and meta-analysis	KRAS/GNAS testing showed good diagnostic performance but significant heterogeneity across studies	Supports molecular testing as adjunct rather than stand-alone tool
Belfrage et al.	2024	Diagnostic accuracy study	NGS improved identification of mucinous, malignant, or premalignant cysts leading to surgery	Reinforces role of integrated molecular work-up
Ohtsuka et al. (Kyoto Guidelines)	2024	International guideline	Molecular markers recognized as adjunct tools in IPMN evaluation	Confirms guideline-level relevance of molecular testing
Gyimesi et al.	2025	Cohort study	KRAS/GNAS mutations did not significantly improve detection of mucinous cysts after conventional assessment	Highlights variability of molecular testing benefit

From a surgical perspective, the most clinically relevant observation is the incremental diagnostic yield of molecular testing in cytology-negative or non-diagnostic cases. Several patients with histologically confirmed high-grade dysplasia demonstrated non-malignant cytology but positive KRAS/GNAS mutations. Similar findings have been reported in other surgical-pathologic series.[12]

However, the low specificity of KRAS/GNAS mutations highlights an important limitation: molecular positivity alone should not drive surgical decision-making in the absence of concordant high-risk morphological features. Current international guidelines emphasize the need for multimodal risk stratification, integrating imaging findings, clinical features, and adjunct diagnostic tools.[3513]

Recent research has explored expanded next-generation sequencing panels for pancreatic cyst characterization, which may further improve diagnostic accuracy.[14]

Future studies integrating molecular profiling with imaging-based approaches such as radiomics and artificial intelligence may further improve risk stratification and personalized management of pancreatic cystic lesions.[15]

A proposed multimodal diagnostic workflow integrating cytology and molecular testing is presented in Figure 2.

From a surgical perspective, the key clinical question in IPMN management remains the identification of patients who truly benefit from pancreatic resection. Overestimation of malignant

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potential may expose patients to unnecessary surgery, whereas underestimation risks delaying treatment of high-grade dysplasia or invasive carcinoma.

In this context, the integration of molecular testing with established clinical and radiological criteria may represent an important step toward more individualized risk stratification. However, molecular positivity alone should not be interpreted as an indication for surgery, particularly given the high prevalence of KRAS and GNAS mutations in biologically indolent mucinous cysts.

Instead, molecular testing should be considered within a multimodal diagnostic framework combining imaging features, cyst fluid cytology, and clinical risk factors. Such an approach may reduce diagnostic uncertainty and improve selection of surgical candidates.

Future multicenter prospective studies integrating molecular profiling with advanced imaging biomarkers and artificial intelligence–based models may further refine risk prediction and support precision management of pancreatic cystic neoplasms.

Study Limitations

This study has several limitations that should be acknowledged. First, the retrospective single-center design may introduce selection bias and limits the generalizability of the findings. Second, the number of patients undergoing surgical resection was relatively small, reflecting the clinical reality that only a subset of pancreatic cystic lesions ultimately require operative management. Third, molecular analysis was available only in a proportion of resected cases, which may have influenced the estimation of diagnostic performance.

An additional limitation relates to the potential selection bias inherent to surgical series. Diagnostic performance was evaluated only in patients who ultimately underwent surgical resection, which may overrepresent lesions with higher clinical suspicion. However, the use of histologically confirmed surgical specimens provides a robust reference standard for evaluating the complementary roles of cytology and molecular testing.

Despite these limitations, the use of histologically confirmed surgical specimens as the reference standard represents a major strength of the present study and provides clinically meaningful insight into the complementary roles of cytology and molecular testing in the preoperative evaluation of IPMNs.

Conclusions

The integration of cytology and KRAS/GNAS molecular profiling improves the preoperative risk stratification of IPMNs. Cytology remains highly specific but limited in sensitivity, whereas molecular testing provides high sensitivity but limited specificity.

A multimodal diagnostic approach combining morphology, cytology, and molecular testing may therefore optimize clinical decision-making and support tailored surgical management within multidisciplinary pancreatic teams.

Author Contributions: L.M. conceived the study, collected and analyzed the data, and drafted the manuscript. E.A. contributed to data collection and interpretation. S.L. performed endoscopic procedures and contributed to methodological aspects. A.F. performed pathological evaluation. E.J. supervised the study and critically revised the manuscript. All authors approved the final version of the manuscript.

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Institutional Review Board Statement: The study was conducted according to institutional policies and in accordance with the Declaration of Helsinki.

Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Conflicts of Interest: The authors declare no conflict of interest.

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Abbreviations

IPMN	Intraductal papillary mucinous neoplasm
EUS	Endoscopic ultrasound
FNA	Fine needle aspiration
PSC	Papanicolaou Society of Cytopathology
LGD	Low-grade dysplasia
HGD	High-grade dysplasia
NGS	Next-generation sequencing

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