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Acinar Gland Hyperplasia Masquerading as Pancreatic Head Carcinoma: A Case Report on a Diagnostic and Surgical Dilemma

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A B S T R A C T

The preoperative differentiation of benign from malignant pancreatic head masses presents a significant clinical challenge. While pancreatic ductal adenocarcinoma (PDAC) is the primary concern, rare benign pathologies can be radiologically and clinically indistinguishable from cancer, leading to diagnostic uncertainty and complex surgical decisions. A 61-year-old male presented with a classic triad of obstructive jaundice, significant weight loss, and right upper abdominal pain. A contrast-enhanced computed tomography (CT) scan of the abdomen revealed a solid mass in the head of the pancreas, causing concomitant dilation of the common bile and pancreatic ducts—the "double duct sign." These findings were highly suggestive of pancreatic head carcinoma, prompting a decision for surgical intervention. The patient underwent a standard pancreaticoduodenectomy (Whipple procedure). Surprisingly, the final histopathological examination of the resected specimen revealed no evidence of malignancy. The diagnosis was benign acinar gland hyperplasia. The postoperative course was complicated by a delayed post-pancreatectomy hemorrhage from a gastroduodenal artery pseudoaneurysm, which was successfully managed with minimally invasive transarterial embolization (TAE). In conclusion, acinar gland hyperplasia is an exceedingly rare benign condition that can precisely mimic the clinical and radiological features of pancreatic cancer. This case underscores the current limitations of preoperative diagnostics and affirms that aggressive surgical management is justified in patients with a high suspicion of malignancy, as the risk of withholding surgery for a potentially curable cancer outweighs the risk of resecting a benign lesion. Furthermore, it highlights that the Whipple procedure carries a significant risk of life-threatening complications, such as delayed hemorrhage, irrespective of the underlying pathology, necessitating vigilant postoperative care.

1. Introduction

A solid mass in the head of the pancreas represents one of the most formidable diagnostic challenges in modern surgical oncology.[1] The overwhelming majority of these lesions are pancreatic ductal adenocarcinoma (PDAC), a malignancy with a notoriously poor prognosis, making prompt and accurate diagnosis imperative.[2] The standard diagnostic pathway typically involves cross-sectional imaging, primarily contrast-enhanced computed tomography (CT), which can identify a mass and secondary signs of malignancy. The "double duct sign," characterized by the simultaneous dilation of the common bile duct (CBD) and the main pancreatic duct (MPD), is a radiological finding with high specificity for an obstructing lesion in the pancreatic head, most commonly PDAC.[3]

However, a critical diagnostic gray area exists, as a subset of pancreatic head masses are benign lesions that can be clinically and radiologically indistinguishable from their malignant counterparts. This spectrum of "cancer mimics" includes chronic pancreatitis (particularly focal mass-forming types), autoimmune pancreatitis, groove pancreatitis, solid pseudopapillary neoplasms, and various cystic lesions.[4] Misidentifying these benign conditions as cancer can lead to the significant morbidity and mortality associated with major pancreatic resection, such as the pancreaticoduodenectomy (Whipple procedure).[5] Conversely, misdiagnosing a true cancer as a benign entity can deny a patient their only chance at a cure.

Within this differential diagnosis lies acinar gland hyperplasia (AGH), an exceptionally rare and poorly understood benign condition of the pancreas.[6] It is histologically defined by the proliferation of pancreatic acini, forming unencapsulated but well-demarcated lobules that can coalesce into a focal mass. Due to its rarity, AGH is seldom considered in the initial differential diagnosis, and its characteristics on advanced imaging like CT or magnetic resonance imaging (MRI) are not well-defined.[7]

This report presents the case of a 61-year-old male whose clinical presentation and imaging findings were archetypal for pancreatic head carcinoma, leading to a Whipple procedure.[8] The final pathology, however, revealed only benign acinar gland hyperplasia. The case was further complicated by a life-threatening postoperative hemorrhage, underscoring the inherent risks of the surgical intervention.[9,10] The aim of this manuscript is to highlight AGH as a rare but important cancer mimic, discuss the justification for aggressive surgery in the face of diagnostic ambiguity, explore the pathophysiology of AGH, and analyze the management of severe postoperative vascular complications. The novelty of this report lies in its detailed account of a rare benign pathology masquerading as cancer, coupled with the management of a classic, severe surgical complication, providing valuable lessons for surgeons, gastroenterologists, and radiologists.

2. Case Presentation

A 61-year-old male presented to our surgical outpatient clinic with a chief complaint of right upper abdominal pain that had progressively worsened over the preceding week. The patient reported that this pain had been present intermittently for approximately one year but had recently become constant and more severe. Over the past two weeks, he had developed progressive jaundice, noted as a yellowing of his skin and sclera, accompanied by pruritus. He also reported a significant, unintentional weight loss of approximately 10 kg over three months. His history was notable for changes in stool color, which had become acholic (pale and pasty), and dark, tea-colored urine. These symptoms were associated with nausea and a marked decrease in appetite. The patient had no significant past medical history, specifically no history of chronic pancreatitis, heart disease, hypertension, or diabetes mellitus, and was not a regular consumer of alcohol (Table 1).

On physical examination, the patient was visibly jaundiced. His vital signs were stable. The abdominal examination revealed marked tenderness to deep palpation in the epigastrium and right hypochondrium, without guarding or rebound tenderness. There were no signs of ascites, and no palpable abdominal mass or hepatosplenomegaly was appreciated. Courvoisier's sign (a palpable, non-tender gallbladder) was negative.

Initial laboratory investigations were significant for cholestatic jaundice and hepatic injury. The results showed markedly elevated total bilirubin at 13.23 mg/dL, with elevations in serum glutamic-oxaloacetic transaminase (SGOT) to 108 U/L and serum glutamic-pyruvic transaminase (SGPT) to 202 U/L. His hematological parameters and renal function were within normal limits, and his albumin level was adequate at 4.2 g/dL, suggesting a relatively preserved nutritional status despite his weight loss (Table 2).

To further characterize the cause of his obstructive jaundice, a contrast-enhanced multiphasic abdominal CT scan was performed. The scan revealed a poorly defined, hypodense solid lesion in the head of the

pancreas, measuring approximately 3.2 x 2.5 cm. This mass was causing significant upstream dilation of both the common bile duct, to a diameter of 1.6 cm, and the main pancreatic duct, to a diameter of 7 mm, creating a classic "double duct sign". There was no evidence of vascular invasion of the superior mesenteric artery or portal vein. However, several enlarged lymph nodes were noted in the peripancreatic and paraaortic regions, raising suspicion of regional metastasis. No distant metastases, such as liver lesions, were identified. Based on the combination of the patient's age, clinical presentation (painless, progressive jaundice and weight loss), and classic CT findings of a pancreatic head mass with a double duct sign and regional lymphadenopathy, a preoperative diagnosis of resectable pancreatic head carcinoma (T2N1M0) was made.

Given the high suspicion for malignancy and the patient's fitness for major surgery, the decision was made to proceed with a pancreaticoduodenectomy (Whipple procedure) with curative intent. Intraoperatively, the pancreatic head was noted to be diffusely firm and indurated, without a distinct, rock-hard nodule but with an ill-defined fullness that was consistent with the preoperative imaging. There was no gross evidence of vascular invasion or metastatic disease on the liver or peritoneal surfaces. A standard Whipple procedure was completed, involving en-bloc resection of the pancreatic head, duodenum, gallbladder, distal bile duct, and proximal jejunum (Figure 2). Gastrointestinal continuity was restored with a pancreaticojejunostomy, a hepaticojejunostomy, and a gastrojejunostomy.

Table 1. Summary of the patient's clinical findings on admission.

Summary of Patient Information and Clinical Findings

CATEGORY	FINDING / PARAMETER	RESULT	SIGNIFICANCE / NOTE
Demographics	Age / Sex	61 years / Male	*Age is a significant risk factor for pancreatic neoplasms.*
Clinical Presentation	Chief Complaint	Progressive right upper abdominal pain	*Worsening over 1 week; intermittent for 1 year.*
	Constitutional Symptoms	Significant weight loss (~10 kg), nausea, anorexia	*"B symptoms" are highly suggestive of malignancy.*
	Signs of Biliary Obstruction	Jaundice, pruritus, acholic stools, dark urine	*Classic signs of extrahepatic cholestasis.*
	Physical Examination	Visible jaundice, epigastric & RUQ tenderness	*Localizes pathology to the pancreatobiliary region.*
	Past Medical History	Non-contributory	*No history of chronic pancreatitis or alcohol abuse.*
Laboratory Findings	Total Bilirubin	13.23 mg/dL	Markedly High
	SGOT (AST) / SGPT (ALT)	108 U/L / 202 U/L	High
	Hematology	Within normal limits	*No anemia or leukocytosis at presentation.*
	Renal & Nutritional Markers	Creatinine & Albumin normal	*Indicates good baseline organ function.*
Imaging Findings (CT Scan)	Pancreatic Mass	3.2 × 2.5 cm ill-defined, hypodense lesion in pancreatic head	*Typical appearance of pancreatic adenocarcinoma.*
	Key Radiological Sign	"Double Duct Sign"	Positive
	Staging Information	Regional lymphadenopathy; No distant metastasis	*Suggests locally advanced but resectable disease.*
Final Diagnosis	**Post-Surgical Histopathology**	**Benign Acinar Gland Hyperplasia**	

Table 2. Laboratory Test Results at Initial Presentation

PARAMETER	RESULT	REFERENCE RANGE	INTERPRETATION
Hematology Panel			
Hemoglobin	14.9 g/dL	13.5 - 17.5 g/dL	Normal
Hematocrit	41%	41 - 50%	Normal
Leukocytes (WBC)	8.8 thousand/µL	4.5 - 11.0 thousand/µL	Normal
Platelets	364 thousand/µL	150 - 450 thousand/µL	Normal
Clinical Chemistry Panel			
Total Bilirubin	**13.23 mg/dL**	0.1 - 1.2 mg/dL	Markedly High
SGPT (ALT)	202 U/L	7 - 56 U/L	High
SGOT (AST)	108 U/L	10 - 40 U/L	High
Albumin	4.2 g/dL	3.5 - 5.5 g/dL	Normal
Creatinine	0.7 mg/dL	0.6 - 1.2 mg/dL	Normal
Blood Urea Nitrogen (BUN)	15 mg/dL	7 - 20 mg/dL	Normal

The resected specimen was sent for histopathological analysis. Gross examination of the specimen revealed a firm, pale, and vaguely nodular, unencapsulated area within the pancreatic head, corresponding to the area of concern. However, microscopic examination yielded a surprising result: there was no evidence of adenocarcinoma or any other malignancy. Instead, the sections showed well-demarcated, unencapsulated lobules composed of tightly packed pancreatic acini with minimal intervening stroma. The acinar cells themselves showed no significant atypia, and mitotic figures were not observed. These findings were consistent with a benign diagnosis of focal acinar gland hyperplasia. The regional lymph nodes that were resected showed only reactive changes.

Postoperatively, the patient was transferred to the intensive care unit (ICU) for close monitoring (Table 3). His initial recovery was uneventful. However, on postoperative day 7, his abdominal drain output became frankly bloody, and he developed melena (black, tarry stools) with a corresponding drop in hemoglobin levels. This constellation of findings was highly concerning for a delayed post-pancreatectomy hemorrhage (PPH).

An urgent angiography was performed, which identified a 1.5 cm pseudoaneurysm arising from the stump of the gastroduodenal artery (GDA), which was actively bleeding. This is a known, life-threatening complication of the Whipple procedure. A minimally invasive transarterial embolization (TAE) was immediately performed. The interventional radiologist successfully deployed microcoils into the GDA stump, achieving complete occlusion of the pseudoaneurysm and cessation of bleeding.

Following the successful embolization, the patient's condition stabilized. He required blood transfusions but did not need a re-laparotomy. His subsequent recovery was slow but steady. He was eventually discharged home on postoperative day 25. At his 6-month follow-up, he was pain-free, had regained most of his lost weight, and had normal liver function tests.

Table 3. Postoperative Course and Follow-up

TIMELINE	EVENT	DETAILS	OUTCOME / STATUS
Day 0-6	Initial Recovery	Patient transferred to ICU for monitoring. Initial postoperative course was uneventful.	Stable
	Management	Standard postoperative care including pain management, IV fluids, and monitoring of vitals and drain output.	Routine Care
Day 7	Complication Detected	Abdominal drain output became frankly bloody. Patient developed melena (black, tarry stools) and a drop in hemoglobin levels.	Hemorrhage
	Diagnosis	Clinical findings highly suggestive of a delayed Post-Pancreatectomy Hemorrhage (PPH).	Critical Event
Day 7 (Urgent)	Intervention	Urgent angiography performed, identifying a 1.5 cm pseudoaneurysm of the gastroduodenal artery (GDA) stump.	Diagnostic
	Treatment	Minimally invasive Transarterial Embolization (TAE) with microcoils was performed to occlude the pseudoaneurysm.	Successful
Day 8-24	Post-Intervention Recovery	Patient stabilized following TAE. Required blood transfusions. Recovery was slow but progressive without need for re-laparotomy.	Stabilized
Day 25	Discharge	Discharged home in stable condition.	Fit for Discharge
6 Months	Long-Term Follow-up	Patient was pain-free, had regained most of his lost weight, and demonstrated normal liver function tests.	Excellent Outcome

3. Discussion

This case report presents a significant diagnostic and therapeutic conundrum: a patient whose clinical and radiological presentation was a textbook example of pancreatic head cancer, but whose final diagnosis was a rare benign entity.[11] The discussion will focus on three central themes highlighted by this case: the justification for aggressive surgery in the face of diagnostic uncertainty, an in-depth analysis of acinar gland hyperplasia as a cancer mimic, and the management of severe, life-threatening postoperative complications.

The decision to proceed with a pancreaticoduodenectomy in this patient was not merely a choice but a clinical imperative, grounded in decades of experience with pancreatic neoplasms (Figure 1). The patient's presentation—a 61-year-old male with the ominous triad of obstructive jaundice, significant weight loss, and a solid pancreatic head mass—creates a clinical picture where the preoperative probability of pancreatic ductal adenocarcinoma (PDAC) is overwhelmingly high. The addition of the "double duct sign" on computed tomography, a finding with a specificity for malignancy often exceeding 90%, further solidified this presumptive diagnosis. In this context, the presence of regional lymphadenopathy, while not definitively metastatic without tissue confirmation, served as another compelling piece of evidence pointing towards cancer.[12]

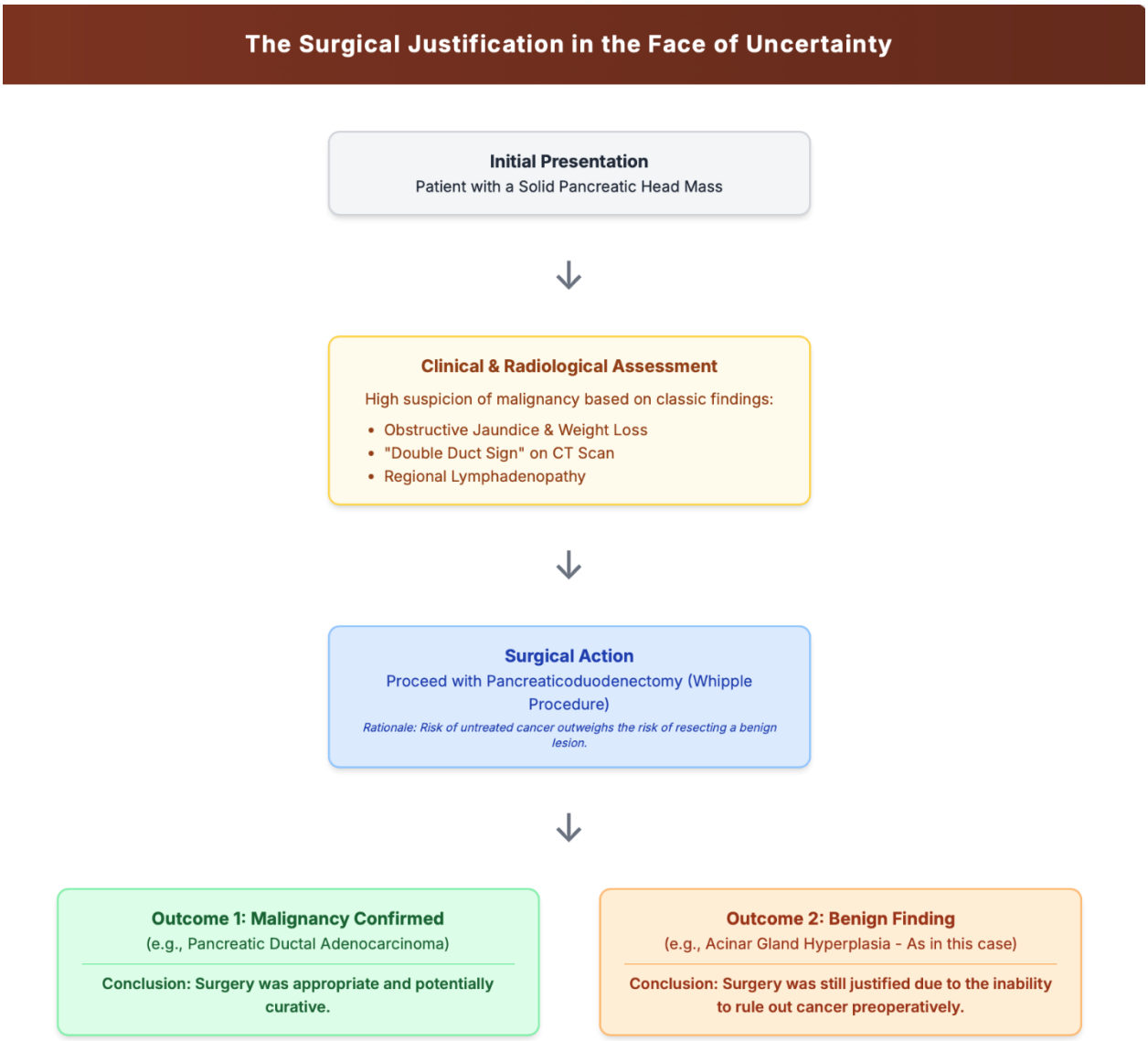

Figure 1. The surgical justification in the face of uncertainty.

In the modern era of personalized medicine, proceeding with major surgery without a definitive tissue diagnosis may seem counterintuitive.[13] However, the biology of pancreatic cancer dictates a unique surgical philosophy. PDAC is an aggressive disease with a narrow window for curative intervention.[14] Any delay in treatment can allow for micrometastatic spread or local vascular invasion, converting a resectable tumor into an unresectable one. Therefore, the primary guiding principle for a surgeon faced with a resectable pancreatic head mass is to avoid the catastrophic error of leaving a potentially curable cancer in situ. The risk of mortality from untreated PDAC approaches 100%, whereas the mortality risk of a Whipple procedure at a high-volume center is typically less than 5%.[15] This stark risk-benefit calculus heavily favors aggressive surgical intervention, even if it means a small percentage of patients will undergo a major operation for a benign condition. This approach is widely accepted as the standard of care globally.

The role of preoperative biopsy via endoscopic ultrasound-guided fine-needle aspiration (EUS-FNA) warrants detailed consideration.[16] EUS-FNA has

become an indispensable tool in the diagnostic armamentarium for pancreatic masses, offering high sensitivity (85-95%) and specificity (~100%) for diagnosing malignancy. A positive EUS-FNA result can confirm the diagnosis of cancer, which is particularly useful for planning neoadjuvant therapy or for patients who are not surgical candidates.[17] However, the procedure's limitations are most apparent when the result is negative or non-diagnostic, as a benign cytology result from an EUS-FNA does not definitively rule out cancer. This imperfect negative predictive value is attributable to several factors. For instance, a simple sampling error, or "geographic miss," can occur if the needle fails to hit the small focus of cancer within a larger area of inflammation or benign tissue.[18] Furthermore, the inherent biology of PDAC, which is characterized by a dense, fibrous stroma, can make it physically difficult to aspirate a sufficient number of malignant cells. The challenge is compounded in cases of well-differentiated tumors, whose cells may lack overt cytological features of malignancy and can be difficult to distinguish from reactive cells. Finally, the diagnostic yield of the procedure is highly operator-dependent, relying heavily on the skill and experience of the endosonographer.

Consequently, in a patient with a high pre-test probability of cancer based on clinical and imaging findings, a negative EUS-FNA result is often viewed with skepticism. Many surgical guidelines and expert consensus statements support proceeding directly to surgery in such cases, as the risk of a false-negative biopsy is too high to ignore. This case powerfully illustrates this principle. Even if an EUS-FNA had been performed and returned benign or non-diagnostic findings, the compelling clinical and radiological evidence would have, and should have, still led to the recommendation for surgical resection. The Whipple procedure, in this context, becomes not only a therapeutic intervention but also the ultimate diagnostic test—an excisional biopsy of the highest order.[18]

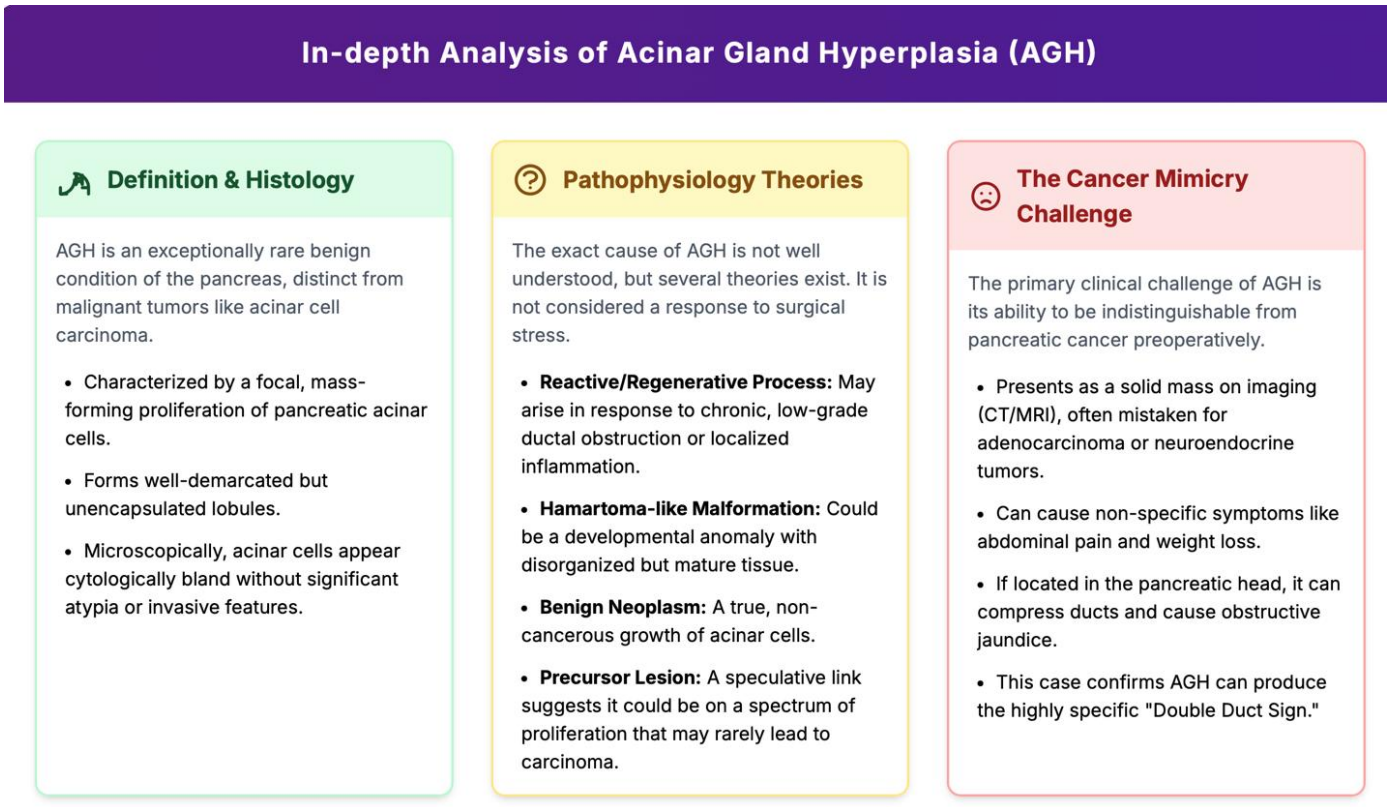

Figure 2. In-depth analysis of acinar gland hyperplasia.

The final histopathological diagnosis of acinar gland hyperplasia (AGH) is the central and most profound teaching point of this report. AGH is an entity of profound rarity, existing at the periphery of pancreatic pathology literature.[18] It is defined as a focal, non-neoplastic proliferation of pancreatic acinar cells that organizes into well-demarcated, unencapsulated lobules, which can coalesce to form a macroscopic mass. This stands in stark contrast to its malignant counterpart, acinar cell carcinoma, which is characterized by infiltrative growth, significant nuclear atypia, high mitotic activity, and a fundamentally different biological behavior.

The pathophysiology of AGH remains enigmatic, with several competing theories. It could be a response to surgical stress is chronologically incorrect, as the hyperplasia was the pre-existing lesion that necessitated the surgery.[19] Among the more plausible theories, the most widely favored is that AGH represents a reactive or regenerative process. This theory posits that the condition is an exuberant, localized response to some form of chronic, low-grade injury or inflammation, where potential triggers like ductal obstruction from a small stone or sludge lead to an increase in growth factors that stimulate acinar cell proliferation—a concept supported by this patient's year-long history of intermittent abdominal pain. Other possibilities exist along a spectrum of benign processes. It could be considered a hamartomatous malformation, essentially a developmental anomaly where acinar cells have over-proliferated in a disorganized but mature fashion. Alternatively, while histologically benign, it might represent a true benign tumor, or acinar cell adenoma, although the lack of a true capsule and the preservation of a lobular architecture make a hyperplastic process more likely. Finally, at the most speculative end of the spectrum is the concept of AGH as a precursor lesion. Drawing from the well-established adenoma-carcinoma sequence seen in other organs, some researchers speculate that a spectrum of acinar cell proliferation may exist, with AGH at one end and acinar cell carcinoma at the other, where it is conceivable that certain genetic or epigenetic events could eventually drive a focus of hyperplasia towards malignant transformation.

The true challenge of AGH lies in its remarkable ability to function as a high-fidelity cancer mimic. The clinical presentation in this case—obstructive jaundice and weight loss—is archetypal for PDAC. While other benign mimics like autoimmune pancreatitis or groove pancreatitis can cause similar symptoms, AGH is rarely, if ever, considered in the initial differential diagnosis due to its obscurity. The radiological findings are equally deceptive. While some reports describe focal acinar proliferations as well-demarcated, hypervascular lesions on imaging (more akin to a neuroendocrine tumor), this case demonstrates that AGH can also present as an ill-defined, hypodense mass causing the "double duct sign."[19] This radiological presentation makes it virtually indistinguishable from PDAC on standard imaging modalities. This report, therefore, makes a crucial contribution by documenting that AGH must be added to the very short list of benign pathologies that can produce this highly specific radiological sign, thereby cementing its status as an ultimate cancer mimic.

This case serves as a sobering reminder that the decision to perform a pancreaticoduodenectomy, even when clinically justified, carries an unavoidable and significant risk of severe, life-threatening complications. The development of a delayed post-pancreatectomy hemorrhage (PPH) is one of a surgeon's greatest fears. PPH is formally classified by the International Study Group of Pancreatic Surgery (ISGPS) based on timing (early <24h vs. delayed >24h), location (intraluminal vs. extraluminal), and severity (Grade A, B, or C). This patient experienced a delayed, Grade C hemorrhage, the most severe form, requiring urgent intervention and intensive care.[18]

The pathophysiology of delayed PPH, particularly from the gastroduodenal artery (GDA) stump, is well-described. During a standard Whipple procedure, the GDA is ligated and divided. The ligated stump lies in close proximity to the newly created

pancreaticojejunostomy anastomosis. One of the most common complications after a Whipple procedure is a leak from this pancreatic anastomosis. When a leak occurs, even if it is small and clinically occult, enzyme-rich pancreatic fluid can escape and bathe the surrounding tissues. The proteolytic and lipolytic enzymes in this fluid, particularly elastase, can begin to digest the wall of the nearby GDA stump. This enzymatic erosion weakens the arterial wall, leading to the formation of a fragile pseudoaneurysm—a contained rupture of the artery. This pseudoaneurysm can then rupture into the gastrointestinal lumen (causing melena, as in this case) or into the peritoneal cavity, resulting in catastrophic hemorrhage.

The management of this complication has undergone a dramatic evolution. Historically, the only option was an urgent re-laparotomy. However, operating on a critically ill patient in the early postoperative period is fraught with peril. The tissues are inflamed and friable, anatomical planes are obliterated, and the patient is physiologically depleted. A re-laparotomy for PPH carries an exceptionally high mortality rate, often exceeding 50%. The approach used in this case—urgent angiography followed by transarterial embolization (TAE)—now represents the gold standard for hemodynamically stable patients with PPH.[19] This minimally invasive technique offers several key advantages. It allows for precise localization of the bleeding source, which can be difficult to find in a hostile, postoperative abdomen. It allows for targeted occlusion of the bleeding vessel using microcoils or other embolic agents, without the systemic physiological insult of a major open surgery. The success of TAE in this patient underscores the critical importance of a multidisciplinary team approach in a high-volume pancreatic surgery center. The immediate availability of skilled interventional radiologists is as crucial to patient survival as the skill of the operating surgeon. The successful management of this life-threatening complication, despite the benign nature of the underlying pathology, validates the gravity of the initial decision to operate and highlights the comprehensive care required to shepherd a patient through the full course of a Whipple procedure.

4. Conclusion

Pancreatic head masses continue to pose a profound diagnostic challenge. This case of acinar gland hyperplasia—an exceedingly rare benign condition—precisely masquerading as a pancreatic carcinoma underscores the limitations of current preoperative imaging. It affirms the long-standing surgical principle that in patients with high clinical and radiological suspicion for pancreatic cancer, aggressive surgical resection is justified to avoid undertreating a potentially curable malignancy. Clinicians, radiologists, and pathologists should be aware that acinar gland hyperplasia, while rare, belongs in the differential diagnosis of solid pancreatic head masses. Finally, this report highlights that the pancreaticoduodenectomy is an operation with a high potential for severe morbidity, such as delayed arterial hemorrhage, which requires prompt recognition and modern, minimally invasive management, irrespective of the final benign or malignant nature of the resected pathology.

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