

CASE REPORT

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ALERT AT 26 MG/DL: A RARE PRESENTATION OF INSULINOMA IN THE PANCREATIC UNCINATE PROCESS

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Abstract

Insulinoma is the most common functional pancreatic neuroendocrine tumor (incidence < 4/million/year), yet diagnosis is frequently delayed by years due to nonspecific symptoms mimicking neurological conditions. Uncinate process location adds surgical complexity from proximity to the main pancreatic duct and superior mesenteric vessels. This case highlights the value of early biochemical investigation and anatomy-driven surgical planning. Clinical evaluation and biochemical workup were performed at Barleti University Polyclinic, Tirana. Endogenous hyperinsulinemia was confirmed by simultaneous fasting measurement of glucose, insulin, C-peptide, and cortisol per established thresholds. Tumor localization was performed with contrast-enhanced abdominal magnetic resonance imaging (MRI) at Villa Maria Hospital, Tirana (T1/T2, DWI/ADC, dynamic post-contrast). Surgical strategy was decided by a multidisciplinary team. The specimen underwent histopathological and immunohistochemical analysis (Ki-67/MIB-1, Chromogranin A, Synaptophysin, Congo Red) per World Health Organization (WHO) 2022 classification. A 33-year-old woman with a two-year history of recurrent hypoglycemic episodes presented with fasting glucose 26 mg/dL, insulin 26.3 µU/mL, and C-peptide 3.64 ng/mL, confirming endogenous hyperinsulinemia; cortisol was normal, excluding adrenal insufficiency. MRI identified a 20 × 16.7 mm hypervascular lesion in the uncinate process with arterial enhancement and delayed washout. Proximity to the main pancreatic duct and superior mesenteric vessels precluded safe enucleation; pancreaticoduodenectomy (Whipple procedure) was performed without complications, with discharge on day seven and normalized glycemia. Histopathology (Biopsy 13777/25) confirmed a well-differentiated pancreatic neuroendocrine tumor, WHO Grade 1, Ki-67 < 2%, Chromogranin A and Synaptophysin positive, clear resection margins (R0), and no lymph node involvement (pN0). Measuring insulin and C-peptide alongside glucose during any hypoglycemic episode is a simple step that can prevent diagnostic delay. When insulinoma arises in the uncinate process, enucleation may be unsafe regardless of tumor size; pancreaticoduodenectomy is the guideline-consistent, curative alternative. The G1, R0, pN0 outcome is associated with 5-year survival of 94–100%, underscoring the importance of timely and complete surgical resection.

Keywords: insulinoma, pancreatic neuroendocrine tumor, hypoglycemia, uncinate process, pancreaticoduodenectomy, histopathology

1. Introduction

Insulinoma arises from the beta cells of the pancreatic islets of Langerhans and is the most common functional pancreatic neuroendocrine tumor (panNET) — though it remains genuinely rare in absolute terms, with an incidence of less than 4 cases per million per year [1]. The great majority are solitary, benign, and surgically curable. What makes them clinically treacherous is the pattern of presentation: symptoms tend to appear gradually, are worst during fasting or physical exertion, and overlap considerably with anxiety disorders, epilepsy, or hypoglycemia from other causes. Many patients spend months or years being reassured rather than investigated [2].

The clinical signature of insulinoma is Whipple's triad — symptoms of hypoglycemia, a documented low plasma glucose at the time of those symptoms, and resolution with glucose correction [3]. In practice, however, this triad is not always recognized even when all three components are present, and tumors located in the pancreatic uncinate process add a further layer of difficulty: their proximity to the main pancreatic duct and superior mesenteric vessels can make the standard, parenchyma-sparing operation unsafe, forcing a more extensive resection.

We report the case of a 33-year-old woman whose recurrent, witnessed hypoglycemia was repeatedly treated symptomatically over two years without hormonal workup, and who was eventually diagnosed at a glucose nadir of 26 mg/dL. The aim of this report is to illustrate, through her case, the value of early biochemical investigation guided by established diagnostic thresholds, and of anatomy-driven surgical decision-making when an insulinoma occupies the uncinate process.

2. Materials and Methods

This is a single-case report describing the diagnostic workup, surgical management, and histopathological confirmation of an insulinoma, assembled from the patient's clinical, laboratory, imaging, and pathology records.

2.1. Clinical and Biochemical Workup

Clinical evaluation and biochemical workup were performed at Barleti University Polyclinic, Tirana. Endogenous hyperinsulinemia was investigated by simultaneous fasting measurement of plasma glucose, insulin, C-peptide, and cortisol, drawn during a symptomatic fasting period. Results were interpreted against the diagnostic thresholds recommended for functioning pancreatic neuroendocrine tumor syndromes — fasting glucose < 55 mg/dL, insulin ≥ 3 μ U/mL, C-peptide ≥ 0.6 ng/mL, proinsulin ≥ 5 pmol/L, beta-hydroxybutyrate ≤ 2.7 mmol/L, and a negative sulfonyleurea screen [4] — with cortisol measured concurrently to exclude adrenal insufficiency as a contributor to the hypoglycemia.

2.2. Imaging and Histopathological Protocol

Tumor localization was performed by contrast-enhanced abdominal magnetic resonance imaging (MRI) at Villa Maria Hospital, Tirana, using a multisequence protocol: T1- and T2-weighted imaging, T2 fat-saturated sequences, T1 VIBE without contrast, diffusion-weighted imaging (DWI/ADC), post-contrast dynamic sequences, subtraction images, and Cholangio-MRI. Surgical strategy was decided by a multidisciplinary team on the basis of tumor size, location, and its relationship to the main pancreatic duct and adjacent vasculature. The resected specimen underwent formal histopathological examination together with immunohistochemical analysis for Chromogranin A, Synaptophysin, the Ki-67 proliferation index (MIB-1 antibody), and Congo Red staining, graded according to the World Health Organization (WHO) 2022/2023 classification of pancreatic neuroendocrine neoplasms, which separates well-

differentiated tumors (panNET G1, G2, G3) from poorly differentiated neuroendocrine carcinomas using the Ki-67 index and mitotic count [5,6].

3. Results

3.1. Clinical Presentation and Biochemical Findings

The patient was a 33-year-old woman who presented electively to Barleti University Polyclinic, Tirana, seeking a definitive evaluation after two years of recurring episodes of weakness, dizziness, profuse sweating, and, on several occasions, complete loss of consciousness. Between episodes she felt persistently hungry and had gained nearly 20 kilograms, a change she attributed to overeating out of fear of the episodes. She had visited emergency departments multiple times; on those occasions blood glucose was measured around 60 mg/dL, she was given intravenous saline, and was told to eat something sweet whenever she felt weak. No hormonal workup was initiated and no referral was made at any of these visits.

Fasting blood samples were drawn for comprehensive biochemical analysis (Table 1).

Table 1. Fasting biochemical results, symptomatic period.

Parameter	Result	Reference range
Fasting blood glucose	26 mg/dL	70–100 mg/dL
HbA1c	4.84%	4.0–5.6%
Insulin (fasting)	26.3 μ U/mL	< 3 μ U/mL during hypoglycemia
C-peptide	3.64 ng/mL	0.6–2.0 ng/mL
Cortisol	205.6 nmol/L	150–600 nmol/L

A glucose of 26 mg/dL in the fasting state, accompanied by an insulin level of 26.3 μ U/mL and a C-peptide of 3.64 ng/mL, was unambiguously consistent with endogenous hyperinsulinemia; the elevated C-peptide excluded exogenous insulin administration. Cortisol at 205.6 nmol/L was normal, excluding adrenal insufficiency as a contributor. The results were reviewed by a multidisciplinary team, and the decision to proceed directly to localization imaging was made without delay.

3.2. Imaging, Surgical Course, and Histopathological Findings

Abdominal MRI with contrast showed a liver of normal size, structure, and echogenicity, with a single simple cyst (6.5 mm) in segment VII; the gallbladder, bile ducts, portal and biliary anatomy, spleen, adrenal glands, kidneys, and aorta were all unremarkable, with no free intraperitoneal fluid. Within the pancreas, a well-defined nodular lesion measuring 20 × 16.7 mm was identified in the uncinate process, with an exophytic growth pattern: hyperintense on T2, hypointense on T1, with restricted diffusion on DWI. Post-contrast sequences demonstrated early, intense arterial enhancement with washout in the delayed phase — the hallmark pattern of a hypervascular neuroendocrine tumor. The main pancreatic duct was of normal caliber throughout, and no regional lymphadenopathy was detected. Impression: a 20 × 16.7 mm lesion in the uncinate process with imaging characteristics fully consistent with insulinoma.

Following MRI confirmation, the patient was referred to the surgical team. The initial consideration was enucleation, as is standard practice for a benign-appearing insulinoma of this size. In this case, however, enucleation was judged not to be safely feasible: the tumor's location deep within the uncinate process placed it in close anatomical proximity to the main pancreatic duct and the superior mesenteric vessels, precluding a safe dissection plane. A pancreaticoduodenectomy (Whipple procedure) was therefore

performed. The operation proceeded without intraoperative complications, the postoperative course was uneventful, and the patient was discharged on day seven in good condition, with blood glucose levels fully normalized.

The surgical specimen was submitted for histopathological examination (Biopsy No. 13777/25; received July 17, 2025; approved August 1, 2025). Macroscopic examination — comprising an 18 cm intestinal fragment with attached pancreatic tissue ($9 \times 5 \times 2$ cm) and 29 cm of adipose tissue — revealed a single, well-circumscribed, beige-colored nodule with a maximum diameter of 1.5 cm; the difference between the MRI measurement (20×16.7 mm) and this pathological measurement is within the expected range of post-fixation tissue shrinkage. Resection margins were clear on all sides. Microscopic examination showed a proliferation of basophilic and oncocytoid cells in acinar and solid islet formations with vesicular nuclei, and a focal zone of necrosis. Immunohistochemistry showed strong, diffuse positivity for Chromogranin A and Synaptophysin, confirming neuroendocrine lineage; the Ki-67 proliferation index (MIB-1), assessed across more than 500 cells, was below 2%, classifying the tumor as WHO Grade 1. Congo Red staining was negative, excluding amyloid deposition. All four sampled lymph nodes (superior mesenteric and celiac trunk regions) were reactive, without metastatic involvement (Table 2).

Table 2. Histopathological and immunohistochemical report, Biopsy 13777/25.

Feature	Finding
Specimen	Intestinal fragment (18 cm) with attached pancreas ($9 \times 5 \times 2$ cm) and adipose tissue (29 cm)
Macroscopic lesion	Solitary, well-circumscribed, beige-colored nodule; maximum diameter 1.5 cm
Resection margins	Histologically clear (R0)
Cellular morphology	Basophilic and oncocytoid cells; acinar and solid islet architecture with vesicular nuclei
Necrosis	Focal necrotic zone identified
Chromogranin A	Positive
Synaptophysin	Positive
Ki-67 index (MIB-1)	$< 2\% \rightarrow$ WHO Grade 1 (G1)
Congo Red	Negative (amyloid excluded)
Lymph nodes	4 reactive nodes (superior mesenteric + celiac trunk); no metastatic involvement — pN0
Final pathological diagnosis	Well-differentiated panNET, WHO G1, Ki-67 $< 2\%$ — indolent insulinoma. RO. pN0.

Final pathological diagnosis: well-differentiated pancreatic neuroendocrine tumor (panNET), WHO Grade 1 (G1), Ki-67 $< 2\%$, resection margins clear (R0), no lymph node metastasis (pN0) — findings consistent with indolent insulinoma, and in complete concordance with the preoperative biochemical and radiological diagnosis.

4. Discussion

A glucose of 26 mg/dL in a young woman who had been circulating through emergency departments for two years is, frankly, a system failure. The individual pieces were all there — recurrent hypoglycemia, weight gain, sweating, episodes of loss of consciousness — but no one put them together. This case is unfortunately not an outlier: diagnostic delays of two to five years are consistently reported in the insulinoma literature [1,2], and the reasons are predictable. Hypoglycemia in a nondiabetic patient without obvious cause is uncommon enough that it rarely prompts a structured hormonal workup in an emergency setting. The symptoms themselves fall into two broad, overlapping categories (Table 3): adrenergic and cholinergic activation, triggered by falling glucose, produces the classic neurogenic

symptoms of sweating, tremor, palpitations, and hunger, while deepening hypoglycemia produces neuroglycopenic symptoms — cognitive slowing, behavioral change, confusion, and, in severe cases, seizure or loss of consciousness, as this patient experienced. The weight gain she described, and attributed to fear-driven overeating, is itself a characteristic but underappreciated feature of insulinoma, reflecting compensatory eating driven by persistent hunger together with the anabolic effects of chronic hyperinsulinemia [3].

Table 3. Distinguishing signs and symptoms of insulinoma.

Neurogenic	Neuroglycopenic
Adrenergic: palpitations, tremor, anxiety/arousal. Cholinergic: diaphoresis, hunger, paresthesia, circumoral tingling	Blurred vision, cognitive impairment, behavioral changes, confusion, disorientation, memory loss, seizure, stupor

Epidemiologically, this presentation sits squarely within the expected pattern: the reported incidence of insulinoma is 0.7 to 4 cases per million per year, with a modest female predominance, and women tend to present slightly later than men — in their sixth decade rather than their fifth [2,7]. Around 90% of insulinomas are benign, and in most cases, including this one, no clearly identifiable predisposing cause is found; somatic mutations in the *YY1* and *KRAS* genes are identified in up to 30% of sporadic cases, and the multiple endocrine neoplasia type 1 (*MEN1*) locus on chromosome 11q13 — most often associated with multiple, syndromic insulinomas — may also be implicated in up to a third of apparently sporadic tumors [1,2]. There was no clinical suggestion of *MEN1* in this patient's history.

Biochemically, the results left little room for doubt. A simultaneously drawn glucose of 26 mg/dL, insulin of 26.3 μ U/mL, and C-peptide of 3.64 ng/mL represent a clear violation of normal glucose–insulin homeostasis. Under normal circumstances insulin secretion is tightly coupled to serum glucose, falling as glucose falls; insulinoma cells lose this feedback mechanism entirely, secreting insulin autonomously regardless of prevailing glucose levels and thereby driving glucose into peripheral tissues while suppressing hepatic glucose production [1,8]. An intact, elevated C-peptide at that glucose level is definitive evidence of this endogenous, autonomous secretion — it excludes both exogenous insulin use and factitious hypoglycemia at a stroke. The cortisol level was normal, and the clinical severity of hypoglycemia in insulinoma more broadly is known to vary considerably between patients, depending on tumor size, the functional capacity of the neoplastic cells, and the patient's own metabolic counter-regulatory reserves [8,9].

From an imaging standpoint, the MRI performed exactly as expected for insulinoma: avid arterial enhancement with delayed washout is the radiological fingerprint of these hypervascular tumors, and it identified the uncinate lesion clearly. Contrast-enhanced computed tomography (CT) is typically the first-line localization study, identifying insulinomas in roughly 54–80% of cases; MRI performs similarly overall but can detect up to 85% of lesions when dedicated pancreatic sequences are used, while endoscopic ultrasound offers the highest sensitivity (81%) and specificity (90%) among noninvasive modalities and provides the clearest information about a tumor's relationship to the main pancreatic duct, information that directly shapes the surgical approach [1,10,11] (Table 4). What the MRI in this case also made clear was that this was not going to be a straightforward enucleation: the uncinate process is anatomically one of the most challenging locations for pancreatic surgery, sitting nestled between the superior mesenteric vessels anteriorly and the aorta posteriorly, with the main pancreatic duct running in close proximity. For enucleation to be feasible and safe, the tumor must be sufficiently superficial and distant from the duct, a threshold that the anatomy here did not support.

Table 4. Imaging modalities in insulinoma localization.

Modality	Sensitivity	Specificity	Notes
CT	54%	75%	Most common first-line study
MRI	54%–85%	65%	~85% detection with gadolinium enhancement
EUS	81%	90%	Best tool for duct proximity assessment
ASVS	93%	86%	Arterial stimulation and venous sampling
SSTR SPECT	53%	50%	Preferred in aggressive/metastatic disease
SSTR PET	39%	14%	Preferred in aggressive/metastatic disease
GLP-1R SPECT	83%	40%	Preferred in indolent/localized disease
GLP-1R PET	87%	94%	Preferred in indolent/localized disease

Abbreviations: ASVS, arterial stimulation and venous sampling; CT, computed tomography; EUS, endoscopic ultrasound; GLP-1R, glucagon-like peptide-1 receptor; MRI, magnetic resonance imaging; PET, positron emission tomography; SPECT, single-photon emission computed tomography; SSTR, somatostatin receptor.

The histopathological grading framework is worth setting out explicitly, since it underpins the prognostic weight of this patient's result. The WHO 2022/2023 classification separates well-differentiated tumors (panNET G1, G2, G3) from poorly differentiated neuroendocrine carcinomas using the Ki-67 proliferation index and mitotic count, with insulin immunostaining itself no longer mandatory once the biochemical diagnosis has been secured [5,6] (Table 5). It bears emphasizing that grade and Ki-67 alone do not reliably distinguish indolent from aggressive insulinomas: aggressive forms tend to be larger at presentation, express ARX and alpha-1-antitrypsin rather than insulin and PDX1, and frequently carry ATRX and DAXX mutations, the alternative lengthening of telomeres (ALT) phenotype, and chromosomal instability, whereas indolent insulinomas, like the one described here, remain closely related to normal beta cells in their molecular profile [3,12,13].

Table 5. WHO 2022/2023 classification for pancreatic neuroendocrine neoplasms.

Differentiation	Name	Grade	Ki-67 (%)	Mitotic count (2 mm ²)
Well differentiated	NET G1	G1	< 3	< 2
	NET G2	G2	3–20	2–20
	NET G3	G3	> 20	> 20
Poorly differentiated	NEC (G3), small & large cell type	G3	> 20	> 20

Abbreviations: NEC, neuroendocrine carcinoma; NET, neuroendocrine tumor.

The decision to proceed with pancreaticoduodenectomy rather than enucleation deserves explicit justification, because enucleation is well-established as the preferred approach for small, superficially located insulinomas — for tumors under 2 cm that lie at least 2–3 mm from the main pancreatic duct, it preserves pancreatic parenchyma, avoids long-term endocrine and exocrine insufficiency, and can be performed laparoscopically or robotically in experienced centers [7,14,15]. But "preferred" does not mean "always appropriate." Complications of pancreatic surgery, particularly postoperative pancreatic fistula (POPF), hemorrhage, and delayed gastric emptying, are not trivial: in large series, Clavien-Dindo grade ≥ 3 complications occurred in 32% of pancreaticoduodenectomies, 20% of distal pancreatectomies, and 25% of enucleations, and published meta-analytic data confirm that for tumors deeply embedded in the parenchyma or in proximity to the duct, enucleation carries POPF rates exceeding 46–50% — comparable to or worse than formal resection, and without the oncological security of clear margins [16,17]. When

enucleation is not safely achievable for a lesion in the pancreatic head or uncinate region, as was the case here, pancreaticoduodenectomy is the anatomically logical and guideline-consistent alternative [14,15].

The excellent postoperative course, discharge on day seven, no fistula, normalized glucose, reflects what is achievable with appropriate surgical expertise and patient selection. And the histopathology provided the final confirmation the case needed: a WHO G1 tumor, Ki-67 well below 2%, R0 margins, pN0, the most favorable possible pathological scenario, associated with a 5-year survival of 94–100% [7]. For a 33-year-old woman who had spent two years losing consciousness in hospital corridors and being handed a sweet, this is the outcome that matters.

5. Conclusions

Insulinoma can remain undiagnosed for years even when its biochemical and clinical signatures are effectively present at every emergency visit. The key diagnostic step, measuring insulin and C-peptide alongside glucose during a symptomatic episode, costs relatively little and requires nothing more than clinical awareness. We hope this case encourages that awareness.

Anatomical location drives the surgical decision as much as tumor size does. The uncinate process presents specific challenges that can make enucleation unsafe regardless of tumor dimensions, and when that is the case, pancreaticoduodenectomy is the appropriate and curative choice. Histopathological confirmation completes the workup and provides the prognostic information the patient deserves. In this case — G1, R0, pN0 — the news could not have been better.

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References

1. Zhuo F, Menon G, Anastasopoulou C. Insulinoma. [Updated 2025 Jan 22]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2026 Jan–. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK544299/>
2. Giannis D, Moris D, Karachaliou GS, Tsimigras DI, Karaolanis G, Papalampros A, et al. Insulinomas: from diagnosis to treatment. A review of the literature. J BUON. 2020;25(3):1302–14.
3. de Herder WW, Hofland J. Insulinoma. [Updated 2023 Apr 4]. In: Feingold KR, Adler RA, Ahmed SF, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2000–. Available from: <https://www.ncbi.nlm.nih.gov/books/NBK278981/>
4. Hofland J, Falconi M, Christ E, Castaño JP, Faggiano A, Lamarca A, et al. European Neuroendocrine Tumor Society 2023 guidance paper for functioning pancreatic neuroendocrine tumour syndromes. J Neuroendocrinol. 2023;35(8):e13318. doi:[10.1111/jne.13318](https://doi.org/10.1111/jne.13318)
5. La Rosa S, Hong SM, Ohike N, Brosens LAA, Mete O, de Herder W. Insulinoma. In: Klimstra DS, Osamura RY, editors. WHO classification of tumours: endocrine and neuroendocrine tumours. 5th ed. Lyon (France): International Agency for Research on Cancer; 2023.

6. Rindi G, Mete O, Uccella S, Basturk O, La Rosa S, Brosens LAA, et al. Overview of the 2022 WHO classification of neuroendocrine neoplasms. *Endocr Pathol.* 2022;33(1):115–54. doi:[10.1007/s12022-022-09708-2](https://doi.org/10.1007/s12022-022-09708-2)
7. Hofland J, Refardt JC, Feelders RA, Christ E, de Herder WW. Approach to the patient: insulinoma. *J Clin Endocrinol Metab.* 2024;109(4):1109–18. doi:[10.1210/clinem/dgad641](https://doi.org/10.1210/clinem/dgad641)
8. Minn AH, Kayton M, Lorang D, Hoffmann SC, Harlan DM, Libutti SK, et al. Insulinomas and expression of an insulin splice variant. *Lancet.* 2004;363(9406):363–7. doi:[10.1016/S0140-6736\(04\)15438-X](https://doi.org/10.1016/S0140-6736(04)15438-X)
9. Rizza RA, Haymond MW, Verdonk CA, Mandarino LJ, Miles JM, Service FJ, et al. Pathogenesis of hypoglycemia in insulinoma patients: suppression of hepatic glucose production by insulin. *Diabetes.* 1981;30(5):377–81. doi:[10.2337/diab.30.5.377](https://doi.org/10.2337/diab.30.5.377)
10. Christ E, Wild D, Forrer F, Brändle M, Sahli R, Clerici T, et al. Glucagon-like peptide-1 receptor imaging for localization of insulinomas. *J Clin Endocrinol Metab.* 2009;94(11):4398–405. doi:[10.1210/jc.2009-1082](https://doi.org/10.1210/jc.2009-1082)
11. Yang Y, Shi J, Zhu J. Diagnostic performance of noninvasive imaging modalities for localization of insulinoma: a meta-analysis. *Eur J Radiol.* 2021;145:110016. doi:[10.1016/j.ejrad.2021.110016](https://doi.org/10.1016/j.ejrad.2021.110016)
12. Hackeng WM, Brosens LAA, Dreijerink KMA. Aggressive versus indolent insulinomas: new clinicopathological insights. *Endocr Relat Cancer.* 2023;30(5):e220321. doi:[10.1530/ERC-22-0321](https://doi.org/10.1530/ERC-22-0321)
13. Hackeng WM, Schelhaas W, Morsink FHM, Heidsma CM, van Eeden S, Valk GD, et al. Alternative lengthening of telomeres and differential expression of endocrine transcription factors distinguish metastatic and non-metastatic insulinomas. *Endocr Pathol.* 2020;31(2):108–18. doi:[10.1007/s12022-020-09611-8](https://doi.org/10.1007/s12022-020-09611-8)
14. Howe JR, Merchant NB, Conrad C, Keutgen XM, Hallet J, Drebin JA, et al. The North American Neuroendocrine Tumor Society consensus paper on the surgical management of pancreatic neuroendocrine tumors. *Pancreas.* 2020;49(1):1–33. doi:[10.1097/MPA.0000000000001454](https://doi.org/10.1097/MPA.0000000000001454)
15. Sada A, McKenzie TJ, Vella A, Levy MJ, Halfdanarson TR. Interventional vs surgical procedures in localized/nonmetastatic insulinomas (ablation vs surgery). *Endocr Relat Cancer.* 2023;30(6):e220362. doi:[10.1530/ERC-22-0362](https://doi.org/10.1530/ERC-22-0362)
16. Alogakos M, Nakanishi H, Athanasiadis DI, Farsi S, Suresh S, Witkowiak MM, et al. Enucleation for insulinoma: consolidating evidence through systematic review and meta-analysis. *Surg Endosc.* 2025;39(10):6352–65. doi:[10.1007/s00464-025-12099-0](https://doi.org/10.1007/s00464-025-12099-0)
17. Heidsma CM, Tsilimigras DI, van Dieren S, Rocha F, Abbott DE, Fields R, et al. Indications and outcomes of enucleation versus formal pancreatectomy for pancreatic neuroendocrine tumors. *HPB (Oxford).* 2021;23(3):413–21. doi:[10.1016/j.hpb.2020.06.015](https://doi.org/10.1016/j.hpb.2020.06.015)

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