

complications. Consequently, she underwent EUS-guided radiofrequency ablation (RFA). Immediate post-procedure outcomes demonstrated near-complete resolution of the hypoglycemic episodes. Diazoxide was subsequently stopped. Outpatient continuous glucose monitoring confirmed sustained normoglycemia and marked symptom resolution, with no procedure-related complications.

CONCLUSION

The absence of a pancreatic lesion on CT demands persistent clinical suspicion in cases of biochemically proven hypoglycemia. EUS remains paramount for detecting occult lesions missed by standard imaging. Importantly, EUS-RFA serves as a highly effective, tissue-sparing alternative to surgical resection for insulinomas, especially when conventional surgery poses prohibitive anatomical risks.

EP_A124

RARE PROGRESSION OF MICROPROLACTINOMA TO MACROPROLACTINOMA: A CASE REPORT

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INTRODUCTION

Prolactinomas are the most common functioning pituitary adenomas, accounting for 50% of all pituitary tumors. Microprolactinomas (<10 mm) are the most frequent subtype and usually follow a benign course, with tumor progression reported in only 5% of cases. We report a rare case of microprolactinoma that progressed to macroprolactinoma over 16 years.

CASE

A 39-year-old female was initially diagnosed with a microprolactinoma at the age of 23 during evaluation for irregular menses since menarche. Baseline pituitary magnetic resonance imaging (MRI) at that time revealed a lesion measuring 2 × 2 × 0.8 mm. She was treated with bromocriptine for 2 months but subsequently lost to follow-up. The patient had been married for 10 years without conceiving and continued to have irregular menses.

She decided to repeat prolactin before seeking fertility treatment after 16 years. Laboratory investigations revealed markedly elevated serum prolactin (>42,000 mIU/L) with suppressed gonadotropins, while thyroid and adrenal axes were normal. She reported no galactorrhea, headache, or visual disturbances and notably did not develop amenorrhea. Visual field assessment was normal.

Repeat pituitary MRI demonstrated that the previously diagnosed microprolactinoma had progressed to a macroprolactinoma, measuring 2.1 × 2.3 × 1.6 cm, with extension into the left cavernous sinus and encasement of the left internal carotid artery, without optic chiasm compression. Oral cabergoline 0.25 mg twice weekly was initiated. She was counselled regarding potential risks of dopamine agonist therapy and advised to use mechanical contraception during treatment. At the 2-week follow-up, she tolerated therapy well. Follow-up imaging and prolactin monitoring were planned at 3 months to assess treatment response and guide fertility planning.

CONCLUSION

This case highlights the rare progression of microprolactinoma to macroprolactinoma, underscoring the importance of long-term monitoring in patients with prolactinoma. A careful balance between tumor control and fertility management is essential for optimizing care in women of reproductive age.

EP_A125

EMPTY SELLA AS A CLUE TO IDIOPATHIC INTRACRANIAL HYPERTENSION PRESENTING WITH PULSATILE TINNITUS AND PROGRESSIVE HEARING LOSS

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INTRODUCTION

Empty sella (ES) is characterized by herniation of the subarachnoid space into the sella turcica, leading to pituitary flattening. Although often considered incidental, ES is increasingly recognized as a radiological marker of idiopathic intracranial hypertension (IIH) which classically presents with headache and visual loss. Importantly, IIH may also present with otolaryngological manifestations such as pulsatile tinnitus, hearing loss, and vertigo. Audiovestibular symptoms have been reported in up to 88.9% of patients with primary ES, with sensorineural hearing loss being the most frequent, while pulsatile tinnitus occurs in approximately 58% of IIH cases.

CASE

A 51-year-old female presented with 3 months of progressively worsening headaches, rapid deterioration of left-sided hearing, and a 20-kg weight gain over 1 year.

Audiometry confirmed moderate left sensorineural hearing loss. Examination revealed a normotensive female with a body mass index of 35 kg/m² and diplopia on left upward gaze without evidence of ophthalmoplegia. The rest of the neurological examination was unremarkable. There was no evidence of papilledema or Cushingoid features. Repeat magnetic resonance imaging brain in 2025 showed persistent partial ES and loss of the posterior pituitary bright spot without an intracranial mass lesion or venous sinus thrombosis. There was no biochemical evidence of hypopituitarism or Cushing's syndrome. Autoimmune markers were unremarkable. Lumbar puncture demonstrated a mildly elevated opening pressure of 23 mmHg with normal CSF composition.

Based on the clinical and radiological findings, early IIH was diagnosed. She was treated with acetazolamide, and prescribed tirzepatide for weight reduction, resulting in improvement in headache and tinnitus.

CONCLUSION

IIH can manifest with partial ES and audiovestibular symptoms related to raised intracranial pressure. Early recognition is important, and IIH should be suspected in patients with pulsatile tinnitus or progressive sensorineural hearing loss. Acetazolamide and weight management remain the mainstay of treatment, with emerging evidence supporting a potential role for glucagon-like peptide-1 receptor agonists in reducing intracranial pressure.

EP_A126

THE TWO-YEAR PARADOX: A "PANCREATIC ADENOCARCINOMA" REVEALED AS METASTATIC INSULINOMA

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INTRODUCTION

Pancreatic neuroendocrine tumors (PNETs) are rare, comprising less than 3% of all pancreatic neoplasms. These tumors are broadly classified as functioning or non-functioning. Insulinoma is the most common functioning PNET. Non-functioning PNETs often present significant

diagnostic issues and may be misdiagnosed as pancreatic adenocarcinoma, especially when immunohistochemical evaluation is omitted during histological examination. We describe a case of metastatic insulinoma that was misdiagnosed as a poorly differentiated pancreatic adenocarcinoma.

CASE

A 65-year-old female first presented to a private hospital in 2023 with obstructive jaundice and was found to have a pancreatic head lesion that was causing biliary obstruction. As a result, a biliary stent was placed. She underwent aortocaval lymph node biopsy, and the result showed poorly differentiated pancreatic adenocarcinoma. Nonetheless, she refused surgical and oncological intervention. Even so, she remained clinically stable and maintained good functional status for 2 years.

In 2025, she presented to Hospital Canselor Tuanku Muhriz with recurrent hypoglycemia fulfilling Whipple's triad. The unexpectedly indolent clinical course prompted reassessment of the initial diagnosis. Biochemical evaluation confirmed endogenous hyperinsulinemic hypoglycemia, with inappropriately elevated insulin (11.03 µIU/mL) and C-peptide levels (1,089 pmol/L). Computer tomography of the abdomen revealed multiple hepatic lesions and progressive lymphadenopathy, suggestive of metastatic disease. Re-evaluation of the initial histopathological specimen showed a well-differentiated neuroendocrine tumor (Grade 1, Ki-67 ~2%). Hence, the diagnosis was revised to metastatic insulinoma.

She was referred to the hepatobiliary surgical team for surgical debulking, but the procedure was deemed high-risk and likely to have high mortality due to the extent of the disease. She was managed with diazoxide and long-acting somatostatin analogues for glycemic control.

CONCLUSION

This case highlights the critical importance of diagnostic vigilance when evaluating pancreatic neoplasms. Persistent or unexplained clinical courses, especially when endocrine symptoms arise, should prompt thorough reassessment. Immunohistochemical confirmation is essential to avoid misdiagnosis and ensure optimal patient management.