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.

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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.