Adult-onset focal nodular non-exophytic nesidioblastosis mimicking insulinoma detected by endoscopic ultrasonography: A case report

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Abstract. Endogenous hyperinsulinemic hypoglycemia in adults is most commonly caused by insulinoma, whereas adult-onset nesidioblastosis represents a rare etiology that can closely mimic insulinoma both clinically and biochemically. In the present study, a rare case of focal, non-exophytic, nodular adult-onset nesidioblastosis that was initially diagnosed as insulinoma was reported. A 25-year-old non-diabetic woman presented with a one-year history of recurrent fasting and postprandial hypoglycemia episodes. The patient fulfilled Whipple's triad and a supervised 72-h fast verified endogenous hyperinsulinism. Conventional imaging modalities, including computed tomography and magnetic resonance imaging, failed to reveal a pancreatic lesion. However, endoscopic ultrasonography (EUS) detected a 7.3x7.1 mm non-exophytic, hypoechoic nodular lesion in the pancreatic tail. Based on the above findings, a preoperative diagnosis of insulinoma was considered and the patient underwent spleen-preserving distal pancreatectomy. Histopathological evaluation revealed focal nodular nesidioblastosis, characterized by ductulo-insular complexes, architectural disorganization, absence of a fibrous capsule and hyperplastic islet cells with a low Ki-67 proliferative index. The patient has remained free of hypoglycemic episodes for >18 months postoperatively. This case underscores the diagnostic challenge of distinguishing focal adult-onset nesidioblastosis from insulinoma before surgery, particularly when a small, non-exophytic pancreatic lesion could be identified by EUS.

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Abbreviations: NIPHS, non-insulinoma pancreatogenous hypoglycemia syndrome; CT, computed tomography; MRI, magnetic resonance imaging; US, ultrasonography; SACI, selective arterial calcium injection test; HbA1c, hemoglobin A1c; EUS, endoscopic ultrasonography; PET/CT, positron emission tomography/computed tomography

Key words: hypoglycemia, insulinoma, nesidioblastosis, endoscopic ultrasonography, pancreatectomy

Introduction

Hypoglycemia can be defined by Whipple's triad: Symptoms consistent with hypoglycemia, a low plasma glucose concentration and resolution of symptoms following glucose administration (1). Although hypoglycemia is common among patients with diabetes treated with oral hypoglycemic agents or insulin, endogenous hyperinsulinemic hypoglycemia is relatively uncommon in non-diabetic adults and can represent a diagnostic challenge. The differential diagnosis of hypoglycemia relies on conditions such as alcohol intake, critical illness with organ failure, and pituitary or adrenal insufficiency, as well as endogenous overproduction of insulin or insulin-like growth factors. Distinguishing reactive hypoglycemia from endogenous hyperinsulinemic hypoglycemia is essential (2). The latter is characterized by inadequately elevated insulin and C-peptide levels combined with hypoglycemia (2).

In adults, insulinoma accounts for ~90-95% of cases of endogenous hyperinsulinemic hypoglycemia. This is a neoplastic disease originating from pancreatic β-cells, with an estimated incidence of ~1 per 250,000 individuals per year (2-4). The majority of insulinomas are benign, although ~15% can be malignant (4). Clinically, the majority of patients typically present with fasting hypoglycemia and significant weight gain (4). The diagnosis of hyperinsulinemic hypoglycemia is based on a 72-h fasting test, which is considered the gold standard for diagnosis (4). When imaging findings are inconclusive, more rare endogenous causes should be considered, such as functional β-cell disorders, insulin autoimmune syndrome (Hirata's disease) and administration of insulin secretagogues (2,4). Functional β-cell disorders include a spectrum of conditions collectively referred to as adult-onset nesidioblastosis or non-insulinoma pancreatogenous hypoglycemia syndrome (NIPHS) (2,4).

Nesidioblastosis, first reported in 1938, refers to the non-neoplastic proliferation of insulin-secreting pancreatic β-cells originating from the pancreatic ductal epithelium (5). Although nesidioblastosis is the most common cause of hyperinsulinemic hypoglycemia in infants and children (5), it only accounts for 0.5-5% of adult hyperinsulinemic hypoglycemia cases, with an estimated annual incidence of <0.1 per 1,000,000 individuals (6-9). Clinically and biochemically, nesidioblastosis resembles insulinoma (5). The most common symptoms of nesidioblastosis include dizziness, blurry vision, cold sweating, overeating, easy hunger, night eating, need for sweets, altered consciousness, memory loss, decreased reaction time and seizures (5,6). Both conditions typically fulfill Whipple's triad and can show prompt symptom resolution following oral sugar or intravenous glucose intake (5). Postprandial hypoglycemia can be commonly observed in patients with nesidioblastosis. In the past, postprandial hypoglycemia without fasting hypoglycemia was considered an important distinguishing feature of nesidioblastosis (4). However, it has been reported that certain patients with nesidioblastosis can also experience fasting or exercise-induced hypoglycemia (4). Therefore, the clinical presentation of nesidioblastosis can range from mild and transient hypoglycemia to more severe, permanent hypoglycemia (1).

Although computed tomography (CT), magnetic resonance imaging (MRI) and ultrasonography (US) can effectively localize insulinomas, the above modalities are less useful for diagnosing and localizing adult-onset nesidioblastosis (10). Therefore, nesidioblastosis is typically diagnosed in patients presenting with postprandial neuroglycopenic symptoms, biochemical evidence of hyperinsulinemic hypoglycemia, a negative 72-h fasting test and negative conventional imaging for insulinoma (4,10,11). Selective arterial calcium injection test (SACI) with hepatic venous sampling can also be used to confirm the diagnosis and guide surgical intervention (4,10).

Definitive diagnosis relies on histopathological evaluation, which classifies nesidioblastosis into focal nesidioblastosis, which is characterized by nodular hyperplasia, diffuse nesidioblastosis, affecting the entire pancreas, and atypical forms that do not fit in either form (5,9,12,13). Recently, this condition has been increasingly recognized as a rare complication following bariatric surgery, particularly gastric bypass (6,11).

Dietary modification, pharmacological therapy and surgical intervention are the most common treatment options for nesidioblastosis (11). Surgery is generally considered for patients with severe or persistent refractory hypoglycemia (1). The majority of patients require partial or total pancreatectomy (1,5). However, there are currently no established standards regarding the type and optimal extent of surgical resection (2,5).

In the present study, a rare case of focal, non-exophytic nodular adult-onset nesidioblastosis detected by endoscopic ultrasonography (EUS) and initially misdiagnosed as insulinoma was described.

Case report

Patient. A 25-year-old non-diabetic woman presented to Lokman Hekim University Training and Research Hospital (Sincan, Turkiye) in October 2023 with a 1.5-year history of recurrent neuroglycopenic symptoms, including sweating, limb tremors, loss of consciousness, blurred vision, sweet cravings and nocturnal food intake. The aforementioned episodes occurred during both fasting and postprandial periods. The patient reported multiple recurrent hypoglycemic events that resolved with oral carbohydrate or intravenous glucose administration, two of which fulfilled Whipple's triad. Capillary glucose levels measured in the Emergency Department during symptomatic episodes were 53 and 55 mg/dl.

The patient was not taking any medication and had no history of smoking, alcohol consumption or prior surgery. The patient also had a family history of diabetes mellitus in the patient's mother.

Physical examination was unremarkable. The first laboratory exams at admission revealed plasma glucose levels of 85 mg/dl (reference fasting range, 70-100 mg/dl), hemoglobin A1c (HbA1c) of 4.82% (normal range, 4-5.6%) and an estimated average glucose level of 91.63 mg/dl (normal range, 70-117 mg/dl). Further laboratory tests excluded renal, hepatic, adrenal, pituitary and thyroid dysfunction. Anti-insulin and anti-insulin receptor antibodies were negative, while no evidence of insulin-like growth factor overproduction was reported. During a supervised 72-h fasting test, the patient experienced neuroglycopenic symptoms at 36 h, with plasma glucose levels of 57 mg/dl. Concurrent biochemical examinations showed an insulin level of 8 μIU/ml (reference fasting range, 2-6 μIU/ml) and a C-peptide level of 543 pmol/l (<150 pmol/l during hypoglycemia), confirming endogenous hyperinsulinism. Abdominal US, contrast-enhanced CT and MRI did not reveal any pancreatic lesion. However, EUS identified a 7.3x7.1 mm hypoechoic nodular lesion in the pancreatic tail (Fig. 1). Given the presence of a discrete nodular lesion highly suggestive of insulinoma on EUS, and the limited availability of SACI testing and ^{68}Ga-based positron emission tomography (PET)/CT imaging at the Department of General Surgery, Lokman Hekim University Training and Research Hospital (Sincan, Turkiye), no further functional localization studies were performed.

Subsequently, the patient underwent spleen-preserving distal pancreatectomy. The postoperative course was uneventful and the patient was discharged on postoperative day 5. A minor pancreatic fistula developed postoperatively (based on the detection of elevated amylase and lipase levels in the drainage fluid) but resolved spontaneously within 1 week.

Histopathological examination. Histopathological examination was performed on formalin-fixed, paraffin-embedded (FFPE) tissue specimens. Following routine tissue processing, 4-μm thick sections were obtained and stained with hematoxylin and eosin. Hematoxylin staining was performed for 5 min and eosin staining for 2 min at room temperature (20-25˚C) according to the standard laboratory protocol. Microscopic evaluation was carried out by experienced pathologists using a conventional light microscope. Representative sections demonstrating the characteristic histomorphological features of the lesion were selected for detailed assessment and photomicrographic documentation.

For digital pathological assessment and image acquisition, representative slides were scanned at high resolution using a Leica whole-slide imaging system (Leica Biosystems). Digital slide evaluation, annotation and photomicrograph acquisition were performed using the ViraSight digital pathology software

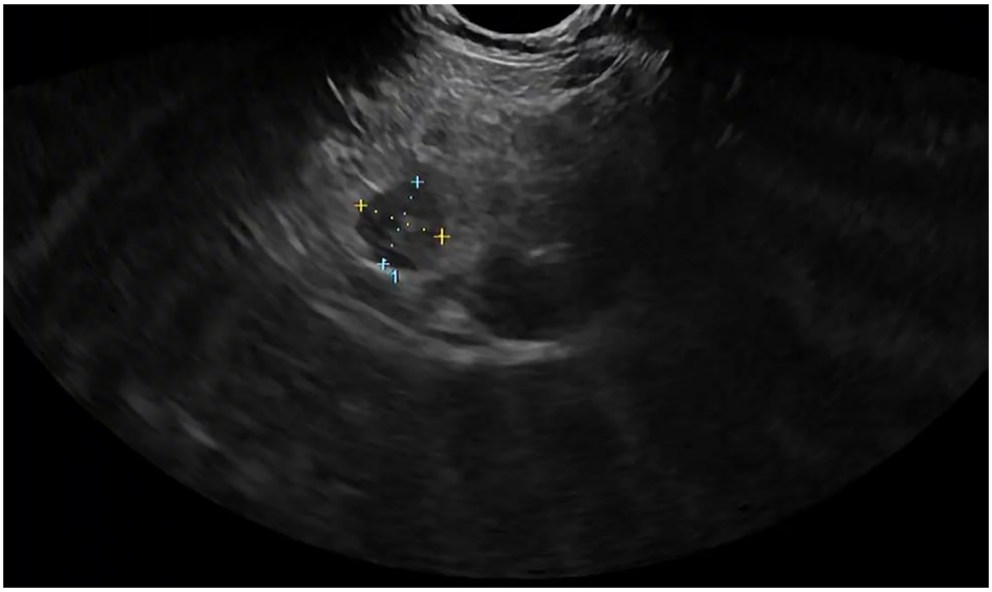

Figure 1. Endoscopic ultrasonography image showing a 7.3x7.1 mm hypoechoic nodular mass in the tail of the pancreas.

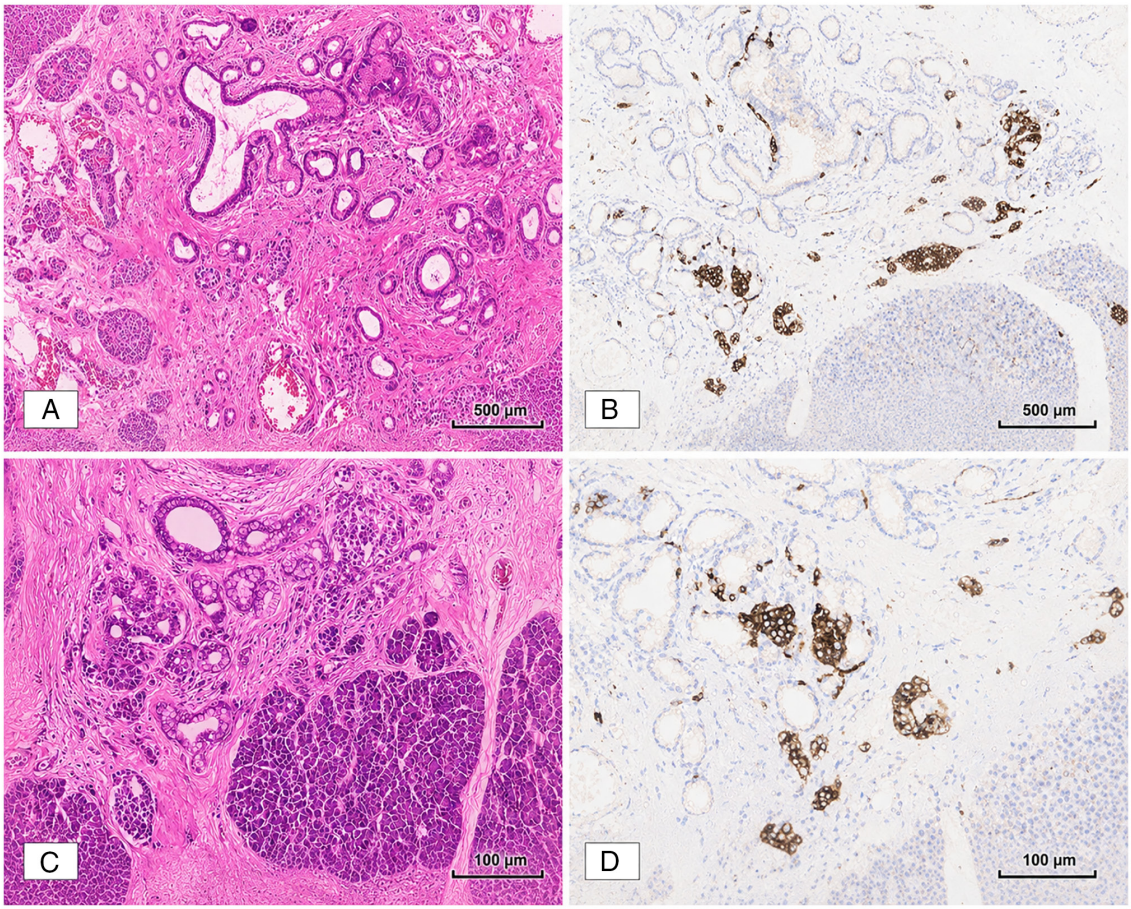

Figure 2. Pathology and IHC analysis. (A) Histology indicates enlarged and irregular hyperplastic islet cells mixed with pancreatic ducts (ductuloinsular complexes) (H&E staining; magnification, x4; scale bar, 500 nm). (B) IHC staining of synaptophysin highlighted the hyperplastic islet cells mixed with pancreatic ducts (ductuloinsular complexes) (magnification, x4; scale bar, 500 nm). (C) Histology at larger magnification (H&E staining; magnification, x10; scale bar, 100 nm). (D) IHC at larger magnification (magnification, x10; scale bar, 100 nm). IHC, immunohistochemistry.

platform. Representative images demonstrating the characteristic histomorphological features of the lesion were selected for publication.

Immunohistochemical analysis. Immunohistochemical staining was performed on 4-μm thick FFPE tissue sections using the Dako Autostainer Link 48 platform (Agilent

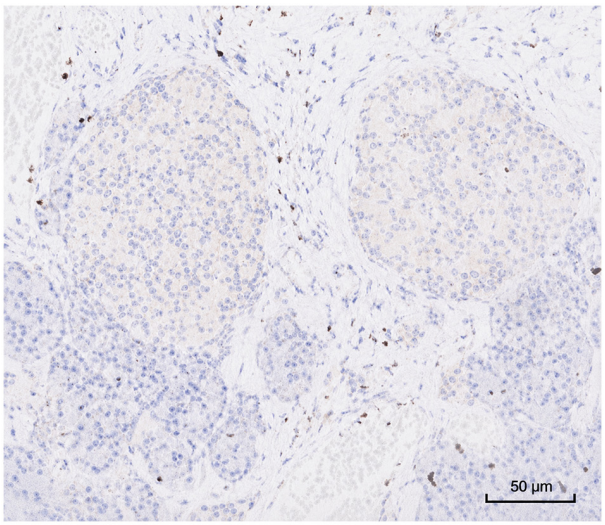

Figure 3. Immunohistochemistry staining of Ki67 highlighted the low proliferation index in the hyperplastic islet cells (magnification, x20; scale bar, 50 μm).

Technologies, Inc.) according to the manufacturer's instructions. Synaptophysin immunostaining was performed using a mouse monoclonal antibody (clone DAK-SYNAP; catalog no. M0776; dilution 1:100) and Ki-67 immunostaining was performed using a mouse monoclonal antibody (clone MIB-1; cat. no. M7240; dilution 1:100) (both Agilent Technologies, Inc.). Primary antibody incubation was performed at room temperature for 20 min. Appropriate positive and negative controls were included in each staining run. Stained slides were dehydrated and covered with mounting medium (cat. no. s3023; Dako; Agilent Technologies, Inc.) and cover-slips.

Synaptophysin expression was evaluated according to the extent and intensity of cytoplasmic staining in lesional cells. The Ki-67 proliferation index was determined by counting positively stained nuclei in hotspot areas and expressed as the percentage of immunoreactive tumor cells.

Histopathological examination of the distal pancreatectomy specimen revealed no evidence of neoplastic proliferation. Hematoxylin-eosin staining and synaptophysin immunostaining demonstrated enlarged, hyperplastic islet cells with irregular architecture mixed with pancreatic ducts, forming ductulo-insular complexes (Fig. 2). Ki-67 immunostaining showed a low proliferative index <1% (Fig. 3). Finally, the patient was diagnosed with focal nodular nesidioblastosis. The patient was followed up by the Departments of General Surgery and Endocrinology for the first month postoperatively, and subsequently by the Department of Endocrinology with check-ups and blood tests every 3 months. At the time of writing this case report, the patient remains asymptomatic, with no recurrence of hypoglycemic episodes for >18 months after surgery.

Discussion

A comprehensive clinical and laboratory evaluation is essential in non-diabetic adults presenting with hypoglycemia (4). The diagnostic criteria for hypoglycemia should include careful assessment of common etiologies, including pituitary or adrenal insufficiency, alcohol intake, medication use (particularly hypoglycemic agents), critical illness with organ dysfunction, prior bariatric surgery and malignancy. Other rare causes of hypoglycemia, including insulin autoimmune syndrome (Hirata's disease) or Doege-Potter syndrome, should also be excluded (1,4). When hyperinsulinemic hypoglycemia is suspected, a 72-h fasting test remains the gold standard, with a 4-6-h oral glucose tolerance test being considered in selected cases (4).

In adults, the most common causes of hyperinsulinemic hypoglycemia include insulinoma, Hirata's disease, use of insulin secretagogues and nesidioblastosis (10). Insulinoma accounts for the vast majority of hyperinsulinemic hypoglycemia cases, while nesidioblastosis is rare, with an estimated incidence of ~1 per 10 million individuals per year (3-5,9,14). Although nesidioblastosis was first described by Laidlaw (8) in 1938, it was first reported in adults in 1975 (10,15). Nesidioblastosis is the most common cause of hyperinsulinemic hypoglycemia in infants, with an incidence of ~1 per 25,000-50,000 individuals (4,12). A recently published review reported a female-to-male ratio of ~1.7/1 in adult patients (2). While focal nesidioblastosis is observed in 30-40% of affected infants, it is uncommon in adults, with only 12 cases reported to date (2,5,12).

Clinically, nesidioblastosis is characterized by postprandial hyperinsulinemic hypoglycemia that is commonly refractory to dietary therapy and difficult to distinguish from insulinomas both clinically and biochemically (9,10). Histopathologically, it is defined by neoformation of Langerhans islets from the pancreatic ductal epithelium (ductulo-insular complexes), and enlarged, hyperchromatic β-cell nuclei (10,11). The underlying pathogenesis remains incompletely understood and can involve several causes, including pancreatic β-cell dysfunction, altered expression of growth factors and/or their receptors, and unidentified genetic variations (5,14). In recent years, adult-onset nesidioblastosis has been increasingly reported after bariatric surgery, particularly following Roux-en-Y anastomosis, with an estimated incidence of 0.1-0.3% (6), and less frequently, after sleeve gastrectomy or gastrectomy with Roux-en-Y reconstruction (6,10). It has been suggested that nesidioblastosis can result from enhanced levels of pancreatic β-cell trophic factors in these patients (6,14). However, it has also been increasingly reported in individuals without a prior history of surgery, a condition referred to as NIPHS (6,10,16).

Distinguishing NIPHS from insulinoma preoperatively is challenging due to the significant similarities in clinical presentation associated with hypoglycemia. Hypoglycemia can be accompanied by a wide spectrum of symptoms, including hunger, palpitations, sweating, tremors, excitement, anxiety, drowsiness, delirium, disorientation, loss of consciousness, confusion, paresthesia, seizures and coma (2,5,6,10,11,13,17). Although the original definition of NIPHS included postprandial neuroglycopenic symptoms with concurrent hypoglycemia and many patients present with postprandial hypoglycemia, others can experience fasting hypoglycemia, exercise-induced hypoglycemia or a combination of these (2,4,13,16). Although fasting hypoglycemia is widely regarded as a hallmark of insulinoma and a positive fasting test has traditionally been considered to exclude NIPHS, a positive fasting test is not adequate for distinguishing both conditions (4). Consistently,

exercise-induced or postprandial hyperinsulinemic hypoglycemia, which can also be observed in certain patients with insulinoma, is not a defining feature of NIPHS (4). Therefore, fasting hypoglycemia and fulfillment of classic Whipple's triad can occur in both NIPHS and insulinoma (13), as shown in the current case. Furthermore, the diagnostic criteria for NIPHS, including postprandial neuroglycopenic symptoms with concurrent detectable hypoglycemia and a negative 72-h fasting test, are not applicable to all patients. Biochemically, NIPHS and insulinoma are indistinguishable, as both can present with hypoglycemia and abnormally elevated insulin and C-peptide levels (5).

Radiological differentiation between these conditions remains particularly challenging. While insulinomas can be typically identified as a well-defined hypervascular lesion on conventional imaging, diffuse nesidioblastosis is commonly not detectable (10). Additionally, small insulinomas (<1 cm) can evade detection on conventional imaging (9). The reported preoperative sensitivity of conventional imaging techniques for focal nesidioblastosis can be comparable to that for insulinoma, with detection rates of 54% for CT, 58% for MRI, 76% for EUS and 85% for SACI testing with hepatic venous sampling (12). To further aid in the differential diagnosis between insulinoma and nesidioblastosis, PET/CT imaging using ^{68}Ga-somatostatin receptors (^{68}Ga-DOTATATE) and ^{68}Ga-glucagon-like peptide-1 receptor analogs (^{68}Ga-DOTA-exendin-4) can be useful. However, these modalities are not widely available (5,12,14). EUS can detect small nodular lesions; however, focal nesidioblastosis can mimic insulinoma when presenting as a discrete mass, as shown in the present case.

In the present case, SACI testing and ^{68}Ga-based PET/CT imaging were not performed due to the unavailability of these advanced imaging modalities at our center. The presence of hyperinsulinemic fasting hypoglycemia, fulfilling Whipple's triad, a positive 72-h fasting test and the identification of a well-defined nodular lesion on EUS strongly supported a working diagnosis of insulinoma and justified surgical intervention. Therefore, based on clinical, biochemical and EUS findings, surgical management was prioritized.

The most common form of nesidioblastosis in adults is the diffuse form, while focal nesidioblastosis is rare and has been reported as exophytic pancreatic lesions (9,18,19). In contrast to previous focal nesidioblastosis cases, herein, the lesion located in the pancreatic tail was not exophytic. Furthermore, no spread was detected in the remaining part of the resected pancreatic tissue and the resolution of hypoglycemia following distal pancreatectomy confirmed the diagnosis of non-exophytic focal nesidioblastosis localized in the distal pancreatic parenchyma.

Histopathologically, nesidioblastosis is distinguished from insulinoma by the absence of a well-defined capsule, preservation of the pancreatic lobular architecture, presence of ductulo-insular complexes, irregularly enlarged β-cell nuclei and a low Ki-67 proliferation index. By contrast, insulinomas typically exhibit a circumscribed neoplastic proliferation with a more uniform cellular architecture. However, definitive criteria for the histopathological diagnosis of adult-onset focal nesidioblastosis are still lacking (13). Goossens *et al* (20) defined focal nesidioblastosis in infants as a well-defined compact nodule with tumor-like features, including frequent ductulo-insular complexes and enlarged β-cells with large nuclei. The histopathological findings in the current case were consistent with those described for focal nesidioblastosis.

Several treatment strategies have been described for adult-onset nesidioblastosis (2). Medical management includes pharmacological agents such as diazoxide, α-glucosidase inhibitors, steroids, calcium channel antagonists and somatostatin analogs, typically combined with a low-carbohydrate diet. However, these approaches are commonly insufficient, and therefore, definitive therapy frequently requires surgical intervention (2,12-14). Notably, medical treatment of secondary nesidioblastosis after bariatric surgery is more effective compared with idiopathic nesidioblastosis (13). Currently, partial (50-60%), subtotal (80-95%) or total pancreatectomy is considered the most effective treatment approach for adult-onset nesidioblastosis (2,12-14). The optimal extent of pancreatic tissue resection remains a controversial issue in the literature. Therefore, insufficient resection can result in an increased recurrence risk, while excessive resection can enhance the risk of type 3C diabetes (2,12,14). If focal nesidioblastosis is suspected, the surgical approach should be guided by the anatomical location of the mass (17). Although partial pancreatectomy can achieve clinical remission, particularly in patients with focal nesidioblastosis, it is effective only in ~50% of patients with nesidioblastosis, while postoperative complications, including bleeding, infection, pancreatic fistula and type 3C diabetes, can also occur (9,12). Even in high-volume centers, the reported rate of postoperative pancreatic fistula ranges from 3 to 45% (12,21). In addition, type 3C diabetes has been reported in up to 40% of patients following near-total pancreatectomy (12,22). Although recurrence of hypoglycemia in adult-onset nesidioblastosis cannot be predicted, current evidence supports distal or subtotal pancreatectomy, which is associated with a ~70% success rate and a 10% risk of developing type 3C diabetes (5,12,14). In patients with persistent hypoglycemia, particularly those who are not eligible for surgical intervention and in whom pharmacological treatment has failed, dietary therapy with uncooked cornstarch, which maintains stable blood glucose levels during fasting, should be considered (12).

In conclusion, the pathogenesis of adult-onset focal nesidioblastosis remains poorly understood, and further studies are needed to uncover its underlying mechanisms. The diagnosis of nesidioblastosis in adults is challenging, requiring a multidisciplinary approach to ensure comprehensive and effective therapy. Although rare, nesidioblastosis should be considered in the differential diagnosis of insulinoma in adults with hyperinsulinemic hypoglycemia. However, preoperative differentiation between focal adult-onset nesidioblastosis and insulinoma remains challenging, particularly in the presence of a pancreatic mass. In such cases, advanced imaging methods, including ^{68}Ga-DOTATATE and ^{68}Ga-DOTA-Exendin-4 PET/CT, can aid in the differential diagnosis. Although medical therapy combined with dietary management can be beneficial in selected cases, definitive treatment is commonly achieved through partial, subtotal or total pancreatectomy.

Focal adult-onset nesidioblastosis is exceptionally rare, with most reported lesions showing an exophytic pattern. By contrast, in the present case, the non-exophytic nature of

the lesion detected by EUS, the patient's young age and the presence of fasting hypoglycemia collectively underscored the uniqueness of this case.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

MP and ÜÖ designed the study and drafted the manuscript. ÜÖ performed the collection, analysis and interpretation of the data. KY and ABÖ performed the surgical procedure. KY and ÜÖ obtained medical images (including MRI, CT scans and endoscopic USG images) and confirmed the authenticity of all the raw data. ABÖ advised on patient treatment and critically reviewed the manuscript and approved the final version for publication. All authors have read and approved the final manuscript, approved the journal to which the article will be submitted and agree to be accountable for all aspects of the work.

Ethics approval and consent to participate

According to institutional policy, ethical committee approval is not required for single case reports.

Patient consent for publication

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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