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Case Report

A Landmark Diagnosis: First Reported Case of MEN1 Syndrome in a Qatari Patient with Literature Review

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Abstract

Background: Multiple Endocrine Neoplasia type 1 (MEN1) is a rare autosomal dominant syndrome, most often involving parathyroid, gastroenteropancreatic, and pituitary tumors. First described in the early 20th century and confirmed in 1997 to result from mutations in the MEN1 gene. Early recognition is essential for timely management and family screening.

Case Presentation: A 44-year-old woman presented for a second opinion on a recently diagnosed functional parathyroid adenoma, with prior workup showing elevated calcium (2.77 mmol/L) and PTH (141 pg/mL), and imaging suggestive of a left parathyroid adenoma plus a suspicious right thyroid nodule. Family history revealed possible pituitary tumor in her mother and thyroid cancer with pancreatic tumor in a cousin, raising suspicion for MEN1. Further evaluation included CT/MRI abdomen showing two small pancreatic tail lesions suspicious for neuroendocrine tumors, and pituitary MRI revealing a 5–6 mm microadenoma. Hormonal testing showed elevated prolactin (577 mIU/L). The diagnosis of MEN1 syndrome was made (parathyroid adenoma, pancreatic neuroendocrine tumors, pituitary microadenoma), and the positive genetic test confirmed the diagnosis. Multidisciplinary discussion recommended parathyroidectomy with surveillance of pancreatic and pituitary lesions.

Conclusion: This case represents the first reported diagnosis of MEN1 syndrome in Qatari patient, highlighting the need to recognize its presence within the population. Early identification is critical, as MEN1 is a rare hereditary syndrome that requires timely detection, multidisciplinary management, and genetic screening of at-risk relatives. Establishing awareness and screening for suspicious cases will improve outcomes and ensure appropriate long-term surveillance.

Keywords: multiple endocrine neoplasia; primary hyperparathyroidism; gastroenteropancreatic tumors

Abbreviations

MEN: Multiple Endocrine Neoplasia
ZES: Zollinger-Ellison Syndrome
PHPT: Primary Hyperparathyroidism
GEP tumors: Gastroenteropancreatic tumors
FNA: Fine Needle Aspiration
CT: Computed Tomography
MRI: Magnetic Resonance Imaging
PTH: Parathyroid Hormone
TSH: Thyroid Stimulating Hormone
FT4: Free Thyroxine 4
ACTH: Adrenocorticotropic Hormone
GH: Growth Hormone

Introduction

Multiple Endocrine Neoplasia type 1 (MEN1) is a rare inherited syndrome affecting the endocrine system, typically characterized by parathyroid tumors (95%), neoplasms of the endocrine gastroenteropancreatic tract (30–80%), and anterior pituitary gland (15–90%) [1]. The first documented case of what is now recognized as MEN was reported by Erdheim in 1903, describing an autopsy of a patient with acromegaly and enlargement of all four parathyroid glands [2]. Approximately two decades later, Cushing and Davidhoff documented the first case presenting with the classic triad of tumors characteristic of MEN1 [3]. In 1953, Underdahl and colleagues published the inaugural review of MEN1 syndrome, detailing 14 cases[4]. In 1954, Wermer was the first to characterize the MEN1 phenotype as following an autosomal dominant inheritance pattern, based on observations in a family where the father and four out of nine children were affected [5]. The original name

"Wermer's syndrome" was eventually replaced by the term MEN1 syndrome, which is now widely used [6]. In 1988, the MEN1 gene locus was localized to chromosome 11q13 through recombinant DNA analysis in two brothers with MEN1 [7]. The gene itself was subsequently identified via positional cloning, and mutations in MEN1 were confirmed as the cause of the syndrome in 1997 [8].

Case Presentation

A 44-year-old female presented to the Surgery Clinic at Al Ahli Hospital seeking a second opinion regarding a recently diagnosed functional parathyroid adenoma, previously evaluated at another medical center. The patient is married, and has children. She had a medical history of lumbar disc prolapse.

Two months prior to her presentation at Al Ahli Hospital, the patient underwent routine blood tests as part of the preparation for initiating medical management for weight reduction. These tests revealed elevated serum calcium, prompting further evaluation of parathyroid hormone (PTH) levels, which were found also elevated. Subsequent assessment at another medical center, including neck ultrasound, Sestamibi scan, and biopsies, confirmed the presence of a parathyroid adenoma and identified a suspicious thyroid nodule.

Adjusted serum calcium was elevated at 2.77 mmol/L (reference range: 2.10–2.60 mmol/L). Parathyroid hormone (PTH) was also elevated at 141 pg/mL (reference range: 12–88 pg/mL). Insulin levels were increased at 42.9 µIU/mL (reference range: 2.6–24.9 µIU/mL). Thyroid-stimulating hormone (TSH), free thyroxine (FT4), and calcitonin levels were within normal limits.

Neck ultrasound revealed multiple hypoechoic nodules in the right thyroid lobe, the largest measuring 1.1 × 0.6 cm near the isthmus, containing small calcifications (TI-RADS 4). A smaller hypoechoic nodule measuring 7.4 × 0.2 cm was also noted. In the left thyroid lobe, a hypoechoic nodule measuring 1.9 × 1.0 × 0.6 cm was identified near the lower pole; in some images, it appeared to be located outside the thyroid gland, raising the possibility of a parathyroid adenoma. An additional small hypoechoic nodule measuring 0.4 × 0.3 cm with tiny calcifications was seen. Bilateral subcentimetre cervical lymph nodes were also observed.

Sestamibi scan demonstrated a focal area of increased tracer uptake localized to the posterior aspect of the left upper thyroid region, measuring approximately 0.8 cm in short axis and bordered by the esophagus, trachea, and thyroid gland. No ectopic parathyroid tissue was identified. The findings were suggestive of a left parathyroid adenoma. (Figure 1)

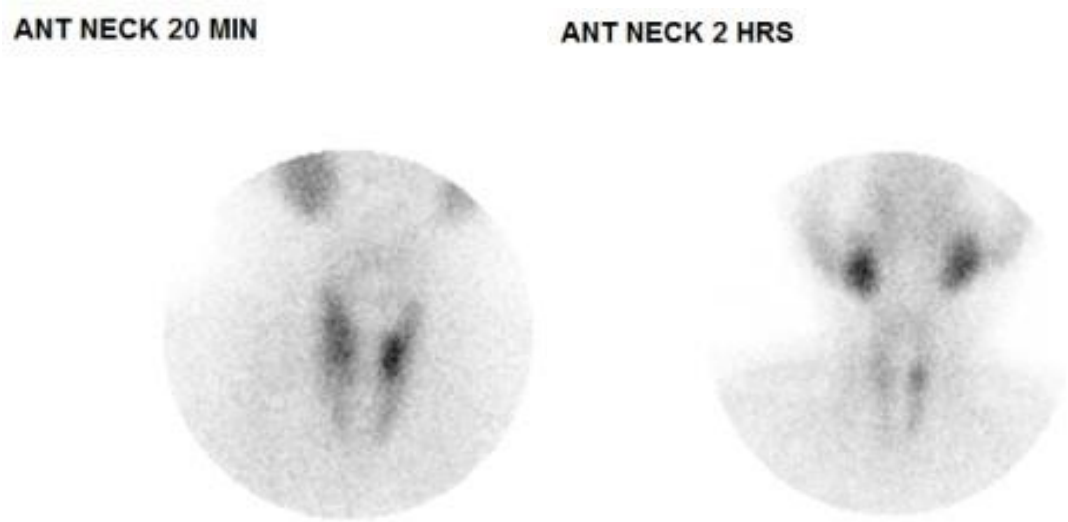

Figure 1: ^{99m}Tc-sestamibi parathyroid scintigraphy of the neck at 20 minutes and 2 hours shows increased focal uptake, suggestive of a left parathyroid adenoma.

Ultrasound-guided fine-needle aspiration of the left-sided nodule revealed parathyroid tissue, confirming the diagnosis of a parathyroid adenoma.

Ultrasound-guided fine-needle aspiration of the suspicious right thyroid nodule revealed non-Diagnostic result (cyst fluid only).

Based on the above investigations, the patient was scheduled for surgical management of the parathyroid adenoma, specifically a left parathyroidectomy under general anesthesia, at her initial medical center. At this stage, the patient presented to our hospital seeking a second opinion. A comprehensive family history assessment was performed and the patient described a tumor in her mother, who passed away many years ago. Although no medical records were available, the tumor's reported location was suggestive of the pituitary gland. Additionally, the patient reported a family history of thyroid cancer and pancreatic tumor in her cousin residing in Bahrain. Given this information, the possibility of multiple endocrine neoplasia (MEN) syndrome was considered.

Neck ultrasound was repeated and revealed a hypoechoic, oblong-shaped soft tissue nodule located at the inferior lateral border of the left thyroid lobe, highly suggestive of a parathyroid adenoma. Another oblong hypoechoic nodule was identified at the junction between the isthmus and the right thyroid lobe, demonstrating vascularity on color Doppler imaging. Additionally, multiple small hypoechoic soft tissue nodules were scattered throughout the right thyroid lobe.

The ultrasound results were reviewed with the radiology team, who considered the right thyroid nodule to be suspicious based on its radiological features. Consequently, a decision was made to perform a repeat ultrasound-guided fine-needle aspiration (FNA) of the large right thyroid nodule, as the previous biopsy performed at HMC was nondiagnostic.

Ultrasound-guided fine-needle aspiration of the suspicious right thyroid nodule was repeated and revealed atypia of undetermined significance.

To evaluate the possibility of multiple endocrine neoplasia syndrome based on her family history, despite the absence of related symptoms, contrast-enhanced CT and MRI scans of the abdomen and pelvis, as well as an MRI of the pituitary gland with contrast, were performed.

Contrast-enhanced CT scan of the abdomen and pelvis revealed 11 mm enhancing lesion in the tail of pancreas, A separate 5 mm lesion on the surface of the pancreas anteriorly, and third enhancing nodule in the tail posterior surface measuring 4.5 mm. (Figure 2)

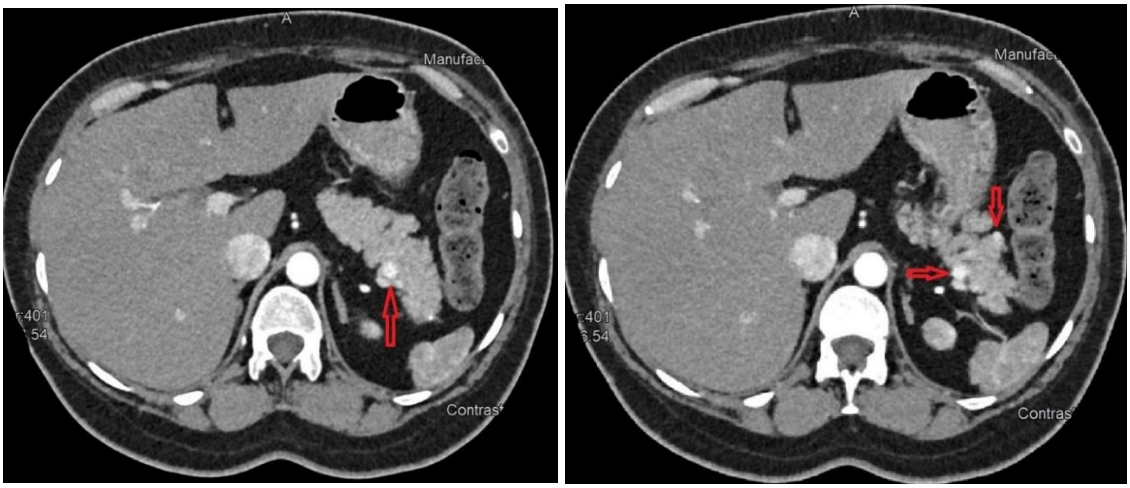

Figure 2: Contrast-enhanced CT scan of the abdomen and pelvis with red arrow indicating the location of pancreatic lesions.

MRI of the abdomen and pelvis with contrast demonstrated an arterially enhancing nodule within the tail of the pancreas, measuring up to 9 mm, suspicious for a neuroendocrine tumor. Additionally, a subtle separate lesion at the same level in the pancreatic tail, corresponding to enhancement seen on the CT scan, is likely indicative of a secondary lesion. (Figure 3)

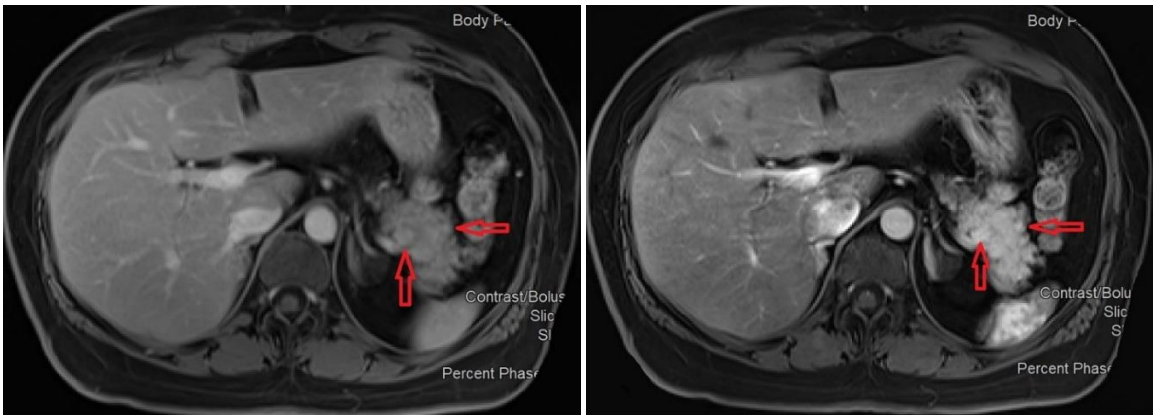

Figure 3: Contrast-enhanced MRI of the abdomen and pelvis with red arrows indicating the location of pancreatic tail lesions.

Brain MRI with dynamic contrast of the pituitary gland revealed a 5 to 6 mm nodule in the left portion of the gland, extending into the posterior half. The nodule exhibited delayed or absent early enhancement, with a well-defined delayed enhancing margin compared to the surrounding pituitary tissue. On delayed images, the lesion showed uniform enhancement, with the pituitary stalk centrally located and aligned, consistent with a microadenoma. (Figure 4)

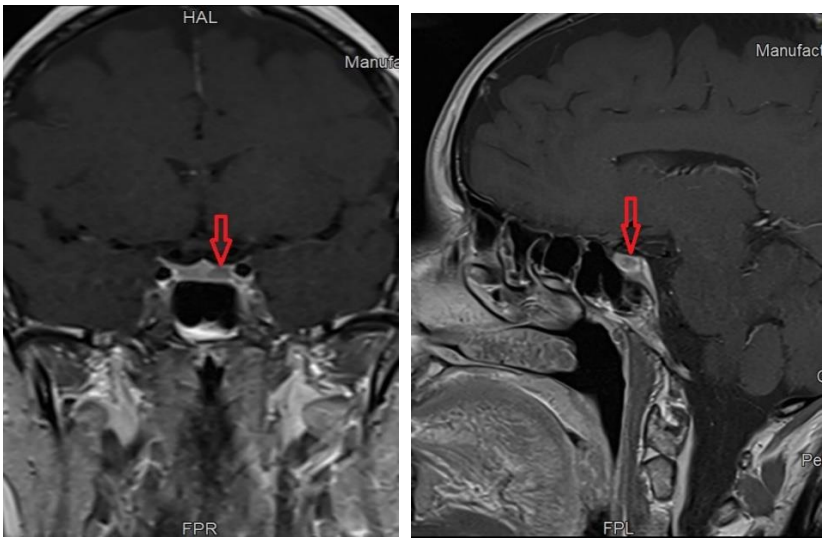

Figure 4: Contrast-enhanced Brain MRI with red arrow indicating the location of pituitary gland lesion.

The patient was diagnosed as MEN 1 Syndrome (Functional parathyroid adenoma, pancreatic neuroendocrine tumors, and pituitary microadenoma). She was referred to the leading academic teaching hospital in Qatar (Hamad Medical Corporation "HMC") for the management.

At HMC, further hormonal tests were conducted: Prolactin was elevated at 577 mIU/L (reference range: 102–495 mIU/L), while ACTH, cortisol, estradiol, FSH, LH, TSH, FT4, IGF-1, and gastrin levels were within normal limits.

Targeted Variant Testing for the *MEN1* Gene "c.1546dup, p.(R516Pfs*15)" was performed, and the result was positive. The case was discussed in a multidisciplinary team meeting, where surgical management of the parathyroid adenoma was recommended. Close monitoring and regular evaluation of the pancreatic and pituitary lesions were also advised.

Discussion

Multiple Endocrine Neoplasia type 1 (MEN1) is a rare inherited syndrome affecting the endocrine system. It affects roughly 1 in 30,000 individuals, occurring equally in males and females, with no preference for any particular ethnic or racial group [9].

In Qatar, two previously published case reports have described MEN1 syndrome in non-Qatari patients: the first involved a 30-year-old man diagnosed at Hamad Medical Corporation in 2021, and the second described a 15-year-old boy diagnosed at Sidra Hospital in 2024 (10,11). Our report is, to the best of our knowledge, the first documented case of MEN1 syndrome in a patient of Qatari nationality.

Most MEN1 patients have a positive family history of the syndrome. The condition follows an autosomal dominant inheritance pattern, meaning that an affected parent carries a 50% risk of passing it on to each child. MEN1 gene mutations are detectable in approximately 70–95% of affected patients [8].

MEN 1 characteristic tumors include: 1) Parathyroid adenoma (90%), 2) Enteropancreatic tumor (30–70%): gastrinoma (40%), insulinoma (10%), nonfunctioning and PPoma (20–55%), glucagonoma (1%), VIPoma (1%), 3) Pituitary adenoma (30–40%): prolactinoma (20%), somatotropinoma (10%), corticotropinoma (5%), nonfunctioning (5%), 4) Associated tumors: adrenal cortical tumor (40%), pheochromocytoma (1%), bronchopulmonary NET (2%), thymic NET (2%), gastric NET (10%), lipomas (30%), angiofibromas (85%), collagenomas (70%), meningiomas (8%) [12].

MEN1 is considered a likely diagnosis in patients who exhibit endocrinopathy in two of the three typically involved organs, or in those with an endocrinopathy in one of these organs accompanied by a first-degree family history of MEN1 [8]. Approximately 50% of affected individuals exhibit clinical signs and symptoms by age 20, with over 95% developing symptoms by age 40 [13].

In our patient case, hyperparathyroidism secondary to a parathyroid adenoma was confirmed by sestamibi scanning, and she had initially been prepared for surgical management at another medical center. However, when she presented to our hospital for a second opinion, certain clinical and familial findings raised suspicion for multiple endocrine neoplasia type 1 (MEN1) syndrome, despite the absence of a clearly documented family history. Notably, her mother had undergone surgery for a pituitary lesion many years earlier, though details of the pathology were unavailable, and her cousin had a history of both thyroid cancer and hyperparathyroidism. These findings prompted further targeted evaluation, which ultimately established the diagnosis of MEN1 syndrome in our patient.

Early detection of MEN1 is crucial for enhancing disease outcomes and survival in both patients and their affected family members [14]. A recent cohort study of Dutch MEN1 patients examining the delay between diagnosis in index patients and their relatives (non-index patients) reported that 10 individuals, representing 4% of non-index cases, died from a MEN1-related condition that arose during or prior to this lag period (before diagnosis) [15]. Patients should be cared for by a multidisciplinary team of specialists with expertise in diagnosing and treating endocrine tumors, to enable appropriate genetic screening of family members and to ensure that affected patients undergo the correct surveillance protocols [12].

In 90% of patients, primary hyperparathyroidism is the initial endocrine manifestation of MEN1 and can occasionally be detected as early as age 8 [16]. Primary hyperparathyroidism (PHPT) is the most prevalent endocrine disorder in MEN1, occurring in nearly all patients by the age of 50 [17]. The symptoms of hyperparathyroidism in MEN1 are closely resembles that of sporadic PHPT, typically involving a prolonged phase of asymptomatic hypercalcemia and low associated morbidity [8].

Endocrine gastroenteropancreatic (GEP) tumors develop in approximately 30-80% of MEN1 patients, representing the second most common clinical manifestation of the syndrome. In contrast to sporadic GEP tumors, those associated with MEN1 are typically multiple and nodular, and tend to appear about ten years earlier than sporadic cases

[18,19] Multiple adenomas can be dispersed throughout the pancreas, sometimes numbering up to 100. Their size varies from microadenomas to macroadenomas exceeding 0.5 cm in diameter. About two-thirds of these tumors are hormonally active, producing excess amounts of gastrin, insulin, somatostatin, glucagon, neurotensin, or vasoactive intestinal polypeptide (VIP), each linked to characteristic clinical syndromes. Among functional pancreatic tumors, gastrinomas are the most common (54%), followed by insulinomas (15%). While non-functional tumors and insulinomas are typically confined to the pancreas, gastrinomas often occur in the surrounding soft tissues and within the duodenal submucosa, but not in the mucosa itself, where the gastrin-producing G cells normally reside. Non-functional tumors of the gastroenteropancreatic (GEP) tract occur in roughly 20% of individuals with MEN1 syndrome [8].

Anterior pituitary adenomas have been reported in 15–90% of patients with MEN1 [20].In MEN1, anterior pituitary adenomas are typically solitary. They are invasive in only 10–15% of cases, and malignant transformation is extremely rare. Clinical manifestations arise from the hormone secreted by the tumor and/or from its mass effect. Pituitary macroadenomas may compress the optic chiasm, leading to bitemporal hemianopia, other visual field deficits, blurred vision, and headaches. Compression of adjacent normal pituitary tissue can result in hypopituitarism. Around 60% of MEN1-related pituitary tumors are prolactin-secreting (prolactinomas), 25% produce growth hormone (GH), 3% secrete adrenocorticotropic hormone (ACTH) causing hypercortisolism, and the remainder appear to be non-functional [8].The average age at diagnosis of MEN1-associated pituitary adenomas is approximately 40 years, which is comparable to that of sporadic isolated pituitary tumors [21].Prolactin-secreting tumors are the most common pituitary tumors in MEN1, and symptoms in women include galactorrhoea, amenorrhoea, and infertility. While symptoms in men include hypogonadism, sexual dysfunction, reduction of libido, impotence, and, more rarely, gynecomastia [8].

Other MEN1-associated endocrine tumors include the Adrenocortical tumors, which may involve one or both adrenal glands, occur in approximately 20–40% of patients with MEN1 (22). Pheochromocytoma occurs in less than 1% of MEN1 patients and is invariably unilateral (20). Thyroid tumors—such as adenomas, colloid goiters, and carcinomas—have been reported in over 25% of individuals with MEN1 [13].

MEN1-associated non-endocrine tumors include the Carcinoid tumors are found in about 10% of MEN1 patients and may arise in the gastrointestinal tract (from type II gastric enterochromaffin-like cells), pancreas, bronchi, or thymus [8].Collagenomas have been observed in over 70% of MEN1 patients and typically appear as multiple skin-colored or occasionally hypopigmented nodules (23). Lipomas are seen in 20–30% of MEN1 cases (23). Leiomyomas are benign tumors arising from smooth muscle, can occasionally develop in the esophagus, uterus, or rectum of MEN1 patients. Meningiomas occur in approximately 8% of MEN1 patients and generally present later in life [24]. Spinal ependymomas are rare in MEN1, reported in about 1% of cases, and are associated with abnormalities at the 11q13 locus [25].

Surgery is considered the preferred and most effective treatment for primary hyperparathyroidism (PHPT) in MEN1 patients. It is recommended for MEN1 patients who are symptomatic for PHPT, and for asymptomatic MEN1 patients who have hypercalciuria, serum calcium levels above 12.0 mg/dl, or a concurrent gastrinoma [8]. The timing of parathyroid surgery typically considers the severity of PHPT symptoms, the circulating levels of PTH and calcium, the presence of other MEN1-related endocrinopathies (particularly Zollinger-Ellison syndrome (ZES)), and the patient's age. Surgical options include subtotal parathyroidectomy (removal of three parathyroid glands and part of the fourth) or total parathyroidectomy (removal of all four parathyroid glands along with thymic tissue to reduce the risk of recurrence and thymic carcinoids). Recurrence of hyperparathyroidism can occur due to increased activity of residual tumor tissue or the development of new tumors in remaining normal, ectopic, or supernumerary parathyroid glands. Because of the high rate of postoperative recurrence and the significant risks associated with neck reoperation, total parathyroidectomy combined with autologous parathyroid grafting into the brachioradialis muscle of the forearm is often the preferred surgical approach for MEN1-associated PHPT [8].

The recommended treatment for non-metastatic gastrinomas is surgical removal. However, approximately 50% of gastrinomas in MEN1 patients have already metastasized by the time of diagnosis, and 30% of these cases prove fatal [26].

Surgery is generally the preferred treatment for insulinomas for several reasons: hypoglycemia is difficult to manage with medication; the pancreatic surgery often reveals one or more incidental macroscopic lesions; removing these visible pancreatic lesions typically cures the hypoglycemic syndrome; and complete surgical removal usually results in a cure. The optimal surgical strategy for MEN1-related insulinomas involves intraoperative localization of nodules larger than 0.5 cm using palpation or intraoperative ultrasound, followed by enucleation of these nodules or pancreatic resection if multiple large, deep tumors are present [27].Limited resection or simple enucleation of nodules is more often associated with disease persistence or recurrence [28].

For other functional (glucagonoma, somatostatinoma, VIPoma, Ppoma) and non-functional GEP tumors, the authors recommend monitoring with endoscopic ultrasound. Pancreatic surgery for asymptomatic MEN1 patients should be considered when the lesion size approaches 2 cm [8].

Treatment of MEN1-associated pituitary tumors follows the same approach as for sporadic pituitary tumors. Pituitary adenomas are categorized as microadenomas (<10 mm) or macroadenomas (>10 mm). For macroadenomas, the preferred treatments are transsphenoidal or endoscopic resection and/or radiotherapy [29].Dopamine agonists (such as cabergoline, bromocriptine, pergolide, and quinagolide) are the first-line therapy for prolactin-secreting microadenomas, while somatostatin analogues are the medical treatment of choice for growth hormone-secreting microadenomas[30].Surgery is the primary treatment for non-functioning pituitary adenomas. Preoperative therapy with potent dopamine agonists or, occasionally, somatostatin analogues can reduce tumor size in 5 to 15 percent of cases [31].

There is no general agreement on how to manage adrenocortical tumors in MEN1. However, larger tumors are believed to carry a greater risk of malignancy, and some experts recommend laparoscopic surgical removal of adrenocortical tumors larger than 3.5 cm in diameter [32].

Surgery is the treatment of choice for bronchial and thymic carcinoids. Thymic carcinoids have a high metastatic rate (60%) and nearly always recur after surgery, with recurrence rates reaching up to 100% within one year postoperatively [33].

Conclusion

This case represents the first reported diagnosis of MEN1 syndrome in Qatari patient, highlighting the need to recognize its presence within the population, as it is a rare inherited syndrome affecting the endocrine system with some associated non-endocrine tumors. Early detection of MEN1 is crucial for enhancing disease outcomes and survival in both patients and their affected family members. Patients should be cared for by a multidisciplinary team of specialists with expertise in diagnosing and treating endocrine tumors, to enable appropriate genetic screening of family members and to ensure that affected patients undergo the correct surveillance protocols.

Author Contributions

Bakhos Alhaddad: Diagnosis of the Case, study concept and design, data collection and analysis, writing the paper.

Abdul Azim Hussain: critical revision of the manuscript, final approval of the paper.

Thair S. Abdulla: image evaluation and interpretation, final approval of the paper.

Mahmoud Zari: critical revision of the manuscript, final approval of the paper.

Declarations

Ethics approval and consent to participate

This case report was approved by the ethics committee, Al Ahli Hospital, Doha, Qatar. A copy of the approval letter is available for review upon request.

Consent

Verbal and written informed consent were obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Competing interests

The authors declare that they have no competing interests.

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