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Case Study

Metastatic Neuroendocrine Neoplasm with An Uncertain Primary Site Managed with Long-Acting Octreotide: A Case Report

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Neuroendocrine neoplasms (NENs) are a heterogeneous group of epithelial malignancies characterized by neuroendocrine differentiation and a broad spectrum of biological behavior, ranging from relatively indolent well-differentiated neuroendocrine tumors to aggressive poorly differentiated neuroendocrine carcinomas. Accurate classification requires integration of histomorphology, immunohistochemistry, proliferative activity, and clinical and radiological findings. A 58-year-old woman was evaluated for a neuroendocrine neoplasm with radiologically suspected metastatic disease. Computed tomography of the chest performed in November 2024 demonstrated a 2 × 1 cm lesion in the right lower lobe of the lung, for which surgical resection was advised; however, the patient declined the procedure. During subsequent follow-up, she developed cough and intermittent vomiting with significant weight loss from 53 kg to 43 kg. Further imaging demonstrated a 6.7 × 6.2 cm lesion involving segments II and IV of the liver, while both adrenal glands were described as bulky and suspicious for metastatic involvement. Biopsy was reported as a neuroendocrine neoplasm, with immunohistochemical positivity for synaptophysin and chromogranin. Review of the original pathology slides remained pending at the time of preparation of this report, and therefore definitive tumor differentiation, grade, proliferative index, and primary site could not be established. Although the pulmonary lesion represented a potential primary site, its relationship to the neuroendocrine neoplasm could not be definitively confirmed. The patient was managed through the oncology day-care unit and was receiving octreotide 30 mg intramuscularly, with the fifth treatment cycle ongoing at the time of reporting. This case highlights the diagnostic complexity of metastatic neuroendocrine neoplasms when the primary site and complete pathological classification remain incompletely established and emphasizes the importance of pathological characterization and correlation with radiological findings during treatment planning.

Keywords: Neuroendocrine neoplasm; metastatic neuroendocrine tumor; octreotide; somatostatin analogue; liver metastasis; adrenal metastasis; uncertain primary site; case report.

INTRODUCTION

Neuroendocrine neoplasms (NENs) are a heterogeneous group of epithelial neoplasms that exhibit neuroendocrine differentiation and can arise in multiple anatomical sites, including the gastrointestinal tract, pancreas, lung, and other organs. They encompass biologically distinct entities ranging from well-differentiated neuroendocrine tumors (NETs) to poorly differentiated neuroendocrine carcinomas (NECs). The current World Health Organization classification separates these major categories according to differentiation,

morphology, and proliferative characteristics; well-differentiated NETs may be further graded according to proliferative activity, whereas NECs are high-grade malignancies by definition. The Ki-67 proliferative index and mitotic activity are therefore important components of contemporary classification and grading. [1]

Etiology of NEN

The etiology of NENs is heterogeneous and depends on the anatomical site and biological subtype. Most cases arise sporadically, although hereditary predisposition is recognized in a subset of patients, particularly in association with syndromes such as multiple endocrine neoplasia type 1, von Hippel-Lindau disease, neurofibromatosis type 1, and tuberous sclerosis complex. NENs may also arise from different populations of neuroendocrine cells distributed throughout the body, accounting for their wide anatomical distribution and clinical heterogeneity. In some patients, the primary tumor remains clinically occult and the disease first becomes apparent through a metastatic lesion. [1,3,5]

Pathophysiology and clinical manifestation

The pathophysiology of NENs is related to abnormal proliferation of cells exhibiting neuroendocrine differentiation. These tumors may retain the ability to synthesize and secrete peptide hormones or biogenic amines, resulting in functional syndromes in a proportion of patients, whereas many tumors are nonfunctioning and present because of mass effect, incidental detection, or metastatic disease. Tumor behavior is strongly influenced by differentiation and proliferative activity. Well-differentiated NETs may demonstrate relatively slow progression, whereas poorly differentiated NECs are characterized by aggressive biological behavior and a high proliferative rate. Somatostatin receptor expression is also clinically relevant because it provides both a potential therapeutic target and an imaging target for functional receptor-based imaging. [1,2,6] NENs may present with nonspecific symptoms, and the clinical manifestations depend on the primary site, hormone secretion, tumor burden, and presence of metastases. Patients with advanced disease may present with weight loss, abdominal or respiratory symptoms, gastrointestinal disturbances, or symptoms related to metastatic involvement. The liver is a particularly important site of metastatic disease in NENs, and hepatic tumor burden may substantially influence prognosis and therapeutic decision-making. [15,16] In some patients, the disease is first recognized as metastatic NEN without an identifiable primary tumor. NENs of unknown or uncertain primary origin represent a recognized clinical challenge, with the primary site remaining unidentified despite conventional imaging and pathological evaluation in a proportion of patients. [3,4,17]

Diagnostic Criteria

Diagnosis of a NEN requires integration of clinical presentation, radiological assessment, histopathological examination, and immunohistochemistry. Histological assessment establishes the morphological nature of the tumor, while neuroendocrine markers such as synaptophysin and chromogranin support neuroendocrine differentiation. However, positivity for these markers alone does not establish the tumor grade or anatomical primary site. Additional pathological assessment, including evaluation of Ki-67 proliferative activity and mitotic rate, is important for classification, while site-specific immunohistochemical markers may assist in identifying the likely origin of a metastatic NEN. [1,2,5] In patients with metastatic disease and an uncertain primary site, cross-sectional imaging, somatostatin-receptor imaging, immunohistochemical profiling, and, in selected circumstances, molecular approaches may be used to identify the most likely primary site and guide management. [3,4,5]

Management

Management of NENs is individualized according to tumor differentiation, grade, anatomical primary site, extent of disease, somatostatin-receptor expression, functional status, symptoms, and rate of disease progression. Treatment options may include surgical resection or cytoreductive procedures for selected localized or metastatic disease, somatostatin analogues for appropriately selected well-differentiated and somatostatin-receptor-positive tumors, targeted therapies such as everolimus or sunitinib in selected settings, cytotoxic chemotherapy for appropriate high-grade or progressive disease,

peptide receptor radionuclide therapy for somatostatin-receptor-positive tumors, and liver-directed therapies in selected patients with hepatic metastases. [6,7,9,12,13,15,16] Somatostatin analogues such as octreotide and lanreotide have both antisecretory and antiproliferative roles. The PROMID study demonstrated delayed tumor progression with octreotide long-acting release in patients with metastatic midgut NETs, while the CLARINET study demonstrated prolonged progression-free survival with lanreotide in patients with metastatic enteropancreatic NETs. [10,11] Despite advances in imaging, pathology, and systemic treatment, establishing the primary site may remain difficult in patients presenting with metastatic NEN. This diagnostic uncertainty can have important therapeutic implications because treatment selection and expected biological behavior vary according to tumor differentiation, grade, primary site, and receptor status. [3,4,7]

2. Case Presentation

2.1 Patient information and clinical presentation

A 58-year-old woman was evaluated in the oncology department for ongoing management of a neuroendocrine neoplasm with radiologically suspected metastatic disease. The initial evaluation included a CT scan of the chest performed in November 2024, which demonstrated a 2 × 1 cm lesion in the right lower lobe of the lung. Surgical resection of the pulmonary lesion was advised; however, the patient was not willing to undergo the proposed surgical intervention. During subsequent follow-up, the patient developed cough and intermittent vomiting. This was accompanied by significant unintentional weight loss, with her body weight decreasing from 53 kg to 43 kg, corresponding to an approximately 19% reduction in body weight. Further radiological evaluation demonstrated a 6.7 × 6.2 cm lesion involving segments II and IV of the liver. The bilateral adrenal glands were described as bulky, with metastatic involvement considered suspicious. The patient was subsequently investigated with tissue sampling. Histopathological examination was reported as a neuroendocrine neoplasm, with immunohistochemical staining positive for synaptophysin and chromogranin. At the time of preparation of this report, review of the original pathology slides was still pending. Therefore, definitive classification according to differentiation and grade could not be established from the currently available documentation. Although a right lower-lobe pulmonary lesion was identified at the initial presentation, the available clinical information does not definitively establish the lung as the primary site. Accordingly, the present case is described as a metastatic neuroendocrine neoplasm with an uncertain primary site rather than a confirmed primary pulmonary neuroendocrine tumor.

2.2 Relevant medical and medication history

The available medication history included Esciflo 250 inhaler, one puff twice daily.

No additional significant chronic medical history has been provided in the available clinical information.

The patient's family history was notable for malignancy in two first-degree relatives. Her mother had carcinoma of the endometrium, while her sister had carcinoma of the pancreas.

This family history was documented as part of the patient's clinical background; however, no hereditary cancer syndrome has been established in the available records.

2.3 Histopathological and immunohistochemical findings

Biopsy examination was reported as consistent with a neuroendocrine neoplasm.

Immunohistochemical examination demonstrated:

* Synaptophysin: positive

* Chromogranin: positive

The expression of synaptophysin and chromogranin supports neuroendocrine differentiation. However, these findings alone do not establish the anatomical primary site or provide sufficient information for definitive tumor grading. Contemporary pathological classification requires integration of morphology, differentiation, proliferative activity, and, where appropriate, additional immunohistochemical

markers. In this patient, Ki-67 proliferation index, mitotic count, definitive differentiation status, and the complete immunohistochemical profile were not available at the time of preparation of the manuscript. The pathology slides were awaiting review. This limitation is particularly relevant because the distinction between well-differentiated NET and poorly differentiated neuroendocrine carcinoma has important therapeutic implications.

3. Diagnostic Assessment

The clinical and radiological evaluation demonstrated abnormalities at multiple sites, including a 2 × 1 cm lesion in the right lower lobe of the lung, a 6.7 × 6.2 cm lesion involving segments II and IV of the liver, and bilateral bulky adrenal glands suspicious for metastatic involvement. Histopathological examination of the biopsy was reported as a neuroendocrine neoplasm, with immunohistochemical positivity for synaptophysin and chromogranin, supporting neuroendocrine differentiation. Based on the available clinical, radiological, and pathological findings, the overall diagnosis was considered to be a metastatic neuroendocrine neoplasm. However, the available records did not establish the anatomical site of origin with certainty. Although the right lower-lobe pulmonary lesion represented a potential primary site, its relationship to the neuroendocrine neoplasm could not be definitively established from the available information. Furthermore, the available pathology report did not provide a documented Ki-67 proliferation index, mitotic count, or definitive tumor grade. Review of the original pathology slides was pending at the time of preparation of this report. Accordingly, the diagnosis is reported as metastatic neuroendocrine neoplasm with an undetermined primary site, pending completion of pathological review.

4. Therapeutic Intervention

The patient was managed through the oncology day-care treatment unit and was not admitted for inpatient treatment. She was receiving octreotide 30 mg intramuscularly and was undergoing her fifth treatment cycle at the time of reporting. The available clinical information does not establish whether the treatment was initiated specifically for hormonal symptom control, antiproliferative intent, or both. Therefore, the indication for octreotide is described conservatively as ongoing systemic management of the neuroendocrine neoplasm. Somatostatin analogues exert their effects through somatostatin receptors and have both antisecretory and antiproliferative roles in selected neuroendocrine tumors. Recent reviews continue to recognize SSAs as a central component of treatment for many well-differentiated neuroendocrine tumors, although their optimal use depends on tumor differentiation, grade, primary site, disease burden, somatostatin receptor expression, symptoms, and treatment goals.

5. Follow-up and Outcome

At the time of preparation of this report, the patient remained under oncology follow-up and was receiving treatment through the oncology day-care unit. She was receiving octreotide 30 mg intramuscularly and was undergoing her fifth treatment cycle. The patient was not admitted for the current treatment and continued her therapy through the day-care setting. At present, objective treatment response could not be assessed, as follow-up radiological imaging after initiation of octreotide was not available for inclusion in this report. Therefore, no conclusion regarding complete response, partial response, stable disease, or disease progression was made. The patient continued to be followed clinically during treatment. Pathological review of the available biopsy slides remained pending at the time of preparation of the manuscript and was expected to provide further information regarding tumor classification, differentiation, proliferative activity, and potentially the site of origin.

DISCUSSION

Rindi et al., in their overview of the 2022 WHO classification of neuroendocrine neoplasms, emphasized the distinction between well-differentiated neuroendocrine tumors (NETs) and poorly differentiated neuroendocrine carcinomas (NECs), with differentiation, morphology, mitotic activity, and proliferative assessment forming important components of classification. [1] In the present patient, biopsy demonstrated a neuroendocrine neoplasm with positive synaptophysin and chromogranin staining. However,

the available report does not document the Ki-67 proliferation index, mitotic count, or definitive tumor grade. Therefore, the tumor cannot currently be assigned to a specific NET grade or categorized as NEC on the basis of the available information alone. Bellizzi highlighted the importance of integrating morphology and immunohistochemistry when diagnosing and classifying neuroendocrine neoplasms. Synaptophysin and chromogranin are established markers of neuroendocrine differentiation, but their positivity does not by itself determine the anatomical primary site or tumor grade. [2] In the present case, positivity for both markers supports neuroendocrine differentiation, while the pending pathology slide review may provide additional information regarding morphology, differentiation, proliferative activity, and potentially the primary site. Thus, the pending review represents an important part of the diagnostic evaluation. Determining the primary site is particularly relevant in metastatic neuroendocrine neoplasms. Juhlin et al. discussed the diagnostic challenges associated with metastatic neuroendocrine neoplasms of unknown primary and emphasized the role of pathological evaluation in identifying clues to tumor origin. [3] Similarly, diagnostic approaches to NENs with an unknown primary may incorporate cross-sectional imaging, immunohistochemistry, functional imaging, and other investigations according to the clinical context. [4] In the present patient, the right lower-lobe pulmonary lesion represents a possible primary site; however, its presence alone does not establish that the neuroendocrine neoplasm originated in the lung. The current evidence therefore supports describing the condition as a metastatic neuroendocrine neoplasm with an uncertain primary site until further pathological and radiological assessment is available. The possibility of a pulmonary primary is nevertheless clinically relevant. Pelosi et al. described the pathological spectrum of pulmonary neuroendocrine tumors and emphasized the importance of appropriate morphological and immunohistochemical classification in distinguishing the different pulmonary neuroendocrine entities. [5] In this patient, the previously identified 2 × 1 cm right lower-lobe lesion was advised for surgical resection, but the patient declined the procedure. Consequently, tissue confirmation from the pulmonary lesion was not available to establish whether it represented the primary neuroendocrine tumor. The liver lesion represents another significant component of the patient's disease. The patient was found to have a 6.7 × 6.2 cm lesion involving segments II and IV of the liver, while the bilateral adrenal glands were described as bulky and suspicious for metastatic involvement. Recent literature on neuroendocrine tumor liver metastases has emphasized that hepatic disease burden and distribution are important considerations when evaluating treatment options. [15,16] Depending on tumor biology, extent of hepatic involvement, resectability, symptoms, and overall disease distribution, treatment of neuroendocrine liver metastases may involve systemic therapy or selected liver-directed approaches, including surgical cytoreduction, ablation, or embolization-based procedures. [15,16] In the present case, the available information does not provide sufficient detail regarding the complete number and distribution of hepatic lesions, vascular involvement, or resectability; therefore, no specific conclusion regarding suitability for liver-directed treatment can be made. The bilateral adrenal abnormalities were described radiologically as bulky and suspicious for metastatic involvement. However, because histological confirmation was not available, these findings should be considered radiologically suspicious rather than confirmed adrenal metastases. This distinction is important when presenting a case report because it separates pathological diagnoses from radiological impressions. The patient is currently receiving octreotide 30 mg intramuscularly and was undergoing her fifth treatment cycle at the time of reporting. Somatostatin analogues have an established role in selected neuroendocrine tumors because of their antisecretory and antiproliferative effects. The PROMID study by Rinke et al. evaluated octreotide long-acting release in patients with metastatic midgut neuroendocrine tumors and demonstrated delayed tumor progression compared with placebo. [10] These findings provide clinical evidence supporting the antiproliferative activity of octreotide in appropriately selected well-differentiated metastatic NETs. However, because the primary site and definitive pathological grade are not yet established in the present patient, the PROMID findings should be considered supportive evidence for the therapeutic role of octreotide rather than direct evidence of efficacy in this individual case. The

CLARINET trial conducted by Caplin et al. further demonstrated prolonged progression-free survival with the somatostatin analogue lanreotide in patients with metastatic enteropancreatic neuroendocrine tumors. [11] Although the drug and patient population differed from the present case, the findings contribute to the broader evidence supporting the antiproliferative role of long-acting somatostatin analogues in selected neuroendocrine tumors. The use of somatostatin analogues is influenced by tumor differentiation, grade, primary site, receptor expression, tumor burden, symptoms, and disease progression. The ASCO guideline on systemic therapy for metastatic well-differentiated gastroenteropancreatic NETs recognizes somatostatin analogues as an important systemic treatment option for appropriately selected patients. [7] Similarly, recent reviews have continued to describe octreotide and lanreotide as important components of the therapeutic approach to selected neuroendocrine tumors. [8,9] In the present patient, octreotide is the documented ongoing systemic treatment, while the underlying pathological characteristics required for more specific therapeutic categorization remain incompletely available. Other treatment modalities may be considered in patients with advanced neuroendocrine neoplasms depending on tumor characteristics and treatment response. The NETTER-1 study by Strosberg et al. demonstrated improved progression-free outcomes with ^177Lu-DOTATATE compared with high-dose octreotide in patients with advanced somatostatin-receptor-positive midgut NETs. [13] Similarly, the RADIANT-4 study by Yao et al. demonstrated improved progression-free survival with everolimus in patients with advanced, nonfunctioning neuroendocrine tumors of gastrointestinal or pulmonary origin. [12] These studies illustrate the expanding range of systemic treatment options for advanced NETs. Nevertheless, their findings cannot be directly applied to the present patient without information regarding tumor differentiation, grade, somatostatin-receptor expression, primary site, and evidence of progression during current treatment. The patient's substantial weight loss is another clinically relevant feature. Her body weight decreased from 53 kg to 43 kg, representing approximately a 19% reduction in body weight, in association with cough and intermittent vomiting. Significant weight loss in patients with advanced malignancy may result from several factors, including reduced oral intake, gastrointestinal symptoms, systemic effects of malignancy, or increased metabolic requirements. In this case, the available information does not establish a single cause for the weight loss. Nevertheless, nutritional status represents an important component of supportive care during ongoing oncology treatment. The family history was notable for endometrial carcinoma in the patient's mother and pancreatic carcinoma in her sister. This history is clinically noteworthy because pancreatic neuroendocrine tumors may occur in the context of hereditary endocrine tumor syndromes. However, the available family history alone does not establish a hereditary syndrome in this patient. Any consideration of genetic evaluation would depend on the eventual pathological classification, a more detailed three-generation family history, and clinical assessment.

CONCLUSION

This case describes a 58-year-old woman with a metastatic neuroendocrine neoplasm and an incompletely established primary site, initially identified in the context of a right lower-lobe pulmonary lesion. The subsequent clinical course was characterized by cough, intermittent vomiting, significant weight loss from 53 kg to 43 kg, a 6.7 × 6.2 cm hepatic lesion involving segments II and IV, and bulky bilateral adrenal glands suspicious for metastatic involvement. Biopsy demonstrated a neuroendocrine neoplasm with positive synaptophysin and chromogranin staining. The patient is currently receiving octreotide 30 mg intramuscularly and is on her fifth treatment cycle through the oncology day-care unit. The case emphasizes the importance of complete pathological characterization, including assessment of differentiation and proliferative activity, and careful correlation of pathological findings with imaging to establish the primary site. The pending pathology-slide review represents an important component of the ongoing diagnostic assessment. Because objective post-treatment imaging and complete pathological classification are currently unavailable, treatment response cannot be conclusively determined at the time of reporting.

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