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Isolated Metachronous Splenic Metastasis Secondary to Gastric Neuroendocrine Carcinoma: Case Report

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AUTHOR AND AFFILIATION

Alia Yassine Kassab[1], Lina Lasri[1], Mehdi Salmane[1], Asaad El Bakkari[1], Hounayda Jerguigue[1], Youssef Omor[1], Rachida Latib[1]

[1] Department of Radiology, National Institute of Oncology, Mohammed V University, Rabat, Morocco

Corresponding author : Alia Yassine Kassab,

ABSTRACT

We report a rare case of isolated metachronous splenic metastasis from poorly differentiated large-cell gastric neuroendocrine carcinoma, contributing to the scarce literature on atypical metastatic patterns. A 66-year-old woman, asymptomatic during routine surveillance, was found to have a solitary arterial-enhancing splenic nodule on imaging, with normal clinical examination and laboratory tests. Total splenectomy was performed, and histopathology confirmed metastatic large-cell neuroendocrine carcinoma consistent with the gastric primary. The patient recovered uneventfully and resumed routine oncologic follow-up. This case underscores that, although extremely uncommon, isolated splenic metastasis should be considered during follow-up of gastric neuroendocrine carcinoma, and that timely splenectomy can provide both diagnostic confirmation and therapeutic benefit, highlighting the importance of vigilant surveillance and recognition of unusual metastatic sites.

KEYWORDS

Large-cell neuroendocrine carcinoma, spleen metastasis, metachronous metastasis, splenectomy, case report

MAIN ARTICLE

INTRODUCTION

Neuroendocrine tumors (NETs) are rare neoplasms most commonly arising in the gastrointestinal tract. Gastric neuroendocrine carcinoma is a particularly uncommon subtype, characterized by aggressive behavior and a high metastatic potential [1,2]. The liver is the predominant site of metastasis, followed by the lungs and bones. In contrast, splenic involvement is extremely rare and is usually detected incidentally, often in the context of widespread multivisceral disease [1,2,4].

Total splenectomy remains the preferred treatment for secondary splenic lesions, with limited data suggesting a post-splenectomy survival of approximately seven years [2,5,7]. Here, we report a unique case of an isolated metachronous splenic metastasis detected during routine follow-up in a patient previously treated for gastric neuroendocrine carcinoma, adding valuable insight into the atypical metastatic patterns of this rare malignancy.

PATIENT AND OBSERVATION

Patient Information

A 66-year-old woman with no significant past medical history, no known genetic disorders, and no relevant family history of malignancy, was diagnosed in 2022 with a poorly differentiated large-cell gastric neuroendocrine carcinoma. She had no notable psychosocial risk factors and was independent in her activities of daily living. Initial treatment included distal gastrectomy with lymph node dissection, followed by adjuvant chemotherapy. The patient tolerated treatment well, with no major complications. She presented for routine follow-up one year after completion of therapy, during which she was asymptomatic and reported no abdominal pain, weight loss, fever, or other systemic complaints.

Physical examination at the time of follow-up was unremarkable. Vital signs were within normal limits. The abdomen was soft, non-tender, with no palpable masses or splenomegaly. Laboratory studies, including complete blood count and liver function tests, were within normal ranges.

Diagnostic Assessment

Imaging studies included:

- **CT scan:** Solitary 1.5 cm nodular lesion on the lateral spleen with arterial-phase enhancement (Figures 1 and 2), ill-defined in the portal phase (Figure 3) and no longer visible in the delayed phase (Figure 4); no other metastatic lesions.
- **Ultrasonography:** Well-circumscribed, hypoechoic splenic nodule consistent with metastasis.

DISCUSSION

Neuroendocrine tumors (NETs) of the gastrointestinal tract and pancreas have shown a notable increase in incidence over the past decade, largely due to advances in imaging and diagnostic techniques. Despite these improvements, metastases are common at the time of diagnosis, occurring in 50–85% of cases [1,2]. Gastric neuroendocrine carcinomas, in particular, are aggressive and have high metastatic potential. Secondary splenic metastases are exceptionally rare, with an estimated incidence of approximately 7% for all primary tumors, and are usually observed in the context of advanced multivisceral disease [2,3,5]. Isolated splenic metastasis, as seen in our patient, is extremely uncommon, with only a handful of cases reported from gastropancreatic NETs in the literature [1–5].

The rarity of splenic metastases has been attributed to several anatomic and physiological factors, including constant splenic blood flow, rhythmic splenic capsule movements, absence of afferent lymphatics, and the immune activity of splenic macrophages, which create a hostile microenvironment for tumor implantation [1,2,3,4,6]. Anatomical considerations, such as the acute angle of the splenic artery, may further reduce the likelihood of tumor emboli reaching the spleen. Hematogenous spread via the systemic arterial route is thought to be the main mechanism, though this typically results in multiorgan involvement [4,5,6]. In our case, the isolated nature of the lesion suggests that unusual hematogenous dissemination or retrograde lymphatic spread may have occurred, but no definitive mechanism explains this exceptional presentation.

Clinically, splenic metastases are often asymptomatic and discovered incidentally during surveillance imaging. Rarely, patients may present with splenomegaly, left upper quadrant

pain, or severe complications such as splenic rupture or thrombosis [2,5,6]. Imaging features vary; lesions can be solitary or multiple and may involve the parenchyma, capsule, or both. Ultrasound typically demonstrates well-circumscribed hypoechoic lesions, CT shows well-defined hypodense nodules with peripheral or arterial-phase enhancement, and MRI usually shows T1 hypointensity and T2 hyperintensity [4–6]. Our patient's lesion was solitary, involved both parenchyma and capsule, and demonstrated full arterial-phase enhancement on CT, consistent with previously reported imaging patterns.

Definitive diagnosis requires histopathological confirmation. Fine-needle aspiration is often inconclusive, leaving splenectomy as the gold standard for both diagnosis and management. In addition to providing histological confirmation, splenectomy may prevent complications and control disease progression [2,5,6]. Novel therapeutic approaches, such as percutaneous radiofrequency ablation, are under investigation but are not yet standard of care.

CONCLUSION

Secondary splenic metastases from solid tumors—whether synchronous or metachronous, solitary or multiple—are rare, and the incidental discovery of an isolated splenic lesion during cancer follow-up presents diagnostic challenges. In patients with gastric neuroendocrine carcinoma, any splenic lesion should raise suspicion for metastasis, despite its uncommon occurrence. Such lesions are often asymptomatic and may vary in size, shape, and density, necessitating further evaluation with imaging modalities such as MRI or 18F-FDG PET-CT. Splenectomy remains the preferred diagnostic and therapeutic approach, providing histopathological confirmation while effectively controlling disease progression. Early recognition of atypical metastatic patterns is essential to guide timely management and optimize patient outcomes.

FIGURES:

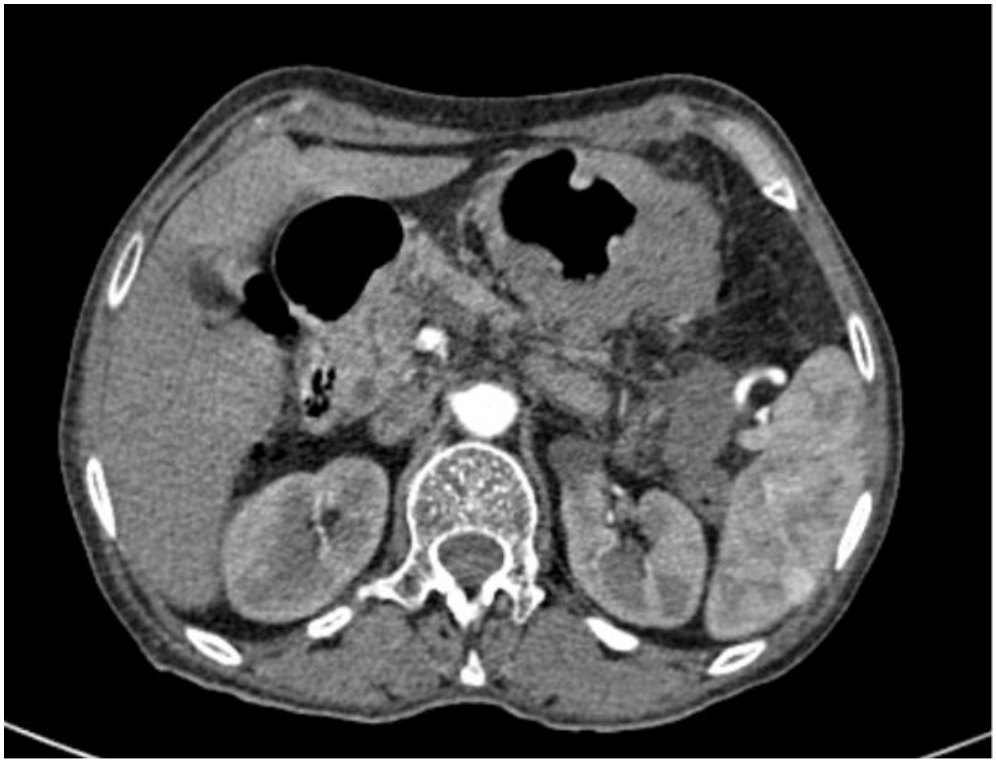

Figure 1: *Axial slice of an abdominal CT scan in the arterial phase, showing a markedly enhancing splenic nodule corresponding to the splenic metastasis, alongside the gastric NET.*

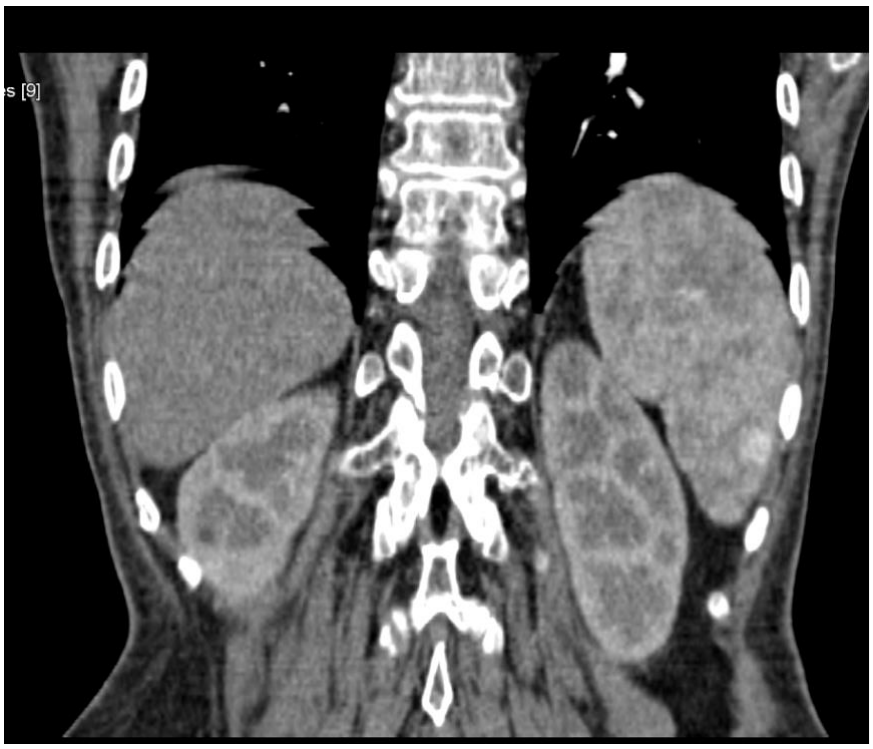

Figure 2: *Coronal slice of an abdominal CT scan in the arterial phase, demonstrating a splenic nodule with pronounced arterial enhancement corresponding to the splenic metastasis.*

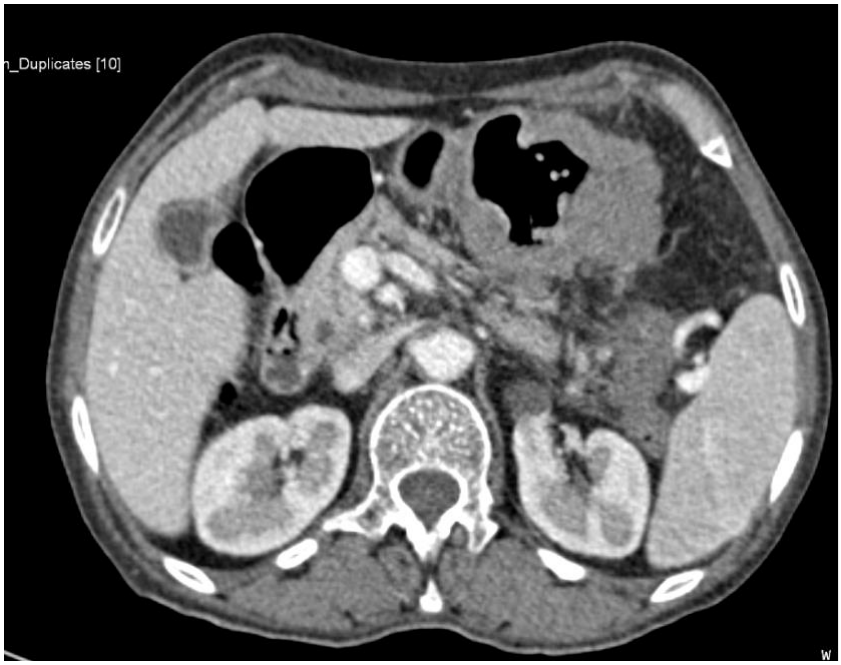

Figure 3: *Axial slice of an abdominal CT scan in the portal phase, with the splenic nodule corresponding to the metastasis faintly visible. The gastric NET is also visible.*

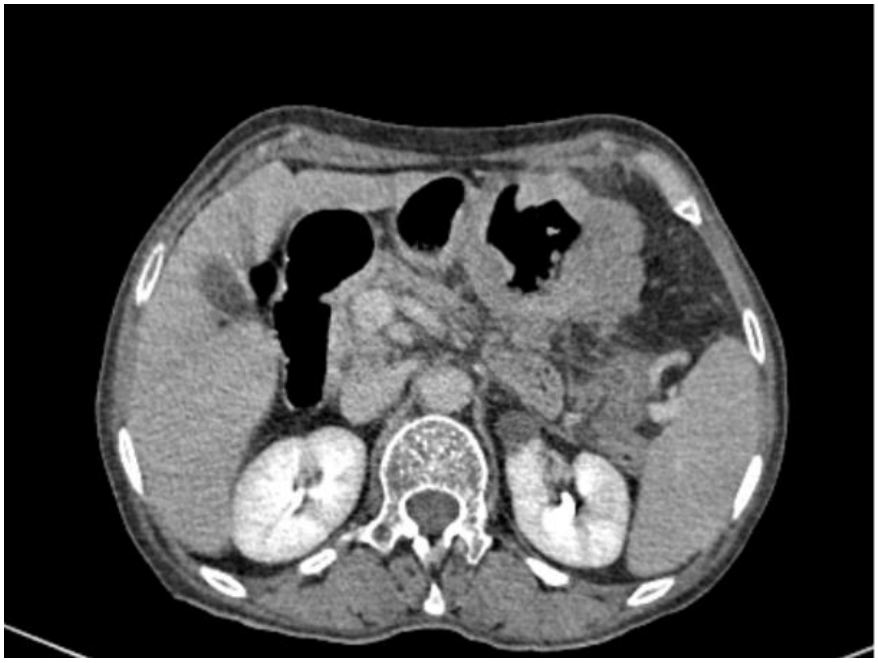

Figure 4: *Axial slice of an abdominal CT scan in the delayed phase, showing the splenic nodule corresponding to the metastasis no longer visible.*

ACKNOWLEDGEMENTS

The authors sincerely thank the patient for her cooperation and consent to share her case for scientific and educational purposes. We also acknowledge the contributions of the multidisciplinary team—radiologists, surgeons, oncologists, and pathologists—whose efforts made this report possible.

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CARE case report guidelines

CARE Checklist of information to include when writing a case report

Topic	Item	Checklist item description	Reported on Line
Title	1	The diagnosis or intervention of primary focus followed by the words "case report"	Page 1, Lines 1-2
Key Words	2	2 to 5 key words that identify diagnoses or interventions in this case report, including "case report"	Page 1, Lines 20-21
Abstract (no references)	3a	Introduction: What is unique about this case and what does it add to the scientific literature?	Page 1, Lines 8-10
	3b	Main symptoms and/or important clinical findings	Page 1, 10-12
	3c	The main diagnoses, therapeutic interventions, and outcomes	Page 1, Lines 12-14
	3d	Conclusion—What is the main "take-away" lesson(s) from this case?	Page 1, Lines 15-19
Introduction	4	One or two paragraphs summarizing why this case is unique (**may include references**)	Page 1-2, Lines 23-35
Patient Information	5a	De-identified patient specific information.	Page 2, Lines 38-44
	5b	Primary concerns and symptoms of the patient.	Page 2, Lines 45-47
	5c	Medical, family, and psycho-social history including relevant genetic information	Page 2, Lines 39-42
	5d	Relevant past interventions with outcomes	Page 2, Line 43
Clinical Findings	6	Describe significant physical examination (PE) and important clinical findings.	Page 2, Lines 48-52
Timeline	7	Historical and current information from this episode of care organized as a timeline	Page 2, Lines 53-64
Diagnostic Assessment	8a	Diagnostic testing (such as PE, laboratory testing, imaging, surveys).	Page 2-3, Lines 65-70
	8b	Diagnostic challenges (such as access to testing, financial, or cultural)	Page 3, Lines 71-74
	8c	Diagnosis (including other diagnoses considered)	Page 3, Lines 75-77
	8d	Prognosis (such as staging in oncology) where applicable	Page 3, Lines 78-80
Therapeutic Intervention	9a	Types of therapeutic intervention (such as pharmacologic, surgical, preventive, self-care)	Page 3, Lines 81-86
	9b	Administration of therapeutic intervention (such as dosage, strength, duration)	Page 3, Lines 81-86
	9c	Changes in therapeutic intervention (with rationale)	Page 3, Lines 81-86
Follow-up and Outcomes	10a	Clinician and patient-assessed outcomes (if available)	Page 3, Lines 87-94
	10b	Important follow-up diagnostic and other test results	Page 3, Lines 87-94
	10c	Intervention adherence and tolerability (How was this assessed?)	Page 3, Lines 87-94
	10d	Adverse and unanticipated events	N/A
Discussion	11a	A scientific discussion of the strengths AND limitations associated with this case report	Page 5, Lines 143-150
	11b	Discussion of the relevant medical literature **with references**.	Page 4-5, Lines 105-142
	11c	The scientific rationale for any conclusions (including assessment of possible causes)	Page 5, Lines 151-157
	11d	The primary "take-away" lessons of this case report (without references) in a one paragraph conclusion	Page 5, Lines 158-168
Patient Perspective	12	The patient should share their perspective in one to two paragraphs on the treatment(s) they received	Page 3, Lines 95-100
Informed Consent	13	Did the patient give informed consent? Please provide if requested	Yes [x] No [ ]