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Case Report

Small Cell Lung Carcinoma Presenting With Paraneoplastic Diarrhoea Mimicking Carcinoid Syndrome Clinically: A Case Report

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ABSTRACT

Small cell lung carcinoma (SCLC) is an aggressive pulmonary neuroendocrine malignancy that rarely presents with carcinoid-like manifestations. We report the case of a woman in her 70s who presented with an 8-week history of profuse watery diarrhoea, hypotension, and acute kidney injury. Serum chromogranin A was markedly elevated, and computed tomography demonstrated a right hilar mass with bilateral pulmonary nodules, raising suspicion for a functional pulmonary carcinoid tumour. However, the 24-hour urinary 5-hydroxyindoleacetic acid level was normal, arguing against classical carcinoid syndrome. DOTA-TATE positron emission tomography showed mild to moderate uptake in the hilar lesion but no significant uptake in the pulmonary nodules. Bronchial washing cytology with immunohistochemistry established the diagnosis of SCLC, demonstrating positivity for CD56, synaptophysin, and thyroid transcription factor-1, with a Ki-67 proliferation index approaching 100%. The patient was treated with carboplatin, etoposide, atezolizumab, and supportive care, resulting in recovery from acute kidney injury. This case highlights that SCLC may present with chronic diarrhoea mimicking carcinoid syndrome and underscores the limitations of biochemical markers and functional imaging. Histopathological examination remains essential for establishing the correct diagnosis and guiding appropriate management.

Key report: Small cell lung carcinoma, lung neoplasms, neuroendocrine tumours, paraneoplastic syndromes, diarrhoea, carcinoid syndrome

INTRODUCTION

Lung neuroendocrine tumours (LNETs) are a family of heterogeneous malignancies showing neuroendocrine morphology and phenotype. [1] These tumours arise from bronchial neuroendocrine cells—specifically APUD (amine precursor uptake and decarboxylation) cells, also known as argentaffin cells. [2] In Australia, neuroendocrine neoplasms (NEN) accounted for 3.2% of cancers diagnosed as of 2020. Of the 4693 NEN cases diagnosed in 2020, 1824 were found to originate from the lungs. NENs were estimated to account for 14% of the lung cancer cases diagnosed in 2020. [3] Small cell lung carcinoma (SCLC) accounts for 15% of all primary lung malignancies, whereas lung Carcinoids account for 2%, and SCLC tends to occur in heavy smokers, whereas lung Carcinoids typically occur in patients of a younger age with less clear association with smoking habits. [1] Here we describe a case of SCLC mimicking Carcinoid syndrome with a presenting complaint of chronic diarrhoea.

CASE REPORT

A woman in her 70s presented to the emergency department with symptomatic hypotension, functional decline, and chronic diarrhoea. For 8 weeks she reported approximately 10 watery bowel movements daily. Over the preceding 2 weeks, she developed faecal urgency, incontinence, and per-rectal bleeding, accompanied by dizziness and general malaise.

One week before presentation, she had seen her general practitioner. Metformin was discontinued without improvement (hence ruling out drug-induced diarrhoea), and a course of antibiotics was prescribed with no effect. She denied recent travel, new medications, fever, weight loss, or other constitutional symptoms. Her medical history was notable for a 40 pack-year smoking history. On presentation, her blood pressure was 103/45 mmHg, heart rate 80 bpm, and oxygen saturation 97% on room air. Physical examination was largely unremarkable with a soft, non-tender abdomen and normal bowel sounds. During assessment, she developed hypoglycaemia, with a blood glucose nadir of 2.6 mmol/L. Management with intravenous normal saline and 5% glucose was used primarily for fluid and blood glucose management.

Investigations

Laboratory tests on presentation demonstrated leucocytosis with a white cell count of 13.4 × 10^9/L (N: 4.0–11.0) and neutrophilia of 10.3 × 10^9/L (N:2.0–8.0). C-reactive protein was elevated at 60 mg/L (N:0–10). Renal function showed acute kidney injury with an estimated glomerular filtration rate of 21 mL/min (N: >60), reduced from a baseline of 50 mL/min. Contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis performed at presentation revealed a 30 mm lesion in the right hilum with pulmonary nodules suspicious for bilateral upper lobe metastases (**Figure 1A, B**). We performed additional imaging for staging, including a CT brain with IV contrast, which showed a stable meningioma seen on previous CT brains, and a CT liver and adrenal glands, which did not detect any further lesions. Given the chronic diarrhoea and suspicion for neuroendocrine malignancy, serum chromogranin A was measured and was elevated at 588.7 µg/L (N <102). A 24-hour urinary 5-hydroxyindoleacetic acid (5-HIAA) level was 8 µmol/day (reference 10–40). The findings of this investigation provide compelling evidence against the diagnosis of classical carcinoid syndrome, even though the patient exhibited symptoms resembling those of carcinoid syndrome, such as chronic diarrhoea. However, her biochemical results do not support this differential diagnosis. A DOTA-TATE PET scan demonstrated mild to moderate tracer uptake in the right hilar and right lower paratracheal anterior mediastinal nodes, suggesting a neuroendocrine tumour. Multiple lobulated nodules in both lungs were non-DOTA-TATE avid (**Figure 2A**). The differences in avidity between the primary and metastatic nodules suggest possible heterogeneity, or the metastatic nodules had higher dedifferentiation leading to lower SSTR expression.

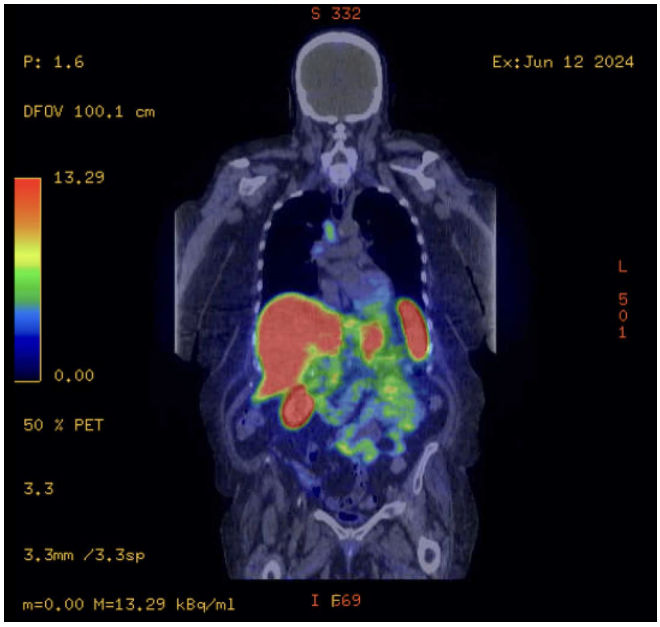

Figure 2: Right hilar DOTA-TATE demonstrating mild to moderate avidity of the right hilar and right lower paratracheal anterior mediastinal nodes, suggestive of a neuroendocrine tumour.

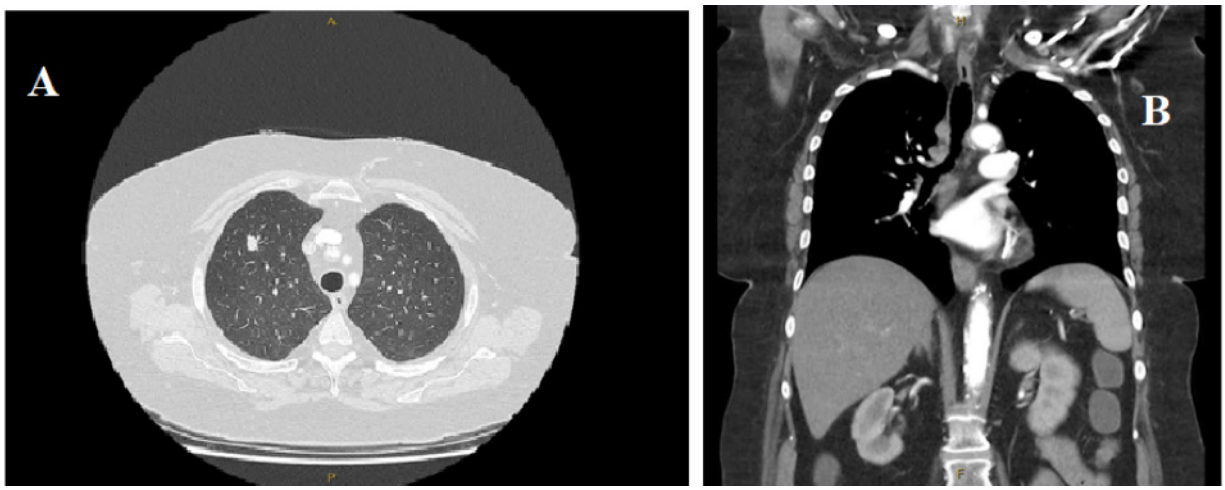

Figure 1: (A) 17 mm pulmonary nodular lesion in the anterolateral aspect of the right upper lobe. (B) Ovoid soft tissue lesion at the right hilum reflecting a markedly enlarged lymph node measuring 28 × 30 mm.

Bronchial washings revealed clusters of atypical cells with stippled chromatin, nuclear moulding, and absent nucleoli. Mitotic figures and pyknotic debris were present. Immunohistochemistry showed positivity for CD56, synaptophysin, and thyroid transcription factor-1 (TTF-1), while chromogranin staining was negative. The Ki-67 proliferation index approached 100%, consistent with SCLC (**Figure 3A, B**). Pancytokeratin staining was not performed. The immunophenotypic profile overall supported the diagnosis of poorly differentiated neuroendocrine carcinoma/small-cell neuroendocrine carcinoma.

The patient was subsequently started on a treatment regimen of carboplatin, etoposide, and atezolizumab, along with dexamethasone to prevent nausea. Her acute kidney injury has also resolved with aggressive fluid resuscitation, hence the impression of pre-renal AKI from severe diarrhoea. She remains under the care of the oncology team where she continues to receive chemotherapy. Given her significant comorbidities including ischaemic heart disease with stents, her age, compounded by the advanced disease stage, she is not a suitable candidate for aggressive treatment such as mediastinoscopy for consideration of surgical resection.

DISCUSSION

Neuroendocrine tumours arise from cells capable of producing neurotransmitters, neuromodulators, or peptide hormones. [3] LNETs are classified by the 2021 World Health Organization thoracic tumour classification into four main subtypes: typical Carcinoid, atypical Carcinoid, SCLC, and large cell neuroendocrine carcinoma (**Table 1**). [4]

Clinical manifestations of LNETs vary according to tumour location, size, and biological behaviour. Many patients present with respiratory symptoms such as cough, dyspnoea, haemoptysis, or recurrent infections. Peripheral lesions may be asymptomatic and discovered incidentally. Most LNETs are non-functioning; however, a minority produce ectopic hormones. Hormone secretion may lead to paraneoplastic syndromes such as ectopic ACTH production causing Cushing's syndrome (frequency of 1.6%–4.5%), antidiuretic hormone secretion causing Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH) (frequency of 7%–16%), or serotonin production leading to Carcinoid syndrome. [5,6]

Carcinoid syndrome classically presents with diarrhoea, flushing, and bronchospasm and is more commonly associated with gastrointestinal neuroendocrine tumours. In contrast, SCLC is a high-grade neuroendocrine carcinoma characterised by small cells with scant cytoplasm, finely granular chromatin, nuclear moulding, and a high mitotic rate with extensive necrosis. [7]

The tumour cells in this case had a Ki-67 index approaching 100%, hence in keeping with SCLC. Bronchial washings were used in this case as, unfortunately, there was difficulty in proceeding with a bronchoscopy due to labile renal function and COVID infection. Although cytology can confirm neuroendocrine differentiation, it is often insufficient for definitive classification of LNETs because architectural assessment is limited, and it may also provide suboptimal material for complete molecular testing; therefore, tissue histology with immunohistochemistry remains the preferred standard for final subtype assignment. [8] In addition, DOTA-TATE uptake in the nodal disease with absent uptake in the pulmonary nodules should be interpreted with caution, as somatostatin receptor expression varies with tumour grade and differentiation. Non–DOTA-avid metastases are compatible with high-grade neuroendocrine carcinoma, including

Table 1: Thoracic World Health Organization terminology and criteria for neuroendocrine neoplasms (2021). [4]

Terminology	Criteria: Mitotic counts per 2 mm^2
Typical carcinoid	<2
Atypical carcinoid	2–10 (or necrosis)
Small cell lung carcinoma and large cell lung carcinoma (neuroendocrine carcinoma)	>10

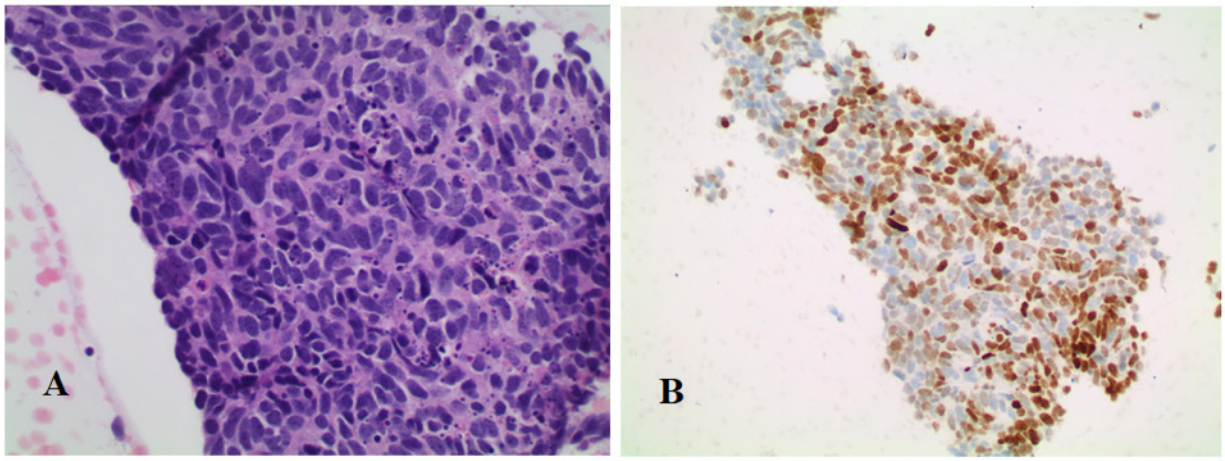

Figure 3: (A) Bronchial washing cytology demonstrating classic features of small cell lung carcinoma with nuclear moulding and finely granular chromatin. (B) Immunohistochemical staining showing Ki-67 proliferation index approaching 100%.

small cell lung cancer, which may demonstrate reduced somatostatin receptor expression. [9] However, DOTA-TATE PET should not be relied upon in isolation to subtype LNETs, and histopathology with immunohistochemistry remains essential for definitive classification. In this case, a low DOTA-TATE uptake in the nodules and high Ki-67 index point towards an advanced, poorly differentiated tumour, therefore increasing the likelihood of SCLC from Atypical Carcinoid.

Chromogranin A is produced by endocrine and neuroendocrine cells. The largest amount of Chromogranin A occurs in chromaffin granules of adrenal medulla and in the dense-core vesicles of sympathetic nerves. [10] Chromogranin A is also a nonspecific marker and may be falsely elevated in the setting of acute kidney injury. [11] In this case, reduced renal clearance likely contributed to the elevated Chromogranin A, limiting its specificity for neuroendocrine tumour burden.

The precise mechanism underlying the diarrhoea remains uncertain, given the 24-hour urinary 5-HIAA levels were within the normal range (hence arguing against classical serotonin-mediated Carcinoid syndrome). Possible explanations for diarrhoea in this case could include alternative mechanisms, such as secretion of other vasoactive peptides or inflammatory mediators, cytokine-driven secretory diarrhoea, or tumour-related malabsorption. Neuroendocrine tumours can occasionally present with carcinoid-like clinical manifestations despite normal urinary 5-HIAA levels, particularly when serotonin is not the dominant secretory product.

In SCLC, watery diarrhoea is sometimes associated with the ectopic secretion of Vasoactive Intestinal Peptide (VIP), sometimes called Verner-Morrison syndrome. There was one reported case of VIPoma syndrome simultaneously occurring with SCLC [12]. VIP was not tested in our case, but remains a possible explanation for diarrhoea in this case.

The mechanism underlying the patient's hypoglycaemia remained uncertain. While severe diarrhoea and poor nutritional intake may have contributed, objective investigations for malabsorption were not performed. Importantly, hypoglycaemia is not a typical feature of classical Carcinoid syndrome. Alternative mechanisms described in association with neuroendocrine and small-cell malignancies include non-islet cell tumour hypoglycaemia mediated by incompletely processed IGF-2 [13]. In this case, there was no radiological evidence of hepatic metastases to suggest impaired gluconeogenesis secondary to liver involvement.

CONCLUSIONS

In summary, this case represented an atypical Carcinoid-like paraneoplastic presentation of high-grade neuroendocrine carcinoma, characterised predominantly by severe diarrhoea in the absence of classical features such as flushing, bronchospasm, or elevated urinary 5-HIAA. Functional imaging supported the presence of a NEN but was insufficiently specific to distinguish low-grade Carcinoid tumour from small-cell neuroendocrine carcinoma. The case therefore highlights the diagnostic limitations of biochemical and functional imaging findings in isolation and underscores the importance of histopathological confirmation in atypical neuroendocrine presentations.

Learning Points

- Chronic diarrhoea may represent a paraneoplastic manifestation of lung neuroendocrine tumours.
- Pulmonary neuroendocrine tumours may present without typical respiratory symptoms.
- Histological examination remains essential for definitive diagnosis despite suggestive biochemical markers.
- High-grade neuroendocrine carcinomas (SCLC) can present with functional symptoms traditionally associated with low-to-intermediate grade carcinoids, potentially misleading initial clinical staging.

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CONSENT

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

AUTHORS' CONTRIBUTION

All authors have significantly contributed to the work, whether by following the case at the bedside, conducting literature searches, drafting, revising, or critically reviewing the article. They have given their final approval of the version to be published, have agreed with the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

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CONFLICT OF INTEREST

None.

REFERENCES

1. Trucco GV, Righi L, Volante M, Mauro P. Updates on lung neuroendocrine neoplasm classification. Histopathology. 2024;84(1):67–85. http://doi.org/10.1111/his.15058
2. Australian Institute of Health and Welfare. Cancer data in Australia: Neuroendocrine neoplasms incidence and survival rates and trends. Canberra: AIHW; 2024 [cited 2024 Dec 14]. Available from: https://www.aihw.gov.au/reports/cancer/cancer-data-in-australia/contents/cancer-data-commentaries/neuroendocrine-neoplasms-incidence-and-survival-ra
3. Wong MH, Chan DL, Lee A, Li BT, Lumba S, Clarke SJ, et al. Systematic review and meta-analysis on the role of chemotherapy in advanced and metastatic neuroendocrine tumor (NET). PLoS One. 2016;11(6):e0158140. http://doi.org/10.1371/journal.pone.0158140
4. Tsao MS, Nicholson AG, Maleszewski JJ, Marx A, Travis WD. Introduction to 2021 WHO classification of thoracic tumors. J Thorac Oncol. 2022;17(1):e1–e4. http://doi.org/10.1016/j.jtho.2021.09.017

5. Savu C, Melinte A, Diaconu C, Stiru O, Gherghicanu F, Tudorica SDO, et al. Lung neuroendocrine tumors: A systematicliteraturereview.ExpTherMed.2022;23(2):176. http://doi.org/10.3892/etm.2021.11099
6. Kanaji N, Watanabe N, Kita N, Bandoh S, Tadokoro A, Ishii T, et al. Paraneoplastic syndromes associated with lung cancer. World J Clin Oncol. 2014;5(3):197–223. http://doi.org/10.5306/wjco.v5.i3.197
7. Rekhtman N. Lung neuroendocrine neoplasms: Recent progress and persistent challenges. Mod Pathol. 2022;35(1):36–50. http://doi.org/10.1038/s41379-021-00943-2
8. Keihanian T, Diaz L, Plafsky L, Shergill U, Satiya J, Abraham R, et al. Diagnostic concordance of cytology and histology in samples obtained via endoscopic ultrasound-guided fine-needle biopsy (EUS-FNB). Cureus. 2021;13(6):e15596. http://doi.org/10.7759/cureus.15596
9. Tsuta K, Wistuba II, Moran CA. Differential expression of somatostatin receptors 1-5 in neuroendocrine carcinoma of the lung. Pathol Res Pract. 2012;208(8):470–474. http://doi.org/10.1016/j.prp.2012.05.014
10. Louthan O. Chromogranin A in physiology and oncology. Folia Biol (Praha). 2011;57(5):173–181. http://doi.org/10.14712/fb2011057050173
11. Tramonti G, Ferdeghini M, Annichiarico C, Norpoth M, Donadio C, Bianchi R, et al. Relationship between renal function and blood level of chromogranin A. Ren Fail. 2001;23(3-4):449–457. http://doi.org/10.1081/jdi-100104728
12. Noseda A, Fuss M, De Nutte N, Cogan E, Schmerber J, Corvilain J. Vipoma syndrome simultaneously occurring with small-cell carcinoma of the lung. Arch Intern Med. 1989;149(5):1223. http://doi.org/10.1001/archinte.1989.00390050167043
13. Karamanolis NN, Kounatidis D, Vallianou NG, Alexandropoulos K, Kovlakidi E, Kaparou P, et al. Paraneoplastic hypoglycemia: An overview for optimal clinical guidance. Metabol Open. 2024;23:100305. http://doi.org/10.1016/j.metop.2024.100305