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Acute Sensory Polyneuropathy Revealing Occult Small Cell Lung Carcinoma: A Case Report

R. Laamim[1*], A. Rafik, I. Achour[1], S. El Fathi[1], M. Beaouiss[1], H. Asri[1], A. Benali[1], A. Zegmout[1], H. Souhi[1], H. El ouazzani[1], I. A. Rhorfi[1]

[1]Department of Pneumology, Mohammed V Military Training Hospital, University Hospital Center of Rabat (CHU Rabat), Rabat, Morocco

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***Corresponding author:** R. Laamim
Department of Pneumology, Mohammed V Military Training Hospital, University Hospital Center of Rabat (CHU Rabat), Rabat, Morocco

Abstract **Case Report**

Paraneoplastic neurological syndromes are rare immune-mediated conditions that may exclusively reveal an otherwise asymptomatic malignancy, particularly small cell lung carcinoma. We report the case of a 64-year-old man with type 2 diabetes mellitus and a significant smoking history who presented with a two-week history of progressive diffuse paresthesias without motor deficit or systemic complaints, initially suggestive of diabetic neuropathy. Neurological examination revealed hyporeflexia without objective sensory loss. Laboratory investigations demonstrated an inflammatory syndrome with preserved glycemic control. Electrodiagnostic studies identified a pure sensory axonal polyneuropathy inconsistent with typical diabetic neuropathy. Given the rapid progression of symptoms and the patient's smoking history, chest imaging was performed and revealed a large right hilar mass with mediastinal lymphadenopathy. Bronchoscopic biopsies confirmed small cell lung carcinoma with a high proliferative index. The patient experienced sudden clinical deterioration and died before initiation of oncologic treatment. This case highlights that paraneoplastic neurological syndromes may represent the sole manifestation of an otherwise clinically silent malignancy and should be considered in rapidly progressive or unexplained neurological presentations.

Keywords: Paraneoplastic neurological syndrome; sensory neuropathy; small cell lung carcinoma; paraneoplastic neuropathy; occult malignancy; diabetic neuropathy differential diagnosis.

INTRODUCTION

Paraneoplastic neurological syndromes (PNS) are rare immune-mediated disorders occurring in the context of malignancy and independent of direct tumor invasion, metastasis, or treatment-related toxicity. Their estimated incidence ranges from 1 to 8 cases per 100,000 person-years and they affect fewer than 1% of cancer patients [1]. These syndromes are thought to result from an autoimmune response triggered by aberrant expression of neuronal antigens by tumor cells, leading to immune-mediated neuronal damage.

Small cell lung carcinoma (SCLC) is the malignancy most commonly associated with paraneoplastic syndromes, occurring in approximately 10–20% of cases [2].

Sensory neuropathy represents a well-recognized but uncommon paraneoplastic manifestation, frequently associated with anti-neuronal antibodies such as anti-Hu. Neurological symptoms may precede the diagnosis of the underlying malignancy and may occasionally constitute its initial or sole manifestation. The diagnostic process may be particularly challenging in patients with pre-existing conditions commonly associated with peripheral neuropathy, such as diabetes mellitus, potentially leading to diagnostic delay. Early recognition is therefore crucial, as prompt investigation may allow detection of an otherwise clinically silent tumor.

We report a case of acute pure sensory axonal neuropathy revealing an asymptomatic small cell lung carcinoma in a diabetic patient, emphasizing the need for systematic malignancy screening in cases of atypical or rapidly progressive neuropathy.

CASE PRESENTATION

A 64-year-old man with a history of hypertension, type 2 diabetes mellitus, and a 32 pack-year smoking history presented with progressive sensory complaints.

He reported a two-week history of progressive diffuse paresthesias involving all four limbs. Symptoms initially began in the distal extremities and gradually spread proximally to the elbows and knees, with associated perioral tingling. There was no history of balance disturbance, motor weakness, or neuropathic pain, and no other neurological symptoms were noted. No respiratory symptoms, constitutional features such as fever or weight loss, or other systemic complaints were present.

On examination, the patient was in good general condition, with an Eastern Cooperative Oncology Group (ECOG) performance status of 1. He was eupneic at rest, and prominent bilateral digital clubbing was noted, raising suspicion of an underlying pulmonary pathology. Gait was normal without instability or ataxia.

Neurological examination revealed no objective sensory loss on bedside testing or motor deficit. Deep tendon reflexes were preserved in the upper limbs; the right patellar reflex was decreased, while Achilles reflexes were absent bilaterally. Plantar responses were flexor. Coordination testing was normal, and cranial nerve examination was unremarkable.

Laboratory testing demonstrated a marked inflammatory syndrome, with microcytic hypochromic anemia (hemoglobin level of 12.3 g/dL) and an elevated C-reactive protein level of 86 mg/L. Renal, hepatic, metabolic, and muscle enzyme parameters were unremarkable, and glycated hemoglobin (HbA1c) was 6.3%. Screening for connective tissue diseases, endocrine disorders, and vitamin deficiencies yielded no abnormalities.

Electrodiagnostic studies revealed a length-dependent pure sensory axonal polyneuropathy, predominantly affecting the lower limbs, characterized by reduced sensory nerve action potential amplitudes without motor involvement or demyelinating features.

Given the acute progression of symptoms, associated hyporeflexia, and the patient's significant smoking history, a chest radiograph was performed, revealing a spiculated right hilar opacity. Subsequent chest computed tomography demonstrated a right hilar–basal mass measuring approximately 7 × 3.5 × 9 cm, associated with bulky bilateral mediastinal lymphadenopathy.

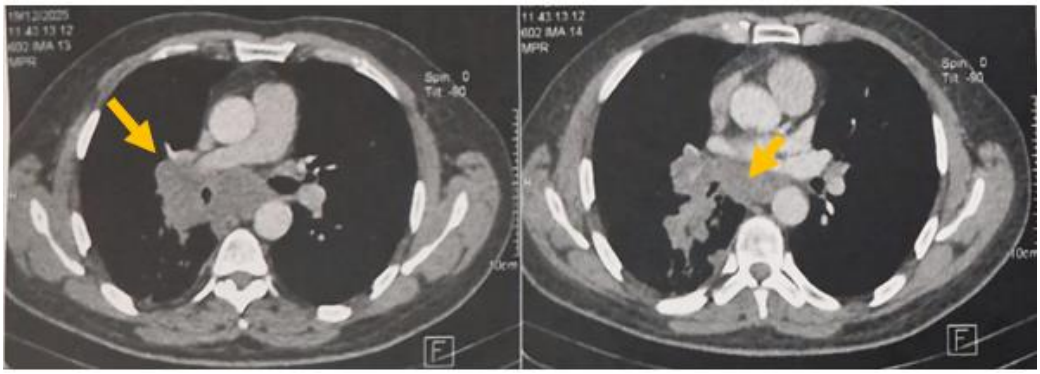

Figure 1: Chest CT demonstrating a right hilar–basal mass with bulky mediastinal lymphadenopathy

Flexible bronchoscopy revealed an endobronchial lesion originating from the basal segments of the right lower lobe, and bronchial biopsies were obtained.

Figure 2: Flexible bronchoscopy revealed an obstructive endobronchial polypoid lesion arising from the basal segments of the right lower lobe

Before histopathological confirmation and prior to initiation of oncological treatment, the patient experienced sudden clinical deterioration leading to unexpected death.

Histopathological analysis later confirmed small cell neuroendocrine carcinoma of the lung, with a Ki-67 proliferation index of approximately 99%.

The final diagnosis was acute pure sensory axonal neuropathy revealing clinically silent small cell lung carcinoma with a rapidly fatal course.

DISCUSSION

Peripheral neuropathy is an uncommon initial manifestation of lung cancer. Although paraneoplastic neurological syndromes occur in fewer than 1% of cancer patients, they are most frequently associated with small cell lung carcinoma (SCLC), a tumor characterized by neuroendocrine differentiation and marked immunogenicity [3,4].

Paraneoplastic neurological syndromes may precede, coincide with, or follow the diagnosis of the underlying malignancy. When neurological manifestations precede tumor detection, the interval is variable, most often ranging from weeks to several months, although longer delays have also been reported [5,6]. In our patient, this interval was limited to four weeks, suggesting an unusually rapid clinical course. The absence of respiratory or systemic symptoms further emphasizes the atypical and insidious nature of this presentation.

The pathophysiology of paraneoplastic neuropathy involves immune-mediated neuronal injury resulting from cross-reactivity between tumor antigens and neural tissues, often associated with onconeural antibodies such as anti-Hu in small cell lung carcinoma, leading predominantly to axonal degeneration and limited reversibility of neurological deficits [3,7]. This mechanism differs from diabetic neuropathy, which results from chronic metabolic and microvascular damage related to prolonged hyperglycemia [8]. However, both conditions may present with similar sensory symptoms and axonal electrophysiological patterns, potentially complicating the initial diagnosis. In our case, the abrupt onset and rapid progression of symptoms, together with relatively well-controlled diabetes (HbA1c 6.3%), argued against classical diabetic neuropathy and prompted further etiological investigation.

The diagnosis of paraneoplastic neurological syndromes remains largely clinical and relies on the combination of a compatible neurological phenotype, exclusion of alternative etiologies, and identification of an underlying malignancy or onconeural antibodies. According to current diagnostic criteria proposed by Graus et al., rapidly progressive neurological symptoms and the presence of a high-risk tumor such as small cell lung carcinoma strongly support a paraneoplastic origin even in the absence of detectable antibodies [9]. The presence of risk factors for malignancy, including significant smoking history, may further strengthen clinical suspicion and justify early oncological evaluation. In our case, the atypical clinical evolution, associated cancer risk factors, and exclusion of common causes prompted targeted oncological investigation, ultimately leading to diagnosis.

Management of paraneoplastic neuropathy primarily relies on treatment of the underlying malignancy. Previous studies have shown that oncologic therapy may stabilize neurological progression but rarely leads to complete recovery because neuronal damage is often irreversible, as emphasized by Darnell and Posner *et al.,*and Antoine *et al.,*[3,7]. Immunomodulatory treatments have demonstrated variable efficacy and remain secondary to tumor-directed therapy, as reported by Zis *et al.,*[10]

These observations emphasize the diagnostic complexity of paraneoplastic neuropathies, particularly when neurological symptoms occur in patients with alternative potential explanations or in the absence of systemic manifestations. Awareness of atypical clinical evolution and rapid symptom progression may help clinicians reconsider the diagnosis and initiate appropriate investigations for an underlying malignancy.

CONCLUSION

This report underscores that paraneoplastic neurological syndromes can precede and even exclusively reveal an otherwise occult malignancy. The occurrence of rapidly progressive or unexplained neurological symptoms should prompt consideration of a neoplastic etiology, even in the absence of systemic or organ-specific signs. Early recognition remains essential to avoid diagnostic delay and enable timely oncological management.

BIBLIOGRAPHIE

1. Binks, S., Uy, C., Honnorat, J., & Irani, S. R. (2022). Paraneoplastic neurological syndromes : A practical approach to diagnosis and management. *Practical Neurology, 22*(1), 19–31. https://doi.org/10.1136/practneurol-2021-003073
2. Hean, V., Camdessanché, J. P., Cathébras, P., & Killian, M. (2020). Paraneoplastic subacute sensory neuropathy with triple positive antineuronal antibodies associated with small-cell lung cancer. *BMJ Case Reports, 13*, e235668. https://doi.org/10.1136/bcr-2020-235668
3. Darnell, R. B., & Posner, J. B. (2003). Paraneoplastic syndromes involving the nervous system. *New England Journal of Medicine, 349*(16),

1543–1554. https://doi.org/10.1056/NEJMra023009

4. Maddison, P., & Newsom-Davis, J. (2008). Paraneoplastic neurological autoimmunity and small-cell lung carcinoma. *Journal of Neuroimmunology, 201–202*, 43–50.
5. Honnorat, J., Antoine, J. C., Derrington, E., Aguera, M., & Belin, M. F. (1996). Antibodies to a subpopulation of glial cells and a 66 kDas developmental protein in patients with paraneoplastic neurological syndromes. *Journal of Neurology, Neurosurgery & Psychiatry, 61*(3), 270–278. https://doi.org/10.1136/jnnp.61.3.270
6. Villagrán-García, M., Farina, A., Muñiz-Castrillo, S., Wucher, V., Dhairi, M., Timestit, N., *et al.,*(2023). Revisiting anti-Hu paraneoplastic autoimmunity : Phenotypic characterization and cancer diagnosis. *Brain Communications, 5*(5), fcad247. https://doi.org/10.1093/braincomms/fcad247
7. Antoine, J. C., & Camdessanché, J. P. (2017). Paraneoplastic neuropathies. *Current Opinion in Neurology, 30*(5), 513–520. https://doi.org/10.1097/WCO.0000000000000475
8. Feldman, E. L., Callaghan, B. C., Pop-Busui, R., *et al.,*(2019). Diabetic neuropathy. *Nature Reviews Disease Primers, 5*, 41. https://doi.org/10.1038/s41572-019-0092-1
9. Graus, F., Vogrig, A., Muñiz-Castrillo, S., Antoine, J. C. G., Desestret, V., Dubey, D., *et al.,*(2021). Updated diagnostic criteria for paraneoplastic neurologic syndromes. *Neurology : Neuroimmunology & Neuroinflammation, 8*(4), e1014. https://doi.org/10.1212/NXI.0000000000001014
10. Zis, P., & Hadjivassiliou, M. (2019). Treatment of paraneoplastic neuropathies. *Current Treatment Options in Neurology, 21*(8), 42.