

PERIPHERAL FACIAL PALSY IN SMALL CELL LUNG CANCER WITH MASTOID METASTASIS

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ABSTRACT

Introduction: Peripheral facial palsy is commonly associated with benign self-limited conditions. In rare circumstances, it may be the manifestation of underlying malignancy.

Case description: We present an unusual case of a 53-year-old woman with previously diagnosed small cell lung cancer (SCLC), who developed sudden-onset peripheral facial palsy. Imaging studies revealed mastoid metastasis with facial nerve involvement and venous sinus thrombosis, despite a normal MRI performed just one month prior. Despite ongoing treatment, the disease showed rapid progression culminating in the patient's death.

Discussion: Metastatic involvement of the mastoid is extremely rare in SCLC. Such metastases can occur via haematogenous or meningeal spread, resulting in complex clinical phenotypes with mastoid involvement, including facial palsy and venous sinus thrombosis among others.

Conclusion: This case emphasises the importance of individualised assessment in peripheral facial palsy and vigilance regarding unusual metastatic patterns in cancer patients. The rapid clinical deterioration despite appropriate treatment underscores both the aggressive nature of SCLC and the critical need for more effective treatment strategies.

KEYWORDS

Small cell lung cancer, mastoid metastasis, facial nerve palsy, venous sinus thrombosis

LEARNING POINTS

- Small cell lung cancer's neuroendocrine nature drives metastasis to unexpected territories, including the rarely affected mastoid region.
- In patients with peripheral facial palsy and malignancy risk factors, prompt imaging should be considered, even when other suspicious features are absent.
- Small cell lung cancer evolves rapidly in spite of treatment, requiring vigilance for metastatic complications despite reassuringly normal recent examinations.





INTRODUCTION

Peripheral facial palsy is a frequent neurological condition in clinical practice, with Bell's palsy being the most common aetiology. The diagnostic approach focuses on clinical presentation, with most guidelines recommending against routine diagnostic imaging^[1]. Small cell lung cancer (SCLC) is characterised by aggressive behaviour and early metastasis^[2]. Nevertheless, mastoid metastasis is extremely rare^[3,4]. Such metastatic involvement may present as peripheral facial palsy, hearing loss, vestibular symptoms or venous sinus thrombosis, depending on the involved structures. This case highlights the importance of considering unusual metastatic sites in cancer patients presenting with neurological symptoms, including isolated cranial neuropathies.

CASE DESCRIPTION

A 53-year-old woman presented to the emergency department with sudden-onset right facial asymmetry noticed upon awakening. She denied other symptoms such as otalgia, fever, pain, auditory or vestibular symptoms. A physical examination revealed right peripheral facial palsy (House-Brackmann grade V) without other neurological deficits.

Her medical history was significant for active smoking (39 pack-years), lung emphysema and recently diagnosed SCLC six months prior, when she presented with a cough and weight loss. Initial staging with thoraco-abdominal-pelvic (TAP) computed tomography (CT) showed a right upper lobe lung mass with mediastinal lymph node involvement and

multiple hepatic metastases (*Fig. 1*). A lung biopsy confirmed SCLC with a Ki-67 proliferation index of 85%. She started first-line chemotherapy with carboplatin and etoposide, later combined with atezolizumab^[2]. During the fourth cycle, she developed a hypersensitivity reaction to etoposide, requiring protocol adjustment.

Following the development of facial palsy, a cranial CT scan revealed a soft tissue density right mastoid lesion with bone erosion, with possible facial nerve and venous sinus involvement (*Fig. 2*). Thrombosis of ipsilateral sigmoid and lateral venous sinuses was confirmed and better characterised by MRI (*Fig. 3*). Surprisingly, she had a normal brain MRI result just one month prior to this presentation (*Fig. 4*).

The treatment plan was modified to include dexamethasone 8 mg/day, enoxaparin 1.5 mg/kg/day and palliative radiotherapy to the mastoid region. However, despite supportive care measures, the patient's condition deteriorated rapidly. She passed away two months after the facial palsy presentation and eight months after her initial cancer diagnosis, highlighting the aggressive nature of the disease and poor prognosis associated with SCLC.

DISCUSSION

Peripheral facial palsy is a frequent neurological condition, with idiopathic Bell's palsy being the most common cause. Secondary aetiologies include infections, trauma and rarely, neoplasms^[3,4]. SCLC represents an aggressive malignancy with early metastatic potential, typically to the brain, liver



Figure 1. CT TAP showing: A) Right upper lobe mass with contrast (arrowhead) and centrilobular emphysema (arrow); B) Necrotic adenopathy; C, D) Hepatic metastases.





Figure 3. Post-gadolinium T1 MRI showing abnormal enhancement and filling defects of right transverse and sigmoid sinuses (arrowheads), in (A, C, D) axial and (B) coronal



Figure 4. Normal mastoid and venous sinuses on post-gadolinium T1 MRI one month prior to presentation.

and adrenal glands. Mastoid metastases, especially those manifesting with facial palsy, remain extremely rare^[4]. Both haematogenous and meningeal spread have been described. Slow blood flow in the sinusoidal capillaries of the mastoid bone marrow creates an ideal environment for tumour cell deposition^[4]. SCLC's neuroendocrine nature, with its expression of neuronal markers, may confer an additional advantage, enhancing both survival in circulation and colonisation of the mastoid microenvironment^[5].

This case highlights the importance of individualised assessment in peripheral facial palsy, and the need for vigilance regarding unusual metastatic patterns in SCLC patients. The concomitant venous sinus thrombosis illustrates the complex pathophysiology of central nervous system metastasis. The rapid clinical deterioration, despite appropriate treatment, emphasises the disease's poor prognosis and underscores the critical need for more effective treatment strategies.

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