

Pontine metastases as a cause of dysphagia in lung carcinoma

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Dysphagia is an unusual symptom in the clinical course of lung carcinoma. When it appears, it is necessary to differentiate between regional dissemination, drug toxicity, opportunistic infection and, most rarely, metastatic dissemination to the brain stem. Magnetic resonance imaging (MRI) is the best diagnostic option to exclude this last possibility. We present a male patient with progressive dysphagia 15 months after the diagnosis of an oat-cell lung carcinoma. Cerebral MRI revealed a pontine lesion, probably of metastatic origin.

Key words: dysphagia, pontine metastases, magnetic resonance.

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INTRODUCTION

The appearance of dysphagia in the clinical evolution of lung cancer requires that other possible diagnosis be discarded, such as local or regional dissemination, toxicity secondary to the treatment received, development of opportunistic infections and metastases in the brain stem; rare findings described in this clinical condition. The frequency of dysphagia in lung carcinoma varies between 1 and 2% in initial clinical presentation and up to 6-7% in the full clinical picture¹.

CLINICAL CASE

Male patient of 60 years of age diagnosed in August 2001 as having small cell carcinoma (oat cell type) in the principal left bronchus. The disease was limited,

situated at least 2 cm from the carina, in contact with the oesophagus but without signs of infiltration. Associated were left hilar ganglia infiltrated into the dense mass. The liver and the supra-renal glands showed no signs of dissemination from the lesion. Among the clinical antecedents there appeared to have been acute non-Q myocardial infarction, and resection of the head of pancreas because of chronic pancreatitis in 1989. Chemotherapy was implemented and consisted of alternate three-weeks with carboplatin VP16 and cyclophosphamide-adriamycin-vincristine for three cycles, from August to December 2001. The result was minor radiological response. Radiotherapy treatment was deferred while awaiting an increased response. Three further cycles were administered together with cisplatin-VP16 every three weeks from January to March 2002. A further partial response was achieved following which the patient received loco-regional radiotherapy, 60 Gy on the tumour bed and mediastinum. Holocranial radiotherapy was deferred.

Two months after completing the treatment, the patient was admitted to hospital with a clinical picture of progressive dyspnoea of 1 month's duration and on slight or moderate effort, difficulty in swallowing and non-productive cough, difficulty in talking and with decrease in strength in the lower right extremity. Physical exploration showed a right hemiparesia, decrease in movement of the tongue and isolated rhonchus. In November 2002 a brain scan was performed using MRI (figs. 1 y 2). An intra-axial infra-tentorial mass was identified at the level of the inferior and right paramedial pons, with contra-lateral extension and into the medula oblongata, heterogeneous, infiltrative, with uneven uptake of intravenous contrast medium, annular, with central non-contrast medium necrotic zones suggestive of a metastatic origin. There was no evidence of other masses at the supra-tentorial level or signs of dissemination to the meninges. The assessment of disease extent was completed with a thoraco-abdominal CT scan which showed a residual lung lesion without adenopathy involvement and the presence of an important left lung pleural discharge, possibly of tumoural origin. Palliative holocranial radiotherapy was administered with symptom improvement, and support measures.

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Fig. 1. Cerebral MRI, axial T2FSE. Intra-axial pontine lesion, heterogeneous, hyper-intense on T2-weighted imaging, with necrotic centre.

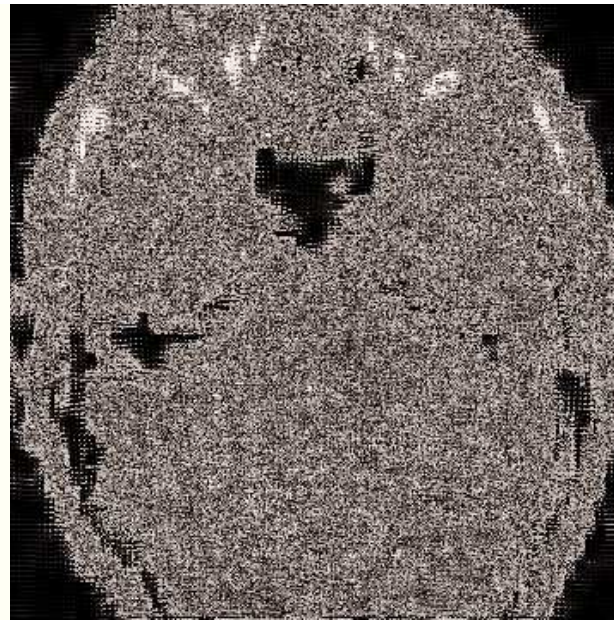


Fig. 2. Cerebral MRI, axial T1FSE post-gadolinium. Intra-axial pontine lesion, heterogeneous, infiltrative, with increased uptake of contrast material, peripheral, and necrotic non-enhanced centre of the lesion.

The clinical picture developed into a complete right hemiparesia, dysphagia towards solids at first and later liquids, and in a depressive state. Finally, he developed acute pulmonary oedema, with tachypnea and tachyrrhythmia and died on 23 December 2002.

DISCUSSION

Carcinoma of the lung usually manifests clinically with respiratory symptoms, with difficulty in swallowing in many cases, varying between 1%-2% in the initial presentation and between 6%-7% in the full clinical picture¹⁻².

Among the possible causes of dysphagia in lung cancer⁵ the following may be encountered:

- Mediastinum disease: the most frequent cause of dysphagia resulting from the direct invasion of the principal left bronchus or by peri-oesophageal or subcarinal adenopathies. As well, this can occur from the development of a secondary achalasia that interferes in oesophageal movement.
- Cervical adenopathies which occur between 15 and 20% of cases.
- Lesions of the brain stem, usually from involvement of the ambiguous nucleus, with development of bulbar paralysis. It is possible that this could be related to radiotherapy treatment received.
- Metastases in the gastrointestinal tract; 2%-10% of cases, the most frequent being the oesophagus.
- Associated systemic pathology, basically dermatomyositis.

- Developing of a second primary tumour.
- Infections of the oropharyngeal and oesophageal tracts, typically by *Candida*.
- Oesophageal toxicity induced by radiotherapy.
- Para-neoplastic hypercalcaemia⁴.

Lung cancer presents with metastases in the brain in about 30% of cases, typically supra-tentorial. Within the infra-tentorial site, the most frequent is in the cerebellum. Metastases in the brain stem are rare⁵.

Intra-parenchymatous metastases are the most common form of involvement. The most frequent primary tumours, in descending order, are lung, breast, melanoma, gastrointestinal tract, kidney, and unknown origin. Frequently, the brain is the only site of the metastases, especially common in lung cancer and melanoma⁶. Solitary metastasis is especially common in melanoma, lung and breast cancer. The primary malignant pulmonary tumour that manifests initially as brain metastases is adenocarcinoma in 76% of cases. This frequency is followed by small cell carcinoma at 20%, undifferentiated large cell carcinoma and squamous cell carcinomas at 2%⁷. The incidence of cerebral metastases is lower in patients with non-small cell lung cancer compared to those with small cell lung cancer⁸.

CT and MRI techniques identified a heterogeneous mass, infiltrative, necrotic, with peripheral uptake of contrast medium, annular, indistinguishable from a primary brain tumour, but with a known primary malignant tumour of the lung, the findings are very suggestive of metastatic origin^{6,9,10}.

The finding of a lesion in the brain stem in the clinical evolution of a pulmonary carcinoma is a complication that is observed very infrequently. The usual presentation is progressive dysphagia.

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