Metastasis from transitional cell carcinoma of urinary bladder as cystic pulmonary lesion

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ABSTRACT Pulmonary metastases from transitional cell carcinoma usually present as multiple nodules, solitary mass, or interstitial micronodules but rarely manifested as cystic pulmonary lesions. We report an atypical case of multiple cavitating pulmonary metastases from transitional cell carcinoma of urinary bladder. Infectious disease is ruled out by sputum examination, laboratory exams and failure of response to antibiotic therapy. In closure, the need to exclude a second primary tumor led us to perform a fine-needle aspiration biopsy by which metastatic transitional cell carcinoma was diagnosed. Inadequacy of blood supply with necrosis may be advocated as possible mechanism of cavitations of the lesion.

Key words: pulmonary metastasis; cavitary shadow; bladder cancer

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Introduction

Pulmonary metastases may present with unusual radiologic appearances; thus, it may be more difficult to distinguish these lesions from non-malignant pulmonary disease. Here, we report a case of multiple cavitated pulmonary metastases from transitional cell carcinoma of urinary bladder. Yet, the possible mechanism of cavity formation is discussed.

Case description

A 69 year-old man with complaints of productive sputum and haemoptysis was admitted to our unit. Two years before, he underwent a transurethral resection of a bladder tumor with the diagnosis of transitional cell carcinoma. A radical cystectomy and bilateral distal ureterectomy with creation of an ileal conduit were performed.

Subsequent follow-ups over 6 months were all negative until the most recent Computed tomography (CT) scan of the chest revealed bilateral cavitated lesions (Fig 1). The physical and laboratory examinations were negative. Systemic screening showed no other lesions.

Because cavitated nodules were considered unusual for metastatic transitional cell carcinoma, firstly we supposed an infectious disease. However, detection of acid-fast organisms,

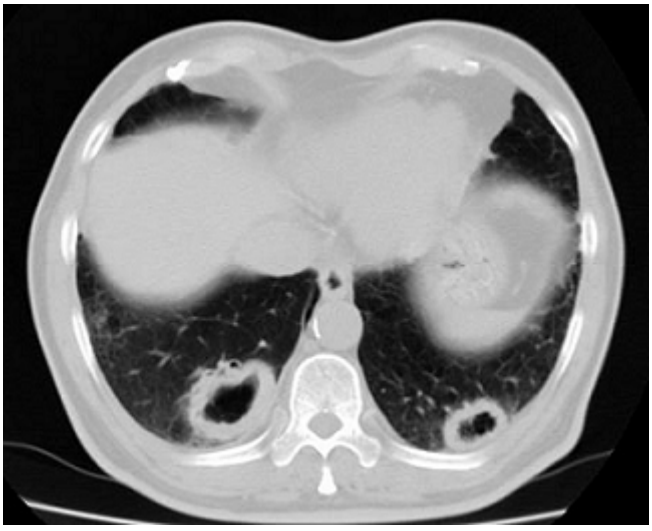

Fig 1. Computed tomography (CT) scans of the chest revealed bilateral cavities, measuring up to 5 cm in the long axis and from 2 to 5 mm in wall thickness. These cavities were distributed peripherically in the lower lobe and were well demarcated from the surrounding parenchyma of the lung. Only air is contained within the cavity spaces.

The authors do not have any conflict of interest.

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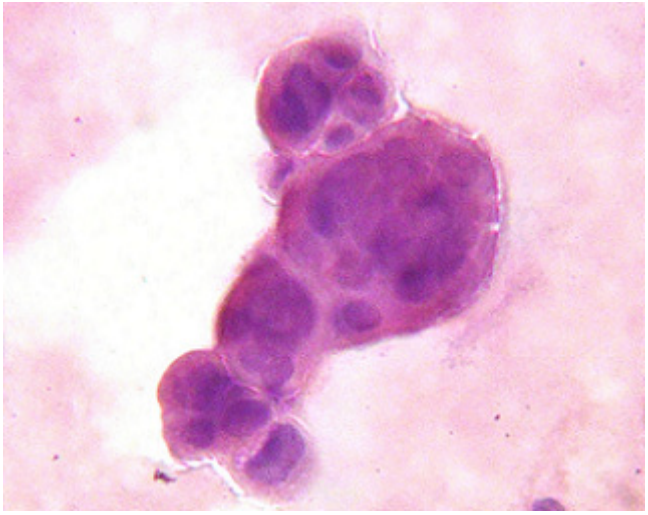

Fig 2. Microscopic findings of the lesion show malignant cells which resemble transitional cell carcinoma (HE original magnification x 400).

fungi or other pathogens was negative in sputum cultures. Repeated sputum cytology did not show the presence of neoplastic cells. Bronchoscopy revealed no abnormalities and material culture were sterile. Any response to antibiotic therapy was found. Yet, the patient had negative c-ANCA and negative p-ANCA, normal Water's graphic and normal ENT examination.

In closure, the need to exclude a second primary tumor led us to perform a fine-needle aspiration biopsy (FNAB) by which metastatic transitional cell carcinoma was diagnosed (Fig 2). The cytological findings closely resembled those of the previously resected bladder tumor with the presence of necrotic tissue. Therefore, the patient was referred to the oncology unit for systemic chemotherapy.

Discussion

The differential diagnosis of multiple pulmonary cavities is not extensive. More common causes include bacterial, fungal, parasitic infections and immunologic disorders such as Wegener's granulomatosis or rheumatoid necrobiotic nodules (1).

The frequency of cavitation tumors in lung were 2-5% and almost 80% were squamous cell carcinoma (2). Cystic pulmonary lesions caused by metastasis are rare (3). Dodd and Boyle, in a series of 574 cases of malignant tumors of the lung, reported that cavitation was encountered in only 4% of pulmonary metastases (4). Pulmonary metastases from transitional cell carcinoma usually present as multiple nodules, solitary mass, or interstitial micronodules but rarely manifested as cystic pulmonary lesions.

In our case, initially we supposed the lesions due to an infection rather than a metastatic disease but this hypothesis was then ruled out. Following admission numerous non invasive examinations were performed including sputum cytology, sputum culture for tuberculosis and common bacteria, antibodies against fungus, and intradermal Mantoux. All were negative. Additionally, Wegener's granulomatosis and Rheumatoid disease were excluded in the light of laboratory test and patient's medical history. Despite several studies report the useful of radiological exams in differentiating benign from malignant cavitated lesions (1,5), in our case the CT findings of the lesions including the wall width and the inner and outer margins did not suggest any differential diagnosis.

Thus, the need to exclude a neoplastic nature led us to perform a FNAB by which metastatic transitional cell carcinoma was diagnosed. At least three mechanisms have been proposed to explain cavity formation in metastatic pulmonary lesions (6): (A). Infiltration of malignant cells into the walls of a pre-existing benign pulmonary bulla. However it was excluded because no pre-existing bullae appeared during the 4-year follow-up period and our patient did not suffer of pneumothorax which may suspect an emphysematous disease. (B). Infiltration of malignant cells into the walls of air sacs formed by cystic distension of small airways through the ball-valve effect of the tumor. Haesegawa and collegues have reported a case of bilateral multiple thin-walled pulmonary due to a metastatic manifestation of sarcoma (6). The check-valve mechanism is confirmed by pathologic studies which show the presence of microscopic cavitary metastases. These lesions are considered to be an early stage in the development of macroscopic thin-walled cysts. Though, the walls of the cavities in this case are not thin but thick and the cystic change of air sacs, suggestive of a check-valve mechanism, can not be detected on cytological study. Thus, this mechanism is also unlikely. (C). Rapid tumor growth that exceeds the blood supply with central necrosis and resultant cavitation of the lesions may be advocated in our case. First, the lesions are large enough to indicate that inadequacy of blood supply was responsible for their necrosis and subsequent cavitation. Then, the cytological patterns show the evidence of necrosis and thrombosis in the metastatic foci.

In closure, some authors report that the tumor may produce degradative enzymes which play a role in cavitation of the neoplasm with digestion of the malignant tissue and its subsequent expectoration through the connected airways. However, the mechanism mentioned is rejected in the present case. First, our patient has metastatic cavities with thick walls while thin-walled cavity is the result of debris expectoration into the airway as reported by Baba et al. and by Kawabata et al (7). Second, during clinical course the repeated cytology of the sputum did not show the presence of necrotic substance or cancer cells which is the result of discharge of the necrotic material as reported by Dolgoff and Hansen (8).

In conclusion, the following case is described in order to call attention to metastatic disease in the differential diagnosis of pulmonary cavities. Although cavitation in pulmonary metastases is not as frequent as in primary tumors, cystic lesions in patients with a known gallbladder should be interpreted with particular caution. Early biopsy should be performed to confirm the metastatic nature of the cavitary lesion. It may prevent unnecessary medical treatment and probably result in improved survival due to early institution of management.

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