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CASE REPORT

Endobronchial pulmonary metastasis in malignant tenosynovial giant cell tumor: A rare cause of central airway obstruction

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ABSTRACT

Tenosynovial giant cell tumor (TGCT) is a rare mesenchymal neoplasm arising from the synovium, tendon sheath, or bursa. Although the localized and diffuse forms of TGCT are typically considered benign, malignant TGCT characterized by marked histologic atypia and increased mitotic activity may exhibit aggressive clinical behavior. Pulmonary metastasis from malignant TGCT is rare and, in previously reported cases, pulmonary involvement has predominantly been described as parenchymal nodules or pleural lesions. In this article, we describe a rare case of endobronchial pulmonary metastasis occurring approximately 72 months after the initial diagnosis of malignant TGCT originating from the tendon sheath of the knee.

Keywords: Endobronchial metastasis, malignant, orthopedic oncology, pulmonary metastasis, tenosynovial giant cell tumor.

Tenosynovial giant cell tumor (TGCT) is a rare mesenchymal neoplasm of the synovium, tendon sheath, or bursa.[1,2] Although the localized and diffuse types of TGCT are typically considered benign, malignant TGCT characterized by marked histologic atypia and increased mitotic activity may exhibit aggressive clinical behavior.[3]

Malignant TGCT is uncommon in orthopedic oncology practice, but may follow an aggressive course with unusual metastatic patterns during long-term follow-up, highlighting the need for multidisciplinary awareness. Pulmonary metastasis from malignant TGCT is rare and, in the limited cases reported in the literature, pulmonary involvement has predominantly been described as parenchymal nodules or pleural lesions.[4] Based on a targeted review of the literature, no cases of malignant TGCT presenting with endobronchial involvement, central airway obstruction, or airway disease requiring bronchoscopic diagnosis or intervention have been reported to date.

In this article, we describe a rare case of endobronchial pulmonary metastasis occurring approximately 72 months after the initial diagnosis of malignant TGCT originating from the tendon sheath of the knee.

CASE REPORT

A 51-year-old female patient with no known comorbidities was diagnosed with malignant TGCT originating from the tendon sheath of the left knee after evaluation for knee pain. She underwent surgical management alone, with complete resection and negative margins achieved according

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to orthopedic oncology principles; no adjuvant systemic therapy or radiotherapy was administered. On longitudinal follow-up, a lesion involving the patella was identified approximately two years after the primary surgical resection, followed by the development of right inguinal lymph node metastasis in the fourth year. Histopathological evaluation of the patellar lesion confirmed malignant transformation. Subsequent assessments of the primary tumor bed demonstrated no evidence of local recurrence.

Approximately six years after the initial diagnosis, the patient presented to an outside institution with progressive dyspnea and respiratory distress. With a presumptive diagnosis of acute central airway obstruction, an emergent tracheostomy was performed and mechanical ventilatory support was initiated. She was subsequently referred to our center for further evaluation of the airway obstruction and advanced management. Due to respiratory failure, the patient was admitted to the intensive care unit (ICU) at our center. Due to concomitant acute kidney injury (estimated glomerular filtration rate, 13 mL per min), contrast-enhanced imaging could not be performed. Non-contrast chest computed tomography (CT) revealed an endobronchial soft-tissue lesion involving the distal right main bronchus, along with diffuse bilateral pulmonary involvement (Figure 1). Initial management focused on medical stabilization in the context of infectious complications, and the patient received broad-spectrum antimicrobial therapy. After clinical stabilization, diagnostic and therapeutic rigid bronchoscopy was performed to determine the etiology of the endobronchial lesion and to assess airway patency. During the procedure, copious secretions and crusts were noted within the trachea

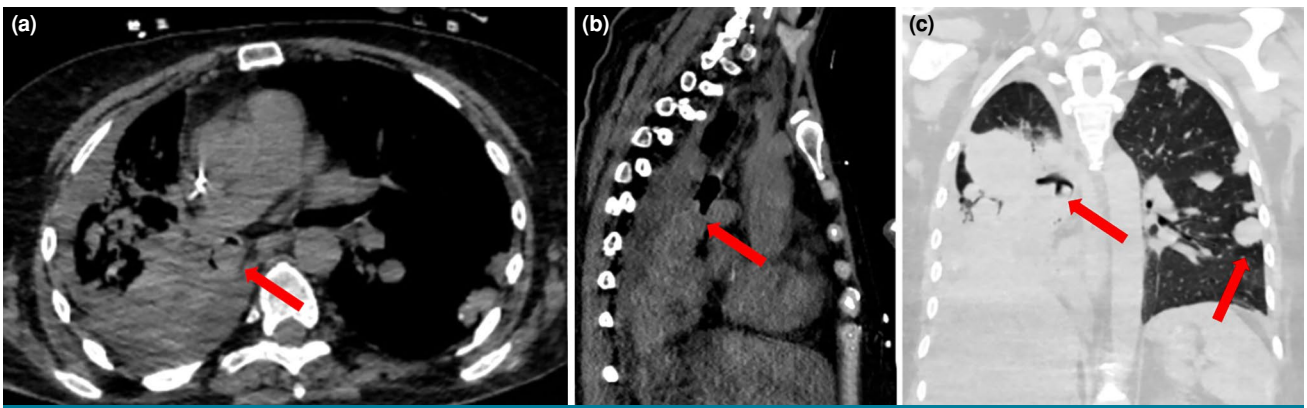

FIGURE 1. Chest CT demonstrating an intraluminal lesion causing significant airway obstruction. **(a)** Axial CT image reveals a hyperdense endobronchial mass occupying the bronchial lumen (red arrow). **(b)** Sagittal reconstruction shows the longitudinal extent of the lesion along the airway (red arrow). **(c)** Coronal image demonstrates both the obstructing endobronchial mass in the right bronchus and multiple parenchymal metastatic lesions in the contralateral lung (red arrows).
CT, computed tomography.

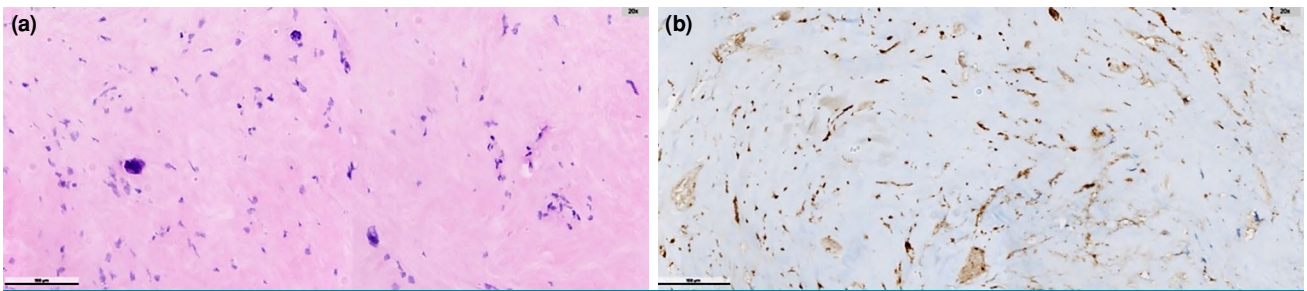

FIGURE 2. Histopathologic and immunohistochemical features of the endobronchial lesion. **(a)** Hematoxylin and eosin staining demonstrates infiltrative nodular tissue composed of markedly atypical cells embedded within extensive areas of hyalinization, consistent with malignant transformation (original magnification, ×200). **(b)** Immunohistochemical staining shows diffuse cytoplasmic expression of CD163 in the atypical tumor cell population, supporting histiocytic differentiation and confirming metastatic malignant tenosynovial giant cell tumor (original magnification, ×200).

and main airways and were removed by suctioning. The lumen of the right intermediate bronchus was found to be nearly completely obstructed by a polypoid, friable mass. Argon plasma coagulation (APC) was applied to the lesion, followed by mechanical resection, resulting in restoration of airway patency. On postprocedural surveillance bronchoscopy, the right intermediate bronchus remained patent; however, residual tumoral tissue was observed within the bronchus intermedius, along with narrowing at the entrances of the right lower lobe bronchi due to extrinsic compression.

Histopathological examination of biopsy specimens obtained by rigid bronchoscopy revealed nodular tissue composed of cells with marked cytologic atypia, accompanied by areas of hyalinization and necrosis. On immunohistochemical analysis, the tumor cells were positive for CD163 and CD68 and negative for cytokeratin, S100, and TTF-1. These histopathological and immunohistochemical findings confirmed the diagnosis of endobronchial pulmonary metastasis from the patient's known primary malignant TGCT (Figure 2).

During the subsequent course, the patient developed a pneumothorax with a prolonged air leak, which was managed with tube thoracostomy. Given the presence of advanced metastatic disease, diffuse bilateral pulmonary involvement, and a performance status of Eastern Cooperative Oncology Group (ECOG) 2-3, active systemic therapy was not considered appropriate by the medical oncology team. The patient was managed with supportive and palliative care in the ICU. Although further evaluation was planned, she elected to return to her home country before completing the diagnostic workup.

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

DISCUSSION

Tenosynovial giant cell tumor encompasses a heterogeneous group of synovial-based neoplasms with variable clinical behavior, ranging from indolent localized lesions to aggressive malignant forms with metastatic potential.[1,5] While localized TGCT is typically benign and treatable with surgical excision, diffuse and malignant forms exhibit aggressive behavior with high rates of local recurrence. In contrast to diffuse-type disease, malignant TGCT represents true sarcomatous transformation with an inherent potential for distant metastasis.[6] Pulmonary metastasis from malignant TGCT is exceedingly rare. In a recent comprehensive review by Shaik et al.,[4] a total of 35 cases with lung metastases reported between 1968 and 2024 were analyzed. The majority of these cases involved malignant TGCT (71%) or diffuse-type TGCT (23%), with pulmonary metastases predominantly manifesting as parenchymal nodules and/or pleural involvement. Notably, no endobronchial involvement was described in the cases included in the review through 2024, nor has a similar pattern of involvement been reported in the subsequently published literature.[4]

The available literature also highlights the diagnostic complexity of malignant TGCT. Earlier clinicopathological series have emphasized that malignant TGCT may show marked cytologic atypia, increased mitotic activity, necrosis, and infiltrative growth, and that immunohistochemical and clinicopathological correlation is essential for distinguishing it from other sarcomas and primary pulmonary malignancies.[7-9] Against this background, the present case differs from previously reported thoracic metastatic patterns by demonstrating an endobronchial mass causing clinically significant central airway obstruction. This distinction is important, since endobronchial involvement may present with acute respiratory compromise and may require urgent bronchoscopic intervention for both diagnosis and airway palliation. Therefore, in patients with a known history of TGCT, the development of acute or progressive respiratory symptoms should prompt consideration of rare metastatic patterns in addition to primary pulmonary malignancies.

In our case, rigid bronchoscopy was essential for diagnostic confirmation of endobronchial involvement as well as for palliative management of acute central airway obstruction. Debulking of the endobronchial mass using APC and mechanical resection restored airway patency and enabled histopathological confirmation of the diagnosis. The absence of published reports on bronchoscopic diagnosis or management of TGCT-related pulmonary metastases underscores the clinical significance of this case. Histopathological and immunohistochemical findings are essential for differentiating metastatic TGCT from primary bronchogenic carcinoma, with CD68 and CD163 positivity and cytokeratin and TTF-1 negativity supporting a nonepithelial origin.[7-9] Accordingly, in patients with a history of TGCT, the evaluation of endobronchial lesions should integrate

clinical history, radiological findings, and immunohistochemical profiling.

There is no established consensus regarding the optimal management of malignant TGCT, and the prognosis in advanced metastatic disease is usually poor.[1,10] Although surgical resection remains the mainstay of treatment, systemic therapies have demonstrated limited efficacy, particularly in the setting of extensive pulmonary metastases.[1,6] In the present case, widespread bilateral lung involvement and poor performance status precluded the use of systemic treatment options.

In conclusion, this case illustrates that malignant TGCT may give rise to delayed and unexpected distant metastases years after apparent local disease control. Endobronchial involvement causing central airway obstruction may occur and can be effectively managed with rigid bronchoscopy, highlighting the need for long-term orthopedic follow-up with multidisciplinary awareness.

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REFERENCES

1. Stacchiotti S, Dürr HR, Schaefer IM, Woertler K, Haas R, Trama A, et al. Best clinical management of Tenosynovial Giant Cell Tumour (TGCT): A consensus paper from the community of experts. Cancer Treat Rev 2023;112:102491. doi: 10.1016/j.ctrv.2022.102491.
2. Mastboom MJL, Palmerini E, Verspoor FGM, Rueten-Budde AJ, Stacchiotti S, Staals EL, et al. Surgical outcomes of patients with diffuse-type tenosynovial giant-cell tumours: An international, retrospective, cohort study. Lancet Oncol 2019;20:877-86. doi: 10.1016/S1470-2045(19)30100-7.
3. Sikaria S, Heim-Hall J, Diaz EH, Williams R, Sankhala K, Laabs B, et al. Partial response of a rare malignant metastatic diffuse tenosynovial giant cell tumor with benign histologic features, treated with SCH 717-454, an insulin growth factor receptor inhibitor, in combination with everolimus, an MTOR inhibitor. Target Oncol 2014;9:73-9. doi: 10.1007/s11523-013-0267-8.
4. Shaik AA, Panigrahi MK, Patro M, Sushmita V, Mishra P. Localized type tenosynovial giant cell tumor with metastases to lungs and pleura: A case report and literature review. J Med Case Rep 2024;18:452. doi: 10.1186/s13256-024-04768-w.
5. Bliss BO, Reed RJ. Large cell sarcomas of tendon sheath. Malignant giant cell tumors of tendon sheath. Am J Clin Pathol 1968;49:776-81. doi: 10.1093/ajcp/49.6.776.
6. Cassier PA, Gelderblom H, Stacchiotti S, Thomas D, Maki RG, Kroep JR, et al. Efficacy of imatinib mesylate for the treatment of locally advanced and/or metastatic tenosynovial giant cell tumor/pigmented villonodular synovitis. Cancer 2012;118:1649-55. doi: 10.1002/cncr.26409.
7. Bertoni F, Unni KK, Beabout JW, Sim FH. Malignant giant cell tumor of the tendon sheaths and joints (malignant pigmented villonodular synovitis). Am J Surg Pathol 1997;21:153-63. doi: 10.1097/00000478-199702000-00004.
8. Li CF, Wang JW, Huang WW, Hou CC, Chou SC, Eng HL, et al. Malignant diffuse-type tenosynovial giant cell tumors: A series of 7 cases comparing with 24 benign lesions with review of the literature. Am J Surg Pathol 2008;32:587-99. doi: 10.1097/PAS.0b013e318158428f.
9. Al-Ibraheemi A, Ahrens WA, Fritchie K, Dong J, Oliveira AM, Balzer B, et al. Malignant tenosynovial giant cell tumor: The true "Synovial Sarcoma?" a clinicopathologic, immunohistochemical, and molecular cytogenetic study of 10 cases, supporting origin from synoviocytes.. Mod Pathol 2019;32:242-51. doi: 10.1038/s41379-018-0129-0.
10. Atik E, Özgür T, Kalaci A, Tuna B. Tenosynovial giant cell tumor in an unusual localization. Acta Orthop Traumatol Turc 2013;47:139-41. doi: 10.3944/aott.2013.2773.