World Journal of Clinical Cases

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World J Clin Cases 2024 April 16; 12(11): 1857-1862

DOI: 10.12998/wjcc.v12.i11.1857

ISSN 2307-8960 (online)

EDITORIAL

Primary pulmonary meningioma and minute pulmonary meningothelial-like nodules: Rare pulmonary nodular lesions requiring more awareness in clinical practice

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Specialty type: Pathology

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's scientific quality classification

Grade A (Excellent): 0
Grade B (Very good): B
Grade C (Good): 0
Grade D (Fair): 0
Grade E (Poor): 0

P-Reviewer: Handra-Luca A, France

Received: November 28, 2023
Peer-review started: November 28, 2023
First decision: January 17, 2024
Revised: January 25, 2024
Accepted: March 21, 2024
Article in press: March 21, 2024
Published online: April 16, 2024

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Abstract

In this editorial, we comment on an article by Ruan *et al* published in a recent issue of the *World Journal of Clinical Case*. Pulmonary meningothelial proliferative lesions, including primary pulmonary meningiomas, minute pulmonary meningothelial-like nodules, and metastatic pulmonary meningiomas are rare pulmonary lesions. These lesions are difficult to differentiate from lung cancers based on clinical and imaging manifestations. Herein, we briefly introduce the clinical, imaging, and pathological characteristics of these lesions and discuss their pathogenesis to strengthen the current understanding of pulmonary meningothelial proliferative lesions in clinical diagnosis and therapy.

Key Words: Pulmonary meningothelial proliferation; Primary pulmonary meningioma; Minute pulmonary meningothelial-like nodule; Lung neoplasm; Rare pulmonary nodular lesion

Core Tip: Primary pulmonary meningiomas (PPM) and minute pulmonary meningothelial-like nodules (MPMN) are rare pulmonary lesions, which are difficult to differentiate from lung cancers due to similarities in clinical and imaging manifestations. To avoid misdiagnosis and overtreatment, PPM and MPMN should be considered in the differential diagnoses of pulmonary nodules of difficult clinical and/or imaging procedure diagnosis, particularly of asymptomatic patients. Differentiating PPM and MPMN from other pulmonary nodule diseases and assessing the malignancy of PPMs are difficult using only imaging-procedures, and histopathological examination thus remains the gold standard diagnostic procedure. Enhancing our understanding of MPMNs and PPMs and elucidating their pathogenesis will aid in the accurate differentiation of these lesions, to improve the clinical management of patients.

Citation: Liu LD, Zhang KX, Zhang HN, Zheng YW, Xu HT. Primary pulmonary meningioma and minute pulmonary meningothelial-like nodules: Rare pulmonary nodular lesions requiring more awareness in clinical practice. *World J Clin Cases* 2024; 12(11): 1857-1862
URL: https://www.wjgnet.com/2307-8960/full/v12/i11/1857.htm
DOI: https://dx.doi.org/10.12998/wjcc.v12.i11.1857

INTRODUCTION

Pulmonary meningothelial proliferative lesions, including primary pulmonary meningiomas (PPMs), minute pulmonary meningothelial-like nodules (MPMNs) and metastatic pulmonary meningiomas (MPMs), are rare pulmonary lesions[1]. PPMs and MPMNs are usually discovered incidentally during physical examination or lung surgeries performed for other indications. In recent years, pulmonary nodules have become more commonly, due to the popularization of regular physical examinations and the development of medical imaging technology, thus raising the attention of clinical physicians and pathologists. Consequently, the incidence of MPMNs and PPMs have also significantly increased. Additionally, MPM is very rare, and diagnosis depends on whether the patient has a medical history of intracranial or spinal meningioma. These lesions are difficult to differentiate from lung cancers based solely on clinical and imaging manifestations. Furthermore, due to differences in biological behavior and treatment methods, it is also necessary to differentiate between PPM, MPMN, and MPM. In a recent issue of the *World Journal of Clinical Cases*, Ruan *et al*[2] reported a rare case of MPMN and explained its histomorphology and immunohistochemical features. In this editorial, we review the recent developments in the clinical, imaging, and pathological characteristics of PPM and MPMN and discuss their pathogenesis, in order to strengthen the current clinical understanding of pulmonary meningothelial proliferative lesion diagnosis and treatment.

CLINICAL FEATURES OF PPM AND MPMN

Meningiomas are common tumors of the nervous system originating from meningothelial cells of the arachnoid layer, often occurring in the intracranial or spinal canal. Meningiomas occurring in tissues and organs not covered by the meninges are termed ectopic meningiomas. PPM is defined as a primary meningioma in the lung tissue[3] and is classified as a rare condition under the category of lung tumors of ectopic tissues according to the World Health Organization (WHO) Classification of Thoracic Tumors[4].

Patients with PPMs and MPMNs usually present with no obvious symptoms, other than some atypical respiratory symptoms, although this can depend on tumor size, location, and accompanying diseases[5-7]. PPMs are usually found through imaging examinations[5,8]. Patients with PPMs are mainly middle-aged or older women[1,7,9]. The vast majority of PPMs are benign, well-circumscribed and solitary, with a favorable prognosis[5,10-12]. Therefore, the major therapeutic method for PPM is surgery, such as wedge resection or lobectomy[8,10,13,14]. It has also been suggested that a conservative approach, using close medical monitoring, is the best option for patients when the location, size, and number of smaller nodules challenge the accessibility for biopsy or resection[13,15]. Malignant PPMs are prone to recurrence or metastasis, leading to a poor prognosis[3,16,17]. Conversely, some reports have indicated that the prognosis of patients with malignant PPM is actually favorable owing to a typically slow progression[6].

IMAGING FEATURES OF PPM AND MPMN

Radiologically, PPMs most commonly appear in the form of pulmonary nodules[5]. However, the visual features of PPM nodules are not universal, for example, they can appear either as a solitary nodule or in clusters[7,15]. The distribution of nodules is frequently subpleural (less than 1 cm away from the visceral pleura), whereas others are intrapulmonary (greater than 1 cm away from the visceral pleura), with these two presentations accounting for 89.1% and 10.9% of cases, respectively[18]. The edges of PPM nodules can also either be close to pulmonary blood vessels or visually away from the bronchi, blood vessels, or pleura[7,18]. There is usually no preferred location for PPM in the lung[19].

PPM nodules usually manifest as a round homogeneous mass with well-demarcated borders on imaging examinations [5,7,13,18]. PPMs can appear with ground-glass opacity and cyst-like structures or calcifications, depending on the changes in the lesion[7,18]. Small, early-stage PPMs can manifest as ground-glass like lesions, which are easily confused with early-stage lung cancers. As the lesions grow, it can become difficult to differentiate them from other benign lung tumors, such as pulmonary hamartoma, sclerosing pneumonocytoma, lipoma, and certain infections (such as cryptococcal infection or virus infection). It is also difficult to differentiate PPMs from aggressive growths or multiple advanced lung cancer nodules[10].

Approximately 74.0% of PPMs are less than 3 cm in diameter[7]. The size of benign PPM tumors ranges from 0.4 to 6 cm in diameter (median: 2 cm), whereas malignant PPM tumors range in size from 1.5 to 15 cm in diameter (median: 6.4 cm)[7]. Although the average diameter of malignant PPMs is larger than that of benign PPMs, it is impossible to diagnose them based solely on tumor size. In one PPM case, the tumor measured 9.5 cm × 8.4 cm × 5.3 cm in size, but pathological examination confirmed it was benign[3].

MPMNs are usually smaller than PPMs, ranging from 2.5 to 5.0 mm in diameter on CT scans, with an average of 3.04 ± 1.12 mm[18]. Therefore, MPMNs are difficult to discover during imaging examinations. In addition, some larger nodules may be misrepresented as PPM due to their increasing prevalence[13].

PET/CT has been proven to be more accurate than traditional CT in evaluating solitary pulmonary nodules. Low uptake of 18F-fluorodeoxyglucose (18F-FDG), defined as a maximum standardized uptake value (SUVmax) less than 1.5-2.0, is generally associated with a benign diagnosis[20], however, PPMs can present with various CT manifestations in enhanced imaging[6,7,21]. Therefore, the pattern of enhancement may not help determine whether the PPM is benign or malignant[7,22,23]. PPMs exhibit anywhere from mild to high metabolic activity, and the average value of SUVmax is 4.36, ranging from 0.6 to 12.9, although this factor has no relevance for malignant evaluation[3]. A recent study reported increased 18F-FDG uptake in synchronous benign and malignant PPMs[23]. In patients with a history of cancer, PPMs or MPMNs can be easily mistaken as lung metastases due to deceptive increases in 18F-FDG uptake[24,25]. As such, a single 18F-FDG PET or contrast-enhanced CT examination may not be sufficient to evaluate patients with PPM.

Overall, the imaging manifestations of PPMs are often heterogeneous between different patients. Although accurate diagnoses based on imaging results is of great significance for developing appropriate treatment plans, it is currently impossible to differentiate PPM from other pulmonary nodule diseases or to differentiate between benign and malignant PPM through radiographic observation alone[6,10]. Thus, pathological examination is the most accurate method of diagnosis of PPM[7,8,10].

PATHOLOGICAL FEATURES OF PPM AND MPMN HISTOLOGICAL FEATURES

PPM, MPMN, and MPM have similar morphological and immunohistochemical features[1]. PPMs share the same histomorphology and WHO grading with that of central nerve system (CNS) meningiomas[8,19]. The premise underlying the diagnosis of PPM is that no CNS lesions are found in imaging modalities and that MPM can be ruled out[26]. In histopathological analyses, PPMs are typically categorized as epithelial, fibrous or transitional[13]. Further, they often exhibit spindle cells in whirlpool arrangements, while psammoma bodies can also be observed[5,6,8,13,15,19]. Pseudonuclear inclusions have also been reported in PPM histopathology[5,8,13]. No mitoses or necrosis has been identified in benign PPMs[5,8]. Finally, high proliferative activity with several mitotic figures and a high Ki-67 index may be indicative of malignant PPM[23].

MPMNs are interstitial cellular proliferations first identified in 1960[27]. MPMNs can often appear with no visible abnormal lesions, or sometimes as pale, reddish gray or brown, and regular or irregular solid nodules, with moderate or soft texture, and unclear boundaries[28]. Like PPMs, MPMN can also present as a solitary or multiple nodules. In cases of MPMN which present in a diffuse, miliary like distribution in both lungs (up to tens, hundreds, or uncountable), it is defined as “diffuse pulmonary meningeal epithelial neoplasia”. Under the microscope, MPMNs exhibit elliptical nodules ranging in size from 1.0 to 5.0 mm. Most lesions tend to be subpleural, or located in the pulmonary interstitium, and distributed along the alveolar septa. Tumor cells are spindle shaped or epithelioid and arranged in a nest or vortex shape around small veins. The nuclei are ovoid with delicate chromatin and indistinct nucleoli with a rich and eosinophilic cytoplasm and an ambiguous cell boundary. Pseudoinclusion bodies can be seen in some nuclei, and mitotic figures are rare[1]. It is difficult to differentiate MPMN from PPM because of their morphologic similarity, with an exception to their size differences[29].

Even though histological diagnosis of PPM may not appear challenging according to morphological and immunostaining characteristics, misdiagnosis remains an issue. When PPM contains psammoma bodies, it can be misdiagnosed as metastatic papillary thyroid carcinoma[30]. In addition, approximately 90% of patients with MPMNs have synchronous lung cancer-related lesions, including atypical adenomatous hyperplasia, adenocarcinoma *in situ*, minimally invasive adenocarcinoma, and invasive adenocarcinoma[1].

IMMUNOHISTOCHEMICAL FEATURES

PPM and MPMN often overlap in terms of histological and immunohistochemical phenotypes, which are consistent with the manifestations of CNS meningioma[31]. Specific immunohistochemical staining is useful for diagnosing PPM and MPMN and excluding other types of lung tumors.

Positive immunohistochemical staining for vimentin, epithelial cell membrane antigen, and progesterone receptor (PR) is common in most cases of PPM and MPMN[5,6,8,13,18], whereas markers such as S100, cytokeratin (CK), and CD34 may exhibit variable or inconsistent staining results[1,8,15,19,26]. The expression of thyroid transcription factor-1 and neuroendocrine markers, such as synaptophysin and chromogranin, are negative[6,31]. Tao *et al*[32] previously reported that MPMNs showed strong and diffuse cytoplasmic expression of somatostatin receptor 2A (SSTR2A) and that the expression rate of SSTR2A was higher than that of conventional markers of meningioma.

PATHOGENESIS OF PPM AND MPMN

The incidence of ectopic meningioma is very rare (2%) and can be found in various anatomical sites such as the head and neck region, and specifically in the sinonasal tract, ear and temporal bone, and scalp[33]. However, meningiomas originating from the lungs are even more rare. Although the pathogenesis of PPM and MPMN remains unclear, it is generally believed that ectopic meningioma is derived from pluripotential subpleural mesenchymal cells or heterotopic embryonic rests of arachnoid cells[3]. Some scholars believe that PPMs originate from MPMNs based on their similar morphological, immunohistochemical and ultrastructural features[8,34]. Kraushaar *et al*[35] suggested that PPM can appear as a giant form of MPMN. Presentation of MPMNs as a single lesion lacking genetic alterations may indicate its origin of reactivity. MPMN-omatosis syndrome, defined as the diffuse distribution of MPMN, may represent the transition between reactive and neoplastic proliferations[31].

PPM and MPMN are more common in females. Further, as both PPM and MPMN cells express PR, the occurrence of these tumors may be related to PR activity. Moreover, PR develop from normal meningeal epithelial cells and the expression in meningiomas has been well confirmed, with results indicating the potential presence of lung meningeal epithelioid cells in normal lung tissues[2].

Patients with malignant lung tumors tend to have a higher incidence of MPMN[18,36]. Further, the microenvironment theory suggests that the external environment is the main contributing factor to the occurrence of MPMNs. Therefore, lung diseases may provide a localized microenvironment that promotes the occurrence and development of MPMN[36]. One case report of a patient with a *BRCA2* germline mutation exhibited quadruple neoplasms, including PPM. *BRCA2* mutations can increase tumor sensitivity in patients, but further research is needed to determine whether the *BRCA2* germline mutation could be related to PPMs[37].

CONCLUSION

The accurate diagnosis of PPM and MPMN is challenging due to their rarity. To avoid misdiagnosis and overtreatment in clinical practice, PPM and MPMN should be considered as differential diagnoses of pulmonary nodules of difficult clinical and/or imaging procedure diagnosis, in particular asymptomatic patients. Differentiating PPM and MPMN from other pulmonary nodule diseases and assessing the malignancy of PPMs are difficult using only imaging procedures. As such, histopathological examination remains the gold standard for diagnosing PPM and MPMN. Moreover, as most PPM and MPMN have synchronous lung cancer-related lesions, ground-glass-like changes which PPM and MPMN often exhibit in imaging examinations may cause the primary malignant lung tumor to exhibit metastatic malignancy, leading to delayed or canceled treatment for the primary malignant lung tumor[29]. Wedge resection is so far the best option for the diagnosis and treatment of these lesions, as no recurrence has been reported in benign cases after complete resection. As PPM is usually benign, conservative monitoring by biopsy is also a good treatment strategy, particularly if the locations of the lesions are inconvenient for surgical operation. In these cases, asymptomatic patients can undergo long-term follow-up observation instead. Enhancing our understanding of MPMN and PPM features and elucidating the pathogenesis underlying the development of these lesions could help to increase the clinical accuracy of PPM and MPMN differentiation, thus improving the clinical management of patients.

FOOTNOTES

Author contributions: Liu LD was responsible for investigation and writing the original draft; Zhang KX, Zhang HN, and Zheng YW were responsible for investigation; Xu HT was responsible for conceptualization, writing, reviewing and editing; all authors have read and approve the final manuscript.

Conflict-of-interest statement: Dr. Xu has nothing to disclose.

Country/Territory of origin: China

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S-Editor: Lin C
L-Editor: A
P-Editor: Yu HG

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