Review Article

Diffuse Pulmonary Meningotheliomatosis: A Literature Review of a Rare Diffuse Parenchymal Lung Disease with Unclear Clinical Significance

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Abstract

Introduction: Diffuse pulmonary meningotheliomatosis is a rare disease, with unclear clinical significance and very few reported cases in the literature. In this study, we review the demographics, presentation, imaging, diagnostic workup, and histologic findings of the 25 patients previously published in the literature with an outline of the disease history. **Materials and Methods:** We conducted a review of the literature through July 2016 for studies reporting cases of diffuse pulmonary meningotheliomatosis by searching multiple scholarly databases. **Results:** Of the 25 cases identified 2 were male (8%), and 23 were female (92%). Ages ranged from 37 to 73 with a median age of 59.5 years at diagnosis. 15 (60%) were asymptomatic and imaging abnormalities were discovered incidentally. 8 (32%) had unexplained respiratory complaints. 11 (44%) had history of or active malignancy. 3 (12%) were diagnosed by transbronchial biopsy while the remainder had surgical lung biopsies. **Conclusion:** Diffuse pulmonary meningotheliomatosis should be considered in all patients with diffuse bilateral pulmonary nodules on HRCT. The condition is more prevalent in females and its clinical significance is unclear, although nearly half of those diagnosed had a history of malignancy. CT imaging and surgical lung biopsy are the modalities of choice for diagnosis but transbronchial biopsies have recently been used obtain the diagnosis. Additional research needs to be done to further characterize the nature of this condition and the clinical scenarios in which is presents.

Keywords: *Diffuse parenchymal lung disease, interstitial lung disease, meningotheliomatosis, micronodules, miliary nodules, transbronchial biopsy*

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Introduction

Diffuse pulmonary meningotheliomatosis (DPM) is a diffuse parenchymal lung disease characterized by many disseminated bilateral minute pulmonary meningothelial-like nodules (MPMNs). These meningothelial-like nodules are classically encountered as benign solitary pulmonary nodules containing small epithelioid cell collections nestled within normal pulmonary parenchyma. Although MPMNs are not uncommon coincidental findings on surgical pathological specimens,[1-4] presentation with DPM is exceedingly rare, with very few reports of this syndrome in the published literature. Here, we review the demographics, presentation, imaging, diagnostic workup, and histologic findings.

Materials and Methods

We conducted a review of the literature during July 2016 for studies reporting cases of DPM by searching Medline/PubMed, SCOPUS, Web of Science, and Google Scholar. We used pre-specified Medical Subjects Heading (MeSH) terms (Medline/PubMed), Boolean logic, and custom search strings due to the evolving and variable nomenclature of this uncommon condition. Keywords included: diffuse pulmonary meningotheliomatosis, pulmonary meningotheliomatosis,

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Table 1: Summary of reports of DPM in the literature

Published cases of DPM

Reference (year)	Year	Age/sex (M/F)	Ethnicity	Radiologic abnormality	IHC markers	Biopsy type	Reason for imaging	Respiratory symptoms
Zak and Chabes[6]	1963	37/F	Black	NR	NR	NR	Pnuemonia and endocarditis	Pneumonia symptoms
		53/F	White	NR	NR	NR	Thymoma	NR
		64/F	White	NR	NR	NR	Ulcerative colitis with hemicolectomy complicated by pulmonary emboli (PE)	Dyspnea from PE
Park *et al.*[20],A	2002	56/F	NR	Bilateral nodules with lower lobe predominance	NR	VATS	Breast cancer	Asymptomatic
Ionescu *et al.*[10]	2004	NR/F	NR	>4 bilateral nodules	(−) Cytokeratin, synaptophysin, 94% s-100 (+) Vimentin 96.9%, EMA 33%	NR	Carcinoid	Asymptomatic
		NR/F	NR	>4 bilateral nodules	(−) Cytokeratin, synaptophysin (+) Vimentin	NR	Carcinoid	Asymptomatic
		NR/F	NR	>4 bilateral nodules	(−) Cytokeratin, synaptophysin (+) Vimentin	NR	Hypersensitivity pneumonitis	Asymptomatic
		NR/F	NR	>4 bilateral nodules	(−) Cytokeratin, synaptophysin (+) Vimentin	NR	Metastatic carcinoma	Asymptomatic
Suster and Moran[15]	2007	51/M	NR	Diffuse bilateral nodules	(−) Cytokeratin AE1/AE3, synaptophysin, CAM5.2 low molecular weight keratin, muscle specific actin, s-100 protein, CD34 (+) Vimentin, EMA	VATS	NR	Cough and dyspnea
		75/F	NR	Diffuse bilateral nodules	(−) Cytokeratin AE1/AE3, synaptophysin, CAM5.2 low molecular weight keratin, muscle specific actin, s-100 protein, CD34 (+) Vimentin, EMA	VATS	Colon carcinoma and mild restrictive ventilator defect	Dyspnea and fatigue
		54/F	NR	Diffuse bilateral nodules	(−) Cytokeratin AE1/AE3, synaptophysin, CAM5.2 low molecular weight keratin, muscle specific actin, s-100 protein, CD34 (+) Vimentin, EMA	VATS	Uterine leiomyosarcoma and restrictive ventilator defect	Spontaneous pneumothorax and dyspnea
		63/F	NR	Diffuse bilateral nodules	(−) Cytokeratin AE1/AE3, synaptophysin, CAM5.2 low molecular weight keratin, muscle	VATS	Coronary heart disease and mild restrictive ventilator defect	Cough and dyspnea

(*Continued*)

Table 1 (*Continued*)

Published cases of DPM								
Reference (year)	**Year**	**Age/sex (M/F)**	**Ethnicity**	**Radiologic abnormality**	**IHC markers**	**Biopsy type**	**Reason for imaging**	**Respiratory symptoms**
					specific actin, s-100 protein, CD34 (+) Vimentin, EMA			
		71/F	NR	Diffuse bilateral nodules	(−) Cytokeratin AE1/AE3, synaptophysin, CAM5.2 low molecular weight keratin, muscle specific actin, s-100 protein, CD34 (+) Vimentin, EMA	VATS	Thyroid, lung, breast carcinoma and mild restrictive ventilator defect	Dyspnea
Mukhopadhyay *et al.*[4]	2009	NR/F	NR	Innumerable bilateral small nodules	(−) Muscle markers and neuroendocrine markers (+) Vimentin, EMA, PR, CD56	SLB	Uterine cancer	Asymptomatic
Kraushaar *et al.*[1]	2010	54/F	NR	Diffuse bilateral nodules	(−) Synaptophysin, pankeratin, S-100, bombesin, CD34 (+) Vimentin, EMA, PR, CD56		Anemia workup with CT enterclysis	Asymptomatic
Sarabia *et al.*[21]	2010	66/F	NR	Diffuse bilateral nodules	(−) Synaptophysin, chromogranin, S-100 (+) Vimentin, EMA, p53, Ki-67 (<2%)	SLB	Breast, rectal cancer	Asymptomatic
An *et al.*[22]	2011	54/F	NR	Diffuse bilateral nodular infiltrates	(+) Vimentin, EMA, PR	VATS	Hepatocellular carcinoma	NR
Lee *et al.*[23]	2013	52/M	NR	Few small bilateral nodules, one 11 mm LLL nodule	(+) Vimentin, EMA, PR, cytokeratin, thyroid transcription factor-1, CD34	VATS	Persistent cough, smoker, erythrocytosis	Cough and dyspnea
Bernabeu Mora *et al.*[18]	2013	59/F	NR	Multiple disseminated minute nodules bilaterally	(+) EMA, vimentin, CD56, and PR (−) Cytokeratin AE1/AE3, chromograinin, synaptophysin, actin, desmin, and HMB-45	TBBX	Chronic diarrhea and uterine cancer s/p TAH-BSO and radiation	Asymptomatic
	2013	68/F	NR	Multiple bilateral	NR	SLB		Asymptomatic

(*Continued*)

Table 1 (*Continued*)

Published cases of DPM								
Reference (year)	**Year**	**Age/sex (M/F)**	**Ethnicity**	**Radiologic abnormality**	**IHC markers**	**Biopsy type**	**Reason for imaging**	**Respiratory symptoms**
Kamiya *et al.*[24]				1–3 mm ground glass opacities and 22-mm tumor with spiculation in the S10 segment of the left lung			Routine health checkup chest X-ray showed abnormalities; pulmonary cryptococcus	
Şen *et al.*[25]	2015	66/F	NR	Diffuse bilateral nodules	(−) Keratin, smooth muscle actin, chromogranin, CD34, and synaptophysin (+) EMA, vimentin, PR, and CD56	VATS	Mild restrictive lung disease	Dry cough
Huang *et al.*[26]	2015	57/F	NR	Diffuse bilateral pulmonary nodules	(−) Cytokeratin, smooth muscle actin, HMB-45, CD 31, S-100, CD34 (+) Vimentin, EMA, PR	SLB	Recent pulmonary embolism follow-up	Asymptomatic
Morresi-Hauf *et al.*[27],B	2015	73/F	NR	Bilateral disseminated military nodules	(−) Synaptophysin, chromogranin, S-100, AE1/AE3, HMB45 (+) Vimentin, EMA, CD56, PR	VATS	Abnormal chest X-ray	NR
		60/F	NR	Bilateral disseminated military nodules	(−) Synaptophysin, chromogranin, S-100, AE1/AE3 (+) Vimentin, EMA, CD56, PR	TBBX Cryo	Nodule follow-up imaging	Dry cough
Gleason *et al.*[19]	2016	63/F	White	Innumerable bilateral micronodules	(+) EMA, vimentin, PR	TBBX	Mild restrictive lung disease	Asymptomatic

M = male, F = female, NR = not reported, TBBX = transbronchial biopsy, VATS = video-assisted thoracoscopic surgery, SLB = surgical lung biopsy. [A] = Japanese text, [B] = German text.

pulmonary meningothelial-like nodules, minute pulmonary meningothelial-like nodules, pulmonary chemodectomatosis, minute pulmonary meningothelial-like nodule-omatasis syndrome, and MPMN-omatosis. We reviewed these studies for demographic and clinical data. Studies were excluded if they were duplicates or if they did not meet criteria for the diagnosis of DPM which we defined as multiple bilateral multilobar diffusely distributed nodules or opacities biopsy-confirmed to be pulmonary meningothelial-like nodules.

Results

Literature search of these terms resulted in the discovery of 29 published articles, of which 15 described cases of DPM. These 15 articles described 25 patients with diffuse bilateral nodules on computed tomography (CT) imaging, and biopsy confirmed pulmonary meningothelial-like nodules consistent with the diagnosis of DPM. Here we summarize the characteristics of these cases and provide a detailed review [Table 1].

Gender and age

Of the 25 patients, two were male (8%), and 23 were female (92%). One study did not report age, but the remaining patients ($N = 21$) were adults aged 37–73, with a median age of 59.5 years.

Presenting symptoms, imaging, and clinical findings

In 15 cases (60%), no respiratory complaints were described (12 asymptomatic and three none reported), and DPM was discovered only as an incidental finding on chest X-ray or chest CT imaging performed for another reason. Chest CT often showed diffuse bilateral ground glass nodules or micronodules. In some cases, these nodules were described as innumerable, miliary pattern, or occasionally having an apical basal gradient with apical predominance [Figures 1 and 2]. Two patients had respiratory complaints explained by concurrent conditions such as pulmonary embolism or pneumonia while the minority of patients (eight cases; 32%) had unexplained respiratory complaints consisting of primarily dyspnea and/or cough. History of or active malignancy including carcinomas of the breast, colon,

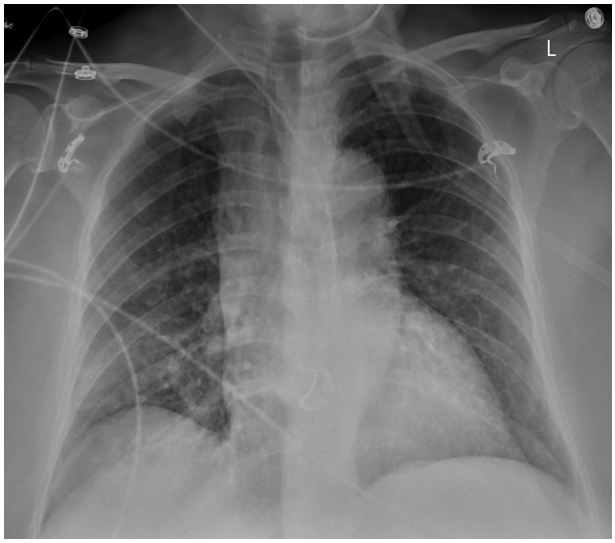

Figure 1: AP portable chest X-ray demonstrating bilateral and diffusely distributed nodular opacities seen in DPM

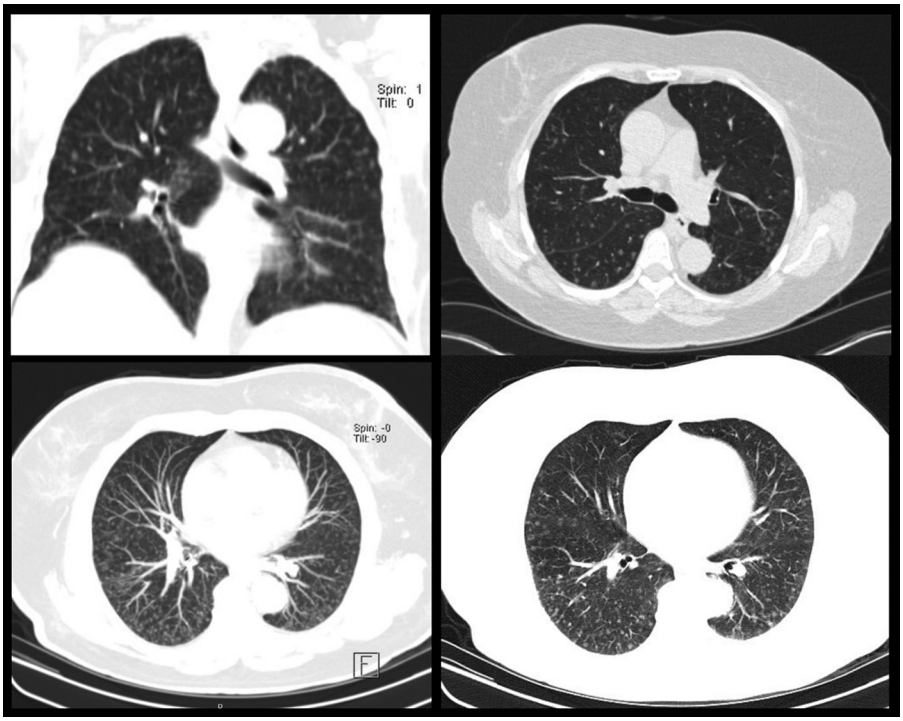

Figure 2: Multiple examples of CT chest imaging demonstrating bilateral randomly distributed nodules seen in DPM

liver, carcinoid tumors, and uterine leiomyosarcoma was documented in 11 (44%) of the patients. Other reported concurrent conditions included pulmonary embolism, pulmonary cryptococcus, hypersensitivity pneumonitis, ulcerative colitis, and infective endocarditis.

Method of diagnosis

At least 14 patients with DPM underwent surgical lung biopsy to obtain a definitive diagnosis. Ten (40%) of these were video-assisted thoracoscopic surgery (VATS) biopsies and the remaining four did not specify specific procedure details. In seven patients (28%), biopsy type was not recorded but was likely surgically obtained. Three patients (12%) were successfully diagnosed by bronchoscopic transbronchial biopsies.

Histological findings

By light microscopy, specimens of lung parenchyma obtained by either surgical biopsy or transbronchial biopsy reveal aggregates of large cells with abundant pale pink cytoplasm usually in a perivenular distribution [Figure 3]. The cells are uniform, oval or spindle, have slightly indented round to oval inconspicuous nuclei, and are separated by thin collagen fibers. Electronic microscopy shows cells with intercellular desmosomes and features reminiscent of meningioma cells. There is absence mitosis, neurosecretory granules, or zymogen granules. Immunohistochemical evaluation shows them to be positive for progesterone receptor [Figure 4], epithelial membrane antigen [Figure 5], and vimentin. They are typically negative for pankeratin, synaptophysin, chromogranin, S100, TTF-1, GATA3, estrogen receptor, AE1/AE3, muscle specific actin, CD34, HMB45, and CD68.

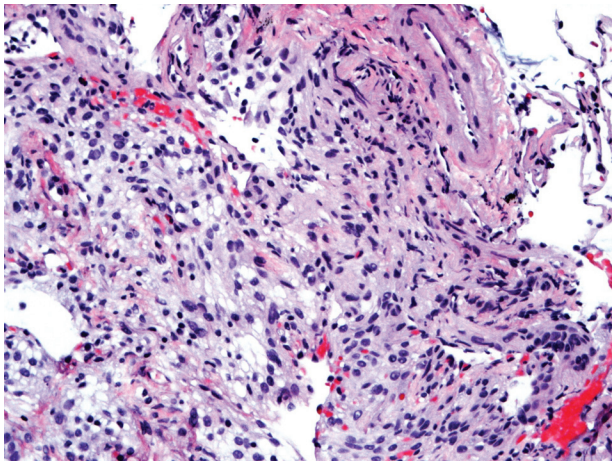

Figure 3: Meningothelial-like body located by vascular spaces in the lung parenchyma and composed of large cells with abundant pink cytoplasm. The nuclei contain inclusions (Hematoxylin and Eosin, 200×)

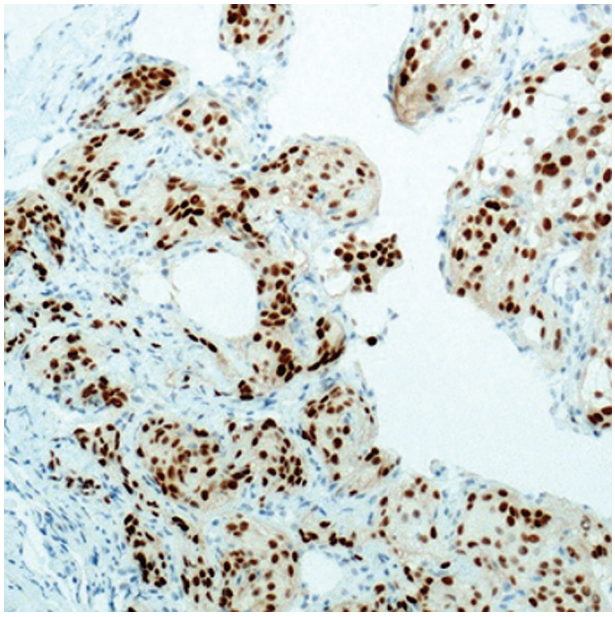

Figure 4: Lung parenchyma with meningothelial-like bodies demonstrating immunohistochemical positivity for progesterone receptor (100×)

Discussion

In 1960, Korn *et al.*[5] first described pulmonary meningothelial nodules as "pulmonary tumors resembling chemodectomas," [Figure 6] stemming from the belief that these nodules originated from oxygen-monitoring chemoreceptors due to geographic predominance near vascular structures. Three years later, the first cases of diffusely distributed or disseminated nodules of this type were described and this presentation was termed pulmonary chemodectomatosis.[6] These nodules were deemed a scientific curiosity discovered incidentally on surgical biopsies without known clinical significance.

Subsequently, the original theory of chemoreceptor origin was nullified as these structures were shown to resemble meningothelial cells on immunohistochemical and ultrastructural analysis.[2,7,8] It was Gaffey *et al.*[2] in 1988 who first coined these lesions as minute pulmonary meningothelial nodules (MPMNs) in response to an extensive immunohistochemical analysis verifying immunoreactivity for epithelial membrane antigen (EMA) and vimentin, two markers described in meningiomas (both intra and extracranial) and lack of reactivity to S-100 protein. These results strongly advocated a meningothelial origin for these structures and have been reproduced in multiple successive reports.[3,9]

Despite our heightened understanding of these nodules, the exact etiology of MPMNs remains elusive. Why some individuals present with diffuse bilateral disseminated nodules (termed DPM) is unknown. Whether this pattern on presentation suggests a different disease entity compared to its isolated counterpart remains unclear. Studies using clonal analysis had previously shown isolated nodules to demonstrate a lack of consistent monoclonal expansion,

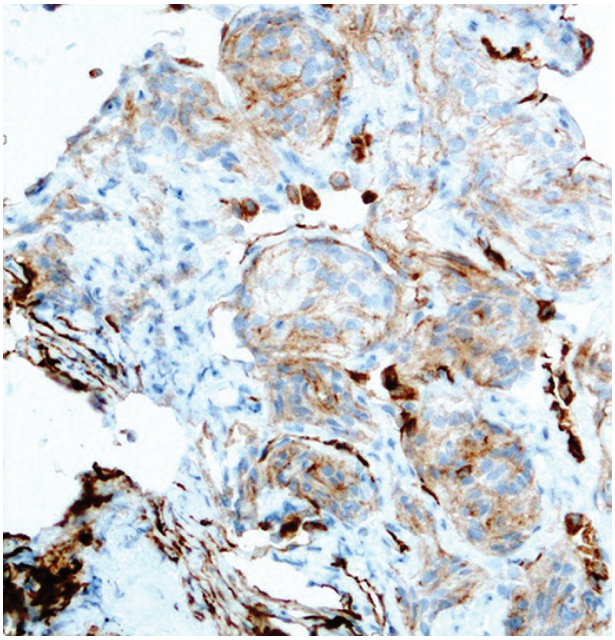

Figure 5: High magnification of the meningothelial-like bodies demonstrating immunohistochemical positivity for epithelial membrane antigen consistent with meningioma cells

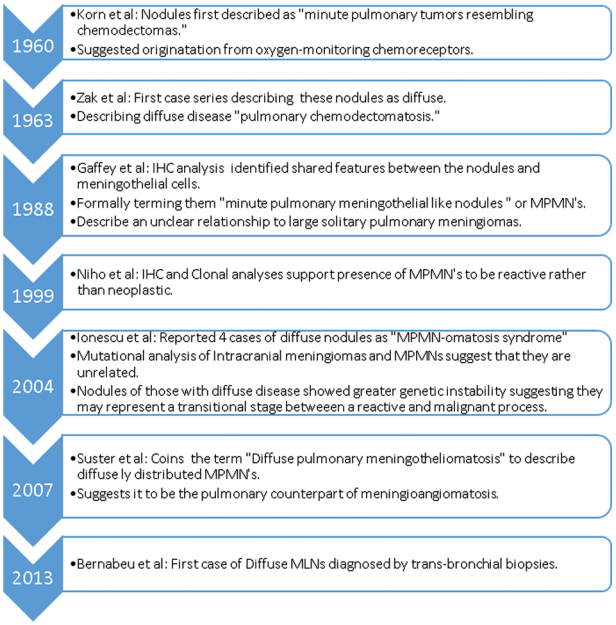

Figure 6: Timeline of DPM highlighting changes in nomenclature and understanding

suggestive for a reactive proliferation as opposed to a neoplastic process.[3] Later Ionescu *et al.* conducted a mutational analysis study comparing intracranial meningiomas to MPMNs which demonstrated that isolated MPMNs lack mutational damage (loss of heterozygosity alterations at differing loci), consistent with the prior conclusions of a reactive etiology. On the other hand, in the four patients with diffuse disease termed "MPMN-omatosis syndrome," Ionescu *et al.*[10] demonstrated a greater genetic instability compared to analysis of samples from those with non-diffuse or single MPMNs, which was hypothesized to represent the transition between a reactive and neoplastic proliferation. The characterization of MPMNs as a neoplastic predecessor has been challenged in the literature by authors observing that MPMNs are not frequently found in those with pulmonary meningioma[2,11,12] with only one reported exception.[13] Additionally, MPMNs have been described in patients with presumably unrelated conditions such as malignancy, pulmonary thromboemboli,[14] and infections, supporting a reactive etiology. Our review of cases only included the very rare diffuse subgroup of MPMNs but identified 44% of cases to have an active or historical malignancy.

In 2007, Suster and Moran[15] described five cases of MPMNs in a widespread bilateral distribution and drew parallels to meningioangiomatosis, a condition of the cerebral cortex and leptomeninges associated with neurofibromatosis type 2. Their observations prompted the current nomenclature "diffuse pulmonary meningotheliomatosis" to describe the rare condition we review [Figure 6].

DPM, just as isolated MPMNs, has a predilection for females (92%), with a median age of 59.5, consistent with prior reports of prevalence in the sixth decade of life. The majority of patients with DPM are asymptomatic at the time of diagnosis (72%), often with incidental findings on imaging done completed for a different purpose. DPM with symptoms of mild restrictive lung disease has been described and in these cases, CT imaging demonstrated findings consistent with interstitial pneumonia patterns, overlapping with fine linear, reticular, and mixed reticulonodular patterns.[15]

The majority of definitive diagnoses of DPM were made by surgical lung biopsy, many by VATS. While VATS has become an increasingly safer modality of biopsy, postprocedural complication rates remain as high as 5%, with persistent air leak being the most common.[16] Despite improvements in complications rates of VATS lung biopsy, transbronchial biopsy remains a safer modality, with a reported complication rate of less than 1%.[17] This is important to highlight as three cases (or 12%) in our review of DPM have been diagnosed by using transbronchial biopsy, the first by Bernabeu Mora *et al.* in 2013.[18] As the majority of patients were asymptomatic at the time of radiographic evidence of DPM, coupled with the uncertainty of clinical significance of this disease entity, it suggests that patients who are free of malignancy may be unnecessarily exposed to the potential risks associated with a more invasive modality of biopsy. This review reveals high rates of active or historical malignancy in those with DPM. Because transbronchial biopsy specimens are not usually adequate to rule out metastatic spread, a surgical lung biopsy may be a more appropriate first step in those with history of malignancy. Anecdotally, in patients with no history of

malignancy and low clinical suspicion for undiagnosed malignancy or normal Positron emission tomography/CT imaging[19] a transbronchial biopsy may be adequate.

Conclusion

DPM should be considered in patients with diffuse bilateral pulmonary nodules on high-resolution computed tomography. This rare condition of widespread MPMNs has unclear clinical significance. It is more commonly discovered in women, and many patients have a history of malignancy suggesting that a workup for undiagnosed malignancy is warranted in those whom DPM is discovered. It is typically diagnosed by surgical lung biopsy, but transbronchial biopsies have shown some diagnostic efficacy and may be an appropriate first step if there is a low index of suspicion for malignant primary or metastatic pulmonary lesions. Additional studies need to be done to better characterize the nature of this rare condition and the clinical scenarios, in which it presents.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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