Arteriovenous malformations of the lung: diagnosis by thin-section HRCT and Cine-MRI

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ABSTRACT: Arteriovenous malformations of the lung: diagnosis by thin-section HRCT and Cine-MRI. E. Yilmaz, A. Akkoclu, A. Gulsen, E. Osma.

We report a case in which a solitary large and multiple group of small pulmonary arteriovenous malformations were demonstrated by computed tomography and magnetic resonance images that could aid diagnosis and therapeutic planning without the need for catheter angiography. Monaldi Arch Chest Dis 2004; 61: 3, 180-182.

Keywords: Lung, arteriovenous malformation, hereditary hemorrhagic telangiectasia, computed tomography, magnetic resonance imaging.

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Introduction

Pulmonary Artery Venous Malformations (PAVMs) are rare and the result of abnormal communication between arteries and veins in pulmonary circulation [1]. Although PAVMs are most often congenital, they can be acquired due to trauma or conditions such as hepatic cirrhosis, mitral stenosis, schistosomiasis and actinomycosis. Symptoms related to PAVMs most commonly occur in middle age and vary widely from being totally absent to severe, cyanosis and congestive cardiac failure [2].

Pulmonary angiography is considered as the standard for assessment of PAVMs. However, Computed Tomography (CT) and Magnetic Resonance (MR) with recent developments in their techniques may be used to identify PAVMs [1, 3, 4]. We report a case of a 19-year-old boy with a solitary large and multiple small PAVMs. Specifically, thin-section high resolution CT and cine-MR with ultrafast gradient-echo sequence could be used to well identify small PAVMs associated with their feeding arteries and draining veins. These techniques, to our knowledge, have been initially reported for that aim and may be particularly useful means of non-invasively approving small PAVMs prior to management decision.

Case Report

A 19-year-old man was admitted to our hospital because of a solitary nodule on his routine chest radiograph. On his physical examination, dyspnea on exertion and mild clubbing of digits were noted. His medical history revealed recurrent episodes of epistaxis at least three times a month. There were no telangiectases of the skin or mucous membranes and no signs of chronic liver disease. Genetic consultation was obtained, and there were features suggestive of hereditary hemorrhagic telangiectasia (HHT) in his father, aunt and grandfather. Laboratory evaluations revealed a hemoglobin concentration of 17.6 g/dl and a white blood cell count of 7,500/mm³. Arterial oxygen saturation (SaO₂) while he was seated was greater than 90% in room air and 92% with 100% oxygen. Pulmonary function tests showed mild hyperinflation and airflow obstruction.

The large nodule on his chest radiograph was a single, 2x2.4 cm rounded opacity with smooth margins in the paracardiac left lung field. Contrast-enhanced CT scans (7-mm collimation, pitch of 1.0 and 50 mL of intravenous iodinated contrast material) showed a well-defined, brilliantly enhancing sac with apparently associated vessels in the lingular segment of the upper left lobe, finding consistent with PAVM (fig. 1). There was a single feeding artery arising from the lingular lobe branch of the left pulmonary artery and venous drainage was by a large vein into the left-superior pulmonary vein. In addition to the solitary large PAVM, CT scans obtained at the lung bases revealed multiple, small nodules of increased attenuation suspecting microPAVMs at the periphery of both lower lobes. To confirm the small lesions, thin-section high-resolution CT (1.3-mm collimation, pitch of 1.0 and overlap of 0.6 mm) was performed and disclosed numerous small PAVMs interconnecting via one or more small feeding arteriovenous communications.
Discussion

Pulmonary arteriovenous malformations are rare and the vast majority are associated with HHT. Although many patients with PAVM remain asymptomatic, a solitary PAVM larger than 2 cm or multiple pulmonary lesions may cause dyspnea, cyanosis or congestive heart failure, and even death in early life. HHT is a systemic fibrovascular displasia with autosomal dominant transmissions that affect arteries of the brain, lung, nose and gastrointestinal system characterised by telangiectasias, arteriovenous malformations, and aneurysms in affected organs [3, 4, 5].

Limited studies have been reported in which the sensitivity of imaging PAVMs have been assessed. Recently, Remy et al [3] evaluated PAVMs with CT of the chest and conventional angiography, both of which were compared to each other by means of detection of PAVMs. Thirty-eight of PAVMs were identified with CT only, a result that indicates the superiority of the CT compared with the angiography. However, in the same series, the anatomic structure of PAVMs was diagnosed with CT in 26% of patients and with pulmonary angiography in 60% of patients. Remy et al [3] suggested that the analysis of the anatomic structure of PAVMs by CT was considered complicated because the arteries that supply and the veins that drain the PAVMs have an undulating course that is often outside the plane of the CT projection. In our

ies and tortuous branches of veins, most of which were peripherally located (fig. 2). Three-dimensional (3D) gadolinium-enhanced MR angiography images (repetition time (TR)/echo time (TE)=5.1/1.44 msec, flip angle=40°, scan duration=22.7 sec) obtained on a 1.5-T superconducting magnet with a synergy body coil in the coronal plane showed a large PAVM with a single feeder originating from the left pulmonary artery and a large vein draining by pulmonary venous circulation (fig. 3). Ultrafast MR imaging was carried out with a commercially available ultrafast gradient-echo sequence; called turbo field-echo; during a breath hold, balanced-weighted images of the thorax in four separate axial planes. The optimal ultrafast gradient-echo sequence included a TR of 2.1 msec, a TE of 1.06 msec, a flip angle of 50°, a matrix size of 96x128, a slice thickness of 10 mm and a turbo factor of 49. A hundred phase image was obtained with typical dynamic duration of 0.118 msec per image and the total scan time of 11.8 sec. Fast images at cine-MR mode demonstrated apparently small PAVMs with the circulation of the feeding and draining vasculatures (fig. 4).

The final clinical diagnosis was PAVMs in HHT, also known as Rendu-Osler-Weber disease. Before the CT and MR scans, the patient had not been known to have HHT. Although catheter angiography for embolization of the large PAVM was planned, the patient rejected any form of therapy and he was discharged from the hospital.
case, the angioarchitecture of the small PAVMs could have been apparently demonstrated using an overlapping thin-section high-resolution CT images.

On the other hand, MRI is inherently sensitive to the detection of flow, and applications dealing with the non-invasive assessment of vascular structures are increasing. The ability to acquire not only morphological but also functional information non-invasively is an important advantage. Early studies in which the role of MR imaging was evaluated for vascular structures in the thorax revealed contrast-enhanced MR angiography as the best technique of choice [6, 7]. Eight patients suspected of having PAVM were recently examined by Maki et al [4] with 3D MR angiography. According to this study, small PAVMs (<5-mm), particularly if they are peripherally located, may be difficult to identify using such a technique due to cardiac and respiratory motions and the susceptibility artifacts related to aerated lung parenchyma. The introduction of faster MR scan times, enabling images to be obtained within a single breath-hold, alleviated some of these problems. In the presented case, we used the ultrafast gradient-echo sequence with a single-slice and multiphase breath holding technique at full expiration, it seems to be the most useful pulse sequence to demonstrate small vascular lesions in this area. We also applied a thin slab and short TE to optimize signal from flowing arterial and venous blood.

Percutaneous transcatheter embolotherapy with coils and detachable balloons is the main treatment of choice in congenital and acquired PAVMs, especially successful in patients with a solitary large PAVM and the risks of which are considered to be acceptable [2, 8]. A PAVM can contain a variety number of feeding and draining vessels, which originate either from the pulmonary or systemic circulation. Knowledge of the size and number of feeding arteries is helpful in preembolization planning. If the feeding artery is greater than 3 mm in diameter, diagnostic angiography and embolization can be performed [4]. In our case, a large PAVM had single feeding artery originating from the left pulmonary artery and a large vein draining into the left inferior pulmonary vein, as termed a classical type of PAVM. Both 3D-MR angiography and conventional enhanced CT allowed the identification of not only the lesion but also the size of a supplying artery in the patient's large PAVM. Surgical resection may be contemplated like a definitive treatment in patients where PAVMs are progressive and multiple. Besides, a conservative approach is probably justified only for diffuse PAVMs and lung transplantation may be an alternative for such cases [1, 5], despite the occurrence of extremely slow growth of the small and untreated PAVMs. Therefore, once the PAVMs have been diagnosed, they need periodical follow-up [2, 3].

We conclude that both thin-section high resolution CT and ultrafast cine-MR examinations may be useful for detection of small PAVMs. Additionally, these techniques also provide demonstration of angioarchitecture of small PAVMs as well as follow-up of untreated patients, thus eliminating the necessity for catheter angiography when treatment is not considered or the patient is not willing to accept any invasive approach.

References

1. White RI. Pulmonary arteriovenous malformations: how do we diagnose them and why is it important to do so? Radiology 182: 633-635: 1992.
2. Tong AK, La Hei ER, Currie B. Congenital pulmonary arteriovenous malformations. Pediatr Surg Int 18: 168-170: 2002.
3. Remy J, Remy-Jardin M, Wattinne L, Deffontaines C. Pulmonary arteriovenous malformations: evaluation with CT of the chest before and after treatment. Radiology 182: 809-816: 1992.
4. Maki DD, Siegelman ES, Roberts DA, Baum RA, Gefter WB. Pulmonary arteriovenous malformations: three-dimensional gadolinium-enhanced MR angiography-initial experience. Radiology 219: 243-246: 2001.
5. Suchin CR, Whitman GJ, Chew FS. Pulmonary arteriovenous malformation. AJR 167: 648: 1996.
6. Bertheneze Y, Howarth NR, Revel D. Pulmonary arteriovenous fistula: detection with magnetic resonance angiography. Eur Radiol 8: 1403-1404: 1998.
7. Burke CM, Safai C, Nelson DP, Raffin TA. Pulmonary arteriovenous malformations: a critical update. Am Rev Respir Dis 134: 334-339: 1986.
8. Coley SC, Jackson JE. Venous sac embolization of pulmonary arteriovenous malformations in two patients. AJR 167: 452-454: 1996.