Magnetic Resonance Imaging in the Diagnosis of Pulmonary Infarction*

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We report for the first time, to our knowledge, MRI features which could differentiate noninvasively pulmonary infarction from pneumonia. Three subjects with angiographically proven pulmonary infarction showed high T1 weighted MRI signals located in the embolic territory. Three patients with pneumonia and one patient with emboli, but without infarction, did not have these T1 weighted images.

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Differential diagnosis between pulmonary infarction and pneumonia is a frequent problem in general practice. Pulmonary infarction occurs in about 15 percent of pulmonary emboli. Its definition is radiologic or anatomic.¹ In clinical situations, only the radiologic definition is helpful: a parenchymal opacity lies in the territory of the thromboembolism and is characterized by its grossly triangular shape with a pleural base and an apex pointing to the hilum. Such shadows are segmental rather than lobar in distribution. Pleural effusion is common. Anatomically, these radiologic findings are associated with alveolar hemorrhage, with or without parenchymal necrosis.

The presence of alveolar hemorrhage in cases of pulmonary infarction prompted us to test the diagnostic value of MRI in this pathologic abnormality. Acute or evolving pulmonary hemorrhage produces different views on MRI.² Isointense T1 weighted images with hyperintense T2 weighted images represent early or acute hemorrhage. High T1 signals are caused by aging hemorrhage, because of methemoglobin formation.³ We hypothesized that the presence of alveolar hemorrhage in cases of pulmonary infarction could produce such a hyperintense T1 weighted signal. We report herein the results of MRI in seven patients with clinically suspected pulmonary thromboembolism.

MATERIALS AND METHODS

Seven patients (four men and three women; mean age, 47 ± 9 years) presented with a history or clinical signs (or both) compatible with pulmonary thromboembolism. Six patients had alveolar opacities on their standard chest roentgenograms. One had a normal chest x-ray film. In these seven patients, we performed the following investigations: phlebography; pulmonary angiography; and thoracic MRI without contrast material, with T1 and T2 weighted images on frontal and sagittal sections. The MRI was performed with a 0.5 tesla superconductive system. (Magniscan; General Electrics) Spin echo sequences were obtained with constant electrocardiographic triggering for T1 weighted (500-750/29-30 [repetition time/echo time, both in milliseconds]) and T2 weighted (1,500-2,000/40-80-100) images. A thoracic CT scan or a perfusion lung scan was obtained in some patients. The alveolar hemorrhage was assessed by the presence of a hyperintense T1 weighted signal in a lung area.

Phlebography and angiography were performed within a mean of six days after admission to the hospital but within a period of time after initial symptoms ranging from 1 to 27 days. Thoracic MRI was realized between 2 and 19 days (mean, 9 days) after admission.

RESULTS

In three patients with radiographic opacities on the chest x-ray film, pulmonary thromboembolism was angiographically confirmed. The abnormal radiographic territory, corresponding to the angiographic territory of the embolism, produced a hyperintense signal on T1 weighted MRI.

Patient 1 was a 50-year-old woman without predisposing conditions for thromboembolism. She presented first with a right upper lobe pneumonia treated with ampicillin for 15 days. Two weeks after her hospitalization, while clinical and radiographic signs of pneumonia decreased, she complained abruptly of right basal pleuritic pain. At this time the chest roentgenogram showed parenchymal consolidation of the right lower lobe, with a little right-sided pleural effusion. Pulmonary angiography revealed a filling defect without complete obstruction of blood flow in the interlobar artery and on the level of the right basilar branch fork; MRI visualized an area of a hyperintense T1 weighted signal in the posterobasal segment of the right lower lobe anterior to a pleural effusion.

Patient 2 was a 61-year-old woman who presented with dyspnea and left-sided pleural pain, which had appeared suddenly four days before admission, and hemoptyis, which occurred on the day preceding the hospitalization. Horizontal trabecular opacities were seen above the left hemidiaphragm. A scintigraphic subsegmental perfusion defect superimposed the ra-
diologic opacity. Pulmonary angiography detected a complete obstruction of the left basilar artery, with a trailing edge, demonstrating the presence of a blood clot; MRI revealed an area in the left lower lobe with a hyperintense T1 weighted signal.

Patient 3 was a 57-year-old homosexual man with HIV infection known for one year. He presented with a three-month history of fatigue and progressive weight loss (2 kg [4 lb]). He complained of right-sided pleural pain, cough, hemoptysis, and fever (38°C [100.4°F]) with shiver. A truncated homogeneous consolidation of the posterior segment of the right upper lobe was seen on the chest roentgenogram (Fig 1). A few trabecular horizontal opacities were seen above the right hemidiaphragm. Bronchoalveolar lavage performed in the right upper lobe recovered no pathogens. The findings from phelebography were normal. Angiography revealed a complete obstruction of the truncus anterior and a marginal filling defect in the right interlobar artery; MRI visualized the presence of a high T1 weighted signal in both territories (Fig 2).

In three other patients with symptoms and radiologic signs compatible with pulmonary infarction, pulmonary angiography demonstrated the absence of embolism. In these cases, thoracic MRI did not show any high T1 weighted signal. Our final diagnosis was infectious pneumopathy.

One patient with dyspnea and normal chest x-ray films underwent phelebography, angiography, and thoracic MRI. The findings in this case showed a pulmonary embolism without any abnormal signal in the lung parenchyma on thoracic MRI.

**DISCUSSION**

Until now, thoracic MRI was evaluated as a method of diagnosing pulmonary emboli by identifying the thrombus in the pulmonary arteries.4-7 Our findings are consistent with the possibility of diagnosing pulmonary emboli with infarction by thoracic MRI. The presence of alveolar hemorrhage in a case of pulmonary infarction appears on thoracic MRI like a territory with high T1 weighted signals. This noninvasive technique could be used to select patients for angiography, especially if an abnormal perfusion lung scan defect cross-matches with a roentgenographic opacity.

Motion and changes in scanning volume due to respiration could decrease the spatial resolution of MRI, especially in the lower lung fields, but should not change the signal of aging hemorrhage. Hemorrhagic lesions such as pulmonary parenchymal confluences, Goodpasture’s syndrome, invasive pulmonary aspergillosis,8 pulmonary sequestration,8 or high protein fluids such as mucus could produce similar images, but the clinical circumstances are different.

We speculate that in the early stages of thromboembolism, before methemoglobin formation, MRI of the lungs produces only isointense T1 weighted signals; but since the interval between the embolic episode and any increase in roentgenographic density ranges from 24 hours to 7 days,1 we believe that in cases of pulmonary infarction, parenchymal lesions are already constituted of hemorrhage with methemoglobin formation. Determination of the specificity and sensitivity of MRI in pulmonary emboli with infarction needs further investigation.

**CONCLUSION**

Thoracic MRI seems to provide useful indications in the diagnosis of pulmonary infarctions and could constitute an intermediary step before angiography or a diagnostic tool if angiography is contraindicated.

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**REFERENCES**


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