Assessment of Lung Ventilation, Gas Trapping and Pulmonary Perfusion in patients with Asthma during Inhaled Corticosteroid Withdrawal

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INTRODUCTION

Asthma is characterized by chronic inflammation and obstruction of the small airways that results in impaired pulmonary ventilation. Visualization of the alveolar and bronchial spaces with use of conventional MR imaging is difficult due to low proton density and increased magnetic susceptibility [1]. Hyperpolarized helium-3 (^{3}He) provides a technique for imaging ventilation in asthmatics and has shown small ventilation defects characteristic to asthma [2,3]. Furthermore, regional lung perfusion can be measured using Gd-chelate injection followed by ultra-fast MRI with short echo time [Hatabu et al]. However it is unclear as to how ventilation-perfusion MRI relates to established pulmonary function tests. The long-term objective of this study is to demonstrate correlations between MR and conventional assessments of pulmonary function following a gradual reduction of inhaled corticosteroids (fluticasone). The specific objective of this work is to develop a protocol for evaluation of lung ventilation, gas trapping and lung perfusion in subjects with asthma.

MATERIALS AND METHODS

Hyperpolarized ^{3}He MR Lung ventilation imaging was performed in seven asthmatic volunteers using a 1.5 T MR scanner with broadband capabilities (Signa LX, GE Medical systems, Milwaukee, WI). A vest RF coil (IGC-Medical Advances, Milwaukee, WI) tuned to receive at the resonant frequency of helium was used. A helium polarizer (IGI.9600, Amersham Health) used spin exchange optical pumping to create hyperpolarized ^{3}He. Each MR session consisted of localization; fast spin echo (FSE) and ^{3}He flip angle calibration scans followed by static imaging, dynamic imaging of inhalation and exhalation and 3D perfusion MRI using gadodiamide (Omniscan, Amersham Health) using a 3D PR-TRICKS [Vigen et al.] A spoiled GRE sequence was used with the following parameters ±15.63 KHz BW, 128 x 128 by 16 matrix, FOV (32-38) cm x (24-29) cm, and slice thickness 10mm. ECG and pulse oximetry was monitored throughout as well as prior to and after imaging. One 1-liter dose of hyperpolarized ^{3}He with a net activity of 4.5 mMol was inhaled for each image. Static images were acquired during breath hold of up to approximately 15 seconds. Dynamic images were acquired to identify gas trapping. Images were evaluated for defects on a regional basis, each lung was divided into three regions: apical, middle, basilar and percentage ventilation defects were noted. Regions corresponding to ventilation defects in the perfusion image were evaluated to identify physiologic shunting. Subjects were imaged at baseline and following withdrawal of inhaled corticosteroids.

RESULTS AND DISCUSSION

Static images demonstrated peripheral ventilation abnormalities at baseline and during exacerbation following ICS withdrawal (Figure 1). Significant variability in the number and size of ventilation defects was observed over two separate baseline studies on semi-quantitative analysis. Dynamic MRI during forced exhalation of the gas shows regional gas trapping associated with early closure of small airways, which to our knowledge has not been demonstrated previously using MRI (Figure 2). Dynamic perfusion images may be acquired and co-registered with the ventilation maps to assess the presence of V/Q abnormalities (Figure 3).

CONCLUSIONS

We have presented an imaging protocol for comprehensive visualization of pulmonary function using He-3 ventilation imaging and 3D perfusion MRI using Gd-chelate injection followed by dynamic proton MRI. The protocol is intended to provide a complete picture of the lung physiology at crucial time points in the corticosteroid reduction scheme including: ventilation of the airways (static and dynamic ^{3}He), and pulmonary perfusion. Ventilation defects, gas trapping, and 3D perfusion have been demonstrated. The corticosteroid withdrawal study of subjects with asthma and quantitative assessments of reproducibility and correlation to pulmonary function measures is ongoing.

REFERENCES

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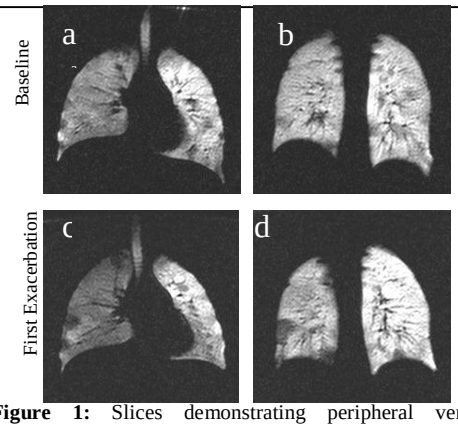

Figure 1: Slices demonstrating peripheral ventilation abnormalities at baseline (top row (a,b)) and 10 weeks later at first exacerbation (bottom row (c,d) for a typical case. Ventilation abnormalities appear more severe at first exacerbation.

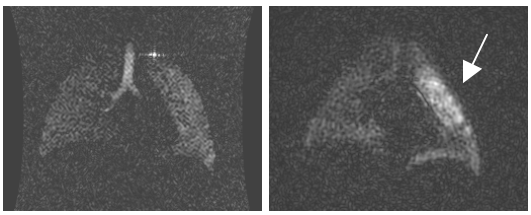

Figure 2: Dynamic expiration phase images from the same subject as in Fig. 1 at (a) Baseline and (b) first exacerbation. Note the region of intense residual signal suggesting gas trapping (arrow).

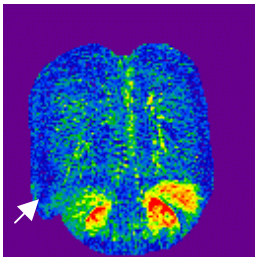

Figure 3: Perfusion map corresponding to Figure:(1d) showing decreased perfusion in area with the most severe ventilation defect (arrow). Other areas of perfusion were normal.