Dear Editor

We read with interest the recent paper by Chaudry et al in which the authors describe an obstructive ventilatory defect in children with sickle cell disease (SCD) which was not linked to bronchial hyper-responsiveness, as assessed by methacholine challenge testing, or to eosinophilic inflammation. Their results further demonstrate that the airflow obstruction seen in children with SCD may not solely be due to asthma. Indeed, in children with SCD, we have shown that pulmonary capillary blood volume is increased compared to matched controls, and is correlated with airflow obstruction as assessed by impulse oscillometry. In SCD adults, we previously demonstrated that prominent central vessels were often present on high-resolution CT (HRCT) with correlations between reductions in FEV1 and FVC and prominence of central vessels. We have subsequently carried out a detailed study of vascular abnormalities on HRCT using two quantitative measures of vessel dilatation (the segmental artery-bronchus (A/B) ratio and the total cross-sectional area of all pulmonary vessels less than 5 mm in diameter (CSA<5 mm%)). Increases in segmental A/B ratio and CSA<5 mm% were independently linked to reductions in FEV1, VC and FEF25–75, and to increased respiratory system resistance and RV:TLC. Small vessel dimensions correlated with reduced haemoglobin concentration and oxygen saturation and increased cardiac output, lactate dehydrogenase level, reticulocyte count and serum bilirubin, suggesting interactions between haemolysis, anaemia, hypoxia and pulmonary function abnormalities. We, therefore, suggest that alterations in pulmonary vascular volumes due to anaemia in SCD patients contribute to their lung function abnormalities.

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Contributors AG and AL wrote the letter.

Competing interests None.

Provenance and peer review Not commissioned; internally peer reviewed.

To cite Greenough A, Lunt A. Thorax 2014;69:1051.
Received 12 June 2014
Accepted 23 June 2014
Published Online First 15 July 2014

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