Background Rheumatoid arthritis (RA) is a systemic inflammatory condition associated with increased cardiovascular risk which is not fully explained by traditional risk factors. Endothelial dysfunction and increased aortic stiffness may mediate some of the increased risk. Additionally, there may be direct vascular inflammation which could directly accelerate atherosclerosis. We hypothesised that patients with RA exhibit a subclinical aortic vasculitis which can be reversed with anti-tumour necrosis factor (TNF) therapy.

Methods The aortas and carotid arteries of 15 patients with severe rheumatoid arthritis were imaged before and after anti-TNF therapy using 18F-fluoro-deoxyglucose positron emission tomography (FDG-PET) with CT co-registration. Tracer uptake was analysed in various arterial segments by measuring maximum standard uptake values (SUV) and subsequently corrected for blood uptake to obtain a target to background ratio (TBR). Carotid to femoral pulse wave velocity (PWV) as a measure of aortic stiffness, disease activity and inflammatory biomarkers were also measured.

Results Mean baseline aortic TBR was 2.07±0.20. Following anti-TNF α therapy, there was a significant reduction in abdominal aortic TBR (−0.18±0.27, p=0.03) and in the most diseased segment in the whole aorta (−0.48±0.59, p=0.01). TBR was also reduced in all other aortic segments and the proportion of hot slices (defined as TBR>1.9) was reduced by 31%, but these did not reach statistical significance. There was no change in carotid TBR following treatment. Aortic PWV was reduced by 0.43±1.0 m/s (p=0.1) and there was a significant correlation between a reduction in aortic PWV and abdominal TBR (R=0.57, p=0.03) and between aortic PWV and proportion of “hot” slices (R=0.66, p=0.01). There was a concomitant reduction in serum CRP (−8±12 mg/L, p=0.02) and disease activity (DAS28 −1.41±1.51, p=0.002).

Conclusions This study demonstrates for the first time that patients with RA have high aortic and carotid FDG uptake, suggesting subclinical vasculitis. Moreover, they exhibit a reduction in FDG uptake following anti-TNF therapy, which correlated with a reduction in aortic stiffness. These results suggest that subclinical vasculitis could be the mechanism behind the increased cardiovascular risk and that effective treatment of inflammation may help to reduce the cardiovascular risk in this patient population.

Conclusion In the current study use of PHHE after brief, bed-side training greatly improved the diagnostic accuracy of medical students and junior doctors, over and above history, physical examination and ECG interpretation.