

WALKING POSTER PRESENTATION

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Cardiovascular magnetic resonance characterisation of pericardial and myocardial involvement in patients with tuberculous pericardial constriction with and without HIV co-infection

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From 19th Annual SCMR Scientific Sessions
Los Angeles, CA, USA. 27-30 January 2016

Background

Tuberculosis pericarditis (TBP) is the most common cause of a large pericardial effusion in the developing world, accounting for 70% of effusions in a case series from South Africa; and has a high mortality related to pericardial tamponade, constrictive pericarditis, arrhythmias and heart failure. Manifestations of TBP include pericarditis with pericardial effusion, effusive-constrictive and constrictive pericarditis. There has been a dramatic resurgence in TBP in the context of co-infection with the human immunodeficiency virus (HIV). Almost 100% of pericardial effusions in those infected with HIV in sub-Saharan Africa are due to tuberculosis, compared with 50-70% in those HIV-uninfected and less than 5% in industrial nations. In patients with TBP, co-infection with HIV is associated with increased heart failure, haemodynamic instability, electrocardiographic (ECG) ST elevation and mortality, suggesting an aggressive myopericarditis in the context of HIV co-infection. However, little is known about myocardial involvement in patients with TBP. Cardiovascular magnetic resonance (CMR) can assess non-invasively cardiac function, myocardial oedema, inflammation and fibrosis. We hypothesised that HIV co-infection would be associated with increased myocardial pathology on CMR in patients with TBP.

Methods

The purpose of this study was to assess cardiac and pericardial structure and function in patients with TBP

with and without HIV co-infection and to assess the relationship of LV function with other imaging biomarkers. 72 patients with TBP (37 male (51.3%), mean age 40 ± 14.3) were included in the study. Of these, 35 were HIV-infected (17 male (48.6%), mean age 34 ± 8) and 37 were HIV-uninfected (20 male (54.1%), mean age 51 ± 16). Assessments included clinical examination, ECG, echocardiography, serum and pericardial biomarkers and CMR (biventricular volumes and function, oedema, and late gadolinium enhancement - LGE).

Results

HIV-infected TBP patients were younger ($p < 0.001$), had lower serum haemoglobin ($p < 0.001$) and were more likely to have NYHA class III and IV symptoms ($p < 0.001$). There were no differences on ECG and echocardiography between HIV-infected and -uninfected TBP patients. There were also no differences in global systolic function and myocardial signal intensity ratio on STIR imaging between HIV-infected and -uninfected TBP patients. Focal fibrosis on LGE was found more commonly in those with HIV infection ($p < 0.001$). Pericardial effusions were frequent (>50%) in both groups of TBP patients. Determinants of LV ejection fraction in TBP included heart rate, LV size, E/A ratio, pericardial LGE and pericardial thickness (all $p < 0.01$).

Conclusions

HIV co-infection is associated with increased focal myocardial fibrosis in TBP patients suggesting increased myocardial inflammation in those with HIV co-infection. In the future, it will be important to assess the prognostic significance of these data.

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Published: 27 January 2016

doi:10.1186/1532-429X-18-S1-Q29

Cite this article as: Ntusi *et al.*: Cardiovascular magnetic resonance characterisation of pericardial and myocardial involvement in patients with tuberculous pericardial constriction with and without HIV co-infection. *Journal of Cardiovascular Magnetic Resonance* 2016 **18**(Suppl 1):Q29.

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