World Trade Center Particulate Matter Associated Cardiopulmonary Dysfunction and Injury: Incorporating Echocardiography in a Murine Model

A. Veerappan, A. Oskuei, S. Vaidyanathan, G. Crowley, Y. Wadghiri, A. Nolan; New York University School of Medicine, New York, NY, United States.

Corresponding author's email: arul.veerappan@nyulangone.org

RATIONALE Vascular changes are known to occur early in the development of obstructive airways disease. Vascular serum biomarkers and an elevated pulmonary artery (PA)/aorta ratio are associated with the development of WTC-associated lung disease. The vascular remodeling and the early detection of cardiopulmonary vascular dysfunction due to WTC-PM exposure are not well understood. Clinically relevant non-invasive whole animal imaging may facilitate our understanding of end-organ dysfunction in our murine model of WTC-PM exposure. This study is designed to quantify noninvasive measures of the cardiopulmonary functional changes that may lead to permanent cardiopulmonary vascular remodeling and dysfunction during secondary to WTC-PM. METHODS C57BI/6 wild type (WT) mice (n=8/group), aged 8-10 weeks aspirated 200µg-PM (WTC-Aggregate, PM53) or PBS. 24-h after exposure all mice underwent noninvasive in vivo echocardiography (Fujifilm VisualSonics Vevo 3100) to monitor the development of cardiopulmonary functional changes. Trichrome (Gomori) stain for collagen analysis was performed on 4% paraformaldehyde fixed cardiac tissue sections (5 µm) using imageJ program. RESULTS 24 hours after PM exposure there was significant reduction in PA flow (velocity) and there was a notch that occurred during the pulmonary ejection time (PET) in PM exposed mice (Figure 1A, red arrow) when compared to PBS controls, Figure 1B. In addition, there was significant reduction in pulmonary acceleration time (PAT) and PAT/PET in WT-PM exposed mice when compared to PBS controls, Figure 1C-E. Systolic arterial pressure (SAP) and mean pulmonary arterial pressure (MPAP) were significantly increased in PM exposed mice compared to PBS, Figure 1C/F. Cardiac output and stroke volume were significantly decreased in PM mice compared to controls, Figure 1G-H. Finally, PM exposed mice showed a statistically significant increase in collagen deposition in cardiac tissues including vessels in heart, Figure 1J-L. CONCLUSIONS PM exposure and PM associated disease which include vascular disease burden are global health concerns. Earlier detection of vascular disease is much warranted in order to prevent cardiovascular related deaths. Acute PM exposure caused cardiopulmonary dysfunction and collagen accumulation in the cardiac tissue. These echocardiography results indicated that there may be features of early pulmonary vasculopathy and collagen formation in cardiovascular tissues after WTC-PM. Further, the noninvasive experiments showed that there is a possible evaluation for the early detection of cardiopulmonary vascular dysfunction due to WTC-PM exposure.