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THE MYOCARDIUM OF FETUSES WITH ENDOCARDIAL FIBROELASTOSIS CONTAINS A PAUCITY OF B AND T CELLS COMPARED WITH NORMAL CONTROLS

ACC Poster Contributions
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Background: Endocardial fibroelastosis (EFE) and dilated cardiomyopathy (DCM) in fetuses born to mothers with anti-Ro/La antibodies have been found to contain myocardial B and T lymphocytes. We hypothesize that fetuses with EFE + idiopathic DCM without antibodies may also exhibit increased immune cellular activity.

Methods: Fetuses were identified from the pathology database. Paraffin tissue sections of the right and left ventricle and interventricular septum were stained for T cell (CD3, CD4, CD7, CD8), B cell markers (CD19, CD20, CD79.a) and CD45 (all cell lineages) and analyzed digitally.

Results: N=14 fetuses between 19 - 41 weeks gestation were included. None of the mothers had autoimmune disease or anti Ro/La antibodies. N=5 had EFE + DCM, 4 had EFE + structural heart disease (hypoplastic left heart 2, aortic stenosis 1, dysplastic tricuspid and pulmonary valves 1), and 5 normal controls (pregnancy termination 2, birth asphyxia 2, motor vehicle accident 1). A total of 203 sections were analyzed. Normal controls had demonstrable B and T cell populations diffusely within the myocardium. The EFE + DCM group had less B cells than the control group (2.0 vs. 6.0 cells/section, p=0.005, specifically CD79a: 3.0 vs. 8.0 cells/section, p=0.004)). The EFE + heart disease group had less B cells (2.5 vs. 6.0 cells/section, p=0.08, specifically CD20: 1.0 vs. 4.0 cells/section, p=0.002) and less T cells (4.0 vs. 7.0 cells/section, p=0.04, specifically CD3: 4.0 vs. 12.5 cells/section, p<0.001) than the control group. Both the EFE + DCM group and the EFE + heart disease group had fewer CD45 cells compared to controls (31.0 vs. 73.5 cells/section, p=0.002 for the EFE + DCM group and 40.0 vs. 73.5 cells/section, p=0.007 for the EFE + heart disease group).

Conclusion: The myocardium of fetuses with EFE + DCM or structural heart disease contain smaller numbers of B and T lymphocytes compared with normal controls, suggesting a different pathophysiology from antibody-mediated disease. This suggests that targeted anti-inflammatory therapies in the 2nd and 3rd trimester may not improve outcome.