

P172. Stenotic Bicuspid Aortic Valve Lesions Are Associated With Increased Inflammation And Neovascularization.

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OBJECTIVES: The pathogenesis of bicuspid aortic stenosis (AS) is considered to be similar to tricuspid AS, only that it occurs at an earlier age. This study tested the hypothesis that severe bicuspid AS evolves from a more aggressive inflammatory process, with increased macrophage and neovessel content when compared to severe tricuspid AS.

METHODS: Immunohistochemistry on 22 aortic stenotic valves obtained at the time of aortic valve replacement was performed for quantification of macrophage/T cell infiltration (CD-68+CD3), and neovessel content (CD-34). Bicuspid (n=12) patients were 15 yrs younger than tricuspid (n=10) patients (61 ± 8 versus 75 ± 8 yrs; $P=0.0001$). Other variables including risk factors, serum creatinine, HbA1c, incidence of coronary disease, ejection fraction, peak gradient, and aortic valve area were similar in both groups.

RESULTS: Total number of macrophages and T cells was increased in bicuspid AS when compared to tricuspid AS (414 ± 32.9 versus 101 ± 9.9 ; $P=0.009$). The number of neovessels was also increased in bicuspid AS when compared to tricuspid AS (275 ± 100 versus 153 ± 4.2 ; $P=0.009$) (Figure). Macrophages and T cells correlated with neovessel content in bicuspid AS ($R=0.85$; $P<0.0001$) but not in tricuspid AS ($R=0.4$; $P=NS$).

CONCLUSIONS: The pathogenesis of severe bicuspid aortic stenosis involves an aggressive inflammatory process, with increased macrophage/T cell, and neovessel content when compared to tricuspid AS. Therefore, aggressive medical therapy to decrease macrophage and neovessel infiltration may provide improved benefit in bicuspid AS. Clinical trials are needed to confirm this hypothesis.

