**CHANGING PATHOLOGY OF THE AORTA: FROM ACUTE TO CHRONIC DISSECTION**

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*Background:*The natural history of the progression of aortic dissections from acute to chronic is not well understood.

*Objectives:*The aim of the study was to evaluate the radiographic, echocardiographic and histopathologic changes from an acute to a chronic dissection, depending on time after onset of symptoms. We concentrate on typical “flap” dissection rather than variant dissections.

*Methods:*One hundred fifty-eight patients with diagnosed aortic dissection were analyzed by computed tomographic (CT) imaging, transesophageal echocardiography (TEE) and/or microscopic histopathology. Among these, 74 were Stanford Type A (mean age 65±13years, 64% male) and 84 Type B dissections (mean age 58±13years, 64% male).

*Results:*Among Type B dissections, a growth rate of 6.78mm/year was found with a significantly higher rate within the acute and subacute stages. Thereafter, the rate of growth stabilized in the chronic phase at 1.88mm/year. Flap-thickness increased at 0.61mm/year, showing a similar early dynamic as for diameter. Flap mobility decreased over time. No longitudinal extension or new branch involvement were noted after the initial event. Among Type A dissections, the flap showed comparable behavior. Due to the limited follow-up because of widespread early surgery in these patients, a natural growth rate could not be determined. Furthermore, no significant histologic changes were seen among Type A dissections over time, possibly related to these patients going to early surgery. However, Type B dissections showed increased fibrosis, as well as elastin fragmentation, over time as the lesion progressed from acute to chronic.

*Conclusions:*In the acute to chronic post-dissection transition, stabilizing after 2 months, (1) the aorta grows more rapidly early after dissection than later; (2) the flap thickens and becomes less mobile; (3) the aortic dissection did not commonly extend longitudinally or involve a new branch vessel; (4) histologically, increased wall fibrosis is noted, along with increased elastin fragmentation over time.