

About inflammatory activation during aortic dissection

To the Editor,

We have read with great interest the article entitled "Effect of inflammation on the biomechanical strength of involved aorta in type-A aortic dissection and ascending thoracic aortic aneurysm: An initial research" which was published in *Anatol J Cardiol* 2018; 20: 85-92 (1). In this study, the authors aimed to explore the degree of systemic and local vessel inflammation in aortic dissection (AD), to detect their influence on vessel tensile properties, and to compare them with those in aortic aneurysm (AA). They hypothesized that a correlation exist between inflammation and decrease in vessel strength in AD.

In the study, aortic vessel specimens were obtained from normal-looking nondissected aortic tissues near the dissected aortic lesions in the type-A AD group; whereas in the AA group, aortic vessel specimens were obtained from the aortic aneurysm walls. The onset time till surgery was 502.9 ± 280.1 h in the AD group. Eighteen (90%) patients had chest and back pain before surgery. Dissection type was the Stanford type A. Thus, all the ascending aorta or the entire aorta may be affected until the iliac artery. According to these results, we can conclude that patients with AD have acute AD, but AA patients don't have. This is the main cause of the significant inflammatory mediator increase in the AD group, compared with the AA group. To prevent this structural mistake of the study design, authors may consider comparing patients with chronic AD with those with nondissected AA. In acute AD, impaired multiple organ perfusion due to dissection of the entire aorta will aggravate systemic inflammatory response independently from the effect of dissection in itself of the aortic vessel tissue. They also assumed that the degree of inflammatory response directly affects tissue strength. They concluded that the serum concentrations of IL-6 and TNF- α may somehow be used to indicate the decrease in biomechanical strength of the affected aorta in AD. It is a known fact that dissected tissues are the most fragile tissues. It is not possible to perform graft anastomosis to dissected aorta without teflon-felt reinforcement. In this study, the authors did not compare tissue strength after the increase in inflammation in the circulation as indicated in the aim of the study. There are no data regarding patients with AD with low and high inflammatory responses in the blood to compare this difference in the aortic tissue. We would be pleased if the authors could clarify these conflicting study results.

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Reference

1. Bai Z, Gu J, Shi Y, Meng W. Effect of inflammation on the biomechanical strength of involved aorta in type A aortic dissection and ascending thoracic aortic aneurysm: An initial research. *Anatol J Cardiol* 2018; 20: 85-92. [CrossRef]

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Author's Reply

To the Editor,

Thanks for reading our manuscript entitled "Effect of inflammation on the biomechanical strength of involved aorta in type-A aortic dissection and ascending thoracic aortic aneurysm: An initial research" (1). Because you have asked some questions in the letter, we would like to introduce some more detailed information.

First, you asked if we should compare patients with chronic AD with those with nondissected AA. AD is a critical illness that has a very high mortality rate in its acute course due to aorta rupture, and urgent or emergent surgical treatment should be performed in this phase. As mentioned in the manuscript, the acute inflammation of the involved aorta walls was the main cause of invulnerable aorta and aorta rupture, and it also made graft anastomosis in surgical treatment more difficult. In an earlier article, we had proven that the inflammation biomarkers of AD, such as IL-6 and TNF- α , present time-dependent changes in its course and may rise to their peak levels in a few days after onset and decline slowly to near-normal levels after the acute phase (2). Because AA progress had not involved an intensive inflammation response and the biomarkers were usually in the normal range, we thought that there should be no significant difference between the patients with subacute or even chronic AD and AA, both in the degree of inflammation and the incidence of surgical implications and mortality. Therefore, we declined performing a comparison between them. As you had mentioned, it would be more appropriate to add the comparison of them. We may consider this in future studies.

Second, you mentioned that "the authors did not compare tissue strength after the increase in inflammation in the circulation, as indicated in the aim of the study" and that "there are no data regarding patients with AD with low and high inflammatory responses in the blood to compare this difference in the aortic tissue." For the former, because the involved patients with AD in our