

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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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

study had already presented a significant inflammatory response both in the tissue and circulation and because the samples were all one-time-point collected, we could not directly compare the change in the tissue strength in one individual before and after inflammation onset. This was also a limitation of our human study. Thus, we had only tested the correlation between inflammation (both in the circulation and vessel tissue) and tissue strength in involved patients, and the significant correlation was shown in the manuscript. For the latter, it is a known fact that there is a chronic inflammatory response in the AD aortic wall before the intima tear, and the implosive acute inflammatory response induced by the blood flow impact occurred after the intima tear. Thus, we thought that all AD-related aortic vessels suffered from severe local tissue inflammation. The following circulatory or systemic inflammation of AD started with the release of inflammation biomarkers from the dissected aorta just after onset, and it might be aggravated by impaired multiple organ perfusion (mainly gastrointestinal tract and kidneys) due to dissection of the entire aorta during AD progress. The severity of circulation inflammation of AD may vary among individuals due to differences in the dissected area or involved organs. In our involved patients, there was no significant impaired organ perfusion due to dissection because no patients suffered from gastrointestinal ischemia and renal failure. However, four of 20 patients suffered from respiratory failure before surgery. Which we thought should be acute lung injury induced by local accumulation of inflammation biomarkers. It was obvious that there were many factors that might have caused uncertainty with regard to a direct correlation between aortic and circulatory inflammation. In such an initial research with a small sample size, we could not eliminate all interference variables; therefore, we declined performing the correlation test. In future research with more patients and more influence factors included, the correlation test might be appropriate.

Because our manuscript was an initial research with a small sample size and simple testing and statistical analysis, the results may sometimes be viewed with subjectivity, one-sidedness, and superficiality. We wish to introduce our research to interested cardiovascular surgeons and researchers, and we accept the criticisms and suggestions of colleagues.

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Discordant results about QT prolongation in patients with Turner syndrome

To the Editor,

We have read the paper entitled "Evaluation of the Tp-Te interval, Tp-Te/QTc ratio, and QT dispersion in patients with Turner syndrome" with great interest (1). The authors stated that patients with Turner syndrome have a longer QTc; however, the numbers of patients in the control group were insufficient. The control group in the study included 35 patients, and the mean QTc was 392.06±13.21. In previous studies with a larger population, the mean QTc of patients was longer than that in the present study. For example, in the previous studies for ages 12–15 years, the mean QTc was 426 for 10,709 female population, whereas for ages 16–19 years, the mean QTc was 423 for 14,453 female population in the large study (2). This raises suspicion about selection bias in the control group. Furthermore, the selection of an inappropriate control group is a common problem in this type of observational study. Inappropriate control group can result in inconsistency with real population statistics. In addition, even if we accept that an accurate control group was selected by the authors, the effect of a small increase in QTc on mortality rate is unclear. We cannot exclude the chance factor for statistical significance (p value) because of the small sample size and small number of patients in the control group of the authors' study. Moreover, in the discussion part, there is not enough data and causality for the prevention of sudden death in patients with Turner syndrome.

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