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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Author`s Reply

To the Editor,

A review of the literature regarding the QTc values of patients in the control group revealed the following observations: Trolle et al.'s (1) study had a control group with a mean age of 38.9±12.4 years, with mean QTc values of 389.1±20.1; Demirol et al.'s (2) study had a control group with a mean age of 12±3.5 years, with mean QTc values of 390±25.1; Olivares López et al.'s (3) study had a control group with a mean age 11.45±2.58 years, with mean QTc values of 391.73±17.7; Ergul et al.'s (4) study had a control group with a mean age of 4.3 (6 days–16 years) years, with mean QTc values of 385±58; Küçük et al.'s (5) study had a control group with a mean age of 60 years, with mean QTc values of 384±43.2; Braschi et al.'s (6) study, which shows reference ranges for non-invasive ventricular repolarization parameters for various patients, had 3 groups: group 1–child (1 day–11 years), group 2–adolescent (12–19 years), group 3–adult (20–64 years). Group 1 had a mean QTc value of 401.7±25, group 2 401.9±21.3, and group 3 407.3±19.8; Akın et al.'s (7) study had a control group with a mean age of 8.8±2.4 years, with min QTc of 371.3±24.7 and max QTc of 411.33±24.6; Ogawa et al.'s (8) study in Japan entitled "The Maximum QTc of Holter Electrocardiography in a Pediatric Population" had a QTc value of 380 (368–390) for 10–12-year-old girls and 397 (380–410) for 13–15-year-old girls; and Krasemann et al. (9) had 7 groups in their study entitled "Changes of the corrected QT interval in healthy boys and girls over day and night," wherein the sixth group with patients aged 12–16 years had a QTc value of 400±20.

Our control group with patients aged 13.17±2.85 years had a mean QTc value of 392.06±13.21, which is not different from those in the 9 studies mentioned above but clearly different from the Brazilian study. Regional factors may be the cause of this difference; therefore, everyone including us use control groups of same population we studied. We indicated that our study population was small and that studies with a larger population are necessary along with the other limitations in the study limitations section.

Our study did not evaluate mortality, and our results indicate the differences only between the study and control groups. Because QTc prolongation can cause sudden and we did find longer QTc in our study population, we only mention that the increased QTc may cause harm and to confide in that we suggested further investigation.

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