Peer review file
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Comment 1: Introduction. »STORBE guildline checklist« was meant to be »STROBE checklist«; this statement is in my opinion unnecessary.

Reply 1: Thank you for your comment. We have deleted this statement in the revised manuscript.
Changes in the text: Page 7, line 8.

Comment 2. Methods. In this chapter, definitions of the normal coronary arteries and obstructive coronary lesions should be explicitly stated. CAE is usually defined as coronary artery with a luminal diameter exceeding the diameter of normal adjacent segment by 1.5 times and involving >50% of the total lentgh of the vessel (DiazZamidio et al. 2007). There are so many exclusion criteria that a huge effort should have been involved to exclude all of them. It is not clear why total coronary occlusions were not allowed. The number of patients undergoing PCI/CABG after the index angiography should be explained as they had to be treated with antiplatelet agents. Finally, what was the statistical power of the sample of 300 patients, 100 of them in each group?

Reply 2: Thank you for your valuable comments.

1) We have revised the definitions of NCA and obstructive coronary lesions in the revised manuscript.

Changes in the text: Page 9, lines 8-10

2) Because our study focused on the primary dilatation of the coronary artery, there were several exclusion criteria which exclude secondary coronary artery dilatation. Total coronary occlusions were not allowed in this study, because we thought that they could be one type of mechanical factor for compensative dilatation. Besides, total occlusion lesions could prevent the distribution of the contrast medium into the distal vessels, thus we could not confirm whether there were dilatations in the distal vessels.
3) We did not include any patients who underwent PCI/CABG because the previous surgery might lead to a secondary coronary artery dilatation.

Remarked in the text: Page 8, lines 7-9

4) Despite this study's clinical topic, this was an experimental basic study that aimed to investigate the pathogenesis of coronary artery dilatation. Thus, it may be not necessary to calculate statistical power of the sample size.

Changes in the text: Page 8, line 3, Page 9, line 1.

Comment 3. Results. The authors should specifically include the coronary arteries involved with CAE/CAA and the accompanying obstructive coronary artery disease.

Reply 3: Thank you for your insightful comment. We have specifically mentioned this data in the Results section and in Table 5.

Changes in the text: Page 15, lines 21-22.

Comment 4. Discussion. The authors should explain why there had been no significant differences in hs-CR and IL-6 levels among the CAE and CAA groups if the inflammation hypothesis was to be held. Higher Gensini and Syntax scores simply suggest more extensive coronary artery disease and not the absence of the disease. Coronary atherosclerosis is usually explained as a low-level inflammation of coronary arteries. Finally, coronary dilatation is supposed to be the result of expansive vessel remodelling in coronary atherosclerosis or other diseases such as the arterial hypertension.

Reply 4: Thank you for your comments.

1) To our knowledge, this study is the first to reveal differences between CAE and CAA. Further, we aimed to compare the differences between CAA and CAE and to investigate their pathogenesis. Although there was no difference in the hs-CRP and IL-6 levels, we found that ANCA, endothelial-1, matrix metallopeptidase-9, and tumor necrosis factor-α values were significantly higher in CAE. Thus, we concluded that CAE was closely related to inflammation. Because we could find no related data that was previously reported, further study will provide additional data for the different types of inflammation factors that may play a role in CAA and CAE.

Changes in the text: Page 20, lines 9-14.

2) In our study, we excluded patients with coronary artery dilatation that appeared in the proximal segment of the coronary artery stenosis. We found instances of coexistence with coronary artery stenosis (but not in the adjacent segment) for a total of 75 patients in the CAA group, and 43 patients in the CAE group. The percentage of patients with concomitant stenosis was significantly higher in the CAA group than in the CAE group ($P < 0.001$). We also found that the Gensini and SYNTAX scores were significantly higher in the CAA than in the CAE group ($P < 0.05$). Thus, we suspected that CAA may be closely related to atherosclerosis due to the significant correlation between atherosclerosis and CAD. Our additional experiments confirmed this hypothesis. We found that ABI was significantly lower in the CAA and CAE groups than that in the NCA group ($P < 0.05$), further, low-density lipoprotein/high-density lipoprotein was significantly higher in the CAA compared with the NCA group ($P < 0.05$), and the detection rate of carotid artery thickening was significantly higher in the CAA than in the CAE and NCA groups ($P < 0.05$).

Remarked in the text: Page 21, lines 17-22~Page 22, line 1

3) We have already excluded other diseases such as arterial hypertension that could result in secondary coronary artery dilatation.

Remarked in the text: Page 8, lines 8-10.

Comment 5. Remaining questions. The authors may want to comment on genetic factors implicated in pathogenesis of CAE (e.g. ACE DD polymorphism).

Reply 5: Thank you for this suggestion. We have modified our text as advised.
Changes in the text: Page 23, lines 8-10.