

## Angiogenin and osteopontin and coronary collateral circulation

To the Editor,

We read the publication on “The association between serum angiogenin (AGN) and osteopontin (OPN) levels and coronary collateral circulation in patients with chronic total occlusion” with a great interest. Gürses et al. (1) concluded that “AGN and OPN are associated with better developed coronary collateral circulation and may have therapeutic implications for the promotion of coronary collateral development” and also noted that “the underlying mechanisms remain largely unknown”. We would like to share our ideas on this report. First, the results in this study inconsistent with those reported previously, which demonstrated that AGN level was associated with complications of acute coronary syndrome and AGN was not a marker for revascularization or collateral circulation formation (2). The adjustment for the background of the patients in both the groups might help increasing the clarity of the findings. Certainly, many possible background conditions might affect the result of AGN investigation. For example, the underlying genetic hemoglobin disorders might result in a high AGN level regardless of the occurrence of the acute coronary syndrome or collateral circulation formation (3). In addition, the use of an ACE inhibitor, which is a common drug in the patient with cardiovascular disease, can also affect the level of OPN (4). We agree with the conclusion by Gürses et al. (1) that there might be an unknown mechanism linking AGN or OPN and collateral circulation formation, but there is a possibility that there might be interference or background factors that can alter the AGN or OPN level.

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### Author's Reply

To the Editor,

We thank the authors for their interest and comments on our manuscript titled “The association between serum angiogenin (AGN) and osteopontin (OPN) levels and coronary collateral circulation in patients with chronic total occlusion” (1).

The authors have claimed that the results of our study were not compatible with the findings of a previous study. However, Tello-Montoliu et al. (2) primarily investigated the prognostic role of AGN in acute coronary syndrome (ACS) patients. They did not explore or comment on the relationship between AGN and coronary collateral circulation. In fact, Tello-Montoliu et al. (2) emphasized that AGN may have a role in the development of microvessels in the core of atherosclerotic plaques as “a potent angiogenic growth factor” and thereby affect the prognosis of ACS patients. In addition, there is strong evidence in the literature regarding the angiogenic potential of AGN, which further support our findings (3, 4).

The authors have also claimed that there may be some background conditions that might affect the result of AGN investigation, such as “genetic hemoglobin disorders” or “use of some drugs”. In the Methods section of our paper, we had already explained that we excluded patients with systemic diseases. In addition, when the prevalence of these genetic disorders is considered, it is unreasonable to assume that these can significantly affect the results of a study with 122 participants. We had also provided data for the medications of participants in Table 3, and there was no difference with respect to the use of renin-angiotensin system (RAS) blockers between poor and better developed collateral groups. Therefore, a confounding effect of RAS blockers does not exist in our study.

From our point of view, the authors' claims those attribute our findings to “interference or background factors” are speculative and do not have a firm basis.

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