in TTC. In addition, the cited article is about different ECGs in recurrent episodes of 2 different territories of left ventricle in the same patient with TTC. Our patient did not have recurrent episodes and ST-segment elevation was limited to high lateral leads. Rather than basal pathology, apicobasal gradient of left ventricular myocardial edema in TTC is more likely to produce ST-segment elevation and reciprocal changes.

8) Posterobasal hypokinesia detected by strain and strain rate analysis before discharge was possibly the last segment, because 1 month later, transthoracic echocardiography showed no wall motion abnormality. However, we did not perform strain and strain rate analysis during that visit. That analysis could have better confirmed improvement of left ventricle systolic dysfunction. On the other hand, CMRI revealed no scar, fibrosis or infarction, just mild myocardial edema.

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Cholesterol embolization syndrom

Dear Editor,

The recent report on cholesterol embolization syndrome was very interesting. Two cases were reported by Dizman et al. Indeed, this condition can be severe and can have serious complications, including neurological and lung problems. In the present case, it was questionable whether the problem was iatrogenic. It was noted that the syndrome is a “complication of peripheral endovascular interventions.” In such cases, if the practitioner is able to recognize and identify the iatrogenic problem early, early man-
agement by “corticosteroid and cyclophosphamide therapy” can provide favorable outcome.[5] In general practice, there must be a tool to monitor such complications following intervention.

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Authors’ reply

To the Editor,

We sincerely appreciate this contribution to our article. We clearly stated in the article that the most important risk factor for cholesterol embolization syndrome (CES) is advanced atherosclerosis, and that CES may occur spontaneously without any invasive procedure.[1] Thus, it is not possible to prove that CES is caused by peripheral endovascular intervention, but it is a fact that invasive cardiovascular procedures are the most important risk factor for CES.

CES is a kind of inflammatory disease that involves multinuclear cells, eosinophilia, and activation of the complement system. Therefore, it is logical to suggest that anti-inflammatory treatments may be helpful in the treatment of CES. As highlighted in the comment letter, there are several case series noting the favorable effects of corticosteroids and cyclophosphamide in the treatment of CES.[2,3] However, serious side effects of these immunosuppressive agents such as increased risk of malignancies, pulmonary, and cardiac toxicities should be kept in mind. Further prospective and randomized studies are needed to clarify these promising results.

In our opinion, rarity of CES possibly sets a barrier to the development of monitoring methods following intervention. Monitoring the peripheral signs in addition to laboratory findings of high-risk patients might be helpful in clinical practice.

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