Dear Editor,

We appreciate Dr Pareek et al for their interest in our article.1 In their letter, the authors suggest that heart failure (HF) should be included in the endpoints of the OXI trial. In addition, they provide more evidence on the pleiotropic effects of hydroxychloroquine regarding its beneficial effects on lipids and glycemic parameters.

As the authors point out, HF is a frequent complication after myocardial infarction (MI). In a cohort study by Gerber et al,2 the cumulative incidence of HF was 14.7–29.8% at 5 years after the first MI depending on the angiographic extent of the coronary artery disease.2 In another register study, HF developed in 12.6% of the patients after being discharged from their first MI during a median follow-up time of 3.2 years.3 As Pareek et al point out, hydroxychloroquine might induce mild bradycardia and thus it might be beneficial in patients with HF. However, no clinical evidence supports this theory. On the contrary, case reports have shown that hydroxychloroquine has cardiotoxic effects that might cause cardiomyopathy and HF in rare cases after prolonged use.5,6 Thus, it would be worth considering HF as an endpoint because at the moment we do not know if the net effect of hydroxychloroquine treatment would be positive or negative in terms of the development of HF.

As the study protocol of the OXI trial does not include echocardiographic evaluations, we will consider performing it to the participants at baseline and during the control visits. Possible clinical signs of HF will also be assessed regularly. Measuring the heart rate for the evaluation of bradycardia effect of hydroxychloroquine is integrated in the study protocol, as electrocardiographic monitoring of each patient will take place at every visit.

Conflict of interest: none declared.

References


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