

The impact of chronic total occlusion in non-infarct-related coronary arteries



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We read with great interest the excellent article entitled “Prognostic impact of non-culprit chronic total occlusions in infarct-related cardiogenic shock: results of the randomised IABP-SHOCK II trial” by Saad et al¹. The authors conducted a retrospective analysis to show that chronic total occlusion (CTO) in a non-infarct-related coronary artery (non-IRA) was an independent predictor of one-year mortality and 30-day ventricular arrhythmias in patients with cardiogenic shock (CS) complicating acute myocardial infarction (AMI). These findings complement previous findings that non-IRA CTOs were also arrhythmogenic, which translated into higher all-cause mortality²⁻⁴.

Our group recently conducted a systematic review and meta-analysis of published studies on the impact of CTOs on arrhythmic and mortality outcomes⁵. We found that the presence of IRA-CTOs was associated with higher risk of ventricular arrhythmia or appropriate implantable cardioverter-defibrillator therapy compared to non-IRA CTOs. However, the study population in the meta-analysis did not include patients with CS complicating AMI. The

study by Saad et al illustrated that non-IRA CTOs carried a negative impact on prognosis in AMI patients with CS, identifying a highly vulnerable group of patients¹. Studies conducted in Japan and Australia also reported higher rates of mortality in these AMI patient populations^{6,7}. There are two main reasons that can explain these findings. Firstly, non-IRA CTOs reduce the supply that can be delivered to the region supplied by the IRA, increasing the risk of myocardial ischaemia to this region and beyond⁸. Secondly, these patients show more extensive adverse remodelling, which can compromise ventricular haemodynamics and increase the propensity to arrhythmogenesis. Successful percutaneous coronary intervention for CTOs has been associated with a lower risk of death, stroke, and coronary artery bypass grafting and less recurrent angina pectoris⁹. However, CTO revascularisation in the setting of CS is very difficult to perform and is likely to have a deleterious effect. In addition, the recent CULPRIT-SHOCK trial showed no benefit with immediate complete revascularisation compared with culprit lesion-only PCI in STEMI patients

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with CS¹⁰. Thus, treatment other than revascularisation, such as mechanical circulatory support or a wearable cardioverter defibrillator, may be even more aggressive in a patient with CS and a concomitant CTO in a non-IRA. Larger-scale prospective trials are needed to explore their benefits in this selected patient population.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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