Editorial

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# The Concurrent Chronic Total Occlusion in a Non-Infarct Artery Strongly Associate With Poor Long-Term Prognosis in Patients With Acute Myocardial Infarction and Multivessel Coronary Disease

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Refer to the page 95-99

Recently, growing evidence demonstrates that recanalization of chronic total occlusion (CTO) lesions exerts a beneficial effect in terms of improvement in left ventricular function and long-term survival by alleviating residual/recurrent angina, and reduces the need for coronary artery bypass grafting.[1)2)] However, it remains unclear whether successful recanalization of a CTO in the non-infarct-related artery (non-IRA) could improve clinical outcomes in patients with acute ST-segment elevation myocardial infarction (STEMI). Angiography before primary percutaneous coronary intervention (PCI) has shown that multivessel coronary artery disease (MVD) is present in 40% to 65% of patients with STEMI and is associated with higher morbidity and mortality after reperfusion therapy.[3-5)] A concurrent CTO in a non-IRA is present in 12% to 13% of patients with STEMI.[3)5)]

In terms of primary PCI for STEMI, previous studies demonstrated that the effect of MVD on mortality is mainly determined by the presence of a CTO in a non-IRA.[4)6)] The presence of CTO lesions is a risk factor for incomplete PCI revascularization, which may, in turn, increase mortality compared to complete revascularization. Tajstra et al.[7)] described in a cohort of 1658 STEMI patients that the effect of MVD on mortality was primarily due to the presence of a CTO in a non-IRA, which was found to be a strong and independent predictor for both early mortality (within 30 days after STEMI) and late mortality (from 30 days to 5 years after STEMI). In contrast, MVD without a concurrent CTO was found only to be a relatively weak predictor for early mortality. Moreover, MVD without CTO lost its independent predictive value for mortality after excluding patients who died within 30 days after STEMI. However, in the relatively short-term follow-up period of 1 year, the majority of patients died within 30 days.[6)]

Lee et al.[8)] evaluated the impact of MVD with CTO on one-year mortality in patients with acute myocardial infarction (MI) including non-STEMI (about 50%). Results of this study are in line with those of the previous study which demonstrating that patients with MVD had a higher one-year mortality compared with SVD patients.[5)] Patients with MVD were older and had a more frequent history of diabetes, hypertension or previous MI, and a lower left ventricular ejection fraction as compared to SVD patients. Therefore, the mortality difference may be explained by a higher prevalence of associated risk factors in patients with MVD. In this study, CTO lesion was present in 20% of patients with MVD. CTO-PCI was attempted in 68% patients, while successful opening was obtained in 66% of the attempted cases. Patients with CTO lesions were older, more often diabetic, and had a more unfavorable Killip class compared with patients without the lesion. In the 88 patients with a CTO lesion, there were 11 deaths, all of which were of cardiac death. However, the presence of a CTO was not a significant predictor of one-year overall mortality and cardiac mortality of acute myocardial infarction (AMI) patients. This study had several limitations. First, this is a single-center retrospective study, and the number of patients was relatively small. Second, the average follow-up period was only 1 year, whereas a more prolonged follow-up is needed to investigate whether such beneficial effects persist over time. Finally, the study population was not uniform since patients with non-STEMI were about 52% of study subjects.

The high mortality rate of STEMI patients with a concurrent CTO

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may be partly explained by the greater risk profile of those with CTO. These patients tend to suffer from diabetes and have a previous MI, lower left ventricular ejection fraction, lower baseline Thrombolysis in Myocardial Infarction (TIMI) flow grades, and cardiogenic shock on admission more often than patients without CTO.[4)6)9)] Lexis et al.[10)] have demonstrated that the presence of CTO in a non-IRA after STEMI is associated with worse reperfusion markers and larger enzymatic infarct size. Another explanation of the underlying mechanism for the increased mortality in patients with STEMI with concurrent CTO could be that in patients with CTO, PCI was less successful. As well, a final TIMI flow grade <3 is strongly associated with a worse prognosis.[11)]

It was more equently present in the CTO group. Other factors that might contribute to adverse outcomes in patients with CTO include the lack of a compensation mechanism for the decrease in the left ventricular ejection fraction in AMI. Patients with MVD and concomitant CTO have a lower residual left ventricular ejection fraction and experience less improvement in left ventricular systolic function, strongly influencing survival.[6)]

Recently, Yang et al.[12)] demonstrated that successful staged revascularization (ranging 7-10 days) of a CTO in the non-IRA was associated with improved survival and reduced major adverse cardiac event in patients with acute STEMI treated with primary PCI. The pathologic process in STEMI involves the entire coronary tree and can lead to the destabilization and rupture of multiple atherosclerotic plaques, resulting in a significantly increased risk of death and repeated ischemic events.[13)] Muitivessel PCI in the prothrombotic milieu of the hyperacute phase of infarction could result in more adverse thrombotic events.[14)] Owing to the paucity of data regarding the optimal treatment of patients with STEMI and MVD, the need for, and timing of, subsequent revascularization of diseased non-IRA vessels remains controversial. Current practice guidelines in the acute setting recommend revascularization of diseased non-IRA vessels only in the presence of hemodynamic or electrical instability.[15)] Given the complexity of CTO angioplasty which requires a skilled and experienced operator staged revascularization of a CTO in the non-IRA after STEMI seems to be a reasonable method of treatment. Several studies have reported beneficial effects and demonstrated additional increases in left ventricular function after recanalization of a CTO, but only in the presence of viable myocardium.[16)17)] An adequately powered randomized controlled trial is warranted to investigate a possible benefit of opening a CTO early after STEMI. The ongoing Evaluating XIENCE V and left ventricular function in PCI on Occlusions after STEMI (EXPLORE) trial[18)] will investigate the effects of opening a concurrent CTO on outcomes in this high-risk subgroup of patients with STEMI.

## References


1. Valenti R, Migliorini A, Signorini U, et al. Impact of complete revascularization with percutaneous coronary intervention on survival in patients with at least one chronic total occlusion. *Eur Heart J* 2008;29:2336-42.
2. Joyal D, Afilalo J, Rinfret S. Effectiveness of recanalization of chronic total occlusions: a systematic review and meta-analysis. *Am Heart J* 2010;160:179-87.
3. Moreno R, Conde C, Perez-Vizcayno MJ, et al. Prognostic impact of a chronic occlusion in a noninfarct vessel in patients with acute myocardial infarction and multivessel disease undergoing primary percutaneous coronary intervention. *J Invasive Cardiol* 2006;18:16-9.
4. van der Schaaf RJ, Vis MM, Sjauw KD, et al. Impact of multivessel coronary disease on long-term mortality in patients with ST-elevation myocardial infarction is due to the presence of a chronic total occlusion. *Am J Cardiol* 2006;98:1165-9.
5. van der Schaaf RJ, Timmer JR, Ottervanger JP, et al. Long-term impact of multivessel disease on cause-specific mortality after ST elevation myocardial infarction treated with reperfusion therapy. *Heart* 2006;92:1760-3.
6. Claessen BE, van der Schaaf RJ, Verouden NJ, et al. Evaluation of the effect of a concurrent chronic total occlusion on long-term mortality and left ventricular function in patients after primary percutaneous coronary intervention. *JACC Cardiovasc Interv* 2009;2:1128-34.
7. Tajstra M, Gasior M, Gierlotka M, et al. Comparison of five-year outcomes of patients with and without chronic total occlusion of noninfarct coronary artery after primary coronary intervention for ST-segment elevation acute myocardial infarction. *Am J Cardiol* 2012;109:208-13.
8. Lee JH, Park HS, Ryu HM, et al. Impact of multivessel coronary disease with chronic total occlusion on one-year mortality in patients with acute myocardial infarction. *Korean Circ J* 2012:42:95-99.
9. Conde-Vela C, Moreno R, Hernández R, et al. Cardiogenic shock at admission in patients with multivessel disease and acute myocardial infarction treated with percutaneous coronary intervention: related factors. *Int J Cardiol* 2007;123:29-33.
10. Lexis CP, van der Horst IC, Rahel BM, et al. Impact of chronic total occlusions on markers of reperfusion, infarct size, and long-term mortality: a substudy from the TAPAS-trial. *Catheter Cardiovasc Interv* 2011;77:484-91.
11. The effects of tissue plasminogen activator, streptokinase, or both on coronary-artery patency, ventricular function, and survival after acute myocardial infarction. The GUSTO Angiographic Investigators. *N Engl J Med* 1993;329:1615-22. Erratum in: *N Engl J Med* 1994;330:516.
12. Yang ZK, Zhang RY, Hu J, Zhang Q, Ding FH, Shen WF. Impact of successful staged revascularization of a chronic total occlusion in the non-infarct-related artery on long-term outcome in patients with acute ST-segment elevation myocardial infarction. *Int J Cardiol* 2011 Aug 25 [Epub]. http://dx.doi.org/10.1016/j.ijcard. 2011.07.074.
13. Goldstein JA, Demetriou D, Grines CL, Pica M, Shoukfeh M, O'Neill WW. Multiple complex coronary plaques in patients with acute myocardial infarction. *N Engl J Med* 2000;343:915-22.
14. Barrett TD, Hennan JK, Marks RM, Lucchesi BR. C-reactive-protein-associated increase in myocardial infarct size after ischemia/reperfu-

sion. *J Pharmacol Exp Ther* 2002;303:1007-13.
15. van der Schaaf RJ, Claessen BE, Vis MM, et al. Effect of multivessel coronary disease with or without concurrent chronic total occlusion on one-year mortality in patients treated with primary percutaneous coronary intervention for cardiogenic shock. *Am J Cardiol* 2010;105:955-9.
16. Sirnes PA, Myreng Y, Mølstad P, Bonarjee V, Golf S. Improvement in left ventricular ejection fraction and wall motion after successful recanalization of chronic coronary occlusions. *Eur Heart J* 1998;19:273-81.
17. Chung CM, Nakamura S, Tanaka K, et al. Effect of recanalization of chronic total occlusions on global and regional left ventricular function in patients with or without previous myocardial infarction. *Catheter Cardiovasc Interv* 2003;60:368-74.
18. van der Schaaf RJ, Claessen BE, Hoebers LP, et al. Rationale and design of EXPLORE: a randomized, prospective, multicenter trial investigating the impact of recanalization of a chronic total occlusion on left ventricular function in patients after primary percutaneous coronary intervention for acute ST-elevation myocardial infarction. *Trials* 2010;11:89.


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