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INCIDENCE AND PREDICTORS OF LEFT VENTRICULAR MURAL THROMBUS AFTER ST-ELEVATION MYOCARDIAL INFARCTION IN THE CONTEMPORARY ERA OF PRIMARY PCI

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Background In patients presenting with ST elevation myocardial infarction (STEMI), early and aggressive reperfusion therapy increases myocardial salvage and improves survival. Left ventricular mural thrombus (LVT) is more likely to occur after full thickness infarction. The aim of this study was to define the incidence of LVT and its predictors in the contemporary era of effective primary percutaneous coronary intervention (PCI).

Methods We retrospectively analysed 1220 patients with STEMI admitted directly to our centre for primary PCI (1 January 2009–31 August 2012). Patients received dual antiplatelet and anticoagulant therapy at the operator's discretion (unfractionated heparin, bivalirudin and/or abciximab). A transthoracic echocardiogram (TTE) was performed in all cases prior to discharge. Echo-contrast was used when appropriate.

Results LVT was detected by TTE in 41 patients (3.9%). There were no significant differences in baseline demographic in patients with and without LVT. LV systolic function, an anterior location of infarction and apical akinesis all predicted LVT. Univariate analysis also demonstrated a relationship between LVT and ejection fraction (EF). Mean EF in patients with LVT was 34% (95% CI 31.7 to 36.6); without LVT EF was 48% (95% CI 47% to 48.6%) p<0.01. 37 patients with LVT (90%) presented with an anterior MI. Apical akinesis was noted on TTE in all the patients with LVT irrespective of the main location of the MI. After diagnosis of LVT patients were treated with warfarin for 3–6 months. There was no difference in mortality at discharge between the two groups. Neither chest pain onset-to-balloon time, final TIMI flow post-PCI, use of thrombectomy catheter nor abciximab use predicted the occurrence of LVT.

Conclusions In the contemporary era of primary PCI, the incidence of LVT in patients with STEMI is significantly lower than the previous literature suggests. Formation of LVT is predicted by anterior location of MI, reduced LV systolic function and the presence of an apical akinetic segment.

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