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Why thrombus aspiration in acute STEMI fails: could plasminogen activator inhibitor 1 be the culprit?

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THEME: Coronary Interventions

TOPIC(S): STEMI

AIMS

To determine whether the use of thrombus aspiration (TA) during primary percutaneous coronary intervention (PCI) affects plasminogen activator inhibitor 1 (PAI-1) activity levels during acute phase ST elevation myocardial infarction (STEMI).

METHODS AND RESULTS

A total of 87 consecutive acute STEMI patients with symptoms duration of <12 hours were enrolled in the study. Enrolment period was from May 1, 2009 to March 23, 2010. Blood samples were collected prior to PCI and after exactly 24 hours. All PCIs were performed using transfemoral approach, according to contemporary guidelines. Dual antiplatelet therapy consisted of aspirin and clopidogrel. Eptifibatide as well as TA were indicated by interventional cardiologist on duty. Repeated aspirations were allowed. Thrombus burden was determined by thrombolysis in myocardial infarction (TIMI) thrombus grade, and was documented for the initial diagnostic angiogram (TG1), and, in patients with occluded coronary artery, after restoring antegrade flow by means of guidewire advancement or small balloon dilatation (TG2). Endpoints were established after 5 years, and were defined as follows: primary as death, secondary as composite of death, stroke and recurrent acute coronary syndrome. Study population was predominantly male (72.3%), aged 61.1 ± 12.2 , with high prevalence of hypertension (66.8%) and active smoking status (48.3%). TA was performed in 12 patients (13.8%). In TA group, higher PAI-1 activity in second sample (7.32 vs 4.60 U/mL, $p=0.019$) and higher PAI-1 activity rise (3.25 vs 1.20, $p=0.003$) were detected, while there was no difference in first sample values. Use of balloon angioplasty was not associated with the difference in PAI-1 activity rise. However, when patients were grouped according to interventional procedures employed (none, balloon angioplasty only, TA only, both), the difference was significant, with the highest value in the group treated with both balloon angioplasty and TA ($p=0.015$). TIMI thrombus grades (TG1 and TG2) did not correlate with PAI-1 activity rise (for TG1 $p=0.289$, for TG2 $p=0.596$). Both TG1 and TG2 values showed no significant difference in groups of patients with or without use of TA (for TG1 $p=0.054$, for TG2 $p=0.966$). There were no differences in primary and secondary endpoints at 5-year follow-up in groups of patients with or without use of TA.

CONCLUSIONS

TA was associated with higher PAI-1 activity rise in acute STEMI patients treated with primary PCI. The clinical implications of this association should be tested in larger studies, and if proved significant, additional efforts to produce and test PAI-1 antagonists should be employed.