Dokumenttyp: journal article
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Abstract: The Pl(A) polymorphism of the platelet glycoprotein IIIa gene is associated with altered platelet function and response to antiplatelet drugs. We sought to assess whether the Pl(A) polymorphism influences myocardial salvage achieved by reperfusion therapy in patients with acute myocardial infarction. We analyzed 292 patients enrolled in 2 randomized trials that compared stenting plus abciximab with thrombolysis (alteplase alone or alteplase plus abciximab) in acute myocardial infarction. Patients were genotyped for the Pl (A) polymorphism using polymerase chain reaction with fluorogenic probes. Technetium-99m sestamibi was injected before and 1-2 weeks after reperfusion treatment. The scintigrams enabled the calculation of the initial perfusion defect, final infarct size, and the proportion of initial defect salvaged by reperfusion (salvage index). Clinical follow-up was done up to 18 months after primary treatment. The genotype distribution was as follows: Pl (A2/A2) in 3.4%, Pl (A1/A2) in 24.7% and Pl (A1/A1) in 71.9% of patients. There were no significant differences between Pl(A2) allele carriers and Pl(A1/A1) patients in salvage index (0.4+/-.0.50 vs. 0.4+/-.0.43, respectively, P=0.48), final infarct size (16.8+/-.20.8% vs. 18.4+/-.19.1% of left ventricle, respectively, P=0.46) as well as 18-month mortality (8.5% vs.7.1%, respectively, P=0.69). The lack of
relationship between PI(A2) allele and myocardial salvage was observed for both reperfusion strategies, stenting and thrombolysis. Thus, these findings show that the functional PI(A) polymorphism of platelet glycoprotein IIIa has no influence on the degree of myocardial salvage achieved by reperfusion therapies in patients with acute myocardial infarction.

Zeitschriftentitel / Abkürzung:
Thromb Haemost

Jahr:
2004

Band:
91

Heft / Issue:
1

Seiten:
141-5

Sprache:
eng

Pubmed:

Print-ISSN:
0340-6245

TUM Einrichtung:
I. Medizinische Klinik und Poliklinik

Occurences:
- Einrichtungen > Fakultäten > Fakultät für Medizin > Kliniken und Institute > I. Medizinische Klinik und Poliklinik (Kardiologie) > 2004

entries: