İzmir Eğitim ve Araştırma Hastanesi Tıp Dergisi,2014;18(2):18-26

ARAŞTIRMA MAKALESİ

ST ELEVASYONLU MİYOKARD İNFARKTÜS HASTALARINDA HEMATOLOJİK PARAMETRELER VE BAŞARISIZ TROMBOLİTİK

HEMATOLOGICAL PARAMETERS AND THROMBOLYTIC FAILURE IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION PATIENTS

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ÖZET

Trombolitik ajanlardaki son gelişmelere rağmen reperfüzyon kriterlerini sağlayan hastaların bir kısmında halen infarkt ilişkili damar (İİD) açıklığı sağlanamamaktadır. Hastaneye başvurudaki hematolojik parametrelerin trombolitik tedavi başarısı ile ilişkisi net değildir. Çalışmamızda akut ST elevasyonlu miyokard infarktüsü (STEMİ) hastalarında hastaneye başvuru hematolojik parametreleri ile trombolitik tedavi başarısı arasındaki ilişkinin incelenmesi amaçlanmıştır.

Trombolitik tedavi alan akut STEMİ hastalarından trombolitik tedavi alanlar retrospektif olarak analiz edilmiştir. Başarısız trombolitik, kurtarıcı perkütan koroner girişim ihtiyacı veya hastane içi mortaliteveya koroner anjiyografide İİD'de TİMİ akım skorunun 0 veya 1 olması olarak tanımlanmıştır. Başarılı veya başarısız trombolitik ile sonuçlanan hastaların verileri karşılaştırılmıştır.

Çalışmaya dahil edilen 233 hastadan 152'inde başarılı tromboliz sağlanmıştır. Ortalama platelet hacmi (OPH) ve nötrofil sayısı başarısız trombolitik grubunda anlamlı derecede yüksek saptanmıştır (Sırası ile 9.2±1.3 fl vs 8.6±1.3 fl, p=0.002 ve sırası ile 9.1±4.2 X 10^3/µL vs 7.6±3.6 X 10^3/µL, p=0.006). İki grup arasında nötrofil dışı beyaz kan hücre sayısı ve diğer hematolojik parametreler açısından anlamlı fark saptanmamıştır. İkili lojistik regresyon analizinde nötrofil sayısı ve OPH başarısız trombolizi öngörmede bağımsız faktörler olarak saptanmıştır.

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Akut STEMİ hastalarında, hastaneye başvuru esnasında saptanan yüksek OPH ve nötrofil sayısı anlamlı olarak başarısız tromboliz ile ilişkili bulunmuştur.

Anahtar Kelimeler: Akut ST elevasyonlu miyokard infarktüsü, tromboliz, ortalama platelet hacmi, nötrofil.

ABSTRACT

Despite recent advances in thrombolytic agents, a significant number of patients fulfilling the reperfusion criteria still fail to accomplish complete patency of infarct related artery (IRA). Therelationshipof hospital admission hematological parameters with thrombolytic outcome is not clear. We aimed to evaluate the relationship of hospital admission hematological parameters with thrombolytic outcome in acute ST elevation myocardial infarction (STEMI) patients.

Records of acute STEMI patients receiving thrombolytic treatment on hospital admission were retrospectively analysed. Thrombolysis failure was defined as the need for rescue percutaneous coronary intervention, or in-hospital mortality, or Thrombolysis in Myocardial Infarction flow grade 0 or 1 in IRA in coronary angiography. The data of the patients with successful or failed thrombolysis were compared.

Of the 233 patients, 152 had successful thrombolysis. Mean platelet volume (MPV) and neutrophil count were significantly higher in patients with failed thrombolysis (9.2±1.3 fl vs 8.6±1.3 fl, p=0.002 and 9.1±4.2 X 10^3/µL vs 7.6±3.6 X 10^3/µL, p=0.006). Non-neutrophil white blood cell countand the other hematological indices were not different between two groups. Neutrophil count and MPV were shown as independent factors predicting thrombolysis failure in binary logistic regression analysis.

Elevated MPV and neutrophil count on hospital admission were significantly related to failed thrombolysis in acute STEMI patients.

Keywords: Acute ST elevation myocardial infarction, thrombolysis, mean platelet volume, neutrophil.

INTRODUCTION

Acute STEMI is a major cause of mortality and morbidity (1,2). Rupture of atheromatous plaque and formation of intracoronary thrombus are the underlying pathophysiological processes in STEMI (3).Optimal treatment of acute STEMI is the early and complete mechanical or pharmalogical reperfusion of IRA (1,2). Intravenous thrombolytics are widely used for reperfusion but it is clear that reperfusion fails in a significant proportion of patients. Successful reperfusion criteria defined by clinical parameters and post-thrombolytic electrocardiography does not always necessarily mean a successful thrombolysis and complete reperfusion of IRA. Up to 60% of the patients fulfilling the reperfusion criteria, fail to achieve Thrombolysis in Myocardial Infarction (TIMI) grade 3 coronary flow at 90 minutes (4,5). Failed thrombolysis indicates a worse prognosis (6,8).

Studies have reported the association of hematological parameters with various cardiovascular diseases (9-11).However, less is known about their relationship with thrombolytic outcome in STEMI. Hematological indices may be helpful in early identification of acute STEMI patients who would have failed thrombolysis. In the current study we evaluated the relationship of hematological parameters measured on hospital admission with thrombolysis failure in acute STEMI.

MATERIAL AND METHODS

The study was a retrospective review of patients admitted to our hospital

between January 2009 and December 2011 with a diagnosis of acute STEMI and received thrombolytic therapy.Our study protocol was approved by institutional ethics committee.Acute STEMI was defined as typical chest pain lasting for at least 30 minutes with ST-segment elevation ≥1 mm in at least two consecutive precordial or inferior leads. Exclusion criteria included active infection, chronic inflammatory disease, neoplastic disease, known endocrine or metabolic disease other than obesity or diabetes mellitus (DM), cardiogenic shock on admission, treatment with thrombolytic therapy in the previous 24 hours. We reviewed the medical, angiographic and invasive procedural records of the patients.Patients were divided in two groups based on whether or not thrombolytic therapy was successful. Failure of thrombolytic therapy was defined according to patient's clinical status, post-thrombolytic electrocardiography, and patency of IRA in coronary angiography. The diagnosis of failed thrombolysis was based on one or more of the following criteria:

1- Ongoing chest pain, hemodynamic instability and/or <50% ST-segment resolution on follow-up electrocardiogram at 90 minutes after the initiation of thrombolytic therapy and requiring rescue percutaneous coronary intervention (PCI);

2- In-hospital mortality;

3- TIMI flow grade 0 or 1 in the IRA in coronary angiography.

Demographic data, medical history of cardiovascular (CV) risk factors and CV diseases, clinical and electrocardio-graphic evaluation, concomittant illnesses, in-hospital adverse outcomes, in-hospital mortality, medications, and admission complete blood count prior to the administration of aspirin, clopidogrel, and thrombolytic agents were provided from the medical records. Samples for total blood cell count measurements were taken into standardized tubes containing dipotassiumethylene-dinitro tetra aceticacid (EDTA) and the measurements were determined on Abbott Cell- Dyn 3700 autoanalyser using commercial assay kits (Abbott Diagnostic, CA, USA). Coronary flow was assessed with the TIMI flow grading system (12). Number of diseased vessels, IRA, TIMI flow grade, and use of stents were obtained from the computerized catheterization laboratory database.

All patients were treated with aspirin 300 mg on admission and 100 mg daily thereafter. Loading dose of 300 mg clopidogrel was applied if the patients aged ≤75 years, followed by a maintenance dose of 75 mg/day. If the patient aged >75 years only maintenance dose of 75 mg was given. Streptokinase (SK), tissue plasminogen activator (tPA), tenecteplase (TNK-tPA) were the thrombolytic agents administered to the patients(1). The choice of thrombolytic agent was based on the decision of the physician who prescribed the treatment. Weight based anticoagulation with enoxoparine was applied until revascularization was performed or for the duration of hospital stay up to eight days.

A routine coronary angiography was planned for all patients. Patients with clinical and electrocardiographic signs of failed thrombolysis underwent rescue PCI. Administration of glycoprotein (GP) IIb/IIIa antagonist and the use of thrombus aspiration device were left at the discretion of the managing operator in the catheterization laboratory.

Statistical methods:

Statistical analyses were performed using computer software (SPSS version 17.0, SPSS Inc. Chicago, IL, USA). Chi-square (χ^2) and Fisher's exact tests were used for the comparison of categorical data, while the student's t test and Mann-Whitney U-test were used for the analysis of parametric variables based on the distribution pattern of data. Binary logistic regression was conducted to determine the hematological factors that contribute to thrombolysis failure. A stepwise selection method was used in the binary logistic analysis. Data were expressed as the mean (standard

deviation; SD), median (IR) and n (%) where appropriate. A *p* value < 0.05 was considered statistically significant.

RESULTS

Two hundred and thirty three consecutive acute STEMI patients of whom the average age was 59.8 ± 12.0 and 190 were males (81.5%) were included in the study. According to our thrombolytic failure definition, successful thrombolysis was achieved in152 patients (65.2%). The agents used for thrombolysis were SK (12.9%), tPA (72.5%) and TNK-tPA (14.6%).

Among patients with failed thrombolysis, 29 out of 81 (35.8%) were undergone rescue PCI. Records of 4 patients were missing in the computerized catheterization laboratory database. A routine coronary angiography was planned for 204 patients. Of those, 9 patients died before the procedure while another 4 patients refused it. Sixty four patients with failed thrombolysis (79.0%) had TIMI flow grade 0-1 in the IRA. Among patients with failed thrombolysis a total of 19 patients (23.4%) died.

The main demographic, clinical, and laboratory features of the subjects are shown in Table 1. The mean age of the patients with failed thrombolysis were significantly higher compared to those with successful thrombolysis (62.6±12.2 vs 58.2±11.7, p=0.008). The prevalance of the patients with DM and the prevalance of the patients with Killip class > 1 were also significantly higher in failed thrombolysis group (38.3% vs 19.7%, p=0.003 and 23.5% vs 1.3%, p<0.001, respectively). The two groups were comparable in terms of gender, smoking habit, hypertension, hyper lipidemia, past or family history of CV diseases, and chronic aspirin treatment. Type of thrombolytic regimen, the localization of MI, number of diseased vessels, IRA, acute stent thrombosis, administration of GP IIb-IIIa inhibitor, and the length of the stent were not significantly different between two groups (Table1). In-hospital mortality, procedural complications, and use of intra-aortic balloon pump (IABP) were significantly higher in failed thrombolysis group (p<0.05). Thrombus in the IRA and the use of thrombus aspiration devices during coronary intervention were significantly more common in the failed thrombolysis group (p<0.001, p=0.013, respectively).

MPV and neutrophil count were significantly higher in patients with failed thrombolysis compared to those with successful thrombolysis (9.2±1.3 fl vs 8.6±1.3 fl, p=0.002 and 9.1±4.2 X 10^3/µL vs 7.6±3.6 X 10^3/µL, p=0.006, respectively). Non-neutrophil white blood cell (WBC) count and the other hematological indices were not different between two groups (Table 1). Binary logistic regression analysis determined MPV (odd's ratio 1.396, 95 % confidence interval 1.127-1.730, p=0.02) and neutrophil count (odd's ratio 1.106, 95 % confidence interval 1.029-1.189, p=0.006) as independent predictors of thrombolytic failure.

DISCUSSION

The principal finding of this study is that elevated MPV and neutrophil count on hospital admission were closely related to failed thrombolysis. There are few data in the literature regarding the value of hematological parameters on throm bolytic failure in STEMI. Studies about MPV and neutrophil count in STEMI were mostly conducted among patients who underwent primary PCI (13,14). In these, MPV (14) and WBC count (13) were suggested as valuable parameters in predicting IRA patency and mortality. Furthermore, neutrophil count was shown as the best independent predictor of mortality in patients with acute STEMI among all WBC subtypes (15).

		Successful thrombolysis (n=152)	Failed thrombolysis (n=81)	*p*
Age (mean ± SD)		58,2 ± 11,7	62,6 ± 12,2	**0,008**
Male gender (%)		128 (84,2)	62 (76,5)	0,160
Smoking (%)	Current	89 (58,6)	38 (46,9)	0,232
	Past	5 (3,3)	3 (3,7)	
Diabetes mellitus (%)		30 (19,7)	31 (38,3)	**0,003**
Hypertension (%)		70 (46.1)	37 (45,7)	1,000
Hyperlipidemia (%)		63 (41,4)	25 (30,9)	0,121
History of CVD (%)		37 (24,3)	18 (22,2)	0,749
Family history of CVD (%)		28 (18,4)	9 (11,1)	0,188
Chronic aspirin treatment (%)		36 (23,7)	19 (23,5)	1,000
Decompensated heart failure on admission> Killip Class I (%)		2 (1,3)	19 (23,5)	**0,000**
Thrombolytic regimen (%)	SK	16 (10,5)	14 (17,3)	
	tPA	115 (75,7)	54 (66,7)	0,267
	TNK-tPA	21 (13,8)	13 (16,0)	
Localization of MI (%)	Anterior	54 (35,5)	31 (38,3)	
	Anteroseptal	15 (9,9)	4 (4,9)	
	İnferior	51 (33,6)	26 (32,1)	0,617
	İnferoposterolateral	19 (12,5)	14 (17,3)	
	İnferior and RV	13 (8,6)	6 (7,4)	
Number of diseased vessels (%)	One	65 (42,8)	23 (33,8)	
	Two	40 (26,3)	21 (30,9)	0,456
	Three or more	47 (30,9)	24 (35,3)	
IRA (%)	LAD	68 (44,7)	28 (41,2)	
	Cx	24 (15,8)	9 (13,2)	0,598
	RCA	58 (38,2)	31 (45,6)	
	SVG	2 (1,3)	0 (0,0)	
Acute stent thrombosis (%)		2 (1,9)	2 (3,9)	0,600
Stent length (mm) (mean ± SD)		29,1 ± 19,3	31,1 ± 17,9	0,541

In-hospital morbidity (%)	26 (17,1)	38 (46,9)	**0,000**
In-hospital mortality (%)	0 (0)	19 (23,5)	**0,000**
Procedural complications (%)	8 (5,3)	17 (23,6)	**0,000**
IABP	2 (1,3)	14 (17,3)	**0,000**
Thrombus in the IRA	19 (12,5)	30 (44,1)	**0,000**
Thrombus aspiration device	5 (3,3)	9 (13,2)	**0,013**
GP IIb-IIIa inhibitor administration	14 (13,6)	8 (14,5)	1,000
Haematologic parameters (mean ± SD)			
Hemoglobin (gr/dl)	14,5 ± 1,5	14,1 ± 1,8	0,106
MCV (fl)	85,8 ± 7,1	86,9 ± 4,9	0,197
RDW (%)	15,3 ± 1,4	15,1 ± 1,1	0,339
Neutrophil count ($X10^3/\mu L$)	7,6 ± 3,6	9,1 ± 4,2	**0,006**
Neutrophil (%)	67,9 ± 62,9	67,1 ± 15,5	0,920
Non-neutrophil count ($X10^3/\mu L$)	4,2 ± 2,0	4,3 ± 2,2	0,869
Platelet count ($X10^3/\mu L$)	268,8 ± 65,8	262,6 ± 62,7	0,485
MPV (fl)	8,6 ± 1,3	9,2 ± 1,3	**0,002**

Table. 1 (continue from page 22) Demographic, clinical, and laboratory features of successful and failed thrombolysis groups

CVD: cardiovascular disease; SK: Streptokinase; tPA: tissue plasminogen activator; TNK-tPA: tenecteplase; MI: myocardial infarction; RV: right ventricule; IRA: infarct related artery; LAD: left anterior descending artery; CX: circumflex artery, RCA: right coronary artery; SVG: Saphenous vein graft; IABP: intraaortic balloon pump; GP: glycoprotein; MCV: mean corpuscular volume; RDW: red cell distribution width; MPV: mean platelet volume.

In another study, higher baseline neutrophil count was associated with worse angiographic findings and increased CV mortality in STEMI patients undergoing thrombolysis (16). In previous studies, MPV was suggested as an adjunctive parameter for assessing thrombolysis outcome (17) and IRA patency after thrombolytic treatment (18). Herein,in addition to IRA patency, we also considered the clinical status andpost-thrombolytic electrocardiograp hyfor failed thrombolysis. In our study, MPV and neutrophil count were found as independent predictors of thrombolysis failure.Thus, among hematological parameters evaluation of admission MPV and neutrophil countappeared to have an adjunctive role in identifying patients with failed thrombolysis.

In the present study, a high MPV and an elevated neutrophil count were significantly associated with more often thrombus formation in the IRA. Furthermore, thrombus aspiration devices were significantly used more in patients with failed thrombolysis. Administration of GP IIb–IIIa inhibitor was not different between two groups. It has been hypothesized that the procoagulant activity of circulating WBC could be increased by the adherence of activated platelets to polymorphonuclear WBC (19). Greater thrombus burden in patients with an elevated WBC count in acute MI was demonstrated in a study by Barron et al (20). Larger platelets express more adhesion receptors, such as GP Ib and GP IIb–IIIa (21). A recent study by Huczek et al. (13) showed that only subjects with elevated MPV had a mortality reduction from abciximab among STEMI patients. Higher baseline neutrophil count was associated with diminished benefit of clopidogrel in another study (16). Therapies directed against platelets may indirectly influence the inflammatory cascade (16). Neutrophil count and MPV routinely measured on admission may provide evidence for additional adjunctive antiplatelet treatment.

Ideal treatment of acute STEMI is primary PCI; however, this is not always the case in real world. Thrombolysis still remains the most commonly used treatment (1,4). When thrombolysis fails, rescue PCI is performed. The efficacy of rescue PCI has always been debated and conflicting data on mortality have been reported (2,22). In our study, mortality in patients treated with rescue PCI for failed thrombolysis was 38.1% and overall in-hospital mortality rate was 19/233 (8.2%). Mortality is still high in patients treated with rescue PCI for failed thrombolysis; a trend towards reduced mortality in rescue PCI has been demonstrated in recent studies (22). A recent study by Cetin et al. (23) showed that MPV and platelet distribution width (PDW) were independent predictors of thrombolysis failurein patients with acute STEMI. Ravan et al. (24) have speculated that acute STEMI patients having higher MPV, PDW and WBC countat admission should be considered for stronger antiplatelet treatment to be able to attain a favorable ST resolution and better clinical outcome. Since both of these studies have a retrospective design, further studies evaluating these patients prospectively on the basis of their treatment in future studies should be arranged in order to gain a a better clinical outcome. Therefore, upon hospital admission identifying the patients who will have a high risk for failed thrombolysis becomes important especially forthe patients presenting to a non-PCI-capable hospital. Our study revealed that such patients may be identified for thrombolytic failure via routine admission haematological analysis, hence, they can be considered to betransferred to a PCI-capable center earlier after thrombolytic administration.

Precise determination of MPV by the appropriate anticoagulation method is important. Furthermore, measurement of MPV in EDTA can be unreliable due to platelet swelling which may probably be because of different amounts of EDTA in the blood tubes (25). However, a study revealed that MPV can be measured accurately by both methods of anticoagulation; EDTA and citrate if analysis be performed within 1 h of sampling (26). In our study, same standardized blood tubes were used and

blood samples were analyzed within 1 hour after blood sampling.

CONCLUSIONS

Elevated MPV and neutrophil count on hospital admission might be adjunctive parameters in predicting thrombolysis outcome in acute STEMI patients.Our findings warrant further prospective studies that include larger study cohorts.

Abbreviations:

CV: cardiovascular; DM: diabetes mellitus; EDTA: ethylene-dinitro tetraacetic acid; GP: glycoprotein; IRA: infarct related artery; IABP:intra-aortic balloon pump; MPV: mean platelet volume; PCI: percutaneous coronary intervention; PDW: platelet distribution width, STEMI: ST elevation myocardial infarction; SK: Streptokinase; TNK-tPA: tenecteplase; TIMI: Thrombolysis in Myocardial Infarction;tPA: tissue plasminogen activator;WBC: white blood cell.

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Yazının alınma tarihi: 10.03.2014
Kabül tarihi: 22.04.2014
Online basım: 24.04.2014