The Frequency of Cerebral Microembolism in Acute Myocardial Infarction

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ABSTRACT

Introduction: Stroke is more common in patients with cerebral microembolisms. Frequency of cerebral microembolisms (high intensity transient signals, HITS) in acute myocardial infarction has been reported about 17%. The factors that influence on microembolism after myocardial infarction (MI) are not definitive. Type of MI, Ejection fraction, Hx of Streptokinase is the factors that were studied.

Methods: During three years we studied the frequency of cerebral microembolisms in AMI patients, we studied forty patients with microembolism as a case group and ninety patients without microembolism as a control group. We detected microembolism in patients by transcranial doppler study within 72 houre after myocardial infarction. Two-dimensional echocardiogram was performed for all patients during hospitalization. Excluding criteria were prosthetic heart valves, carotid stenosis >50% and poor window for TCD monitoring.

Results: number of patients who had history of receiving SK were significantly more common in case group in comparison to control group. OR 2.4 CI(1.1-5.2) The frequency was more prevalent in anterolateral MI in comparison to inferior MI.OR=3.3 CI(1.4-7.4). Ejection fraction has no significant effect on frequency of microembolism. OR 0.5 CI(0.2-1.3).Hypokinesia is also a risk factor for increasing risk of microembolism. OR 4.5 CI(1.4.13.8)

Discussion: frequency of microembolism has been increased in patients with history of streptokinase or in the type of Anterolateral MI or wall motion abnormality, so we should be careful for risk of microembolism in this groups.

Key Words:
Microembilism,
Stroke,
Streptokinase

1. Introduction

There are several factors for stroke. Embolism has been reported in 30-33% of patients as the etiology of stroke (Neto et al. 1997). The prevalence of microembolic signals in stroke patients referred for echocardiography is high. David and colleagues reported that microemboli were detected in 11% of patients (David et al. 1997). Microembolism is one of the risk factor of stroke (Tong et al. 1994). They can be detected by transcranial Doppler (TCD) as high intensity transient signal (HITS) (Christensen et al. 1997). Microembolisms arise from prosthetic heart valves, atrial fibrillation, severe heart failure and carotid stenosis (Slivka et al. 1997). Other study have confirmed clinical significance of Microem-

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bolism in patients with carotid disease (Georgiadis et al. 1998).Acute myocardial infarction(AMI) can also increase the frequency of microembolism (Sliwka et al 1998). The reported incidence of stroke during hospitalization for AMI is 0.9% to 2.4%. Thirty-three percent of these cases occur within 24 hours and about 70% within the first week (Nadareishvili et al 1999).

Based on some reports it is assumed that using thermolysis medications such as strepkinase could be a risk factors for microembolisms (Abraham et al. 1994). Therefore the aim of this study was to explore about this hypothesis.

2. Methods

A case-control study was carried out in patients who had been admitted to the coronary care unit in Firoozgar Teaching hospital affiliated to Iran University of Medical Sciences between February 2006 and January 2009. Forty patients with AMI who had microembolisms were considered as a case group and the control group consisted of ninety patients with AMI without microembolism.

AMI was diagnosed according to the established clinical and electrocardiographic criteria that were confirmed by elevation of cardiac enzymes. Treatment of patients with thrombolytic (medications) drugs was based on the decision of cardiologists and they managed patients according to their diagnosis and only if there was any contraindication for thrombolytic therapy, patients did not receive streptokinase. Patients with prosthetic heart valves, atrial fibrillation, carotid stenosis more than 50% and poor temporal window for insonation were excluded.

History of previous stroke or TIA and other past medical history for all patients were collected from their clinical files and records. Neurological exam were performed for all the patients before the TCD monitoring. Two-dimensional echocardographic studies were performed in all patients with 2.5 MH transcducer (Esaote S.P.A.). LVEF and LV wall motion abnormalities (hypokinetic/akinetic) were assessed and echocardiograms were interpreted by experienced cardiologists blind to the result of the TCD recordings.

TCDs were performed with Explorer CVS (DMS) with headset included bilateral 2MH transducer in the first 72 hours after the patients AMI onset. After identification of both middle cerebral arteries (MCA) via temporal windows, the probes were fixed and recording were performed for 30 minutes. Depth of insonation of the MCA at two points of 50-55 mm and 55-60 mm were recorded.

The TCD device was equipped with a software program that could detect online HITS, which were saved on hard disk. During the monitoring, the investigator was present to watch for the patient's movement and to detect microembolisms himself by hearing. The patients with poor temporal window for insonation or without good cooperation were excluded from the study. Microembolisms were identified as a predominantly unidirectional short duration, intensity increase accompanied by a characteristic clicking or chirping sound.11All microembolisms were analyzed again after recording by experienced observer blinded to the clinical presentation of the patient.

Table1. Cerebral Microemboli in AMI, Demographic data
MI indicates myocardial infarction; LV left ventricle;
LVEF left ventricular ejection fraction;
HITS % means percentage of patients with microembolic signals.

Gender	
FEMALE	63(33.1%)
MALE	87(66.9%)
AGE	61.9(12.9)
<60	60(66.2%)
>60	70(53.8%)
MI	
ANT	69(53.5%)
INF	61(46.9%)
LVEF	61.0(12.0)
<65	43(33.1%)
>65	87(66.9%)
WALL MOTION	
HYPOKINETIC	96(73.8%)
NORMAL	36(26.2%)
STREPTOKINASE	
POSITIVE	65(50%)
NEGATIVE	65(50%)
HITS	
POSITIVE	40(30.8%)
NEGATIVE	90(69.2%)

3. Results

Demographic data of population study is shown in table 1. Male was dominant (70%) and near half of patients were more than 60 years old. Approximately 74% had hypokenitic wall motion in their exam. Seventy percent was microembolism negative.

We considered gender, age, type of MI, LVEF, wall motion and using streptokinase as independent variables in our model. Anterolateral MI was shown to be increase the likelihood of having microembolism in comparison with Inferior MI (OR=3.3 CI=1.4-7.4). Hypokenetic wall motion shown to raise the risk of developing of HITS and this difference was statistically significant (P=0.005). Patients who were treated by Streptokinase had more in the microembolism compared with patients who did not treated (OR=2.4 CI=1.1-5.2). This was statistically significant (P=0.03).

4. Discussion

The most important finding of our study is that streptokinase should be considered as a risk factor for microembolisms in AMI patients (p=0.034). Nadershivili and colleagues found similar results (Nadareishvili et al 1999). However it was not statistically significant, they showed that there was a trend toward an increase in the frequency of microembolisms in patients treated with thrombolysis.(22.2% versus 12.7% p=0.285).

The possible explanation of this phenomen could be that thrombolytic therapy may cause disintegration of thrombus in left ventricle or arterial walls (such as carotid plaque). The same explanation was described in another study regarding developing skin necrosis in patients treated by streptokinase These cases demonstrate the potential microembolic events that may occur following the use of thrombolytic agents (Abraham et

Table 2. Frequency of HITS in both patients who treated with SK or didn't receive SK

VARIABLE	Case (HITS POSITIVE) N=40	Control Group (HITS Negative) N=90	Odds Ratio	95% Confidence Interval	P- Value
Gender					
FEMALE	11	32	0.6		0.42
MALE	29	58	Ref	0.3-1.5	
AGE					
<60	23	37	0.6		0.09
>60	17	53	Ref	0.9-4.1	
MI					
ANT	29	40	3.3		0.09
INF	11	50	Ref	1.4-7.4	
LVEF					
<65	10	57	0.5		0.22
>65	30	33	Ref	0.2-1.3	
WALL MOTION					
HYPOKINETIC	36	60	4.5		0.005
NORMAL	4	30	Ref	1.4-13.8	
STREPTOKINASE					
POSITIVE	26	39	2.4		0.03
NEGATIVE	14	51	Ref	1.1-5.2	

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al. 1994) . Cholesterol embolization of atheromatous plaque has previously been implicated in peripheral and visceral infarction following intravenous streptokinase, (Schwartz et al 1987),(Pearson et al 1987) or intravenous tissue plasminogen activator.(Arora et al. 1993) Cholesterol embolization following thrombolysis may arise from an ulcerated atheromatous plaque exposed by clot dissolution. If this is the case, then microembolism by cholesterol and microthrombus represent two pathological features of the same process. Thrombolytic agents may also lyse left atrial thrombus leading to large vessel occlusion. (Blaze et al. 1986)

To obtain a firm clinical and practical conclusion would be difficult from the findings of this study therefore we would suggest further studies with larger sample size.

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