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THE RELATIONSHIP OF ABNORMAL YAWNING WITH CARDIAC ARREST

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ABSTRACT

Objectives: The aim of this study was to investigate the relationship between cardiac arrest and abnormal yawning in patients undergoing primary percutaneous coronary intervention due to ST-segment elevation myocardial infarction. **Methods:** This prospective study included 174 patients who were diagnosed with ST-segment elevation myocardial infarction and underwent primary percutaneous coronary intervention between December 2016 and April 2018. Patients were divided into two groups as cardiac arrest and non-cardiac arrest patients during the percutaneous coronary intervention, so cardiac arrest predictors were investigated. **Results**: Twenty of the 174 patients who were included in the study had a cardiac arrest during the procedure. While abnormal yawning was observed in 13 (65%) of 20 patients with cardiac arrest, 11 (7.1%) patients had abnormal yawning, but no cardiac arrest developed. Abnormal yawning had a statistically significant effect ($p<0.001$) on cardiac arrest in logistic regression analysis and the regression model was estimated with 98.1% accuracy. **Conclusion:** In the present study, we reveal that abnormal yawning which occurs independently of ambient and emotional conditions during the primary percutaneous coronary intervention in patients with ST-segment elevation myocardial infarction may be the precursor of cardiac arrest.

KEYWORDS: ST-Segment Elevation Myocardial Infarction, Abnormal Yawning, Cardiac Arrest.

INTRODUCTION

Acute ST-segment elevation myocardial infarction (STMI) is still an important cause of morbidity and mortality despite all the medical developments. More than seven million people die from coronary artery disease each year, accounting for 12.8% of all deaths.[1] Percutaneous coronary interventions (PCI) are major treatment strategies for STMI patients and reduce mortality and morbidity in case of complications.[2] STMI patients have an important risk factor for cardiac arrest, which can develop in and out of the hospital. There are many risk factors and scoring systems that will predict cardiac arrest in patients with STMI.[3]

Yawning is an involuntarily act with a maximal opening of mouth from jaws, followed by a deep inspiration and a slow expiration through the mouth and nose. It is thought to be caused by lack of sleep, fatigue, hunger, stress, brain hypoxia and social empathy.[4] Despite this, neither its mechanism nor functionality is fully known.[5] It is argued that one of the important functions of yawning is to reverse hypoxia by a deep inspiration and increase metabolism by extracting more CO_2 and that it is done by stimulating the carotid body by hypoxia.[5-6-7] In addition to physiological yawning, the increase in the frequency of yawning independently of ambient conditions is defined as abnormal yawning in the literature.[8-9]

We observed that a cardiac arrest followed an abnormal yawning in patients undergoing primary PCI due to STMI. This observation made us think that abnormal yawning could be a precursor to cardiac arrest. Therefore, we investigated the relationship of abnormal yawning with cardiac arrest in patients undergoing primary PCI due to ST-segment elevation myocardial infarction.

PATIENTS AND METHODS

175 consecutive patients diagnosed with STMI between December 2016 and April 2018 and taken in the catheter laboratory by the same cardiologists were included in this observational prospective study. Patients were divided into two groups: cardiac arrest developed and non-developed during primary PCI. Cardiac arrest was defined as that cardiopulmonary resuscitation and/or defibrillation are needed. Abnormal yawning was defined as 2 or more yawning frequencies per minute in accordance with the literature. Abnormal yawning was

recorded by a second nurse in the lab during the primary PCI.

For patients who apply to our hospital with chest pain for 20 minutes and above and have V2-V3 derivations at EKG, STMI criterion was defined for men as "over 40 years of age >0.2 mV", "under 40 years of age >0.25 mV", and for women as ">0.15 mV" or ">0.1 MV ST elevation at least two other adjacent derivations".[10] All patients were questioned for risk factors during admission to the hospital. At the time of admission, samples were taken from all patients for myocardial damage markers, blood sugar, lipid profile, renal function markers, liver enzymes, hemogram.

Selective coronary angiography was performed in standard positions, using the right or left transfemoral approach and the Judkins technique. Total congestions were performed using the appropriate guide wire. The primary PK procedure was started by inserting a guide catheter as in standard practice. An appropriate dose of anticoagulant was administered. During the procedure, preferably standard heparin was used. If enoxaparin was used before, it was continued. The guide wire into the catheter was passed through the full-clogged lesion. After passing the lesion by wire, balloon pre-dilatation were performed in case of need. If there is no contraindication in the assessment of vein diameter, vasodilation was performed with intra-coronary nitrate. After that, stent placement was performed. Thrombus aspiration was not performed. The drug-eluting stent and bare metal stent were inflated at appropriate pressures according to the size of the lesion and used. Cardiopulmonary resuscitation was performed in patients with cardiac arrest during the procedure.

Statistical Analysis

The data were analyzed for normal distribution using Kolmogorov-Smirnov test. Two independent samples t-test were used for quantitative variables, and Chi-square test and Fisher exact test were used for qualitative comparisons. The effect of estimating variables on cardiac arrest was analyzed by Logistic regression analysis. Statistical parameters were expressed as n (%) and mean±SD. Statistical significance was accepted as $p<0.05$. IBM SPSS version 22 (IBM SPSS for Windows version 22, IBM correlation, Armonk, New York, United States) was used for all calculations.

RESULTS

Twenty of the 174 patients who were included in the study had a cardiac arrest during the procedure. The basal characteristic and laboratory characteristics of the groups are summarized in Table 1. While abnormal yawning was observed in 13 (65%) of 20 patients with cardiac arrest, 11 (7.1%) patients also had abnormal yawning but no cardiac arrest was developed ($p<0.001$). Abnormal yawning, in-hospital mortality, LAD lesion presence, and DM presence were found to be significantly higher in the cardiac arrest group. ($p<0.001$, $p=0.007$, $p<0,001$, respectively).

In logistic regression analysis, the effect of estimating variables on cardiac arrest was investigated. The predictive variables in Table 1 account for the 76,4% of the change in cardiac arrest. According to this, abnormal yawning and DM variables have a significant effect on cardiac arrest (Table 2). The regression model predicted cardiac arrest with 98.1% accuracy (Table 3).

Table. 1: Baseline characteristics of study patients.

		Cardiac arrest				
		None		**Occured**		
		n	**%**	**n**	**%**	**p**
Gender (Female/ Male) [b]	*n(%)*	34/120	22,1/77,9	8/12	40,0/60	0,096
LAD critical stenosis [a]	*n(%)*	88	57,1	5	25,0	**0,007***
CX critical stenosis [a]	*n(%)*	36	23,4	8	40,0	0,108
RCA critical stenosis [a]	*n(%)*	77	50,0	7	35,0	0,207
Multivessel disease [a]	*n(%)*	103	66,9	12	60,0	0,541
In-hospital mortality [b]	*n(%)*	4	2,6	5	25,0	**0,001***
Hypertension [a]	*n(%)*	49	31,8	8	40,0	0,463
Diabetes mellitus [a]	*n(%)*	48	31,2	15	75,0	**0,001***
Hyperlipidemia [a]	*n(%)*	19	35,8	10	50,0	0,270
Smoking [a]	*n(%)*	82	54,3	14	70.0	0,184

Abnormal yawning[a]	*n(%)*	11	7,1	13	65,0	**0,001***
Age, years[c]	*Mean±SD*	60,25	±11,66	64,45	±11,18	0,130
Triglyceride, mg/dl [c]	*Mean±SD*	156,86	±113,71	166,95	±123,46	0,713
HDL cholesterol, mg/dl [c]	*Mean±SD*	37,15	±10,53	39,65	±7,51	0,307
Total cholesterol, mg/dl [c]	*Mean±SD*	186,40	±44,06	178,80	±48,14	0,475
LDL cholesterol, mg/dl [c]	*Mean±SD*	114,59	±35,08	104,35	±30,40	0,216
Fasting glucose, mg/dl	*Mean±SD*	156,17	±102,73	163,95	±74,32	0,744
Urea, mg/dL [c]	*Mean±SD*	23,85	±12,73	27,00	±17,96	0,325
Creatinine, mg/dL [c]	*Mean±SD*	0,52	±0,081	0,40	±0,06	0,522
Sodium, mmol/l [c]	*Mean±SD*	138,33	±3,88	139,65	±3,54	0,150
Chlorine, mmol/l [c]	*Mean±SD*	104,07	±4,30	103,72	±3,97	0,743
Potassium, mmol/l [c]	*Mean±SD*	3,87	±0,61	3,84	±0,83	0,833

[a]Chi-Square test; [b]Fisher exact test;a:0,05; [c]Independent Samples t test;* statistical significance of frequency distributionsfrekans, HDL: High-density lipoprotein,LDL: Low-density lipoprotein,LAD **: Left Anterior Descending** Artery **,** CX: **Circumflex** Artery RCA : Right Coronary Artery

Table. 2: Logistic regression analysis.

	B	**Wald**	**Sig.**	**Exp(B)**	**95% CI for EXP(B)**	
					Lower	**Upper**
Gender (Female/ Male)	-2,909	3,737	0,053	0,055	0,003	1,041
Age, years	0,034	0,256	0,613	1,035	0,907	1,180
LAD critical stenosis	24,101	0,000	0,996	.	0,000	.
CX critical stenosis	20,018	0,000	0,997	.	0,000	.
RCA critical stenosis	20,067	0,000	0,997	.	0,000	.
Multivessel disease	-1,725	1,344	0,246	0,178	0,010	3,292
In-hospital mortality	-2,152	0,560	0,454	0,116	0,000	32,604
Hypertension	-0,220	0,022	0,882	0,802	0,044	14,692
Diabetes mellitus	-3,770	4,515	**0,034***	0,023	0,001	0,746
Smoking	-2,756	2,802	0,094	0,064	0,003	1,602
Abnormal yawning	-5,938	6,234	**0,013***	0,003	0,000	0,279
Triglyceride	0,001	0,009	0,925	1,001	0,987	1,015
HDL cholesterol	0,042	0,236	0,627	1,043	0,881	1,235
Total cholesterol	0,026	0,311	0,577	1,026	0,937	1,123
LDL cholesterol	-0,065	2,022	0,155	0,937	0,857	1,025
Fasting glucose	-0,013	1,603	0,205	0,987	0,968	1,007
Urea	-0,004	0,006	0,937	0,996	0,908	1,093
Creatinine,	-0,135	0,014	0,906	0,873	0,093	8,194
Sodium	0,085	0,242	0,623	1,088	0,777	1,524
Chlorine	-0,035	0,056	0,814	0,966	0,722	1,292
Potassium	-0,397	0,238	0,626	0,672	0,136	3,315
Constant	-38,022	0,000	0,997	0,000		

R^2: 0,764; Logistic regression analysis;a:0,05;*Effect is statisticaaly significant

Table. 3: The predictive rates of cardiac arrest in the regression model.

Observed		Predicted		
		Cardiac arrest		Percentage Correct
		None	Occured	
Cardiac arrest	None	142	0	100,0
	Occured	3	15	83,3
Overall Percentage				**98,1**

DISCUSSION

To our knowledge, we have illustrated for the first time in the medical literature that abnormal yawning is independently associated with cardiac arrest in patients undergoing primary PCI with STMI.

Cardiac arrest in acute coronary syndromes is one of the rare but an important complication. A number of scoring systems were used to predict in-hospital cardiac arrest. It is known that systolic blood pressure, age, heart rate, ECG changes and heart failure symptoms help to predict in-hospital cardiac arrest. Jonas faxen et al.[3] display that a 7-point and five-variable risk score in NSTMI can be useful in predicting in-hospital cardiac arrest; In this scoring system, Killip class is 1 point, heart rate below 50 and above 100 are 1 point, 60 age and above is 1 point, ST changes are 2 point, and systolic blood pressure below 100 is 2 points. Our study showed that regardless of the clinical and laboratory data, yawning could predict cardiac arrest.

Yawning may be a defensive mechanism to prevent hypotension and brain hypoxia. Askeyay[6] suggested that yawning was a complex defense system with peripheral and central arc aimed at restoring brain hypoxia. Jorma Matikainan[7] suggests that yawning stimulates the carotid body mechanically being an important physiological function of yawning and that this mechanical stimulation causes increased arousal and wakefulness. The decrease in the amount of O_2 in arterial blood and increase in CO_2 and H^+ ions stimulate the carotid body, which has an important role in the peripheral control of respiration.[11] In addition to these chemical warning mechanisms, another hypothesis suggests that yawning mechanically attempts to correct brain hypoxia by stimulating the carotid body.[7] Again, the PO_2 decrease due to the decrease in heart rate, hypotension, and circulatory disturbances may be a defense mechanism for preventing brain hypoxia by stimulating the carotid body. After stimulation of the carotid body, this stimulation can be continued with mechanical yawning to keep the stimulation going and to reduce the hypoxia.

One of the important points to prevent the reduction in blood pressure is to increase venous return. Intensive yawning decreases the pressure on the thorax and increases the abdominal pressure, so leading venous return to prevent hypotension. During cardiac arrest, yawning appears to be an important defense mechanism to prevent hypotension and brain hypoxia.

Although the role of DM in the development of cardiac arrest is not fully known, it may increase cardiac arrest risk by heart failure, coronary artery disease, and arrhythmia. DM is a risk factor for in-hospital and out-of-hospital cardiac arrest.[12-15] In our study, DM was an increased risk factor in STMI patients for cardiac arrest.

CONCLUSION

During the primary PCI procedure, hemodynamic follow-up of the patient is very important, and it is very valuable to be aware of any significant changes. Prior to the hemodynamic instabilities of patients treated with primary PCI, the onset of abnormal yawning, independent of ambient and emotional conditions, maybe a cardiac arrest stimulant.

REFERENCES

1. WHO Fact sheet N8310, updated June 2011, http://www.who.int/mediacentre/ factsheets/fs310/en/index.html.
2. Levine GN, Bates ER, Blankenship JC, Bailey SR, Bittl JA, Cercek B, et al. 2011 ACCF/AHA/SCAI Guideline for Percutaneous Coronary Intervention: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines and the Society for Cardiovascular Angiography and Interventions. Circulation, 2011; 124: e574-e651.
3. Faxén J, Hall M, Gale CP, Sundström J, Lindahl B, Jernberg T,et all. A user-friendly risk-score for predicting in-hospital cardiac arrest among patients admitted with suspected non ST-elevation acute coronary syndrome – The SAFER-score. Resuscitation, 2017; 121: 41-48.
4. Gupta S, Mittal S. Yawning and its physiological significance. Int J Appl Basic Med Res., 2013; 3: 11-15.
5. Aloe F. Yawning. Arquivos de Neuro-psiquiatria, 1994; 52: 273–276.
6. Askenasy JJ. Is yawning an arousal defence reflex? J Psychol, 1989; 123: 609–621.
7. Matikainen J, Elo H. Does yawning increase arousal through mechanical stimulation of the carotid body? Med Hypotheses, 2008; 70: 488-492.
8. Singer OC, Humpich MC, Lanfermann H, Neumann-Haefelin T Yawning in acute anterior circulation stroke. J Neurol Neurosurg Psychiatry, 2007; 78: 1253-1254.
9. Cattaneo L, Cucurachi L, Chierici E, Pavesi G Pathologicalyawning as a presenting symptom of brain stem ischaemia in two patients. J Neurol Neurosurg Psychiatry, 2006; 77: 98–100.

10. Third universal definition of miyocardial infarction.European Heart Journal, 2012; 33: 2551-2567.
11. Gonzalez C, Almaraz L, Obeso A, Rigual R. Carotid body chemoreceptors: from natural stimuli to sensory discharges. Physiol Rev., 1994; 74: 829-98.
12. Echouffo-Tcheugui JB, Kolte D, Khera S, Bhatt DL, Fonarow GC. Comparison of Survival After In-Hospital Cardiac Arrest in Patients With Versus Without Diabetes Mellitus. Am J Cardiol, 2018; 121: 671-677.
13. Jouven X, Lemaître RN, Rea TD, Sotoodehnia N, Empana J-P, Siscovick DS. Diabetes, glucose level, and risk of sudden cardiac death. Eur Heart J 2005; 26: 2142-2147.
14. Kucharska-Newton AM, Couper DJ, Pankow JS, Prineas RJ, Rea TD, Sotoodehnia N, Chakravarti A, Folsom AR, Siscovick DS, Rosamond WD. Diabetes and the risk of sudden cardiac death, the atherosclerosis risk in communities study. Acta Diabetol, 2010; 47: 161-168.
15. Curb JD, Rodriguez BL, Burchfiel CM, Abbott RD, Chiu D, Yano K. Sudden death, impaired glucose tolerance, and diabetes in Japanese American men. Circulation, 1995; 91: 2591-2595.