Original Article

Gender and Acute ST Elevation Myocardial Infarction (STEMI) A Cross Sectional Analysis

Muhammad Shahid, Muhammad Irfan, Shahzad Majeed Bhatti, Nighat Majeed, Muhammad Rashid Ali and Sami Mumtaz

Objective: To determine the gender difference in various qualitative and quantitative factors among acute ST elevation myocardial infarction (STEMI) suffering patients treated with Streptokinase at tertiary care hospital, Gujranwala, Pakistan.

Methods: This cross-sectional study was carried out at the Department of Cardiology, GMC Teaching hospital, Gujranwala from June 2017 to May 2018. After written consent, the data was collected by purposive sampling. The patients admitted with STEMI of all age groups, belonging to both genders, who were treated with Streptokinase injection were included. Statistical analysis was done using SPSS version 25. Independent sample T test and Chi-square test were used for quantitative and qualitative variables respectively to determine their significant association with gender. Then, binary logistic regression analysis was also performed. The p values were taken statistically significant if < 0.05

Results: Amongst 668 patients, 77.1% were male while 22.9% were female. Female had statistically significantly less time from onset of symptoms till arrival at hospital (p=0.005), higher pulse rate at presentation (p=0.031) and higher diastolic BP at presentation (p=0.003), lower ST segment elevation on ECG both minimum (P<0.001) and maximum (p=0.002) and lower serum creatinine (p=0.033). Male had significantly higher rate of H/O IHD in their male family members of age <55 years (p<0.001) as well as in their female family members of age <45 years (p=0.007). Obesity was significantly more prevalent among female as compared to male suffering STEMI (p=0.037). Binary logistic regression model was statistically significant, p<0.05 and it explained 17.1% (Nagelkerke R2) of the variance in the gender wise grouping of patients and correctly classified 77.1% of cases.

Conclusions: Significant gender difference exists in different parameters among patients who presented with STEMI. Female reached hospital earlier after symptoms onset with higher pulse rate and diastolic BP and relatively lower ST segment elevation on ECG. This may be due to their good stress escape response as compared to male that cashed in term of relatively lower their in-hospital mortality rate. Our male should be addressed for a similar quick response to their symptoms to decrease MI related mortality among them. Among STEMI patients, male had higher rate of H/O IHD in their family member while obesity was relatively more prevalent among female which is a modifiable factor.

Keywords: acute STEMI, gender, cross-sectional study, SPSS.

Introduction

Acute myocardial infarction (MI) is the leading cause of mortality and morbidity in men as well as in women.[1] Its prevalence is higher in men than in women (8.3% in men vs 6.1% in women).[2] This is because female have less coronary atherosclerotic burden than men.[3] Broadly, MI is classified into ST-elevation myocardial infarction (STEMI) and non-STEMI,[4] where in first, transmural myocardial necrosis occurs due to complete occlusion of a major epicardial artery.[5] The most effective treatment for STEMI is the immediate restoring the patency of the occluded artery either by PCI or fibrinolysis.[6] There is clear association between longer delay in reperfusion therapy and worse prognosis.[7] In literature, women were found with longer delay from symptoms onset to medical attension and reperfusion.[8,9] ,They were also found with higher in-hospital mortality as compared to men.[10-12] These majority literature findings were from Western population, local studies on gender disparity are scarce. Therefore, the objective of the present study was to determine the gender difference in various qualitative and quantitative factors among acute ST elevation myocardial infarction (STEMI) patients treated with Streptokinase at tertiary care hospital, Gujranwala, Pakistan.

Methods

This cross-sectional study was carried out at the Department of Cardiology, GMC Teaching hospital, Gujranwala from June 2017 to May 2018. After

written consent, the data was collected by purposive sampling. The patients admitted with STEMI of all age groups, belonging to both genders, who were treated with Streptokinase injection were included in this study. Statistical analysis was performed using the Statistical Package for Social Science (SPSS), version 25. Age, BMI, time from onset of symptoms till arrival at hospital in minutes, door to needle time in minutes, baseline pulse, systolic BP at presentation, diastolic BP at presentation, minimum ST segment elevation, maximum ST segment elevation, serum creatinine conc., serum sodium conc. and serum potassium conc. were the quantitative variable, while gender, history of smoking, hypertension, diabetes mellitus, personal H/O IHD, History of IHD in male family member of age <55years, History of IHD in female family member of age <45years obesity, cardiac wall involved by STEMI, right ventricular involvement, ST segment settlement >50% at 1st post-admission day, and outcome of hospitalization were the qualitative variables. Independent sample T test and Chi-square test for independence were used for quantitative and qualitative variables respectively to determine their significant association with gender. Then, binary logistic regression analysis was also performed. The p values were taken statistically significant if < 0.05.

Results

Amongst 668 patients who presented with STEMI, 77.1% were male while 22.9% were female (Picture 1). As compared to male gender group, female group had statistically significantly less time from onset of symptoms till arrival at hospital (p=0.005), higher pulse rate at presentation (p=0.031) and higher diastolic BP at presentation (p=0.003). These may be due to increased anxiety level or more fear of death in female gender. It was also found that females had relatively lower ST segment elevation on ECG both minimum (P<0.001) and maximum (p=0.002) and lower serum creatinine (p=0.033) **(Table-1).** As compared to females, male had significantly higher rate of H/O IHD in their male family members of age <55 years (p<0.001) as well as in their female family members of age <45 years (p=0.007). On the other hand, obesity was significantly more prevalent among female as compared to male suffering STEMI (p=0.037). **(Table-2)** A binary logistic regression analysis was performed to ascertain the likelihood gender difference in significantly associated qualitative and quantitative factors.

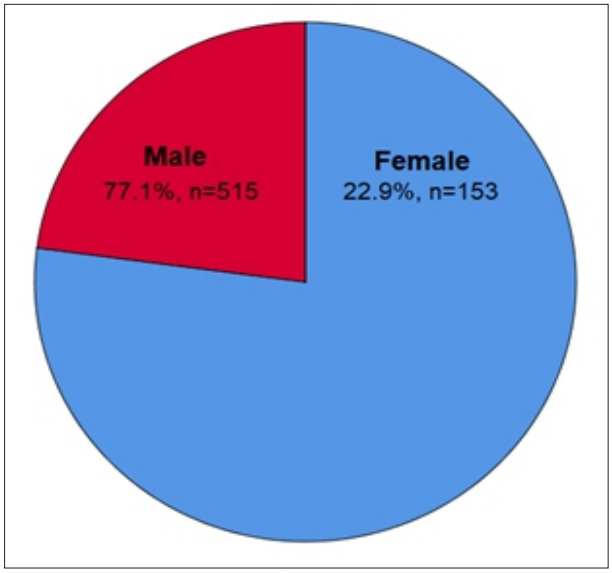

Fig-1: Distribution of acute ST elevation mycaridal infarction (STEMI) among geders (n=668).

Table-1: Associations of various quantitative variables with gender among STEMI suffering patients treated with Streptokinase (n = 668) *.

Quantitative Variables	Gender		Mean Difference	P-value
	Male (Mean±SD)	Femal (Mean±SD)		
Age (years)	53.80±11.90	53.85±1386	-.050	.956
BMI (Kg/m^2)	27.00±4.22	27.258±3.94	-.2575	.502
Time till arrival (minutes)[1]	299.73±392.99	206.93±163.74	92.794	.005
Door to needle time (minutes)	26.43±35.30	32.05±37.29	-5.615	.089
Baseline pulse (per minute)	85.12±19.11	88.90±18.51	-3.779	.031
Baseline systolic BP (mmHg)	130.24±25.09	134.76±29.73	-4.524	.061
Baseline diastolic BP (mmHg)	81.43±15.90	86.31±23.10	-4.872	.003
ST segment elevation, minimum (mm)	2.65±1.60	2.013±1.11	.6413	.000
ST segment elevation, maximum (mm)	5.12±3.42	4.209±2.18	.9074	.002
Serum creatinine (mg/dl)	1.15±0.91	.995±0.26	.1594	.033
Serum Sodium (mEq/L)	137.17±5.42	136.68±5.22	.491	.321
Serum Potassium (mEq/L)	3.78±0.60	3.820±0.80	-.0931	.515

**Independent sample T-test was used; 1=Time from onset of symptoms till arrival at hospital (minutes)*

Table-1: Associations of various quantitative variables with gender among STEMI suffering patients treated with Streptokinase (n = 668) *.

Quantitative Variables	Gender	Male	Female	Total	P-value
History of smoking:	Yes	273 (53%)	89 (58.2%)	362 (54.2%)	0.269
	No	242 (4%7)	64 (41.8%)	306 (45.8%)	
History of diabetes mellitus:	Yes	156 30.3%)	41 (26.8%)	197 (29.5%)	0.421
	No	359 (69.7%)	81 (52.9%)	471 (70.5%)	
History of hypertension:	Yes	289 (56.1%)	72 (47.1%)	361 (54%)	0.053
	No	226 (43.9%)	81 (52.9%)	307 (46%)	
Personal history of IHD:	Yes	156 (30.3%)	37 (24.2%))	193 (28.9%)	0.156
	No	359 (69.7%)	116 (75.8%)	457 (71.1%)	
History of IHD in male family member of age <55years:	Yes	76 (14.8%)	5 (3.3%)	81 (12.1%)	<0.001
	No	439 (85.2%)	148 (96.7%)	587 (84.9%)	
History of IHD in female family member of age <45years:	Yes	72 (14%)	9 (5.9%)	81 (12.1%)	0.007
	No	443 (86%)	144 (94.1%)	587 (87.9%)	
Obesity:	Yes	108 (21%)	45 (29.4%)	153 (22.9%)	0.037
	No	407 (79%)	108 (70.6%)	515 (77.1%)	
Cardiac wall involved by STEMI:	Yes	223 (43.3%)	69 (45.1%)	292 (43.7%)	0.711
	No	292 (56.7%)	84 (54.9%)	376 (92.1%)	
Right ventricular involvement:	Yes	45 (8.7%)	8 (5.2%)	53 (7.9%)	0.176
	No	470 (91.3%)	145 (94.8%)	615 (92.1%)	
ST elevation settled >50% at 1st post-admission day:	Yes	406 (78.8%)	121 (79.1%)	527 (78.9%)	1.000
	No	109 (21.2%)	32 (20.9%)	141 (21.1%)	
Outcome of hospitalization:	Death	21 (4.1%)	4 (2.6%)	25 (3.7%)	0.477
	No Death	494 (95.9%)	149 (97.4%)	643 (96.3%)	

**Chi-square test for independence was used*

Table-3: Binary Logistic Regression Analysis to predict association of various factors with gender among STEMI suffering patients treated with Streptokinase (n = 668) *.

Risk Facotrs	B	S.E.	Wald Statistic	P-value	Odds Ratio	95% C.I. For EXP (B) Lower	Upper
Time till arrival (minutes)1	-.001	.000	7.905	.005	.999	.998	1.000
Baseline pulse (per minute)	.008	.006	2.093	.148	1.008	.997	1.019
Baseline diastolic BP (mmHg)	-.015	.006	6.147	.013	1.015	1.003	1.027
ST segment elevation, minimum (mm)	-.327	.093	12.340	.000	.721	.601	.866
ST segment elevation, maximum (mm)	-.064	.040	2.571	.109	.938	.867	1.014
Serum creatinine (mg/dl)	-.481	.293	2.687	.101	.618	.348	1.099
History of IHD in male family member of age <55years (Yes/No)	2.347	.701	11.213	.001	10.457	2.647	41.312
History of IHD in female family member of age <45years (Yes/No)	-.701	.583	1.447	.229	.496	.158	1.554
Obesity (Yes/No)	-.354	.226	2.454	.117	.702	.450	1.093
Constant	-2.556	.913	7.835	.005	.078		

Cox & Snell R Square = 11.3%, Nagelkerke R Square = 17.1%, 1=Time from onset of symptoms till arrival at hospital (minutes)

The logistic regression model was statistically significant, p<0.05. The model explained 17.1% (Nagelkerke R2) of the variance in the gender wise grouping of patients and correctly classified 77.1% of cases. Male had 10.457 times more likelihood risk of IHD in their male family members of age <55 years as compared to female. Female reached hospital significantly earlier after onset of symptoms (p=0.005) and had more diastolic BP at presentation (p=0.013). Male had 72.1% more minimum ST segment elevation as compared to female **(Table 3).**

Discussion

Amber M Otten et al[13] found that 74% were men and 26% women among 6746 STEMI patients. In another similar study by Prashanth Panduranga and his colleagues,[14] out of 2,465 STEMI patients, 91% were male . In a study of STEMI patients from our own Country, 81% were males and 19% were females.[15] In our study, 77.1% STEM patients belonged male gender. Hence, male suffer STEMI more frequently worldwide. From scientific background, it is proven that female heart is relatively protected from apoptosis and cell death as compared to male heart.[16]

Their cardiomyocytes are able to bear more oxidative stress under similar circumstances.[17] Female have relatively smaller infarct size in which role of female sex hormone, estrogen is documented.[18,19] In our study, male had significantly higher rate of H/O IHD in their male family members of age <55 years (p<0.001) as well as in their female family members of age <45 years (p=0.007). Similarly, Boonchu Srichaiveth et al[20] found family risk factor more in men than in women (p < 0.001). They also found that women had a higher incidence of diabetes and hypertension (46.9% vs. 31.0%, p< 0.001 and 62.1% vs. 45.3%, p<0.001) while male were significantly more smoker (p < 0.001). However, in our study, diabetes, hypertension, and smoking were comparable in both gender. In our rural areas, significant number of female smoke tobacco in the form of "Huka". In a study from Sweden,[9] it was found that female had significantly longer delay until first medical contact (90 vs 66 min, p=0.04) and until ECG (146 vs 103 min, p=0.03). In an international trial "ATLANTIC" comprising of 1862 STEMI patients,(8) gender disparities were analysed. Women had significantly longer delay times from symptom onset to prehospital ECG (median 88 vs 70 min, p<0.01). Women had significantly lower body mass index (BMI) than men (median 25.6 vs 26.5 kg/m^2, p<0.01). Female gender was an independent predictor of short-term mortality (5.7% vs 1.9%, p=0.04). The findings of our study were reverse to the Western studies. In our study, female had statistically significantly less time from onset of symptoms till arrival at hospital (p=0.005). This may be due to longer decision time in our male that resulted higher mortality rate among them (4.1% vs 2.6%). Our male should be addressed for a similar quick response to their symptoms to decrease MI related mortality among them. In our study, Obesity was significantly more prevalent among female as compared to male suffering STEMI (p=0.037). Obesity paradox[21] may have played protective role to yeild less mortality among them in our population. Further studies with large sample size are required to validate these findings about gender discrepancy in our population.

Conclusion

Significant gender difference exists in different parameters among patients who presented with STEMI. Female reached hospital earlier after symptoms onset with higher pulse rate and diastolic BP and relatively lower ST segment elevation on ECG. This may be due to their good stress escape response as compared to male that cashed in term of relatively lower their in-hospital mortality rate. Our male should be addressed for a similar quick response to their symptoms to decrease MI related mortality among them. Among STEMI patients, male had higher rate of H/O IHD in their family member while obesity was relatively more prevalent among female which is a modifiable factor.

Department of Cardiology/Medicine
GMC/DHQ Hospital, Gujranwala
www.esculapio.pk

References

1. Underner M, Perriot J. Tobacco and tuberculosis. Med Press. 2012 Dec; 41 (12 Pt 1): 1171-80. [PubMed]
2. Trosini-Desert V, Germaud P, Dautzenberg B. Exposure to tobacco smoke and bacterial infectious risk. Rev Mal Respir. 2004 Jun; 21 (3 Pt 1): 539-47. [PubMed]
3. Aztatzi-Aguilar OG, Uribe-Ramírez M, Narváez-Morales J, De Vizcaya-Ruiz A, Barbier O. Early kidney damage induced by subchronic exposure to PM 2.5 in rats. Particle and fibre toxicology. 2016 Dec;13(1):68. . [PubMed]
4. Phelan JJ, Basdeo SA, Tazoll SC, McGivern S, Saborido JR, Keane J. Modulating iron for metabolic support of TB host defense. Frontiers in immunology. 2018;9. [PubMed]
5. Bugova G, Janickova M, Uhliarova B, Babela R, Jesenak M. The effect of passive smoking on bacterial colonisation of the upper airways and selected laboratory parameters in children. Acta Otorhinolaryngologica Italica. 2018 Oct;38(5):431.. [PubMed]
6. Kharlamova N, Jiang X, Sherina N, Potempa B, Israelsson L, Quirke AM, Eriksson K, Yucel Lindberg T, Venables PJ, Potempa J, Alfredsson L. Antibodies to Porphyromonas gingivalis indicate interaction between oral infection, smoking, and risk genes in rheumatoid arthritis etiology. Arthritis & rheumatology. 2016 Mar;68(3):604-13. [PubMed]
7. Wang H, Yu M, Ochani M, Amella CA, Tanovic M, Susarla S, et al. Nicotinic acetylcholine receptor alpha 7 subunit is an essential regulator of inflammation. Nature.2003 Jan 23; 421 (6921): 384-8. [PubMed]
8. Kalkman HO, Feuerbach D. Modulatory effects of α7 nAChRs on the immune system and its relevance for CNS disorders. Cellular and molecular life sciences. 2016 Jul 1;73(13):2511-30.. [PubMed]
9. Lin S, Melendez-Torres GJ. Systematic review of risk factors for nonadherence to TB treatment in immigrant populations. Transactions of the Royal Society of Tropical Medicine and Hygiene. 2016 May 1;110(5):268-80.. [PubMed]
10. Catherinot E, Fieschi C, Feinberg J, Casanova JL, Couderc LJ. Mendelian Susceptibility Syndrome to Mycobacterial Infections: Interleukin-12-Interferon-g axis defects. Rev Mal Respir. Nov 2005; 22 (5 Pt 1): 767-76. [PubMed]
11. Nichter M, Padmawati S, Ng N. Introducing smoking cessation to Indonesian males treated for tuberculosis: The challenges of lowmoderate level smoking. Social Science & Medicine. 2016 Mar 1;152:70-9. . [PubMed]
12. Fekih L, Boussoffara L, Abdelghaffar H, Hassene H, Fenniche S, Belhabib D, et al. Effects of smoking on pulmonary tuberculosis. Rev Med Liege. 2010 Mar; 65 (3): 152-5. [PubMed]
13. Leung CC, Li T, TH Lam, Yew WW, WS Law, Tam CM, et al. Smoking and tuberculosis in Hong Kong. Int J Tuberc Lung Dis. 2003 Oct; 7 (10): 980-6. [PubMed]
14. Racil H, Amar JB, Cheikrouhou S, Hassine E, Zarrouk M, Chaouch N, et al. Pulmonary

tuberculosis in smokers. Med Press. 2010 Feb; 39 (2): e25-8. [PubMed]
15. Liu X, Blaschke T, Thomas B, De Geest S, Jiang S, Gao Y, Li X, Buono E, Buchanan S, Zhang Z, Huan S. Usability of a medication event reminder monitor system (MERM) by providers and patients to improve adherence in the management of tuberculosis. International journal of environmental research and public health. 2017 Sep 25;14(10):1115.. [PubMed]
16. Auld AF, Blain M, Ekra KA, Kouakou JS, Ettiègne-Traoré V, Tuho MZ, Mohamed F, Shiraishi RW, Sabatier J, Essombo J, Adjorlolo-Johnson G. Wide variations in compliance with tuberculosis screening guidelines and tuberculosis incidence between antiretroviral therapy facilitiesCote d'Ivoire. PloS one. 2016 Jun 8;11(6):e0157059.. [PubMed]
17. Leung CC, Yew WW, Chan CK, Chang KC, Law WS, Lee SN, Tai LB, Leung EC, Au RK, Huang SS, Tam CM. Smoking adversely affects treatment response, outcome and relapse in tuberculosis. European Respiratory Journal. 2015 Mar 1;45(3):738-45.. [PubMed]
18. Wang JY, Hsueh PR, Jan IS, LN Lee, Liaw YS, PC Yang, et al. The effect of smoking on tuberculosis: different patterns and poorer outcomes. Int J Tuberc Lung Dis. 2007 Feb; 11 (2): 143-9. [PubMed]
19. Agrawal R, Gonzalez-Lopez JJ, Nobre-Cardoso J, Gupta B, Grant R, Addison PK, Westcott M, Pavesio CE. Predictive factors for treatment failure in patients with presumed ocular tuberculosis in an area of low endemic prevalence. British Journal of Ophthalmology. 2016 Mar 1;100(3):348-55. [PubMed]
20. Abal AT, Jayakrishnana B, Parwer S, El Shamy A, Abahussain E, Sharma PN. Effect of cigarette smoking on sputum in adults with active pulmonary tuberculosis. Respir Med. 2005 Apr; 99 (4): 415-20. [PubMed]
21. Jeyashree K, Kathirvel S, Shewade HD, Kaur H, Goel S. Smoking cessation interventions for pulmonary tuberculosis treatment outcomes. Cochrane Database of Systematic Reviews. 2016(1). [PMC free article] [PubMed]
22. Bates MN, Khalakdina A, M Pai, Chang L, Lessa F, Smith KR. Risk of tuberculosis from exposure to tobacco smoke: a systematic review and meta-analysis. Arch Intern Med. 2007 Feb 26; 167 (4): 335-42. [PubMed]
23. Jeyashree K, Kathirvel S, Shewade HD, Kaur H, Goel S. Smoking cessation interventions for pulmonary tuberculosis treatment outcomes. Cochrane Database of Systematic Reviews. 2016(1).. [PubMed]
24. Fisher MD. Practical Pearl: TB Screening and Management-Sept. 2017. [PubMed]