Original Research Article

A study on prevalence of urinary tract infection in patients with acute ST segment elevation myocardial infarction in a tertiary care centre in Chengalpattu district

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Abstract

Introduction: Ischemic heart disease is presently the main source of death worldwide. Based on the information from Framingham Heart and Study the lifetime danger of developing coronary artery disease after age 40 is 49% for males and 32% for females.

Aim of the study: To determine the prevalence of subclinical urinary tract infections and their bacteriological profile in patients with acute ST-segment elevation myocardial infarction.

Materials and methods: It was a prospective study conducted in the General Medicine, Microbiology and Cardiology Department of Karpaga Vinayaga Medical College, Kanchipuram district, Tamil Nadu from January 2020 to April 2020 among 50 patients with acute ST-segment elevation myocardial infarction admitted in IMCU. All patients with ST-elevation myocardial infarction admitted in IMCU were included. WHO definition of myocardial infarction "A history of prolonged chest discomfort ECG changes consistent with ischemia or necrosis, Elevated cardiac enzymes" was considered. All patients with symptomatic UTI, patients who gave a history of previous urethral catheterization, patients with a history suggestive of urinary tract obstruction, patients with a history of recurrent UTI or treated for UTI in the recent past, patients whose USG showed enlarged prostate and/or significant residual volume of urine were excluded from the study.

Results: The overall mean age of the study participants was 58.58±8.53 years with a range of 42 to 77 years. A total of 32 males and 18 females were present in our study. The total number of hypertensives were 64% (n=34), diabetics 56% (n=28) and smokers 54% (n=27). The overall mean

LDL and HDL were 131.36±25.18 mg/dL and 37.74±5.83 mg/dL. The mean number of pus cells observed among the study participants was 7.96±1.17 ranging from a minimum of 6 cells to a maximum of 10 cells. The total mean urea and creatinine among the study participants reported were 31.20±13.48 mg/dL and 1.31±0.38 mg/dL respectively. Total numbers of cystitis were 16.28%.

Conclusion: UTI is positively associated with ST-segment elevation myocardial infarction. Among patients with STEMI, those with UTI more often had hypertension. Patients with combined UTI and hypertension had a higher risk of developing STEMI. Gram-Negative Bacilli especially Escherichia Coli was the commonest organism isolated in STEMI patients with subclinical UTI.

Key words

UTI, Cystitis, Myocardial infarction, STEMI.

Introduction

Ischemic heart disease is presently the main source of death worldwide. Based on the information from Framingham Heart and Study the lifetime danger of developing coronary artery disease after age 40 is 49% for males and 32% for females [1]. Major risk factors are smoking, hypertension, diabetes mellitus, high plasma low-density lipoprotein, and low plasma high-density lipoprotein in addition to non-modifiable risk factors like age, sex, family history, and genes. Several speculations have been proposed to represent the relationship between inflammation and coronary events including endothelial dysfunction, cytokine interaction with coagulation factors, and initiation of proteases that elevate plaque destabilization [2]. In health, a solid physiologic connection between renal and cardiovascular function works to control extracellular liquid volume and blood pressure. Renal failure adjusts the majority of the elements regulating cardiovascular function through direct hemodynamic effects, neurogenic reflexes, and circling hormones [3]. The principal players associated with these interactions are the renin-angiotensin system (RAS), nitric oxide (NO), and the sympathetic nervous system (SNS). Acute kidney injury influences every one of these frameworks independently, with ramifications for cardiovascular capacity. Intense renal disappointment advances cardiovascular flimsiness and heart brokenness. The event of cardiovascular dysfunction in patients with acute renal failure increases the mortality rate. A rise in levels of coursing proinflammatory cytokines is seen as the pathophysiologic mechanism for cardiovascular dysfunction in acute renal failure. Proinflammatory cytokines exert distinct cardio depressant effects. Acute renal failure may prompt an expansion in pulmonary vascular permeability, most likely mediated by proinflammatory cytokines. Hemodialysis treatment itself may add to cardiovascular stress in acute kidney injury patients by prompting hypoxia and hypoperfusion in vulnerable organs, for example, the heart, brain, gut, and kidney [4]. As to coronary hemodynamics minimal extra oxygen can be removed from the blood in the coronaries, so an increase in oxygen utilization requires an increase in coronary blood flow. By decreasing the lumen of the coronary veins (particularly when stenosis lessens the cross-sectional region by 75%) atherosclerosis restricts its fitting increase in perfusion when the demand for the stream is enlarged, as happens during effort or excitement. These outcomes in exertion initiated angina [5]. A shallow disintegration of the endothelium or a forthright plaque rupture or fissure ordinarily delivers the thrombus that causes episodes of unstable angina pectoris or the occlusive and relatively persistent thrombus that causes acute myocardial infarction. The significant hazard factors for atherosclerosis are smoking, hypertension, diabetes mellitus, high plasma LDL, and low plasma HDL notwithstanding non-modifiable hazard factors like age, sex, family history, and genes. There are still subsets of patients where coronary artery disease (CAD) happens without the previously mentioned risk factors [1, 4]. There is expanding

proof to recommend that inflammation assumes a significant job in the pathogenesis of atherosclerosis and along these lines CAD [6]. A few speculations have been hypothesized to represent the relationship between aggravation and coronary occasions including endothelial dysfunction (ED), cytokine connection with coagulation factors, and activation of proteases that advance plaque destabilization [6].

Materials and methods

It was a prospective study conducted in the General Medicine, Microbiology, and Cardiology Department of Karpaga Vinayaga Medical College, Kanchipuram district, Tamil Nadu from January 2020 to April 2020 among 50 patients with acute ST-segment elevation myocardial infarction admitted in IMCU. All patients with ST-elevation myocardial infarction admitted in IMCU were included. WHO definition of myocardial infarction "A history of prolonged chest discomfort ECG changes consistent with ischemia or necrosis, Elevated cardiac enzymes" was considered. All patients with symptomatic UTI, patients who gave a history of previous urethral catheterization, patients with a history suggestive of urinary tract obstruction, patients with a history of recurrent UTI or treated for UTI in the recent past, patients whose USG showed enlarged prostate and/or significant residual volume of urine were excluded from the study. After obtaining institutional ethical committee approved the study was started. The privacy and confidentiality of the patient were maintained. Informed consent was taken from all the patients for the study. A structured pretested validated questionnaire was used. Primary data was collected by the principal investigator by the interview method. A careful history from each patient regarding atherosclerotic risk factors will be taken and thorough clinical examination and investigation like blood counts, blood sugars, S. Urea, S. Creatinine, fasting lipid profile, Routine Urine analysis with cultures, ECG, ECHO, USG were done in all the cases.

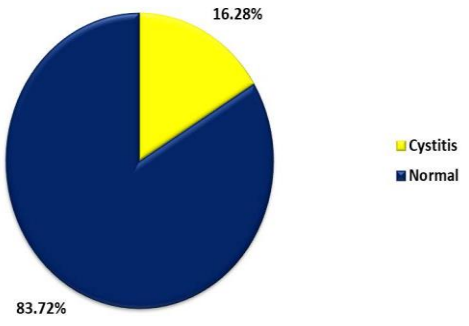
Statistical analysis

Descriptive statistics were reported as mean (SD) for continuous variables, frequencies (percentage) for categorical variables Data were statistically evaluated with IBM SPSS Statistics for Windows, Version 20.0., IBM Corp., Chicago, IL.

Results

The overall mean age of the study participants was 58.58±8.53 years with a range of 42 to 77 years. A total of 32 males and 18 females were present in our study (**Table - 1**). The total number of hypertensives were 64% (n=34), diabetics 56% (n=28) and smokers 54% (n=27). The overall mean LDL and HDL were 131.36±25.18 mg/dL and 37.74±5.83 mg/dL. The mean number of pus cells observed among the study participants was 7.96±1.17 ranging from a minimum of 6 cells to a maximum of 10 cells. The total mean urea and creatinine among the study participants reported were 31.20±13.48 mg/dL and 1.31±0.38 mg/dL respectively. The total number of cystitis was 16.28% (**Figure - 1**). In **Table – 1**, it was seen that females and the number of pus cells were significantly more in the group having cystitis than others. Age was comparable between the groups. EchoEF(%) didn't show any significant difference between the groups.

Figure - 1: Distribution of cystitis among the study participants (N=50).



Discussion

Fifty patients with intense ST-segment elevation myocardial infarction were admitted to

IMCU/ICCU of the cardiology department of Karpaga Vinayaga Medical College, Kanchipuram district, Tami Nadu from January 2020 to April 2020 were included for the assessment. The overall mean age of the study participants was 58.58±8.53 years with a range of 42 to 77 years. A total of 32 males and 18 females were present in our study (**Table - 1**).

Table - 1: Baseline characteristics of study participants (N=50).

Variable	Cystitis (n=7)	Normal (n=43)	p value
Age	60.43±8.10	58.28±8.65	0.54
Gender			
Males	0	32 (25.6)	
Females	7 (100)	11 (74.4)	<0.001
Hypertension	5 (71.4)	27 (62.8)	0.65
Diabetes	5 (71.4)	23 (53.5)	0.37
LDL	131.91±25.33	128±25.90	0.70
HDL	39.29±5.79	37.49±5.86	0.45
Smoking	0 (0)	27 (62.8)	0.002
Pus cells	9.14±0.90	7.77±1.10	0.003
Urea	37.57±10.83	30.16±13.69	0.18
Creatinine	1.12±0.35	1.34±0.38	0.16
EchoEF (%)	54.57±4.79	54.51±4.76	0.97

The total number of hypertensives were 64% (n=34), diabetics 56% (n=28) and smokers 54% (n=27). The overall mean LDL and HDL were 131.36±25.18 mg/dL and 37.74±5.83 mg/dL. The mean number of pus cells observed among the study participants was 7.96±1.17 ranging from a minimum of 6 cells to a maximum of 10 cells. The total mean urea and creatinine among the study participants reported were 31.20±13.48 mg/dL and 1.31±0.38 mg/dL respectively. The number of pus cells was significantly more in the group having cystitis than others. Age was comparable between the groups. EchoEF(%) didn't show any significant difference between the groups.

This perception was like a study already done by John B. Sims, et al. [8] in which he expressed that subclinical UTI was multiple times more common among cystitis than among controls. This is likely a result of the way that in our study UTI was analyzed dependent on urine culture/pus cells rather than checking the number of white blood cells per high power field on urine analysis [9, 10]. Predominantly E.coli was found in our study confined. Among cases, with cystitis, 71.4% were hypertensives contrasted with 62.8 % in those without UTI (p=0.65). Subsequently, among STEMI patients, those with UTI more frequently had hypertension than the individuals who didn't. This observation was similar to the one conducted by John B. Sims, et al. [8]. Other risk factors like diabetes, hyperlipidemia was all the more usually saw in patients with UTI. However, they didn't show any statistical significance. Given the relationship between subclinical proportions of systemic inflammation and CAD development, progression, and instability, it is conceivable that inducers of systemic inflammation, for example, subclinical infections (bacterial or viral) may assume a vital role in instigating STEMI [11, 12]. A likely connection between acute infection and complications of atherosclerosis was first depicted as early as 1897 by Sir William Osler [13]. Various investigations have embroiled infectious agents, for example, Herpesvirus; Cytomegalovirus, C. pneumoniae, Helicobacter pylori, and other infectious microorganisms in the atherosclerotic disease process. Also, acute bacterial respiratory tract infections have been connected to acute myocardial infarction [14].

Chronic and acute infections may trigger plaque rupture through a non-specific inflammatory process. For instance, the presence of raised WBC tally, independent of etiology; was a risk factor for MI in a cohort study [15]. These perceptions propose that systemic inflammation may hasten acute coronary syndrome events. UTI is related to a fundamental host reaction, regardless of whether bacteremia happens. The bacterial connection to uroepithelial cells enacts a cytokine course that incorporates the arrival of IL-1, IL-6, and IL-8 followed by neutrophil and other inflammatory cell recruitment [16]. Neighbourhood cytokine initiation may hence actuate systemic inflammation, advance plaque instability, and thrombosis formation. This mechanism may to some degree clarify the perception in the current study that UTI is related to occurrence STEMI [17]. The limitation of this investigation is that urine was not cultured for organisms other than bacteria. For instance, fungal culture and culture for chlamydia, or mycoplasma were not done as they are not routinely accessible [18]. These examinations could have distinguished more number of UTI cases and in this manner could have added to a more prominent relationship with STEMI.

Conclusion

Subclinical urinary tract infection is more common among patients with the acute coronary syndrome. Underlying infection may precipitate acute coronary syndrome via activation of systemic inflammation. This result should be explored in other data sets, and similar association with other bacterial and viral infections should be examined.

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