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ORIGINAL ARTICLE

Assessment of Pharmacotherapy of Myocardial Infarction Patients at Fauji Foundation Hospital, Peshawar

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ABSTRACT

Objectives: Observation of drug-drug interaction in myocardial infarction at Fauji Foundation Hospital, Peshawar

Methodology: A cross-sectional descriptive study was conducted from May 1, 2022 to July 30, 2022 at Fauji Foundation Hospital, Peshawar. The sample size was 45. The study population was patients admitted to the cardiology unit with a diagnosis of myocardial infarction. A purposive sampling technique was used in the assessment and interview of the patient. Patients suffering from myocardial infarction admitted to hospital during the above-mentioned period were included in this work.

Results: All patients diagnosed with myocardial infarction had major complaints of chest pain and vomiting. There was nausea and sweating in four out of ten cases (80%). Less than half were presented with SOB (shortness of breath) and body aches. Almost two-thirds (60%) of the patients used Glimepiride in the past. Amlodipine and insulin were used by 40% of patients (n =18), while Gibenclamide and metformin were used by 20% of patients (n =9). All the patients had used tablets in the past. Injectables and capsules were used in proportions of 28 and 24 percent, respectively.

Practical implication: This study will provide information to the physician that the major problem with polypharmacy in hospitals is drug-drug interactions.

Conclusion: The study revealed the majority of patients with MI presented with chest pain. The researcher found the main problem with polypharmacy in hospitals is drug-drug interactions. Also, there is a lack of patient counseling, which leads to irrational use of medication.

Keywords: pharmacotherapy; myocardial infarction; polypharmacy; drug-drug interaction

INTRODUCTION

The pathological definition of MI, often known as a heart attack, is the permanent death of myocardial cells brought on by a reduction in blood flow. Chest discomfort, which is usually the most significant of these symptoms and is backed by biochemical test alterations, ECG changes, or results on imaging modalities able to identify myocardial damage and necrosis, are the clinical indicators of MI [1]. In addition to ischemic symptoms, ischemic ECG abnormalities, and imaging evidence, troponin elevation provides the basis for the global definition of myocardial infarction (MI). There are five categories of MIs depending on whether they are spontaneous, related to sudden death, attributable to a blood flow imbalance in the coronary arteries, or related to revascularization treatments. The criteria for the definition are an increase or decrease in troponin levels that take place in a clinical environment [2]. In the United States, cardiovascular disease is the primary factor in both male and female fatalities [3].

Myocardial infarction is the term used to describe the acute ischemia necrosis of a myocardial region or myocardial necrosis brought on by a severe imbalance between coronary blood flow and myocardial demand. Blood flow to a part of the heart is reduced in a myocardial infarction, which damages the heart muscle [4].

Using thrombolytics to halt the progression of myocardial necrosis has gained interest as a result of this work. Nowadays, unless there is a contraindication, percutaneous coronary intervention (PCI) and thrombolytic therapy are regarded as first-line treatments. The American College of Cardiology (ACC) and the American Heart Association (AHA) have a committee that examines the literature and releases practice guidelines on a regular basis to help health care professionals decide the best course of action for patients with STEMI (ST segment elevation myocardial infarction) [5].

Chest pain or discomfort, which may travel to the arm, shoulder, neck, back, or jaw, is the most common or noticeable sign of MI. The majority of patients experience right and left ventricular infarction, and left ventricular inferior wall MI commonly follows. Sometimes the discomfort may feel just like heartburn. Other signs could be dizziness, nausea, shortness of breath, sweating, fatigue, or feeling weak. About 30% of people experience unusual symptoms [6]. Epidemiology; Although the death rate or ratio from ischemic heart disease (IHD) has decreased in developed nations, cardiovascular disease is still the leading cause of death in the US as of 2008—one in three [7]. For instance, rates of cardiovascular death in the US have decreased by approximately a third between 2001 and 2011 [8]. Coronary artery disease frequently manifests as MI. According to WHO estimates, ischemic heart disease caused 12.2% of all deaths worldwide in 2004 [9]. The role of lifestyle variables in the emergence of atherosclerosis and myocardial infarction has been highlighted by epidemiological studies. 90 percent of myocardial infarctions in the INTERHEART study25 of over 15 000 patients were caused by modifiable risk factors such as smoking, dyslipidemia, hypertension, abdominal obesity, and diabetes in males (94 percent in women). Future advancements in risk assessment may involve the use of novel imaging techniques like MRI and CT scanning, particularly in the identification of patients at low risk for whom preventative medicine may not be necessary [10].

Signs and Symptoms: The most prevalent or frequent sign of an acute myocardial infarction is chest discomfort, which is frequently described as a pressure, tightness, or squeezing feeling. The left arm is where the pain most frequently radiates, but it can also radiate to the right arm, neck, lower jaw, back, and belly [11]. There may be additional symptoms in addition to discomfort. In addition to these symptoms occasionally occurring without any pain, nausea or vomiting, sweating, and fainting can also be linked with pain [12]. Acute Myocardial Infarction: An acute myocardial infarction often occurs when an artery has plaque, which blocks the coronary arteries that feed blood to the heart. It can also happen when plaques in the coronary artery break or fissure. When a plaque ruptures, blood is exposed to fatty acids and collagen, which causes platelets to become activated. This is the initial stage of thrombosis and fibrin clot production [13]. Coronary atherosclerosis with superimposed luminal thrombus causes more than 80% of acute myocardial infarcts. Coronary spasm, coronary embolism, and thrombosis in normal, nonatherosclerotic blood vessels are uncommon causes of myocardial infarction. Concentric subendocardial necrosis can also happen after prolonged cardiac

arrest with resuscitation due to global ischemia and reperfusion. Although myocyte hypoxia results in specific changes that depend on the length of the vessel's occlusion, the time between the occlusion and reperfusion, and the presence of collateral circulation, myocardial ischemia shares characteristics with other types of myocyte necrosis, such as that brought on by inflammation [14]. The Etiology of MI: Smoking contributes to around 36% of CAD, and obesity contributes to roughly 20% (coronary artery disease). Inactivity is thought to be responsible for 7 to 12 percent of MI cases. About 3% of cases of MI and persistently elevated stress levels can be attributed to less prevalent reasons such as stress-related work or job stress. The type of food we consume is a significant component that can be taken into consideration in cases of myocardial infarction because diet can potentially cause it. Oral contraceptive pill users are more likely to get a myocardial infarction, particularly if other risk factors are also present. Use of NSAIDs (non-steroidal anti-inflammatory medicines) for even a week can raise the risk of MI. Air pollution is another significant concern that is controllable. Acute CVS events such as MI have also been associated with short-term exposure to nitrogen dioxide, sulphur dioxide, carbon monoxide, and other gases. Atherosclerosis and MI can also be linked to a number of acute and chronic infections. These infections include influenza, Chlamydophila pneumoniae, and H. Pylori, among others.

Globally, 17.1 million people die from coronary heart disease (CHD) each year [15]. Thus, CHD is currently the major cause of death worldwide and will probably always be the main factor in mortality trends. In low- and middle-income countries, CHD is the leading cause of death for people under the age of 70 (39%) [16]. Risk of MI is higher among Asians [17, 18]. MI is thought to affect South Asians in the UK at a rate that is 50% higher than that of white people [19]. With a population of more than 187 million, Pakistan is a developing nation in South Asia [20]. According to Kannel's citation of the Framingham study from 1961, smoking, diabetes mellitus, hypertension, and hypercholesterolemia were the main risk factors for the development of CHD (16). In Rahim Yar Khan and Peshawar, Pakistan, there is little information available on MI risk factors [21].

Therefore, the purpose of the current study was to observe drug-drug interactions in myocardial infarction at Fauji Foundation Hospital, Peshawar.

METHODOLOGY

An observational study was conducted from May 1, 2022 to July 30, 2022 in cardiology at Fauji Foundation Hospital, Peshawar. The sample size was 45. The study population was patients admitted to the cardiology unit with a diagnosis of myocardial infarction. A purposive sampling technique was used. Purposive sampling's major objective is to concentrate on particular demographic features that are interesting to you in order to best enable you to respond to your research questions [27].

Data from patients was collected and analyzed for MI, its complications, and management. The sample includes patients from nearby rural and urban areas, referred by accident and emergency or OPD to the cardiology ward.

Inclusion criteria: Patients suffering from myocardial infarction admitted to hospital during the above-mentioned period were included in this work. The majority of the patients in this research had acute MI upon presentation, while a few individuals had MI that was later revealed while being treated in the hospital.

Exclusion Criteria: Patients were also excluded if they had previously had fluctuating chest discomfort.

Data Collection: The data collected in respect of each case, from the hospital treatment chart and/or interview with the patient or the patient's attendants, includes the patient and treatment-related information like name, age, gender, major complaints, present illness history, past medical and surgical history, family history, social history, history of medication, allergies, physical examination, laboratory examination, drug-therapy provided in the hospital, discharge medications and outcomes of treatment.

Data Analysis

- Each patient's medication history was examined for drug allergies, responses, side effects, compliance, and its use in the evaluation and treatment of the patient's present medical conditions.
- Current therapy provided in the hospital was analyzed for its indications and outcomes.
- The whole medication therapy provided in the hospital was analyzed for drug-related problems and their management.
- Any drug information, therapeutic consultation, or patient education and counseling that were provided during rotations were also reported.

RESULTS

Demographic Information:

- More than half (53) percent of the participants
- were female (n=24). Fourscore 80 percent
- of the patient admitted from emergency department.

Chief complaints of the patients

- All patients diagnosed with myocardial infarction had chief complain of chest pain and vomiting. Fourscore (80) percent were present with nausea and sweating. Less than half were presented with SOB and body aches

Table 1:

Demographic details	Total (n=45)	%
Age group		
35-49	9	20
50-65	18	40
66-80	18	40
Gender		
Male	21	47
Female	24	53
Mode of admission		
Emergency	36	80
OPD	9	20

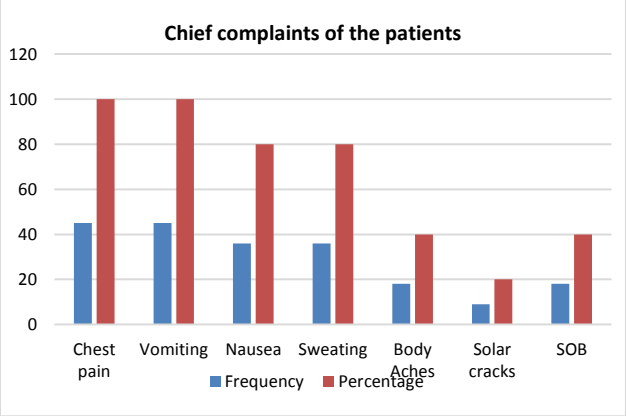

Figure 1:

Generics Statistics in Past Medication History: Almost two third (60) of the patient used Glimepiride in past. patients used Amlodipine and Insulin were 40 percent (n=18) followed by Gibenclamide and metformin twenty percent (n=9)

Table 2:

S.No	Drug Generic	Frequency	Percentage
1	Amlodipine	18	40
2	Glimepiride	27	60
3	Metformin	9	20
4	Insulin	18	40
5	Glibenclamide	9	20

Dosage form based statistics in past medication history: All patients had used tablet in past medication. Injectable and capsule were used 28 and 24 percent respectively

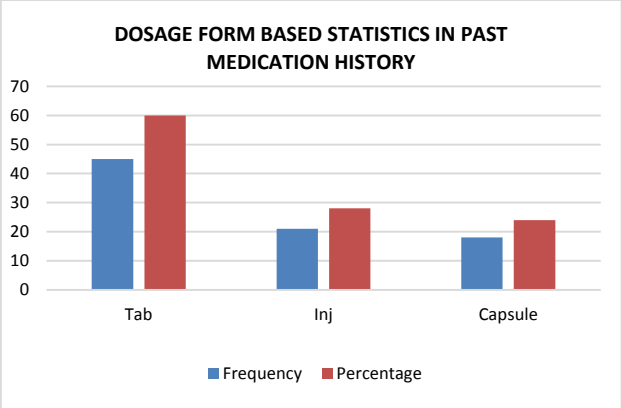

Figure 2:

Table 3: Different Drugs Used in Treatment of Myocardial Infarction.

Generic name	Brand name	Frequency	Percentage
Aspirin	Disprine	27	60
Furosemide	Lasix	18	40
Captopril	Capril	27	60
Enoxaparin	Clexane	36	80
Rosuvastatin	Rast	36	80
Rosuvastatin	Rovista	27	60
Insulin	Humilin	18	40
Glyceryltrinitrate	Cardnit	27	60
Sitagliptin+metformin	Sitamet	15	33.33333
Aspirin/clopidogrel	Abiclot plus	27	60
Glyceryltrinitrate	Cardnit	30	66.66667
Dimenhydrinate	Gravinate	24	53.33333
Aspirin/clopidogrel	Lowplat plus	36	80
Glyceryltrinitrate	Sustac	15	33.33333
Clopidogrel	Lowplate	9	20
Clopidogrel	No clote	21	46.66667
Pantoprazol	Zopent	18	40
Pantoprazol	Neege	27	60
Metoprolol	Merol	24	53.33333
Salbactum & cefoperozone	2 sum	9	20
Striptokinase	Ekinase	24	53.33333

Showing Drug Drug Interactions

Table 4: Aspiririn and clopidrogrel interaction occurred in 27 out of 45 patients, followed by Clopidrogral and pantaprazoole n=21.

S.No	Drug Drug Interactions	Mechanism of Interaction	Frequency (n=45)
1	Aspirin+Clopidogrel	Pharmacodynamics synergism. High dose cause toxicity. Monitor closely.	27
2	Clopidogrel+Pantoprazole	Pantoprazole decrease the effect clopidogrel by affecting hepatic enzyme CYP2C19 metabolism, Use caution.	21
3	Aspirin+Captopril	Pharmacodynamics antagonism. Avoid or use alternate drug. Aspirin reduces the synthesis of vasodilating renal prostaglandins.	15
4	Ceftriaxone+Warfarin	Ceftriaxone increases effects of Warfarin by Anticoagulation	3
5	Enoxaparin+Warfarin	Additive Effect	3
6	Enoxaparin+Captopril	Enoxaparin increase the toxicity of captopril.	6
7	Furosemide Ramipril	Increases toxicity of each other by Pharmacodynamics Synergism	3
8	Nalbuphine+Aspirin	Tramadol provoke withdrawal symptoms in pts. Who are currently opiates dependent.	3
9	Omeprazole+Clopidogrel	Omeprazole decreases effect of clopidogrel by affecting hepatic enzymeCYP2C19 metabolism	15

DISCUSSION

Acute myocardial infarction treatment has significantly changed in recent years. Aspirin has a well-established role as an anti-platelet medication in the treatment of acute myocardial infarction, and it has been proven to lower mortality by itself by 27%. Unfractionated heparin (UFH), aspirin, and low-molecular-weight heparin (LMWH) have all been used to lessen the likelihood of rethrombosis. The GpIIb/IIIa receptor inhibitors stop platelet aggregation. Aspirin and GP IIb/IIIa inhibitors were administered in conjunction in my study at the cardiology ward of Fauji Foundation Hospital, Peshawar and they had positive effects on patients who had myocardial infarction.

For eligible individuals with AMI, thrombolytic therapy is a typically secure and efficient pharmacological approach to treatment. According to a study, thrombolytic treatment has reduced mortality in individuals with STEMI by one third from what it was before (10–15 percent to 6–10 percent). Although practically all of our research supports this [22].

Despite the vast amount of data supporting its use and the lack of disagreement in clinical research on the mortality reduction found, only about 45 percent of patients with acute AMI receive thrombolytic therapy. About 70% of the patients in our trial at Fauji Foundation Hospital, Peshawar received thrombolytic therapy, and they benefited from it.

The risk of death from acute myocardial infarction is reduced by 30–40% when streptokinase is administered within 90 minutes to patients. Therefore, a door-to-needle time of 15 minutes is optimal and testifies to the effective triage of an institution (time taken in the administration of SK after a patient presents in an emergency) [3]. An indicator of our emergency services in our study is a mean door-needle time of 10 minutes.

Clinical trials have been conducted regarding ACE inhibitors, and these have shown a reduction in mortality in patients who were on ACE inhibitors orally. In our study, Ramipril was prescribed to patients, and it shows maximum benefit and reduces mortality in patients. However, I noticed a few patients in my training that were experiencing side effects of this drug. So it is necessary to closely monitor such patients.

Beta-blockers are also used in the management of AMI patients. The rates of morbidity and mortality associated with infarction were reduced with beta blockers before the usage of thrombolytic drugs. These drugs should be prescribed to all patients suffering from AMI and they should be continued unless there is contraindication [3, 23].

By using statins for a longer period of time, patients with cardiovascular disease experience a lower rate of morbidity and mortality. Early intensive treatment with atorvastatin, simvastatin, flavastatin, or pravastatin for patients with STEMI or NSTEMI is supported by some evidence or data [24]. Rosuvastatin was primarily utilized to treat myocardial infarction in the case of our study, and it showed a decrease in morbidity and mortality. However, if a patient has renal issues, they need to be continuously monitored.

An overall in-hospital mortality of 10% as reported. However, it needs to be kept in mind that our hospital, being a referral center, receives proportionately larger number of critically ill patients from peripheral hospitals & medical facilities.

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