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Acute Myocardial Infarction and Hemodynamic Instability

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Abstract: **Background:** acute myocardial infarction with hemodynamic instability. **Aim of the study:** To assess the incidence of hemodynamic instability, factors associated with it, and in-hospital outcome among AMI patients. **Patients and methods:** A case control study of 500 patients with AMI, of them 100 patients have AMI with hemodynamic instability and 400 have AMI with no hemodynamic instability. A number of 345 males and 155 females were included in this study, and their ages ranged between 26 and 87 years. The data was recruited from the emergency department of AI-Yarmook teaching hospital and Ibn Al Nafees teaching cardiovascular hospital between March 2024 and March 2025. All patients diagnosed to have AMI were included in the study; for all of them, we take history, do examination, investigations, ECG, cardiac 24-hour monitoring, CXR, and echocardiography. **Results:** We found that hemodynamic unstable females were more than those who were stable, while in males, hemodynamic stable patients were more than hemodynamic unstable patients. The percentage of males that develop AMI was more than that of females in both groups. Age range was higher in hemodynamic unstable patients. the presence of risk factors (D.M, HPT, hx of IHD, hx of H.F, family hx of IHD) were more common in hemodynamic unstable patients than hemodynamic stable patients, while (hyperlipidemia, and smoking) were more common in hemodynamic stable patients. Anterior wall MI and double wall (multiple infraction sites) was more common in hemodynamic unstable patients; in contrast, inferior wall MI and other types were more common in hemodynamic stable patients. Dysrrhythemia (tachyarrhythmia and bradyrrhythmia) was more common in hemodynamic unstable patients. The ventricular premature beats were the most common type of tachyarrhythmia in both groups. Patients that have no pulmonary congestion until discharge were more common in the hemodynamic stable group, while the development of pulmonary congestion and cardiogenic shock was more common in hemodynamic unstable patients; the mortality rate was higher in hemodynamic unstable patients. Hemodynamic stable patients receive thrombolytics more than hemodynamic unstable patients do; most of the hemodynamic unstable patients were candidate for primary PCI. All hemodynamic unstable patients develop complications, while most of hemodynamic stable patients do not. All post-AMI complications were more common in hemodynamic unstable patients. The patients that develop hemodynamic instability in the 1st 24 hours were more than those who developed it after that. **Conclusion:** Hemodynamic instability complicating AMI carries high morbidity and mortality, mandating intensive monitoring and management that includes invasive hemodynamic monitoring and early coronary revascularization.

Keywords: Acute Myocardial Infarction; Hemodynamic Instability; Acute Coronary Syndrome; Electrocardiogram; and Heart Failure.

INTRODUCTION

In the developed world, acute myocardial infarction remains a significant public health issue, and in developing nations, it is becoming a more significant one. Even though the fatality rate from AMI has decreased by around 30% in the last ten years, about one-third of patients still die from the condition (Liu, X. *et al*., 2024; Smith, S.C. *et al*., 2023). Approximately 50% of AMI-related fatalities happen within an hour of the incident and are caused by arrhythmias, most frequently ventricular fibrillation (Zhao, Y. *et al*., 2023). From a global standpoint, the World Heart Federation's predictions that the burden of illness in developing nations will converge more closely with that currently plaguing rich nations are especially concerning (Yang, Y. and Gao, Y., 2022). The pathological condition known as acute myocardial infarction is almost always caused by the formation of an occlusive thrombus at the site of rupture and erosion for an atheromatous plaque within a coronary artery (Wang, Y. *et al*., 2023; Johnson, C.L. *et al*., 2021; Ahmed, M. *et al*., 2022). The thrombus frequently undergoes spontaneous lysis over the course of the following few days, but irreversible myocardial damage has already occurred by this point (Peterson, E.D. *et al*., 2022). The infarction process progresses over several hours, so most patients present while myocardium salvage and an improved outcome is still possible (Schwartz, C.L. *et al*., 2021). Acute circulatory failure can be identified by physical signs such as hypotension, abnormal heart rate, cold extremities, and periphral cyanosis, as well as by bedside blood pressure measurements (Nguyen, T.T. *et al*., 2021). Hemodynamic instability, or more precisely, circulatory shock, is a state of either perfusion failure or simply one or more measurements that indicate out-of-range but not necessarily pathological values (Kumar, A. *et al*., 2023).

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Hemodynamic instability, or more precisely, circulatory shock, is a state of either perfusion failure or just one or more measurements that show acute circulatory failure, physical indications that are out of range but not necessarily pathogenic (Fitzgerald, J.R. *et al*., 2024). Hypotension, an irregular heartbeat, chilly extremities, peripheral cyanosis, bedside blood pressure measurements, right-sided filling pressure, and decreased urine output are all indicative of shock (Saha, S. *et al*., 2020). There is no need for invasive hemodynamic monitoring. Because the circulation status in patients with clinically uncomplicated AMI can be determined by careful clinical evaluation, this usually entails monitoring heart rate and rhythm, taking repeated measurements of systemic arterial blood pressure, getting chest roentgenograms, carefully and repeatedly auscultating the lung fields in pulmonary congestion, measuring urine flow, and looking for signs of adequate perfusion in the skin and mucous membranes. The most crucial life-saving measure in cardiogenic shock is early revascularization with coronary artery bypass grafting or percutaneous coronary intervention (Bansal, S. *et al*., 2023; Mansour, A. *et al*., 2022).

Patients under 75 years of age, those who have had a prior MI, and those who get treatment within six hours of the beginning of symptoms report the highest short-term improvement. With medical care, patients over 75 years of age have a higher chance of survival (Chung, M. *et al*., 2024). The use of coronary artery bypass graft is limited since it necessitates substantial surgical as well as medical resources that has a high risk of operation for these patients who are very sick (Urbano, L. *et al*., 2022). Nonetheless, the majority of patients with cardiogenic shock under 75 years of age should have early revascularization, according to outcome studies. For optimum outcomes, primary PCI should be done within 90 minutes of the start of chest pain, although it can also be done within 18 hours of the start of shock and within 36 hours of the start of chest pain (Lee, J.H. *et al*., 2023). Mortality rates decrease when PCI is carried out two to three hours following the onset of symptoms (Choi, E.K. *et al*., 2021). Crucially, in patients receiving primary PCI for STEM, the interval between the start of symptoms and balloon inflation is substantially connected with 1-year mortality after controlling for baseline variables (Zhang, J. *et al*., 2023).

METHODOLOGY

The current study comprised 500 AMI patients among March 2024 and March 2025. A total of 400 individuals with hemodynamically stable angina and 100 with hemodynamically unstable angina were eliminated. The proportion of data collected from patients who were hemodynamically unstable as opposed to the proportion of data collected from patients who were hemodynamically stable was used as a control group. The AL-Yarmook Teaching Hospital's emergency room and the Ibn Al Nafees Cardiovascular Teaching Hospital provided the data. The study included 345 male participants and 155 female participants. The patients' ages ranged from 26 to 87 years.

To be diagnosed with AMI, patients are required to fulfill a minimum of two of the following requirements:
1. Common ischemic chest pain (discomfort) that lasts for at least half an hour.
2. ECG alterations, ST-segment elevation, or new LBBB.

* AMI was split into two groups according to the ECG results that were related to them:
- AMI with ST-segment elevation.
- AMI without ST-segment elevation.
3. Increased myocardial necrosis indicators (CK-MB, Troponins).

In order to collect data for the current study, a brief history and comprehensive examination are conducted for each patient with an AMI diagnosis. This includes information about the patient's name, age, gender, history of smoking, diabetes mellitus, hypertension, and IHD, H.F., co-morbid diseases, medications, such as exposure to cocaine or other sympathomimic drugs, and family history of IHD.

Every hour during the first 24 hours, a systemic examination is performed on all patients, including vital signs (blood pressure, heart rate, and breathing rate) and Cardiogenic shock manifests clinically as volume overload (dyspnea, rales) and inadequate cardiac output combined tissue hypoperfusion (hypotension, clouded sensorum, chilly mottled skin, acidosis, oligurea). ECG, cardiac monitoring, chest x-ray, transthoracic echocardiography, as well as investigations (Hb & PCV, lipid profile during the first 24 hours of admission, RBS, renal function test, liver function test, and serum cardiac biomarkers) are performed on all research participants. Patients listed in the research who are candidates for PCI and those who

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are candidates for thrombolytics have received them.

All patients who are referred to the ward and CCU are monitored until they are discharged. Complications that arose following AMI in the current study's subjects during the hospital stay are described. Since it is used to evaluate wall motion abnormality, ejection fraction, mitral regurgitation, pericardial effusion, mechanical free wall rupture, and papillary muscle rupture, interventricular septum rupture, and right ventricular infarction, transthorasic echocardiography is a crucial tool in the diagnosis and detection of complications. The majority of the patients in the study underwent this procedure.

If the patient has poor perfusion signs and symptoms (cold extremities, mottling, oligurea, clouded sensorum, central pallor), evidence of volume overload (dyspnea, rales), and a systolic blood pressure of less than 90 mm Hg for more than an hour despite a fluid challenge, they are considered hemodynamically unstable.

Since pulmonary wedge pressure, along with cardiac index examinations, were unavailable in our emergency room or in critical care units, we rely on the clinical picture as well as the shock index value to determine whether a patient is hemodynamically unstable. Shock index is calculated for all patients (heart rate/systolic blood pressure), and a patient is in shock when it is greater than 1 (roughly 0.5 and may reach 1).

RESULTS

Table 1: Age & gender distribution in hemodynamically stable and unstable patients

	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
Gender Male	285	71.25	60	60.0	0.030*
Female	115	28.75	40	40.0	
Age (years) < 40 years	21	5.25	1	1.0	0.0005*
40--	67	16.75	3	3.0	
50--	99	24.75	25	25.0	
60--	123	30.75	36	36.0	
70--	74	18.5	25	25.0	
≥ 80 years	16	4.0	10	10.0	
Mean±SD (Range)	60.04±11.84 (26-86)		66.04±11.39 (30-87)		

Table 2: Risk factors in hemodynamic stable & unstable patients.

	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
Diabetes Yes	88	22.0	24	24.0	0.668
No	312	78.0	76	76.0	
hyperlipidemia Yes	104	26.0	21	21.0	0.302
No	296	74.0	79	79.0	
Hypertension Yes	152	38.0	42	42.0	0.463
No	248	62.0	58	58.0	
History of IHD Yes	173	43.3	62	62.0	0.0008*
No	227	56.7	38	38.0	
History of H.F. Yes	11	2.75	4	4.0	0.512
No	389	97.25	96	96.0	
Smoking Yes	184	46.0	44	44.0	0.719
No	216	54.0	56	56.0	
Family history of IHD: Yes	120	30.0	45	45.0	0.004*
No	280	70.0	55	55.0	

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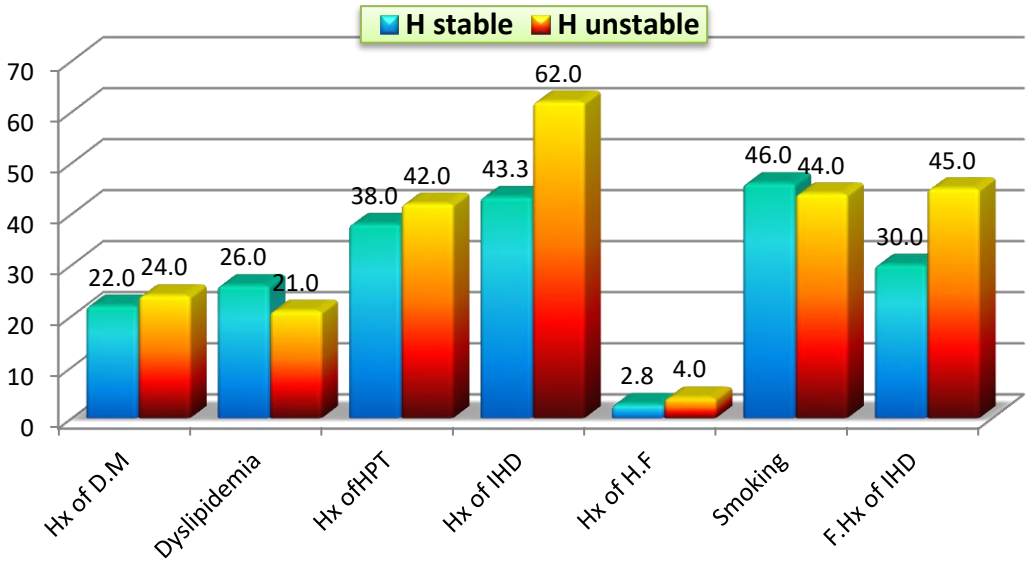

Figure 1: Risk factors

Table 3: Myocardial infarction site in hemodynamically stable and unstable patients

Infarction site	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
Anterior	145	36.25	44	44.0	0.340
Multiple site infarction	7	1.75	3	3.0	
Inferior	233	58.25	51	51.0	
Others	15	3.75	2	2.0	

Table 4: Dysrrhythemia in hemodynamically stable and unstable patients

	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
Arrhythmia Yes	160	40.0	62	62.0	0.0001*
No	240	60.0	38	38.0	
Bradydysrhythemia					
Sinus bradycardia	13	8.1	3	4.8	0.063
A systole	5	3.1	3	4.8	
1st degree heart block	15	9.4	1	1.6	
2nd degree heart block 1	6	3.8	1	1.6	
Type 2	3	1.9	1	1.6	
3rd degree heart block	2	1.3	4	6.5	
Thachydysrhythemia					
Sinus tachycardia	13	8.1	6	9.7	0.577
Aterial premature contractions	10	6.3	5	8.1	
Supraventricular tachycardia	11	6.9	4	6.5	
Atrial fibrilation	9	5.6	9	14.4	
Ventricular premature beats	41	25.5	14	22.6	
Ventricular Fibrilation	15	9.4	6	9.7	
Ventricular tachycardia	17	10.6	5	8.1	

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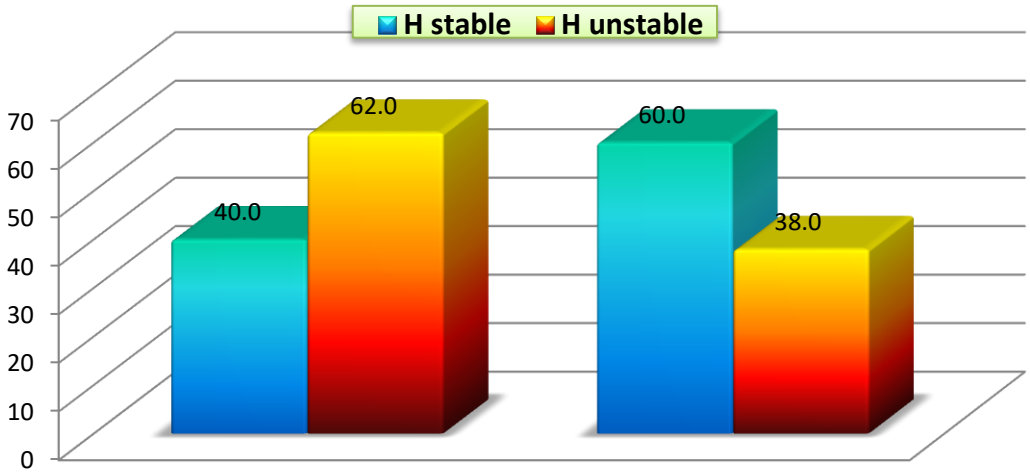

Table 5: In-hospital outcome in hemodynamically stable and unstable patients

	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
In hospital outcome, no H.F	379	94.75	8	8.0	0.0001*
H. F	5	1.25	21	21.0	
Dead	16	4.0	71	71.0	

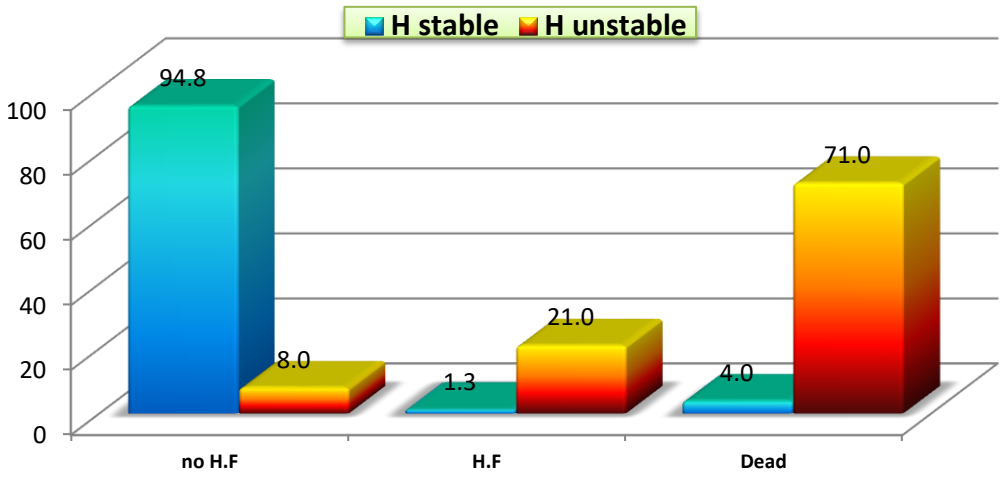

Figure 3: In-hospital outcome

Table 6: Intervention recommended in hemodynamic stable and unstable patients

Intervention recommended		Hemodynamic stability				P value
		Hemodynamic stable		Hemodynamic unstable		
		Number	%	Number	%	
Thrombolytic indication	Yes	224	56.0	15	15.0	0.0001*
No		176	44.0	85	85.0	
PCI indication	Yes	321	80.25	87	87.0	0.119
No		79	19.75	13	13.0	

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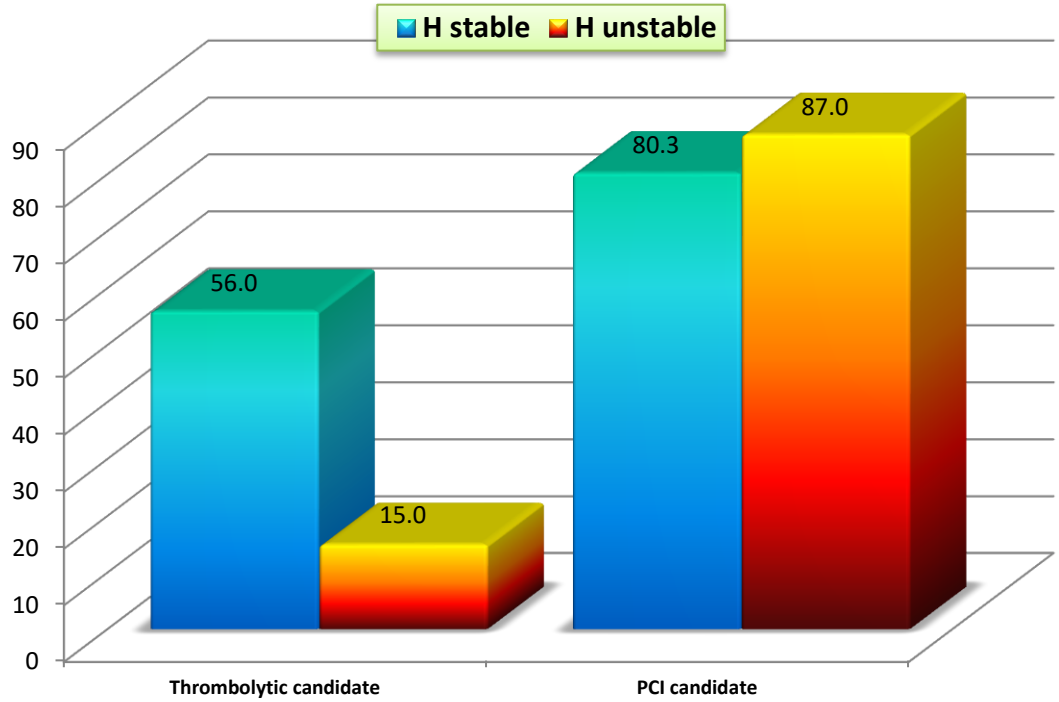

Figure 4: Thrombolytic and PCI candidate patients.

Table 7: Complications in hemodynamically stable and unstable patients

	Hemodynamic stability				P value
	Hemodynamic stable		Hemodynamic unstable		
	Number	%	Number	%	
Complication Yes	71	17.75	100	100.0	-
No	355	88.75	-	-	
Complication Pericarditis	23	5.9	7	7.0	0.638
Post MI angina	26	6.5	12	12.0	0.063
Cardiac failure	13	3.3	80	80.0	0.0001*
Stork	4	1.0	2	2.0	0.411
Mechanical	31	7.75	19	19.0	0.0008*
Dysrrhythemia	49	12.25	62	62.0	0.0001*
Cardiogenic shock	4	1.0	35	35.0	0.0001*

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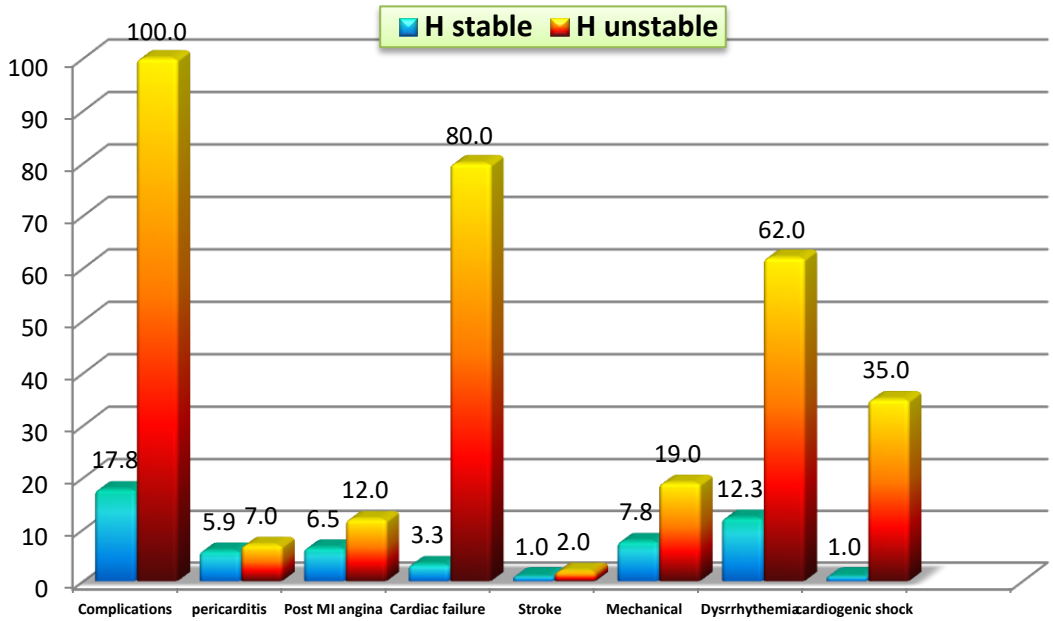

Figure 5: complications

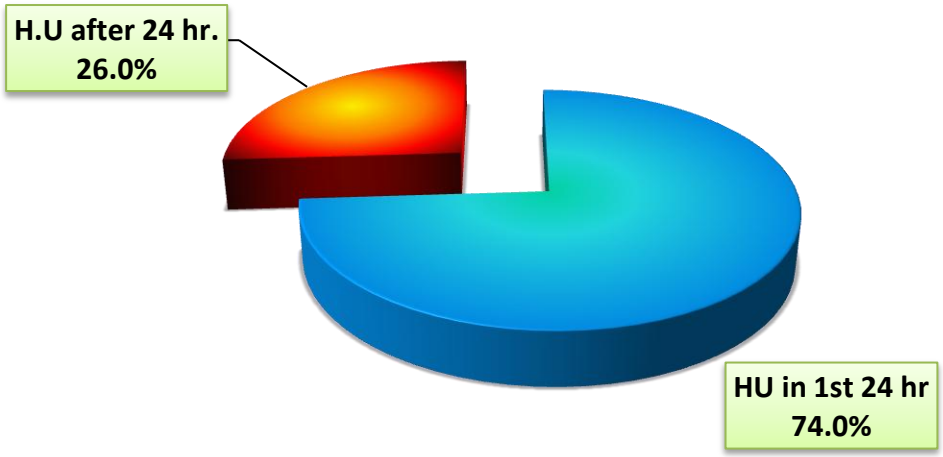

Figure 6: onset of Hemodynamic instability.

DISCUSSION

Acute myocardial infarction (AMI) consequences, which can result in high rates of morbidity and death, are significantly influenced by hemodynamic instability (Owens, T. and Smith, A.M., 2022). Of the 500 patients in this trial, 100 had hemodynamic instability from the time of hospital admission until their discharge, whereas the other 400 did not. Males are more likely than females to get AMI, according to the study, in both hemodynamic stable and unstable groups (Vasudevan, J. *et al*., 2023). AMI was more common in hemodynamically unstable patients than in hemodynamically stable patients, and it was more common in hemodynamically unstable females than in hemodynamically stable ones (Patel, P. *et al*., 2021).

There was no significant difference in the history of diabetes mellitus between the two groups, nor between patients who were hemodynamically stable and those who were unstable (Huang, Z. *et al*., 2020). Additionally, there was no discernible difference in hyperlipidemia between the two groups. Ventricular premature beats were the most prevalent form of tachydysrhythmia in both hemodynamically stable and unstable individuals. Hemodynamically unstable patients were more likely to have anterior wall MI than hemodynamically stable patients, whereas hemodynamically stable patients were more likely to have inferior wall MI. Compared to hemodynamic stable patients, who had a 1.75% incidence of double wall MI, hemodynamic unstable patients had a 3% incidence (Alghatrif, M. *et al*., 2022; Bohm, C.A. *et al*., 2020; Jaeger, S.C. *et al*., 2023).

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There was no discernible difference in papillary muscle rupture (PMI) between individuals who were hemodynamically stable and those who were unstable. There was no discernible difference between hemodynamically stable and unstable individuals' post-MI angina or ischemia (Singh, M.B. *et al*., 2024). Nonetheless, there were notable differences in the two groups' histories of heart failure. After AMI, 1% of patients who were hemodynamically stable and 2% of patients who were hemodynamically unstable experienced a stroke; these rates are not statistically significant (Salgado, A. *et al*., 2023). Ten percent of AMI deaths had mechanical problems, such as ventricular free wall rupture, interventricular septum rupture, or papillary muscle rupture, which often happened one to five days after infarction. For the majority of the patients in the research, echocardiography was the preferred diagnostic test (Ashrafian, H. *et al*., 2020).

Patients with hemodynamic instability differed significantly from those with dysrhythmia. 35.0% of hemodynamically unstable patients, or 7.0% of the study's total population, experience cardiogenic shock. The majority of these patients only receive medical care, and many pass away hours to days after shock. Cardiogenic shock occurs in 90% of patients when they are in the hospital, compared to 10% of patients when they are admitted (Murray, C.J.L. *et al*., 2021; Taleb, A. *et al*., 2024).

CONCLUSION

High morbidity and fatality rates associated with hemodynamic instability complicating AMI necessitate rigorous monitoring and treatment, including invasive hemodynamic monitoring as well as early coronary revascularization.

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