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RESEARCH ARTICLE
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HIGH DEGREE AV NODAL BLOCK IN ACUTE ST ELEVATION MYOCARDIAL INFARCTION ASSOCIATED 30-DAYS MORTALITY AND NEED OF PERMANENT PACEMAKER AND ITS CORONARY ANGIOGRAPHY FINDINGS

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INTRODUCTION:

Acute myocardial infarction (AMI) is a grave clinical condition and remains a leading cause of mortality all over the world. It occurs due to loss of blood supply to part of the heart muscle resulting in tissue injury as a result of oxygen deprivation [1]. Myocardial infarction is considered part of a spectrum referred to as acute coronary syndrome (ACS). The ACS continuum represents ongoing myocardial ischemia or injury and it consists of unstable angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI) [2]. Worldwide, more than 3 million people suffer from STEMIs and 4 million develop NSTEMIs a year [3]. STEMIs occur about twice as often in men as women [4]. Thus, only extensive damage that includes most of the ventricular septum and the anterior wall may interrupt the conduction of the left bundle [5]. Regarding conduction disturbances and infarct location, the clinical management of patients with conduction abnormalities after an MI depends in part upon the location of the infarct. Second or third-degree AV block associated with inferior wall MI is located above the His bundle in 90 percent of patients [4-5]. AV block associated with anterior MI is more often located below the AV node [5]. Conduction defects complicating acute myocardial infarction have been associated with an adverse prognosis in reports which antedate the widespread use of thrombolytic therapy [6]. Acute coronary syndrome is often complicated by electrical conduction disorders. Characteristics of conduction disorders such as high-degree AV block (HDAVB) have been well described in ST-elevation myocardial infarction (STEMI) patients [5-6]. In contrast, the incidence of HDAVB following non-ST-elevation myocardial infarction (NSTEMI) has only been reported in 2 studies (0.4% and 1.9%), which was lower than that in STEMI population (3.2% to 3.5%) [7]. The rate complete AV block after STEMI varies from 2.9 to 12.8%. In Pakistan the rate of complete AV block after STEMI have been reported to be 2.9–11.8% [8-9]. The presence of conduction defects complicating acute myocardial infarction (MI) is relatively frequent and is associated with increased short and long term mortality

rates.

Atrioventricular (AV) nodal conduction defect is a common complication of acute Myocardial Infarction (MI) which is accounted as 7.3% [10]. Mobitz type I (Wenckebach), and complete heart block are commonly seen, since the SA node, AV node, and His bundle are primarily supplied by the RCA [8-9]. Less commonly, anterior MI produces first degree AV block below the level of the AV node, a situation that should be presence of a widened QRS complex [10]. High degree AV block: High (second or third) degree AV block occurs in approximately 9.8 percent of patients with an inferior MI who receive thrombolytic therapy [11]. Other studies reported atrioventricular nodal conduction defects as 50%, 5.3% in patients presenting with acute myocardial infarction [12-13]. Inferior MI is typically associated with the more benign second-degree AV block of the Wenckebach type (Mobitz type 1); Mobitz type II is uncommon in this setting, generally occurring with anterior MI. Mobitz type I block is usually transient, resolving in most cases within five days. Rarely, RCA occlusion produces complete heart block (CHB) that is usually transient but may persist. The latter finding suggests concurrent involvement of the left coronary system, resulting in poor collateral flow [14-15]. The rationale of this study is to evaluate the prevalence of atrioventricular nodal conduction defects in patients with ST elevation Myocardial infarction 30 days mortality and patients requiring permanent pacemaker. As AV block along with STEMI is associated with higher morbidity and mortality. As above mention studies shows significant variations in prevalence of atrioventricular nodal conduction defects [9,12-13]. The results of this study will provide the current and concreate statistics of AV nodal conduction defects and help the clinicians to design a protocol and make some clinical recommendations in our routine practice guidelines for management of these high risk group patients in order to reduce the morbidity and mortality of the community.

OBJECTIVE

To determine the frequency of atrioventricular nodal conduction defects in patients presenting with ST elevation Myocardial infarction associated 30 day mortality and requirement of permanent pacemaker.

OPERATIONAL DEFINITIONS

ST-ELEVATION MYOCARDIAL INFARCTION (STEMI)

Will be diagnosed in patient with any two of the following criteria;

- Typical chest pain >20 minutes (retrosternal pain with radiation to left arm or shoulder, aggravates on exertion or emotional stress, relieved with rest or nitroglycerin)
- New ST elevation in at least two contiguous leads >2mm in men or >1mm in women in leads V2 to V3 and/or of>1mm in other contiguous chest leads or limb leads.

ATRIOVENTRICULAR NODAL CONDUCTION DEFECTS It will be defined as a prolonged PR interval (>0.20 seconds) in rhythm strip. Mobitz type I or Wenckebach second degree AV block will be diagnosed when there will be progressive lengthening of the PR interval until a normally occurring P wave will not be followed by a QRS complex. Mobitz type II second degree AV block will be diagnosed when p waves not followed by QRS complex occasionally or repetitively with consistent PR interval. Third degree AV block will be diagnosed when there will be no AV nodal conduction and the P waves will be completely dissociated from the QRS complexes. Presence of any of above defects will be considered AV nodal conduction defect.

- Patients with preexisting documented AV blocks, sick sinus syndrome, and other reversible causes of AV block such as hyperkalemia will not be considered.

EFFECT MODIFIERS

- **HYPERTENSION:** Patients with documented history of hypertension and on anti-hypertensive drugs for at least 6 (either control / uncontrolled) months will be labeled as hypertensive.

▪ **DIABETES MELLITUS:** Patients with documented history of diabetes and on anti-diabetic medications for at least 6 months (either control / uncontrolled) will be labeled as diabetic.
▪ **SMOKING:** If an individual smoke > 10 pack- year either currently smoking for > 1 years or quit smoking since < 6 months will be marked as smoker.

▪ **FAMILY HISTORY OF CAD:** It will be defined as ischemic heart disease in the father or a brother diagnosed before age 55 years and in the mother or a sister diagnosed before age 65 years.

MATERIAL AND METHODS

STUDY DESIGN: Descriptive Observational Study.
SETTING: This study will be conducted in Department of Adult Cardiology, National Institute of Cardiovascular Diseases (NICVD), Karachi, Pakistan.

DURATION OF STUDY: 06 months from the date of approval of ERC

SAMPLE SIZE: By using W.H.O sample size calculator using frequency atrioventricular nodal conduction defects as (5.3%)[13] in patients with STEMI. Confidence level=95%, Margin of error (d)=1.5% then the estimated sample size will be n=1111 patients.
SAMPLING TECHNIQUE: Non-Probability, Consecutive Sampling.
SAMPLE SELECTION
INCLUSION CRITERIA

- Age >18 years.
- Either Gender
- Patients presented with ST-Elevation Myocardial Infarction (as per the operational definition).

EXCLUSION CRITERIA

- Patients refuse to give consent for participation in the study

Data Collection

Data collection for the study will be started after approval from the ethical review committee of the institution. The study will include subjects presenting with STEMI at the adult cardiology department, NICVD, Karachi and fulfilled the inclusion and exclusion criteria. Prior to inclusion the purpose, and benefits of the study will be explained to all participants and verbal informed consent will be taken by the principal investigator from all patients regarding their participation in the study and publication of study findings. Demographic and clinical characteristics of the patients will be recorded such as age (years), gender, smoking, diabetic mellitus, family history of CAD, and hypertension will be recorded as per the operational definitions.

Standard 12 lead ECG will be done in order to assess outcome variable i.e. AV nodal conduction defects in accordance with operational definition based on presenting ECG of the patient. All the collected data will be recorded on predesigned proforma (provided in annexure A). In-hospital outcomes will be recorded for all the patients. All the survived patients with atrioventricular nodal conduction defects will be followed up to 30-days of the index hospitalization and placement of PPM and mortality will be recorded.

DATA ANALYSIS

Data will be entered and analysis using SPSS version-23 (IBM Corp. Released 2012. IBM SPSS Statistics for Windows, Version 21.0. Armonk, NY: IBM Corp). Shapiro-Wilk test will be applied to check the hypothesis of normality for continuous variables and will be expressed using appropriate descriptive statistics such as Mean± SD, Median (IQR), maximum and minimum. Frequency (%) will

be calculated for categorical variables. Effect modifiers like age groups, gender, BMI, smoking status, hypertension, family history of CAD and diabetes mellitus will be controlled through stratification. Post stratification, appropriate Chi-square test or Fisher exact test will be applied. Taken level of significance $\alpha = 0.05$ and consider two-sided $P \leq 0.05$ as criteria of statistical significance.

Results

A total of 1109 patients were included (**Figure 1**). The mean age was 64.1 ± 14.0 years, and 257 were females (23.2%). Primary percutaneous coronary intervention was performed in 1032 (93.1%) (including 39 with rescue percutaneous coronary intervention) and 77 (6.9%) received thrombolytic therapy alone. HAVB was documented in 95 patients (8.6%).

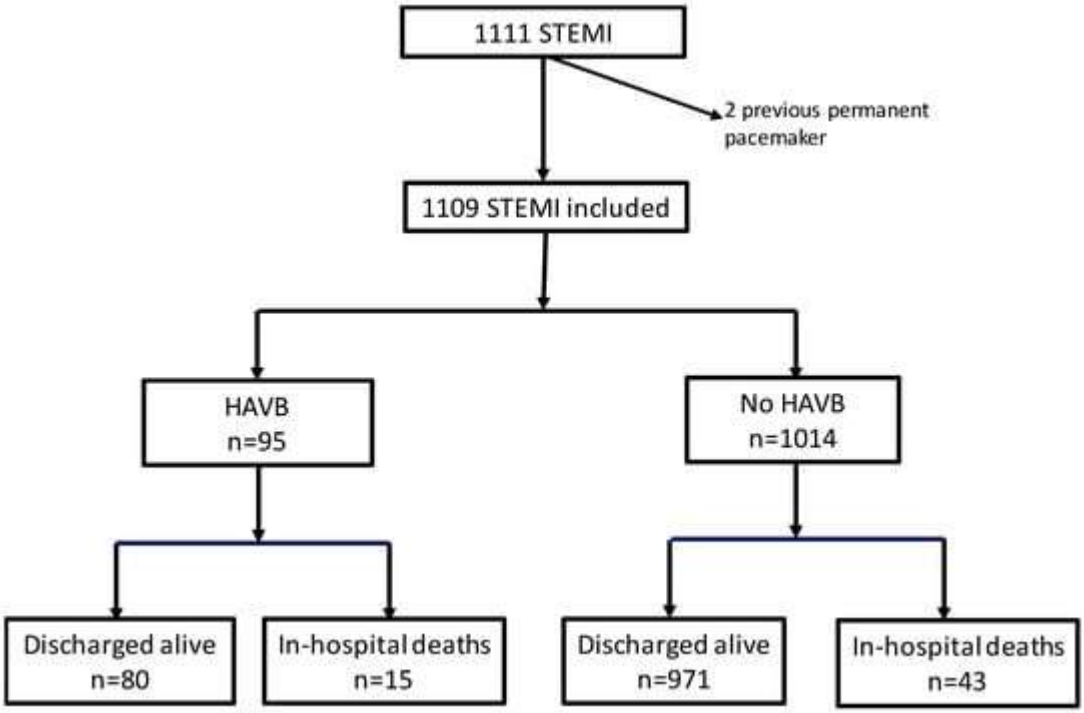

Figure 1. Flow chart of the enrollment of the study participants. STEMI: ST-segment elevation myocardial infarction. HAVB High degree atrioventricular block

Baseline characteristics according to the incidence of HAVB are depicted in **Table 1**. HAVB patients were older, more frequently women, and presented comorbidities (atrial fibrillation, chronic heart failure, active cancer) and dependency more frequently than patients without HAVB. Patients with HAVB had a longer hospital stay and more post-infarction complications: cardiogenic shock, atrial fibrillation, ventricular arrhythmias, pericardial effusion, major bleeding, acute renal failure, infection, and higher mortality. Major bleeding was similar in both groups. There were no significant differences in the incidence of HAVB in patients treated with fibrinolysis compared to those treated with primary percutaneous coronary intervention.

Table 2 presents coronary angiography findings. The right coronary artery was the culprit vessel in 88% of HAVB patients. Compared with patients without HAV, those with HAVB had a higher incidence of right ventricular infarction. The independent predictors of HAVB by multivariable analyses were: right coronary artery culprit lesion, male sex, creatinine value, and age (**Table 3**)

Table 1 **.Baseline Characteristics**

Age, years ± SD	63.4 ± 13.8	70.9 ± 14.3	0.23
Female sex	224 (20.2%)	33 (34.7%)	0.005
Hypertension	543 (53.6%)	58 (61.1%)	0.16
Diabetes mellitus	210 (20.1%)	27 (28.4%)	0.08
Dyslipidemia	466 (45.9%)	46 (48.4%)	0.61
Active smoker	458 (45.2%)	34 (35.8%)	0.08
Body mass index	27.8 ± 4.4	27.1 ± 4.0	0.93
Chronic Obstructive Pulmonary disease	71 (7.0%)	8 (8.4%)	0.61
Previous atrial fibrillation	35 (3.5%)	12 (12.6%)	<0.001
Chronic heart failure	35 (3.5%)	12 (12.6%)	<0.001
Previous cardiac surgery	86 (8.5%)	9 (9.5%)	0.001
Chronic kidney disease	67 (6.6%)	14 (14.7%)	0.004
Peripheral artery disease	46 (4.5%)	2 (2.1%)	0.27
Dependent	25 (2.5%)	8 (8.4%)	0.001
Active cancer	86 (8.5%)	9 (9.5%)	0.006
Infarct site			
Anterior	452 (44.6%)	5 (5.3%)	<0.001
Inferior, lateral or posterior	560 (55.2%)	89 (93.7%)	
Left bundle branch block	2 (0.2%)	1 (1.0%)	
Hours to reperfusion	4.5 ± 3.9	4.6 ± 4.1	0.96
Creatinine (g/dL)	0.97 ± 0.52	1.30 ± 0.84	<0.001
Systolic blood pressure (mmHg)	135.3 ± 29.1	105.8 ± 27.1	0.79
Diastolic blood pressure (mmHg)	77.6 ± 17.6	61.5 ± 15.1	0.10
Left ventricular ejection fraction (%)	46.0 ± 12.1	46.3 ± 11.3	0.26
Ventricular fibrillation	72 (7.1%)	18 (18.9%)	<0.001
Killip > II	185 (18.2%)	45 (47.4%)	<0.001
Hospital stay (days)	6.8 ± 14.1	10.3 ± 18.9	0.006
Complications			
Cardiogenic shock	137 (13.5%)	37 (38.9%)	<0.001
Right ventricle infarction	60 (5.9%)	41 (43.2%)	<0.001
Atrial fibrillation post-STEMI	63 (6.2%)	13 (13.7%)	0.05

Ventricular arrhythmias post-STEMI	35 (3.5%)	10 (10.5%)	0.001
Pericardial effusion	23 (2.3%)	2 (2.1%)	0.93
Major bleeding	38 (3.7%)	7 (7.4%)	0.09
Acute kidney injury	88 (8.7%)	22 (23.2%)	<0.001
Infections	41 (4.0%)	13 (13.7%	<0.001
In-hospital death	43 (4.2%)	15 (15.8%)	<0.001
Treatment			
Fibrinolysis	107 (10.6%)	13 (13.7%)	0.26
Radial access	757 (74.7%)	41 (43.2%)	<0.001
Complete revascularization at discharge	771 (76.0%)	65 (68.4%)	0.09
Temporary pacemaker	--	37 (38.9%)	--
Permanent pacemaker	--	3 (3.2%)	--
Betablocker at discharge	842 (83.0%)	51 (53.7%)	<0.001
ACE inhibitors at discharge	832 (82.1%)	62 (65.3%)	0.04

Table 2. Coronary angiography findings and invasive management.

	No HAVB (n = 1014)	**HAVB (n = 95)**	***p***
Left main or three vessels disease	175 (17.3%)	17 (17.9%)	0.89
Location of culprit lesion			
Left main	6 (0.6%)	0	<0.001
Descending anterior artery	461 (45.5%)	4 (4.2%)	
Circumflex artery	165 (16.3%)	7 (7.7%)	
Right coronary artery	382 (37.8%)	84 (88.4%)	
Initial TIMI flow			
0	705 (69.5%)	74 (77.9%)	0.29
I	80 (7.9%)	7 (7.7%)	
II	75 (7.4%)	3 (3.2%)	
III	154 (15.2%)	11 (11.6%)	
Type of stent			
Bare metal stent	339 (33.4%)	45 (47.4%)	0.01
Drug eluting stent	672 (66.2%)	50 (52.6%)	

	No HAVB (n = 1014)	HAVB (n = 95)	*p*
Final TIMI 3 flow	861 (84.9%)	66 (69.5%)	<0.001

TIMI = thrombolysis in myocardial infarction.

Table 3 Independent predictors of high-degree atrioventricular block.

	Odds Ratio	Confidence Interval 95%	*p*
Right coronary artery culprit lesion	12.41	7.61–20.21	<0.001
Male sex	1.87	1.20–2.93	<0.001
Creatinine (mg/dL)	1.45	1.07–1.97	0.001
Age	1.03	1.01–1.05	0.001

Independent predictors of in-hospital mortality.

	Adjusted OR	*p* Value Adjusted OR	Crude OR	In-Hospital Mortality
Killip II-IV	5.4 (2.2–13.6)	<0.001	27.6 (13.3–57.1)	22.2%
Hemoglobin (g/dL)	0.8 (0.6–0.9)	0.01	0.65 (0.57–0.74)	19.3% [1]
Age (years)	1.04 (1.00–1.07)	0.03	1.07 (1.05–1.10)	12.2% [2]
Left ventricular ejection fraction (%)	0.93 (0.90–0.96)	<0.001	0.89 (0.87–0.92)	14.8% [3]
Significant pericardial effusion	7.9 (2.0–31.7)	0.004	10.5 (4.3–25.7)	32.0%
Ventricular arrhythmias	7.4 (2.9–18.4)	<0.001	26.5 (13.6–51.8)	48.9%
Final TIMI 3 flow	0.28 (0.13–0.61)	0.001	0.14 (0.08–0.25)	2.9%
Chronic kidney disease	3.8 (1.5–9.9)	0.005	6.1 (3.3–11.3)	21%
HAVB	1.48 (0.51–4.36)	0.47	---	15.8%

Discussion

STEMI treated with urgent reperfusion, HAVB occurred in 9% of the patients and was particularly frequent in elderly males with renal failure. The right coronary artery was involved in almost 90% of the cases. Thirty-nine percent of patients with HAVB required temporary pacing. Patients with HAVB had a poor in-hospital and long-term prognosis that was mainly due to the association of HAVB with age and comorbidity, as HAVB was not an independent predictor of mortality.

Early reperfusion, especially primary percutaneous coronary intervention, reduces the size of the infarct and decreases the impact and incidence of HAVB [**1**]. Different studies described the evolution of the incidence of atrioventricular block over time [**9**,**10**,**20**]. Spencer et al. [**11**] reported an incidence of complete atrioventricular block of 6% in the pre-thrombolytic era and 3% in the thrombolytic era. Similarly, Nguyen et al. [**20**] reported a decrease in the incidence from 5% in 1975 to 2% in 2005. Currently, in the middle of primary angioplasty era, the incidence of HAVB in several studies is less than 4% [**1**,**3**,**4**,**5**,**6**,**7**,**8**,**9**], except in the study Gómez-Talavera et al. [**2**], which found 12.6% of all degrees of AV block in STEMI patients, of which 8% had complete atrioventricular block. The rate

of HAVB we found (8.7%) can be considered high and we consider that these results are explained by the prospective character of the DIAMANTE registry and, possibly, the very high percentage of patients with initial TIMI 0 flow compared with other studies [4,7].

Table S1 shows the comparison with previous series. We only included STEMI patients, while other series focused in all types of acute coronary syndromes [1,6] or only included patients with complete atrioventricular block [6,10]. HAVB can be seen in STEMI patients with inferior or anterior location, although it is more common in patients with inferior STEMI. The exact mechanism underlying atrioventricular block in STEMI remains unclear. However, there are several hypotheses depending on the location. In inferior STEMI, the first mechanism involves cardioinhibitory reflexes (Bezold–Jarisch) arising from vagal efferent arm in the ischemic left ventricular inferoposterior myocardial wall [21] and the second involves the effects of ischemia in the atrioventricular node [22], due to the involvement of the artery that irrigates the atrioventricular node distally from the posterolateral branch of the right coronary artery. In patients with left-dominance circulation, the atrioventricular node branch depends on the left circumflex artery. HAVB in anterior STEMI might result from extensive myocardial damage affecting the bundle branch traveling within the interventricular septum [7]. Therefore, HAVB in anterior STEMI is often preceded by bundle branch block, with an unstable escape rhythm [7].

In our study, there was no significant difference in the incidence of HAVB between patients treated with primary percutaneous coronary intervention and thrombolysis. HAVB was associated with poor revascularization results: 69.5% of patients with HAVB at admission had final TIMI III flow in comparison with 84.9% of patients without HAVB treated with percutaneous coronary intervention. This finding is consistent with the results of the Harmonizing Outcomes with Revascularization and Stents in Acute Myocardial Infarction (HORIZONS-AMI) trial [5], in which TIMI 0/I flow was an independent predictor of the development of HAVB, a marker of poor prognosis.

Regarding the need for pacemakers, the 2017 ESC Guidelines for the Treatment of Acute Myocardial Infarction in Patients with ST-segment Elevation [19] recommend temporary pacing in cases of sinus bradycardia with hemodynamic intolerance or HAVB without stable escape rhythm unresponsive to positive-chronotropic drugs. In our study, two fifths of our HAVB patients required a temporary pacemaker insertion, but only 3% needed permanent pacemaker implantation. These results are consistent with previous data [1,6,23]. Patients who required a temporary pacemaker had a longer delay in receiving reperfusion treatment; in addition to the infarct evolution time, the placement of a temporary pacemaker itself might increase the time delay in the catheterization laboratory. Patients with temporary pacemakers died more than those who did not require temporary pacing. The highest mortality in this group could be attributable to a more extensive myocardial ischemia [1]. We did not find significant differences in the incidence of possible complications related to pacemaker implantation (major bleeding, pericardial effusion, infections) regarding patients who did not require temporary pacing. HAVB was transient and reversible in all patients in whom the left anterior descending coronary artery was the culprit vessel. The resolution of HAVB after percutaneous coronary intervention in our patients suggests that the conduction block could be attributable to ischemia rather than necrosis of the intracardiac conduction system [24].

The presence of HAVB in patients with STEMI is considered an unfavorable prognostic marker. In-hospital mortality rates are significantly higher compared to patients without HAVB; in our study, for example, in-hospital mortality in STEMI with HAVB was 16%. Patients with HAVB had a poor in-hospital outcome, including a high risk of cardiogenic shock, left ventricular dysfunction, right ventricular infarction, and ventricular arrhythmias. Although HAVB used to be identified as an independent predictor of mortality [1,6,8], in our study, this was not the case, in line with recently published series [4,7,25]. Probably this is related to the fact that HAVB is not directly responsible for the increase in mortality, being just a marker of a large infarct size and a risky baseline profile. Our findings support this hypothesis, as patients with HAVB more frequently presented biventricular systolic dysfunction and cardiogenic shock.

In the HAVB group, the mortality was higher in anterior compared with inferior STEMI, as in previous publications [3,7]. This is most likely explained by more extensive infarctions when left anterior descending artery is the culprit lesion [3], and, possibly, the differences in the underlying pathophysiology of atrioventricular block in anterior and inferior STEMI as discussed above.
Regarding the impact HAVB on long-term prognosis both in the thrombolytic era and in the angioplasty era, the data are scarce and the results in some studies were discordant [7,11,26]. In DIAMANTE, patients with HAVB had a low long-term survival, but HAVB was not an independent predictor of long-term mortality, which is consistent with the results of Kim et al. [7].
Observational studies are vulnerable to selection bias and unidentified confounding factors, and so, our study had some limitations that must be recognized. As in any registry, the fact that some complications were not reported cannot be ruled out. Furthermore, we only included patients with HAVB at the time of admission, excluding those who could develop HAVB during hospitalization. We did not assess the time of HAVB occurrence or the details related to the implantation and time to explant of temporary pacemakers. Another limitation was the absence of peak values of cardiac biomarkers. We did not carry out a detailed analysis of the technical aspects of the coronary angiography and revascularization procedure that could impact the final vessel flow.

5. Conclusions

In a modern cohort of STEMI treated with urgent reperfusion, HAVB occurred in 9% of the patients and was particularly frequent in elderly males with chronic kidney disease. The right coronary artery was involved in almost 90% of HAVB cases. HAVB was not an independent predictor of in-hospital mortality.

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Proforma

Verbal consent obtained: ☐Yes ☐ No
Case #: _____
Age: _____
CP to ER Time: _____

Co-morbid conditions

☐ DM ☐ HTN ☐ Hyperlipidemia ☐ Obesity ☐ Tobacco
☐FHx IHD ☐ Others: _____

Type Of MI: ☐ Anterior ☐ Inferior ☐ Posterior ☐ Lateral

TIMI score: _____

Reperfusion: ☐ PPCI ☐ Thrombolysis ☐ None

Type of Block: ☐ 1st degree ☐ 2nd degree Type 1 ☐ 2nd degree type 2
☐ 3rd Degree
Number of vessels involved ☐ Normal ☐ SVD ☐ 2VD ☐ 3VD
Culprit coronary artery ☐ LM ☐ LAD ☐ RCA ☐ LCX

☐ Ramus ☐ Diagonal

TIMI (pre procedural) Flow ☐ 0 ☐ I ☐ II ☐ III

TIMI (Post procedural) Flow ☐ 0 ☐ I ☐ II ☐ III

Hospital Stay in days: _____

In hospital Mortality: ☐Yes ☐ No

For patients with AV block

PPM Placed: ☐Yes ☐ No

Day on which reverted to normal sinus rhythm: _____

Day on which required permanent pacemaker: _____

30 day follow up ☐Yes ☐ No