Original Article

Clinical and Angiographic Findings of Complete Atrioventricular Block in Acute Inferior Myocardial Infarction

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Abstract

Introduction: The angiographic findings and prognosis of patients with complete atrioventricular block (AVB) complicating acute inferior myocardial infarction (MI) remain unclear. Materials and Methods: The clinical and angiographic findings of 70 consecutive patients with complete AVB were compared with those of 319 patients with inferior MI without AVB (control group) admitted within the same study period. Results: Patients with complete AVB were older (68 ± 12 vs 63 ± 13 years; *P* = 0.004) and clustered with clinical features indicative of larger infarct size, such as right ventricular infarction, cardiogenic shock, or low left ventricular ejection fraction (LVEF). The onset of the complete AVB was observed within 24 hours in 62 (88.6%), preceded by second-degree AVB in 26 (37.1%) and the escape QRS complex was wide in 8 (11.4%) patients. In patients with complete AVB, a dominant right coronary artery occlusion was found in >95% of cases and in-hospital mortality was increased (27.1% vs 10.7%; *P* = 0.000), especially in those with widen QRS escape rhythm (75.0%). Reperfusion therapy had a positive impact on the natural course of complete AVB. Conclusions: Complete AVB in acute inferior MI was associated with advanced age and larger infarct size. Complete AVB was virtually always caused by dominant right coronary artery occlusion. The in-hospital mortality was significantly higher, but improved by reperfusion therapy. No permanent pacemaker is performed at a mean follow-up of 47 months.

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Key words: Coronary angiography, In-hospital mortality

Introduction

Complete atrioventricular block (AVB) complicates inferior wall myocardial infarction (MI) in 11% to 15% of cases.[1-3] It usually clusters with conditions indicative of poor clinical status, such as right ventricular infarction,[4] cardiogenic shock,[5,6] and atrial fibrillation,[7] probably related to its association with a larger infarct size.[3,8] The pathophysiological mechanism underlying AVB remains unclear, with Bezold-Jarisch reflex,[9] AV node ischaemia,[10] and accumulation of intracellular metabolites being proposed as possible aetiological factors. In-hospital mortality rate is invariably high[3,5,6,11,12] although long-term clinical outcome does not appear affected.[3,5,6] The purpose of this study was to examine the incidence and characteristics of AVB complicating inferior wall MI, its clinical and angiographic correlations, and the effects on long-term electrical and clinical prognosis.

Materials and Methods

Patients

From January 1997 to March 2006, 550 consecutive patients admitted to our Coronary Care Unit with a discharge diagnosis of inferior wall MI were analysed. Inferior wall MI was defined as typical chest pain of longer than 30 minutes, ST-segment elevation of ≥1.0 mm in ≥2 inferior leads (II, III, aVF), and elevation in serum creatinine kinase (CK) or troponin T level of more than twice of upper limit of normal. Patients were classified into 5 subgroups according to their rhythm in the electrocardiogram (ECG): Persistent atrial fibrillation group (n = 22), no AVB group

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(n = 319), first-degree AVB group (n = 114), second-degree AVB group (n = 25), and complete AVB group (n = 70). The clinical characteristics, short- and long-term outcome, and angiographic findings were compared between patients with complete AVB and no AVB (n = 319), the latter group served as a control group. Resolution of the AVB was defined as the reversion of the second-degree or complete AVB to either first-degree or no AVB. The timing of resolution was calculated from the time of admission to the time of resolution. Echocardiography and left ventricular ejection fraction (LVEF) measurement were performed by cardiologists on day 1 or 2 after admission.

ECG Recording

Standard 12-lead and right precordial lead ECGs were recorded at a paper speed of 25 mm/s and a voltage of 10 mm/mV at the time of admission, then every 6 hours for 2 consecutive days, then daily till discharge. Cardiac arrhythmias were documented by telemetry. Widened QRS-complex was defined as QRS duration of ≥120 ms. The ECGs were examined by 2 independent investigators unaware of patients' clinical information. In case of discrepancy, decision was made by a third investigator.

Coronary Angiography

Coronary angiography was performed either as an emergency or elective procedure by experienced interventional cardiologists. Cineangiograms were taken in at least 2 orthogonal views for each vessel. The angiographic findings were evaluated by an experienced observer who was unaware of the clinical and ECG data. The severity of the coronary lesions was expressed as diameter stenosis, as assessed by visual estimation of the replayed angiogram. The infarct-related lesion was identified by either (i) total occlusion or a significant stenosis (≥70% diameter narrowing), or (ii) angiographic evidence of an intraluminal thrombus. The angiographic classification of coronary vessels and segments were based on revised American College of Cardiology (ACC)/American Heart Association (AHA) guidelines.[13]

Statistical Analysis

The measurements were presented as mean ± standard deviation for continuous variables, and absolute number or percentage for categorical variables. Statistical significance of differences between groups was analysed by two-sided Student's *t*-test for continuous variables, and Pearson's χ^2 test for categorical variables. Fisher's Exact test was used whenever an expected cell value was <5 for categorical variables. Hazard ratio (HR) and 95% confidence interval (CI) were calculated by multivariate logistic regression model which was based on entry of clinical variables with a *P* value of ≤0.1 from univariate analysis. *P* <0.05 was considered statistically significant. Analyses were performed using SPSS software, version 15 (SPSS, Chicage, IL).

Results

Complete AVB was found in 70 of 550 patients with inferior wall MI (12.7%). These patients had a significantly higher mean sinus rate (84 ± 17 vs 70 ± 15 beats per minute, *P* <0.0001) than the control group; a sinus rate of below 60 beats per minute was only found in 6 (8.6%) patients. The onset of complete AVB was documented within 24 hours (early onset) in 62 (88.6%) patients. AVB was preceded by second-degree AVB in 26 (37.1%) patients. Mobitz type II second-degree AVB was not observed during the phase of second-degree AVB before the development of complete AVB. The escape QRS complex was narrow in 62 (88.6%); temporary pacemaker was performed in 27 (38.6%) patients. Complete AVB resolved in 34 (54.8%) patients within 24 hours. Seven patients (10.0%) died within 24 hours during the hospital course before resolution of second- or third-degree AVB. Complete AVB persisted in 10 patients (14.3%) for more than 3 days, longer than 7 days in 3 (4.3%) patients and the longest was 12 days (Fig. 1).

Clinical Features

The clinical characteristics were summarised in Tables 1 and 2. Patients with complete AVB were significantly older (68 ± 12 vs 63 ± 13 years; *P* = 0.004). Apart from hyperlipidaemia, there was no difference in the prevalence of other cardiovascular risk factors between the 2 groups.

Complete AVB was associated with a higher incidence of right ventricular infarction (52.9% vs 29.2%; *P* = 0.000) and cardiogenic shock (15.7% vs 4.7%; *P* = 0.001). However, no difference was observed in the incidence of new-onset atrial fibrillation and ventricular tachycardia. Despite similar revascularisation strategy (thrombolytic and primary

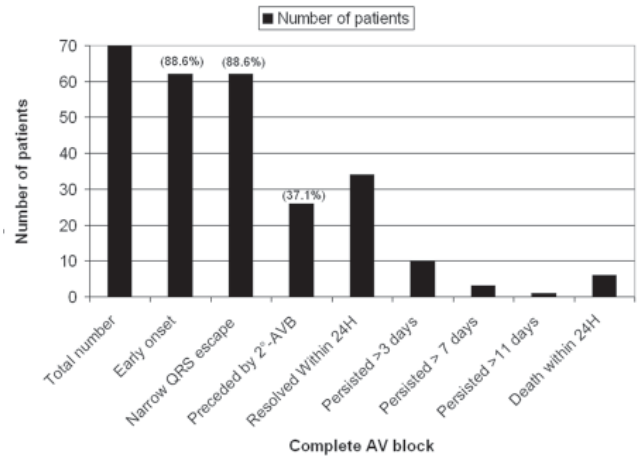

Fig. 1. The onset and severity of complete atrioventricular block (AVB) in patients with inferior wall MI. Percentages of the total number of patients with AVB in parenthesis.

Table 1. Comparison of Demography and Clinical Characteristics between Patients with and without Complete AVB in Inferior Wall MI

	Complete AVB n = 70	No AVB n = 319	*P*
Age (y)	68 ± 12	63 ± 13	0.004*
Male sex (%)	47 (67.1)	242 (75.9)	0.131
Ever-smoker (%)	39 (55.7)	171 (53.6)	0.781
Diabetes mellitus (%)	27 (38.6)	89 (27.9)	0.077
Hypertension (%)	31 (44.3)	130 (40.8)	0.587
Hyperlipidaemia (%)	20 (28.6)	149 (47.2)	0.005*
Symptom onset (hour)	3.4 ± 4.0	3.8 ± 3.8	0.507
Right ventricular infarction (%)	37 (52.9)	93 (29.2)	0.000*
Left ventricular ejection fraction (%)	44.6 ± 12.0	48.7 ± 12.2	0.039*
Peak creatinine kinase level (U/L)	2890 ± 1836	2232 ± 1980	0.0112*

AVB: atrioventricular block

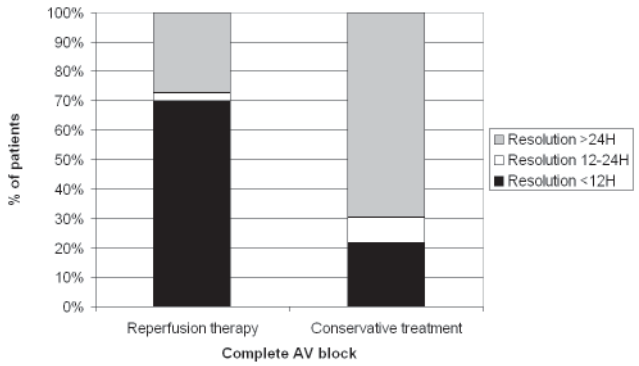

Fig. 2. The effect of reperfusion therapy on the timing of resolution of complete AVB.

angioplasty), complete AVB patients had higher peak CK level (2890 ± 1836 vs 2232 ± 1980 U/L; $P = 0.0112$) and lower LVEF (44.6 ± 12.0 vs 48.7 ± 12.2%; $P = 0.039$); and also less likely to receive beta-blocker (40.0 vs 59.2%; $P = 0.003$) and statin therapy (41.4 vs 54.7%; $P = 0.044$) than the control group (Table 2).

Reperfusion Therapy Versus Conservative Treatment

When patients with complete AVB who received reperfusion therapy (thrombolysis or primary angioplasty) and conservative treatment were compared, the former group was younger, less likely to have diabetes mellitus, more likely to receive beta-blocker and present early, otherwise no difference in the demography and clinical characteristics was observed (Table 3).

The impact of reperfusion therapy on the natural course of complete AVB was shown in Figure 2. Fourteen patients who had either late onset (>24 hours) AVB or death within 24 hours were excluded from this analysis. Reperfusion therapy was significantly associated with early resolution

Table 2. Comparison of Arrhythmias, Treatment, Complications, and Outcomes between Patients with and without Complete AVB Complicating Inferior Wall MI

	Complete AVB n = 70	No AVB n = 319	*P*
Insertion of temporary pacemaker (%)	27 (38.6)	6 (1.9)	0.000*
Atrial fibrillation (%)	12 (17.1)	48 (15.0)	0.660
Ventricular tachycardia (%)	7 (10.0)	19 (6.0)	0.223
Treatment			
Thrombolysis (%)	35 (50.0)	165 (51.7)	0.794
Primary angioplasty (%)	4 (5.7)	36 (11.3)	0.165
Medications			
ACEI (%)	41 (58.6)	219 (69.3)	0.083
Beta-blocker (%)	28 (40.0)	187 (59.2)	0.003*
Statin (%)	29 (41.4)	173 (54.7)	0.044*
Complications			
Re-infarction (%)	6 (8.6)	11 (3.4)	0.097
Cardiac rupture (%)	1 (1.4%)	9 (2.8%)	1.0
Cardiogenic shock (%)	11 (15.7)	15 (4.7)	0.001*
Mortality			
In-hospital (%)	19 (27.1)	34 (10.7)	0.000*
Cumulative 1 year (%)	25 (35.7)	51 (16.0)	0.000*
One-year among survivors (%)	6 (11.8)	17 (6.0)	0.131
Permanent pacemaker (%)	0 (0)	1 (0.3)	1.0

AVB: atrioventricular block

of AVB at different cut-off intervals (Table 4). Four patients who received primary angioplasty showed resolution of AVB within 6 hours after the procedure. The proportion of patients presenting with complete AVB of late onset was lower in the reperfusion therapy group, although it did not reach statistical significance (5.1% vs 19.4%; $P = 0.1265$). The use of reperfusion therapy was associated with a significant reduction in in-hospital mortality (12.8% vs 45.2%; $P = 0.0032$) compared with conservative treatment in patients with complete AVB (Fig. 3).

Angiographic Findings

Coronary angiography was performed in 238 patients (208 patients with no AVB, 30 patients with complete AVB). No difference was observed in the prevalence of left circumflex artery (LCX) dominance in patients with complete AVB and those without AVB. Complete AVB was associated with more dominant right coronary artery (RCA) involvement as the infarct-related artery (96.7% vs 75.4%; $P = 0.0095$), especially with proximal occlusion (63.3% vs 40.4%; $P = 0.018$). The LCX was the infarct-related artery in only 1 patient with complete

Table 3. Comparison of Clinical Characteristics, Medications, and Mortality in Complete AVB Patients who Received Reperfusion Therapy and Conservative Treatment

	Reperfusion therapy n = 39	Conservative treatment n = 31	*P*
Age (y)	65.6 ± 11.0	71.3 ± 11.7	0.041*
Male gender (%)	29 (74.6)	18 (53.1)	0.2358
Ever-smoker (%)	24 (61.5)	15 (48.4)	0.3908
Diabetes mellitus (%)	10 (25.6)	17 (54.8)	0.0247*
Hypertension (%)	13 (33.3)	18 (58.1)	0.0677
Hyperlipidaemia (%)	12 (30.8)	8 (25.8)	0.8491
Presentation <6 hours (%)	36 (92.3)	13 (41.9)	<0.0001*
Presentation >24 hours (%)	0 (0)	11 (35.5)	0.0001*
Left ventricular ejection fraction (%)	47.1 ± 12.3	41.0 ± 10.9	0.085
Peak creatinine kinase level (U/L)	3092 ± 1980	2519 ± 1514	0.264
Medications			
Angiotensin converting enzyme inhibitor (%)	26 (66.7)	15 (48.4)	0.1943
Beta-blocker (%)	21 (53.8)	7 (22.6)	0.0161*
Statins (%)	21 (53.8)	9 (29.0)	0.0657
In-hospital mortality (%)	5 (12.8)	14 (45.2)	0.0032*

Table 4. Comparison of Different Cut-off Intervals of Resolution of AVB between Patients who Received Reperfusion and Conservative Treatment

Resolution	Reperfusion therapy n = 33	Conservative treatment n = 23	*P*
< 8 hours (%)	22 (66.7)	4 (17.4)	0.0004*
<12 hours (%)	23 (69.7)	5 (21.7)	0.0009*
>24 hours (%)	9 (27.3)	16 (69.6)	0.0043*

AVB. This patient had a proximal occlusion of a dominant LCX which supplied 6 obtuse marginal branches as well as the posterior descending artery. Conversely, occlusion of a dominant LCX did not predispose to complete AVB (3.3% vs 9.6%; *P* = NS). Moreover, the prevalence of multi-vessel coronary artery disease (56.7% vs 61.5%; *P* = NS), and concomitant critical left anterior descending artery (LAD) disease, whatever the degree of obstruction, was similar between the 2 groups (Table 5).

Clinical Outcomes

For those 8 (11.4%) patients with a wide QRS complex escape, temporary pacing was performed in 4 patients. Six of them died during the hospital course before the resolution of the AVB. The in-hospital mortality was significantly higher

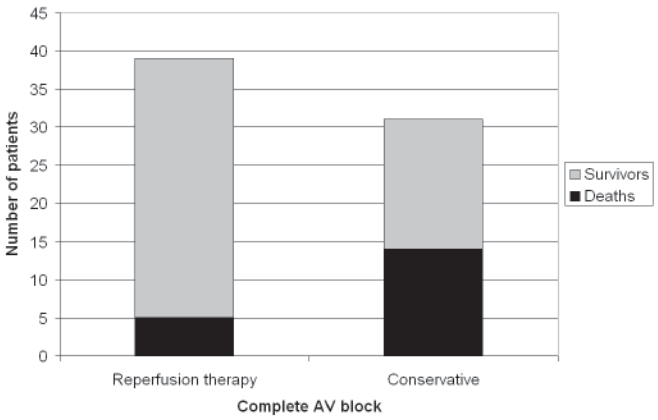

Fig. 3. The effect of reperfusion therapy on the in-hospital mortality of patients complicated by complete AVB.

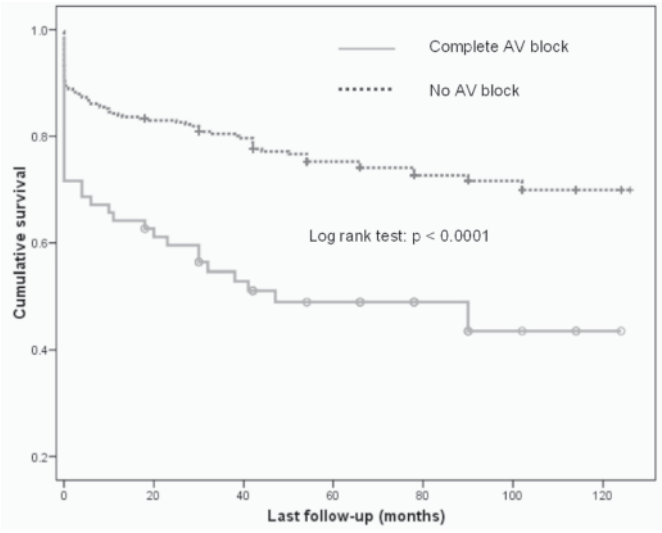

Fig. 4. Kaplan-Meier curve showing the short- and long-term survival of patients who developed complete AVB and those who did not after an inferior wall MI.

(75.0% vs 21.0%; *P* = 0.0039) compared with those with narrow QRS complex escape rhythm. The 2 survivors had complete resolution of the widened QRS before discharge. For those 8 (11.4%) patients who had late onset of compete AVB, the in-hospital mortality (25.0% vs 27.4%; *P* = 1.000) was not different from those with early onset.

Overall, patients who developed complete AVB had higher in-hospital mortality (27.1% vs 10.7%; *P* = 0.000) than controls (Fig. 4). By multivariate analysis, age ≥75 years (HR, 3.47; 95% CI, 0.978-1.105; *P* = 0.015) and complete AVB (HR, 3.94; 95% CI, 0.061-0.785; *P* = 0.013) were found to be independent predictors of in-hospital mortality. However, among the hospital survivors, the 1-year mortality was not increased (11.8% vs 6.0%; *P* = 0.131) (Table 2). No hospital survivor of complete AVB needed a permanent pacemaker at a mean follow-up

Table 5. Comparison of Angiographic Findings of Patient with Complete and no AVB during Inferior Wall MI

	Complete AVB n = 30	No AVB n = 208	*P*
LCX-dominance (%)	1 (3.3)	22 (10.6)	0.3252
Infarct-related artery			
Dominant artery (%)	30 (100)	177 (85.1)	0.0001*
Dominant RCA (%)	29 (96.7)	157 (75.4)	0.0095*
Dominant LCX (%)	1 (3.3)	20 (9.6)	0.4875
Infarct-related lesion			
Proximal RCA (%)	19 (63.3)	84 (40.4)	0.018*
Distal RCA (%)	10 (33.3)	74 (35.6)	0.810
Proximal LCX (%)	1 (3.3)	12 (6.3)	1.0
Distal LCX (%)	0 (0)	37 (17.8)	0.006*
Multi-vessel CAD (%)	17 (56.7)	128 (61.5)	0.096
LAD ≥50% lesion	13 (43.3)	106 (51.0)	0.5579
LAD ≥80% lesion	10 (33.3)	79 (37.9)	0.7718
Proximal LAD ≥50% lesion	10 (33.3)	99 (47.6)	0.2041
Proximal LAD ≥80% lesion	4 (13.3)	40 (19.2)	0.6155

AVB: atrioventricular block; CAD: chronic artery disease; LAD: left anterior descending artery; LCX: left circumflex artery; RCA: right coronary artery

period of 47 months. In the group with no AVB, 1 patient received a pacemaker for sick sinus syndrome, 1 patient had an implantable cardiovertor-defibrillator for recurrent ventricular tachycardia and 1 patient underwent cardiac resynchronisation therapy for congestive heart failure. In the persistent atrial fibrillation group, 1 patient received a permanent pacemaker for symptomatic bradycardia.

Discussion

Our study highlighted several features of complete AVB in acute inferior wall MI. (i) Complete AVB was almost exclusively caused by a dominant RCA (proximal) occlusion. (ii) Wide QRS escape rhythm was associated with very high in-hospital mortality, (iii) No survivor of complete AVB eventually needed a permanent pacemaker.

The AV node receives dual blood supply from the first perforator artery of the proximal LAD and the AV nodal artery from either the distal RCA or the LCX, whichever is dominant. This explains the absence of extensive necrosis in the AV node despite interruption of blood flow to the dominant artery.[14,15] Autopsy studies in patients with AVB have demonstrated an association with large areas of pre-nodal atrial myocardial necrosis, rather than infarction of the AV node proper compatible with larger areas of infarction.[14,15] The proportionate release and accumulation of ischaemic metabolites, for instance, potassium and adenosine is postulated to be the likely cause of the AVB.

The occurrence of complete AVB in acute inferior wall MI is a surrogate marker of a larger infarct size.[3,5,6] In accordance with earlier literature,[7,16] patients in our study who developed complete AVB during the course of acute inferior wall MI were older, and likely to have associated right ventricular infarction, cardiogenic shock, and larger infarct size. They demonstrated a higher in-hospital death but unimpaired 1-year mortality among the hospital survivors. We found that the short-term prognosis was similar in patients with delayed AVB versus those with early onset. This contrasts with the report of Sclarovsky, et al[17] who observed a lower mortality of late AVB compared with early onset. In addition, we documented that patients with second-degree AVB that did not progress to complete AVB remained prognostically similar to those without AVB.[18]

Infarct-related Artery

In contrast to all previous studies, we found that complete AVB occurred almost exclusively in dominant RCA obstruction, especially proximal occlusion which accounted for the larger infarct size and more right ventricular infarction.

Prevalence of Multivessel Disease

Bassan et al[10] described the association of transient complete AVB in inferior wall MI with the presence of concomitant LAD disease of ≥75% diameter narrowing. However, other and larger studies as well as ours have failed to reproduce this result.[19,20]

Reperfusion

The in-hospital mortality rate of complete AVB complicating inferior wall MI has decreased over the last 20 years, probably secondary to the use of thrombolysis and other adjunctive pharmacotherapy. Thrombolytic therapy in acute inferior wall MI has been shown to modestly decrease the incidence of complete AVB in recent studies,[16,21] from 10% to15% in the pre-thrombolytic era to 6% to10%[7] in the thrombolytic era. The small decline is explained by the short therapeutic window (less than 6 hours) in inferior wall MI which limits the number of eligible patient. About one-third of complete AVB occurred after 24 hours (late onset) in early series.[1,3,12] This ratio was reduced to one-fifth in our study. Only about 5% of our patients who received reperfusion therapy developed late onset type of complete AVB compared with 19% in those treated conservatively. The difference was not statistically significant, probably due to the small patient number.

Our study revealed that reperfusion therapy was associated with a significant shortening of the duration of AVB compared with conservative treatment (Fig. 2). This finding is in accordance with a previous study.[11] We found that the use of reperfusion therapy was associated with a

decrease in mortality with an odds ratio of 0.32. Despite these advances, complete AVB still predicts a higher in-hospital death compared with no AVB.

Permanent Pacing

Long-term permanent pacing after complete AVB is uncommon, being reported in less than 1.9% in literature.[22] This relatively low incidence may be related to the ACC/AHA Guidelines that recommend permanent pacing only when complete AVB is persistent.[23] However, the term "persistent" was never clearly defined. Among our hospital survivors, no patient requires permanent pacing in long-term follow-up which is consistent with a cut-off point of 14 to 16 days considered by experts' consensus.[22]

Limitations

This was an observational study which was subjected to a selection and treatment bias. Even multivariate analysis may not completely exclude the effect of the confounding factors. The number of patients with complete AVB who underwent angiography was relatively small. Reperfusion therapy depended on the therapeutic window and could not be randomised according to the presence of complete AVB.

Conclusions

Complete AVB complicated acute inferior wall MI in 12.7% of patients in this study. Complete AVB was mostly of early in onset and short duration (especially with thrombolysis). Complete AVB was more likely to occur in the elderly, and was associated with a larger infarct size. An escape rhythm with a wide QRS complex was associated with a high mortality. Coronary angiography showed that the infarct-related artery was almost exclusively a dominant RCA, especially with proximal occlusion. Reperfusion therapy reduced the proportion of patients with AVB of late onset, shortened the duration of AVB, and improved in-hospital mortality. No patient needed a permanent pacemaker at a mean follow-up of 47 months.

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