

Complete Atrioventricular Block Complicating Acute Anterior Myocardial Infarction can be Reversed with Acute Coronary Angioplasty

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Abstract

Introduction: A retrospective case series of acute anterior myocardial infarction (MI) patients complicated by complete atrioventricular block (AVB) treated with acute percutaneous transluminal coronary angioplasty (PTCA). **Clinical Picture:** Eight patients with anterior MI and complete AVB underwent acute PTCA between 2000 and 2005. Mean onset of complete AVB was 16.6 ± 16.9 hours from chest pain onset. **Treatment:** All patients underwent successful PTCA to the left anterior descending artery. **Outcome:** Complete AVB resolved with PTCA in 88%; mean time of resolution was 89 ± 144 minutes after revascularisation. One patient had permanent pacemaker implanted at Day 12 after developing an 8-second ventricular standstill during hospitalisation but not pacing-dependent on follow-up. The rhythm on discharge for the other surviving patients was normal sinus rhythm. **Conclusion:** This case series suggests that complete AVB complicating anterior MI is reversible with acute PTCA and survivors are not at increased risk of recurrent AVB. Nevertheless, this condition is associated with extensive myocardial damage and high mortality during the acute hospitalisation was not improved with correction of AVB with temporary pacing.

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Introduction

Complete atrioventricular block (AVB) develops in more than 5% of patients with myocardial infarction (MI).¹⁻⁷ These patients have poorer outcomes compared to those without complete AVB.¹⁻⁴ Thrombolysis has been demonstrated to improve the prognosis of such patients, especially those with inferior MI.^{1-3,6-9} Patients with anterior MI, however, remained at high risk of mortality and most required pacemaker implantation.³ With the increasingly widespread availability of acute percutaneous transluminal coronary angioplasty (PTCA) for MI, the prognosis of these high-risk patients would be expected to improve, although limited data have been available to date. We report on a retrospective case series of 8 patients with acute anterior MI complicated by complete AVB who were treated with acute PTCA.

Case Series

The records of all patients with MI treated with acute PTCA at our centre between 2000 and 2005 were examined.

Only patients with anterior MI and complete AVB were included, an example of which is shown in Figure 1. The diagnoses of anterior MI and complete AVB were made using standard clinical definitions. Complete AVB was defined as complete interruption of AV conduction with dissociation of P waves and QRS complexes as well as existence of junctional or ventricular escape rhythm with a rate slower than the atrial rate. Complete AVB was considered present if it occurred during the index hospitalisation. Anterior MI was defined as acute onset of chest pain associated with presence of new or presumably new ST-elevation >0.2 mV in 2 or more contiguous anterior ECG leads (V1-V4) associated with elevated cardiac enzymes within 24 hours of the onset of ischaemic discomfort.

The clinical details for the patients were obtained from our acute MI database and the index hospitalisation records, while follow-up and mortality information were obtained through outpatient and hospitalisation records. The study was approved by the institutional review board. The patients are presented as a case series with their pertinent

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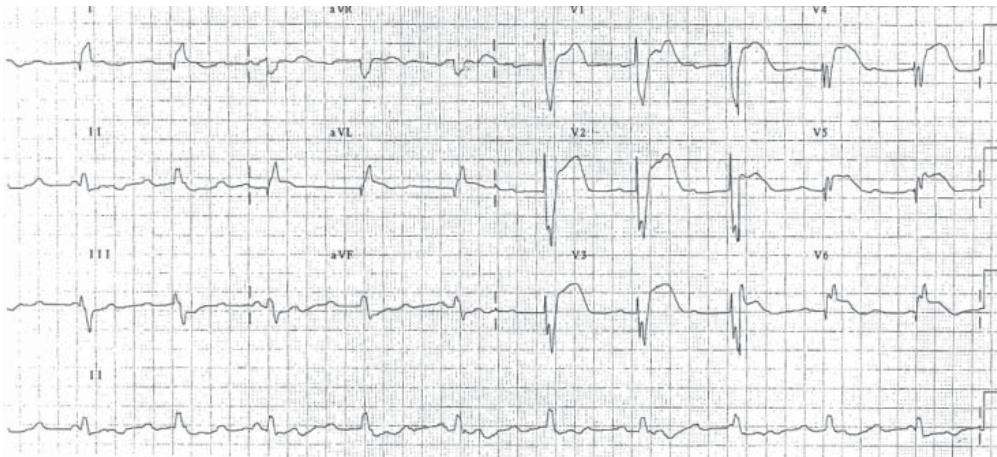


Fig. 1. ECG of anterior AMI complicated by CAVB.

Table 1. Baseline Characteristics of Patients Admitted for Anterior AMI Complicated by CAVB

Patient no.	Age (y)	Sex	Risk factor					Prior IHD	Prior CCF
			Smoking	DM	HTN	HLD	FHX		
1	71	F	N	Y	Y	Y	Y	N	N
2	69	M	Y	N	N	Y	N	N	N
3	62	F	N	N	Y	Y	N	N	N
4	44	M	Y	N	Y	Y	N	Y	N
5	67	M	Y	N	Y	N	N	N	N
6	51	M	N	Y	N	Y	N	N	N
7	65	M	Y	Y	Y	Y	N	N	N
8	69	M	Y	N	N	Y	N	N	N

AMI: acute myocardial infarction; CAVB: complete atrioventricular block; CCF: congestive heart failure; DM: diabetes mellitus; FHX: family history of ischemic heart disease; HLD: hyperlipidaemia; HTN: hypertension; IHD: ischaemic heart disease

details tabulated for reference and comparison. Continuous variables are presented as means ($\pm$ standard deviation) while categorical variables are presented as frequencies and percentages.

Results

Eight patients with anterior AMI and complete AVB who underwent acute PTCA were found. Their baseline characteristics are shown in Table 1. Almost all patients were male, with a mean age of 62.3 ± 9.7 years. All had more than 1 standard risk factor, but only 1 patient had documented pre-existing coronary artery disease prior to the MI.

The clinical characteristics of the patients at presentation are shown in Table 2. The patients were highly unstable, as indicated by their high Killip classification (mean 3.0 ± 1.4) and the presence of cardiogenic shock at presentation or during hospitalisation in 50%. Three patients had complete AVB at presentation and the rest developed it shortly after hospitalisation. The initial electrocardiogram (ECG) of the 5 patients with delayed onset of AVB showed left bundle branch block (LBBB) in 2 patients, right bundle branch block (RBBB)/left anterior fascicular block (LAFB) in 2,

alternating LBBB/RBBB in 1. Mean onset of complete AVB was 16.6 ± 16.9 hours from chest pain onset. Peak cardiac enzyme measurements were: creatine kinase (CK) 4375 ± 2667 U/L, CKMB 459 ± 303 U/L, troponin-T 16.1 ± 5.0 μ g/L. Echocardiography showed a mean left ventricular ejection fraction (LVEF) of $31\% \pm 10\%$.

Mean door-to-balloon time was 239 ± 220 minutes. Angiography showed triple vessel disease in 3 (38%), double vessel disease in 1 (12%) and single vessel disease in 4 (50%). The infarct-related artery was the left anterior descending artery (LAD) in all cases, with the occlusion at the proximal portion in 7 of the 8 patients. All patients underwent successful acute PTCA to LAD.

Seven patients required temporary transvenous pacing due to haemodynamic instability. The complete AVB was transient in 7 (88%), resolving with acute PTCA. Mean time of resolution was 89 ± 144 minutes after revascularisation. The last patient was listed for implantation of a permanent pacemaker (PPM) after developing an 8-second ventricular standstill during hospitalisation. The PPM was inserted on Day 12 of admission. Subsequently, his complete AVB resolved on Day 18, and his discharge rhythm was first degree AVB. During follow-up, the patient was not pacing-

Table 2. Clinical Data of Patients Admitted for Anterior AMI Complicated by CAVB

Patient no.	Killip class on admission	Cardiogenic shock during hospitalisation	Onset of CAVB from chest pain (h)	Bundle branch block on ECG	LVEF (%)	TPW insertion	Time to CAVB resolution from PTCA (h)	Peak CK (U/L)	Peak CKMB (U/L)	Peak troponin T (µg/L)	Death during hospitalisation
1	3	N	13	Left BBB	30	Y	48	1037	94	7.43	Y
2	4	Y	24	Right BBB/LAFB	20	Y	24	7356	843	22.37	Y
3	1	N	48	Right BBB/Left BBB	35	Y	432	6385	626	17.35	N
4	1	N	0	No BBB	22	Y	6	446	51	16.12	N
5	4	Y	24	Left BBB	35	Y	96	3260	317	11.10	N
6	1	N	0	No BBB	50	N	7	4375	459	16.24	N
7	4	Y	0	Left BBB	35	Y	0	4787	443	16.00	Y
8	2	Y	48	Right BBB/LAFB	20	Y	96	7356	843	22.37	Y

AMI: acute myocardial infarction; BBB: bundle branch block; CAVB: complete atrioventricular block; CK: creatinine kinase; LAFB: left anterior fascicular block; LVEF: left ventricular ejection fraction; PTCA: percutaneous transluminal coronary angioplasty; TPW: temporary pacing wire

dependent. The rhythm on discharge for the other surviving patients was normal sinus rhythm.

Four patients died during the index hospitalisation (50% mortality). The cause of death was myocardial pump failure/cardiogenic shock. Death occurred in 1/3 (33%) patients with AVB at presentation and 3/5 (60%) patients who developed AVB later during hospitalisation. During follow-up of 22.4 ± 8.7 months, there was only 1 readmission for congestive cardiac failure. No death, acute coronary syndrome or other events were observed among the other surviving patients.

Discussion

Large community-based studies have demonstrated a reduced incidence of complete AVB complicating acute MI in the thrombolytic era compared to pre-thrombolysis, suggesting the beneficial role of reperfusion on prevention and resolution of complete AVB in acute MI.^{3,4} However, the development of complete AVB is still associated with a higher risk of short- and long-term mortality compared to patients without AVB.^{1-3,6}

The distinction between inferior and anterior MI is important in the setting of complete AVB. AV conduction abnormalities, including complete AVB, commonly complicate inferior MI. This is due to involvement of the blood supply to the AV node, as the AV nodal artery arises from the right coronary artery in most people. The AVB in inferior MI usually resolves promptly with acute revascularisation in most cases, and PPM implantation is usually not required. In contrast, development of complete AVB in anterior MI suggests extensive myocardial damage.⁷ This is reflected in our case series by a high Killip class on admission, high incidence of cardiogenic shock, high peak cardiac enzyme levels, and marked impairment

of LV function. However, information regarding acute revascularisation and outcome in this group of patients is very limited, possibly due to the small numbers of such patients compared to those in the setting of inferior MI. To date, published information regarding PTCA in anterior MI and complete AVB has been confined to case reports.⁹

Acute mortality in our series of patients was high despite temporary transvenous pacing, with cardiogenic shock being the main cause of mortality. The lack of mortality reduction with temporary pacing in patients with anterior MI and complete AVB has also been reported in other studies.^{10,11} This finding suggests that the high mortality is related to the extensive myocardial injury and myocardial pump failure rather than the deleterious effects of complete AVB.

The resolution of complete AVB after acute PTCA in our patients suggests that the conduction block could be attributable to ischaemia rather than necrosis of the intracardiac conduction system.^{9,12,13} Electrophysiological studies in patients who had suffered an anterior MI with complete AVB have demonstrated no permanent AV conduction abnormalities in the majority of them, 4 to 40 months after MI.¹⁴

Prophylactic PPM implantations in such patients have failed to reduce the subsequent risk of sudden cardiac death.¹³ The cause of late sudden cardiac death relates to the occurrence of ventricular tachyarrhythmias rather than the recurrence of AV block. In our case series, AVB resolved completely in the surviving patients within 18 days of MI. Therefore, the decision to implant a PPM should be delayed in such patients who have undergone successful revascularisation to allow time for complete resolution of the AVB. The decision regarding implantation of cardiac defibrillators (ICDs) to reduce sudden cardiac death should

be taken on an individual basis. Survivors do well after discharge with no mortality documented during follow-up, consistent with published data that such patients were not at increased risk of dying after discharge compared to similar patients with anterior AMI patients without.³

Conclusion

Our case series suggests that complete AVB complicating anterior MI is reversible with acute PTCA and survivors are not at increased risk of recurrent AVB. Nevertheless, this condition is associated with extensive myocardial damage and high mortality during the acute hospitalisation which was not significantly improved with correction of AVB with temporary pacing.

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