ST segment elevation myocardial infarction following elective direct current synchronised cardioversion for atrial fibrillation

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ABSTRACT

Atrial fibrillation is the most common arrhythmia in clinical practice. There is no difference in mortality, incidence of stroke or quality of life regardless of whether the rhythm control or rate control strategy is used. However, in certain circumstances, such as when rate control is inadequate, rhythm control is a viable option. We present a 74-year-old Eurasian man with a history of hypertension who presented with new-onset atrial fibrillation and sustained an ST segment elevation myocardial infarction following elective direct current cardioversion.

Keywords: atrial fibrillation, cardioversion, myocardial infarction

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INTRODUCTION

Atrial fibrillation (AF) is a major risk factor for ischaemic stroke, especially in the elderly population.[1] The estimated prevalence of AF and atrial flutter is more than 2.2 million individuals in the United States, with an estimated incidence of more than 75,000 cases per year.[2]

Randomised clinical trials that compare rhythm control versus rate control have failed to show a statistically significant difference in mortality, incidence of stroke or quality of life despite the hypothesised benefits of maintaining sinus rhythm over rate control.[3-7] However, in certain circumstances, such as when rate control is inadequate, rhythm control is a viable option.[8] Rhythm control may be achieved by using either pharmacological agents or elective direct current (DC) cardioversion. Restoring sinus rhythm using DC cardioversion is generally more effective and reliable than pharmacologic cardioversion. Elevation of cardiac markers, namely, troponin, is not a common occurrence following DC cardioversion.[9,10] Therefore, it is deduced that myocardial injury does not commonly occur following DC cardioversion.[9,10] Transient ST elevation does occur following DC cardioversion of AF.[11,12] We report a case of ST elevation with raised troponin T following DC cardioversion for AF that underwent immediate coronary angiogram and primary percutaneous coronary intervention (PCI).

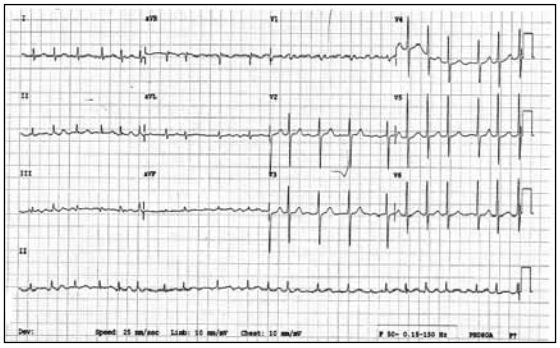

Fig. 1 Electrocardiogram on admission shows atrial fibrillation with rapid ventricular response.

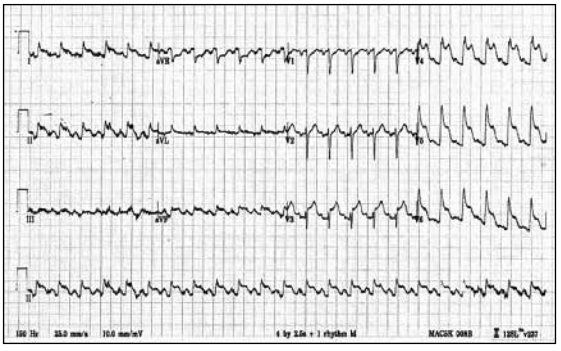

Fig. 2 Electrocardiogram post cardiopulmonary resuscitation shows ST segment elevation in anterolateral leads.

CASE REPORT

A 74-year-old Eurasian man with a history of hypertension and hyperlipidaemia presented at our hospital with palpitation and dyspnoea for the past two

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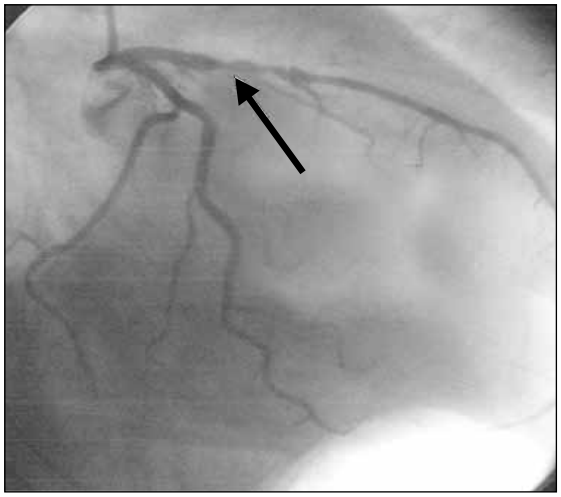

Fig. 3 Diagnostic angiogram shows 90% of stenosis with ruptured plaque in the proximal left anterior descending artery and an area of haziness suggestive of thrombus at the rupture site (arrow).

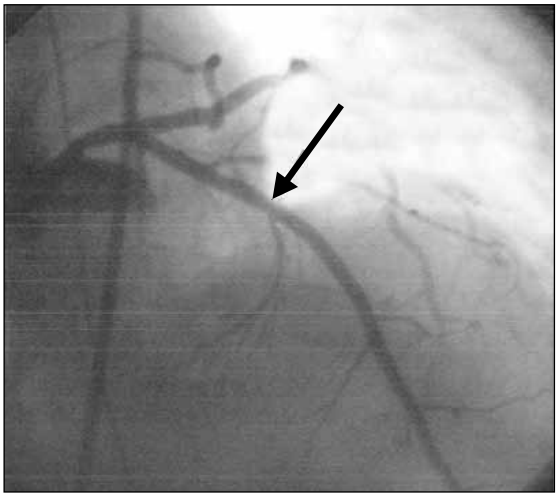

Fig. 4 Post percutaneous coronary angioplasty angiogram after stenting with drug-eluting stent (arrow) (Endeavour resolute 3.0/18 mm).

days. Clinically, his pulse rate was 138 bpm with an irregularly irregular rhythm. The blood pressure was 160/95 mmHg. No other abnormality was detected on systemic examination. The initial electrocardiogram (ECG) revealed AF with a ventricular rate of 127 bpm (Fig. 1). Investigations on hospital admission, which included cardiac markers, renal panels, full blood count, thyroid function and chest radiography, showed normal results. Transthoracic 2D echocardiography revealed no significant valvular pathology and no segmental wall motion abnormality, with a systolic left ventricular ejection fraction of 60%.

A diagnosis of new-onset AF (≤ 7 days) was made. The patient was initially administered an amiodarone infusion, but the medication was stopped after he developed rashes. He was administered subcutaneous low-molecular-weight heparin 1 mg/kg bd, oral beta blocker as well as digoxin from the time of admission. The patient's ventricular rate remained above 100 beats per minute despite the use of the beta blocker and digoxin. He therefore underwent transoesophageal echocardiography (TEE) followed by elective DC cardioversion. Biphasic anterior-posterior cardioversion was performed with directed current at 100 J. Immediately after cardioversion, the patient developed junctional bradycardia interspaced with episodes of sinus arrest. Cardiopulmonary resuscitation was started. During the process, 1.2 mg of atropine and 1 mg of adrenaline were administered. ECG post-CPR showed sinus tachycardia with persistent ST segment elevation in the antero-lateral leads (Fig. 2). Immediate bedside 2D transthoracic echocardiography showed a left ventricular ejection fraction (LVEF) of 20%, with akinesia involving all the mid to apical walls and hypokinesia of the basal walls. A decision was made to perform an immediate primary PCI for ST elevation myocardial infarction.

The patient underwent an angiography, which showed a 90% stenosis with ruptured plaque in the proximal left anterior descending artery (LAD) (Fig. 3). An area of haziness was suggestive of thrombus at the rupture plague site. Primary PCI was performed and the lesion was stented with a drug-eluting stent (Fig. 4). The patient's peak cardiac enzymes post cardioversion were elevated (troponin T 0.672 ng/ml, creatine kinase-MB 6.97 ng/ml) (Table I). He was haemodynamically stable post primary PCI, and his ECG showed sinus rhythm with T inversion over I, II, aVL, aVF, V2 to V6 (Fig. 5). The patient's LVEF improved to 55% on the eighth day of hospitalisation (one day after primary PCI).

DISCUSSION

A diagnosis of new-onset AF (onset duration: approximately 48 hours) was established for this patient who presented with palpitation for two days prior to admission, and his ECG on admission showed AF. The only risk factor for stroke in this patient was hypertension (CHADS2 = 1).

Randomised clinical trials comparing rhythm control versus rate control have failed to show a statistically significant difference in mortality, incidence of stroke or quality of life despite the hypothesised benefits of maintaining sinus rhythm over rate control.[3-7] In view of the fact that this patient

Table I. Serial cardiac enzymes.

Cardiac enzyme	Pre-cardioversion	Post cardioversion		
		1 hour	7 hours	17 hours
CKMB (ng/ml)	2.78	3.29	6.97	3.08
TROP T (ng/ml)	< 0.03	< 0.03	0.672	0.257

CKMB: creatine kinase-MB; TROPT: troponin T

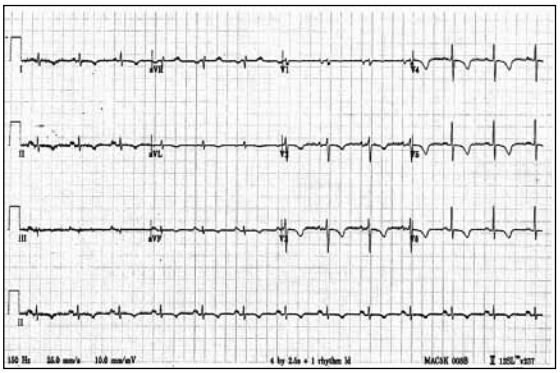

Fig. 5 Electrocardiogram post primary percutaneous coronary intervention shows sinus rhythm with resolution of ST elevation, with T inversion in leads I, II, aVL, aVF, V2 to V6.

presented with new-onset AF (approximately 48 hours), an initial strategy of restoring the sinus rhythm using pharmacologic cardioversion (amiodarone) was chosen with adequate anticoagulant coverage. The patient, however, developed a rash after the initiation of intravenous amiodarone. Amiodarone was stopped and the patient subsequently underwent elective DC cardioversion after TEE showed no intracardiac thrombi. We opted for rhythm control instead of rate control in view of the patient's initial presentation of AF. Rhythm control in the PIAF[5] and HOT CAFE[6] studies showed better improvement in exercise tolerance compared to rate control. Sinus rhythm would also negate the use of long-term anticoagulation. Furthermore, the patient's ventricular rate remained above 100 beats per minute despite the use of digoxin and a beta blocker. Elective DC cardioversion was conducted using a biphasic machine starting at 100 J. Biphasic cardioversion was used instead of uniphasic cardioversion as it requires fever shock, has a lower energy delivery and results in less dermal injury.[13]

Following DC cardioversion, the patient initially went into sinus arrest, necessitating resuscitation. It is possible that the intravenous amiodarone, together with beta blocker and digoxin, may have caused excessive suppression of the sinus pacemaker. ECG post resuscitation showed ST segment elevation at the anterolateral leads as well as raised cardiac markers. A literature review shows that transient ST elevation is not uncommon and comprises about 10% of cases of DC cardioversion of AF,[12,14] but cardiac markers do not increase after DC cardioversion for AF.[9,10] The explanations for this phenomenon include the possibility that a sinus arrest induced the hypotensive episode or plaque rupture as a result of the DC cardioversion or thromboembolic event into the LAD artery. We postulate that the ST elevation myocardial infarction was precipitated by the direct effect of DC cardioversion causing plaque rupture, as the plaque rupture was observed in the proximal LAD artery during angiography. The LVEF recovered to 55% on the eighth day of hospitalisation (one day after primary PCI). The initial deterioration of the global systolic function may have been due in part to stunning. This resolved over time and after the treatment of ischaemia.

In conclusion, ST segment elevation post DC cardioversion of AF usually does not require any treatment as it is transient in nature. We speculate that DC cardioversion causing plaque rupture was the most probable explanation for the ST elevation myocardial infarction. The initial angiographic image showed the presence of an area of plaque rupture together with thrombus formation, which caused the dramatic ST elevation in multiple leads on the surface ECG. Following primary PCI, there was a resolution of the ST elevation followed by T wave inversion in the precordial leads. No pathological Q wave was formed.

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