

Acute Embolic Myocardial Infarction in a Patient with Paroxysmal Atrial Fibrillation Receiving Direct-current Cardioversion

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Coronary embolism with acute myocardial infarction (MI) following direct-current (DC) cardioversion of atrial fibrillation (AF) has rarely been reported. We present the case of a 34-year-old female with severe aortic regurgitation and highly symptomatic paroxysmal AF. Acute embolic MI occurred 4 days after DC cardioversion of AF, although there was no left atrial thrombus detected before this procedure. Insufficient anticoagulation therapy during the post-cardioversion period was the cause, leading to embolic MI. [*J Chin Med Assoc* 2009;72(3):146–149]

Key Words: atrial fibrillation, direct-current cardioversion, embolic myocardial infarction

Introduction

Coronary embolism leading to acute myocardial infarction (MI) in patients with atrial fibrillation (AF) has been frequently reported.¹ However, acute embolic MI following direct-current (DC) cardioversion of AF has rarely been observed.² To the best of our knowledge, this is the second case reported in the literature showing the association of DC cardioversion and acute embolic MI in patients with AF.

Case Report

A 34-year-old female was transferred to our hospital because of an ST elevation inferior MI for 1 day. She denied having any risk factors associated with coronary artery disease. Five years prior to this admission, she had started to suffer from exertional dyspnea and intermittent palpitations. Severe aortic regurgitation (AR) with cardiomegaly and attacks of paroxysmal AF were diagnosed. However, she refused surgical intervention for aortic valve disease. After that, she took

angiotensin-converting enzyme inhibitors, digitalis, and aspirin regularly.

Five days before admission to our hospital, the patient received DC cardioversion in a regional hospital because of highly symptomatic paroxysmal AF, which lasted for more than 2 days. Although there was no cardiac thrombus detected by transesophageal echocardiography (TEE) before DC cardioversion, it was performed after a short period of unfractionated heparin infusion. After successful conversion of AF to sinus rhythm, 2.5 mg of warfarin was prescribed daily. However, the patient did not take the medicine.

Four days after DC cardioversion, sudden onset of chest pain occurred. The chest pain was dull in character and radiated to the jaw. The electrocardiogram (Figure 1A) obtained in the same regional hospital showed sinus rhythm with ST elevation in leads II, III and aVF. Cardiac enzyme data showed elevated values of creatine kinase (CK; 3,180 U/L; reference range, 10–160 U/L) and CK-MB (295 U/L; reference, <16 U/L). Thrombolytic therapy was given immediately with the diagnosis of acute inferior MI. After transfer to our hospital the next day, the chest pain



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Figure 1. (A) Electrocardiogram on the first day of acute inferior myocardial infarction. (B) Follow-up electrocardiogram on the day after thrombolytic therapy.

became mild in severity. Follow-up electrocardiography (Figure 1B) revealed AF and resolution of ST elevation. However, shortness of breath persisted, and the CK level remained high (3,119 U/L). There was no reperfusion arrhythmia observed. Importantly, coronary angiography showed perfectly normal coronary trees (Figure 2), except for a filling defect with TIMI-2 flow at the posterolateral branch of the right coronary artery (see arrow in Figure 2B). These findings strongly supported the diagnosis that coronary embolism was the cause of MI. Due to the coexistence of severe AR

and coronary embolism, the patient received surgical management, including aortic valve replacement and coronary artery bypass grafting.

Discussion

It is well known that after DC cardioversion, thromboembolic complications may occur in AF patients with no demonstrable left atrial thrombus before cardioversion.³ The occurrence of transient “atrial stunning”

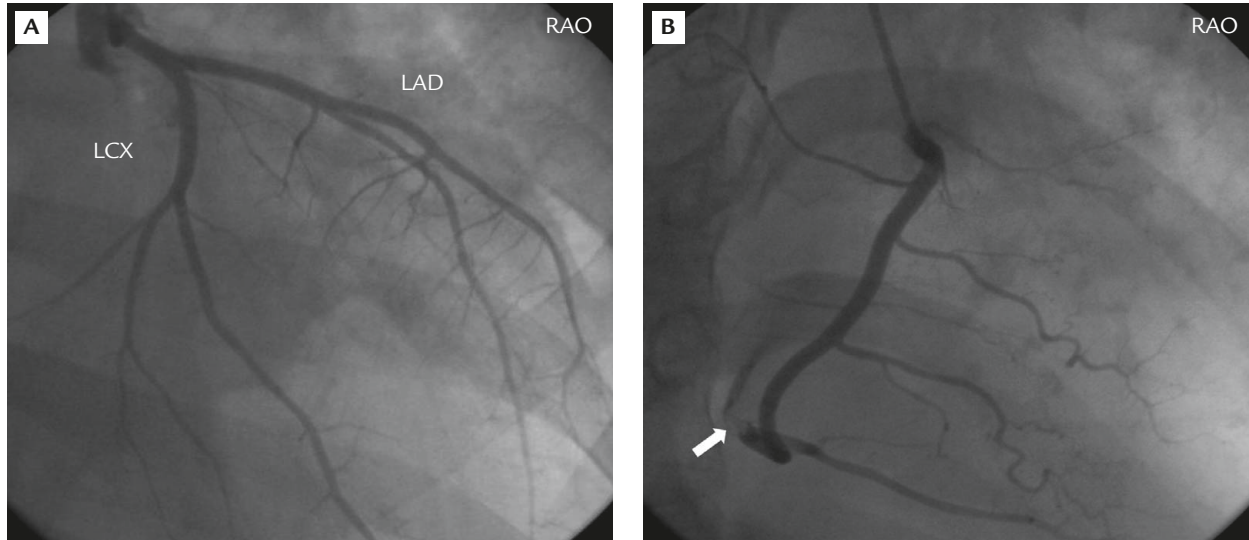


Figure 2. (A) Left coronary angiogram shows normal left anterior descending artery (LAD) and left circumflex artery (LCX). (B) Right coronary angiogram also shows normal right coronary artery but with a filling defect (arrow) at the posterolateral branch. RAO = right anterior oblique view.

during the post-cardioversion period promotes new thrombus formation and emphasizes the importance of adequate anticoagulation therapy at the time of cardioversion.³ For patients with AF of 48 hours in duration or longer (as in this case), anticoagulation therapy (international normalized ratio [INR] of 2.0–3.0) is recommended for at least 3 weeks prior to and 4 weeks after cardioversion.⁴ Alternatively, the use of TEE-guided DC cardioversion with a short period of anticoagulation therapy before and 4-week warfarin therapy after this procedure showed no difference in the rates of embolic events when compared with the aforementioned conventional therapy.⁵ In this case, although no left atrial thrombus was detected by TEE before cardioversion, discontinuation of oral anticoagulation medication during the post-cardioversion period was believed to be the cause, leading to embolic MI.

The standard treatment for coronary embolism with ST elevation MI remains uncertain in current clinical practice. Percutaneous balloon angioplasty may cause distal embolization.⁶ Thrombolysis may not be successful (as in this case) and can cause major bleeding. Kotooka et al⁷ reported that patients with acute MI due to coronary embolism could be successfully treated using a thrombus aspiration device. Thrombus aspiration is now believed to be a feasible and effective strategy for the treatment of embolic MI. However, this method was not tried in this particular case because of surgical planning for both severe AR and coronary embolism.

Although rare, coronary embolism with acute MI may occur in patients with AF receiving DC

cardioversion. This case also emphasizes the importance of adequate anticoagulation therapy during the post-cardioversion period in preventing embolic events, even when no cardiac thrombus has been detected before cardioversion.

Acknowledgments

This study was supported in part by the National Science Council (grants 96-2628-B-010-034 and 96-2628-B-010-035-MY2), and by the Yen Tjing Ling Medical Foundation, Taipei, Taiwan.

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