

## Hemorrhagic pericarditis with cardiac tamponade after percutaneous coronary intervention associated with the use of abciximab

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Glycoprotein (GP) IIb/IIIa inhibitors, such as abciximab, are used as adjunctive therapy for percutaneous coronary intervention (PCI) in high-risk non-ST-elevation myocardial infarction (NSTEMI) and in ST-elevation myocardial infarction (STEMI), although their effects when used for STEMI are less clear. As the use of GP IIb/IIIa inhibitors becomes more widespread, determining the risks associated with them becomes more important. The major risks associated with the use of GP IIb/IIIa inhibitors are the potential for major bleeding and thrombocytopenia. This is the first reported case in Korea of hemorrhagic pericarditis resulting in cardiac tamponade associated with the use of abciximab, a commonly used GP IIb/IIIa inhibitor, following PCI.

**Key Words:** Abciximab; Pericarditis

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### INTRODUCTION

The integrin GP IIb/IIIa receptor is the final common pathway for platelet aggregation. Abciximab is an anti-integrin Fab fragment of a human-mouse chimeric monoclonal antibody with high affinity and a slow rate of dissociation from the GP IIb/IIIa platelet receptor<sup>1)</sup>. Intravenous glycoprotein (GP) IIb/IIIa inhibitors were first used in the setting of PCI in an attempt to reduce abrupt vessel closure and urgent revascularization<sup>1, 2)</sup>. Most cases of bleeding associated with intravenous glycoprotein inhibitors have occurred in patients who underwent PCI, and bleeding primarily occurred at the femoral artery access site<sup>1)</sup>. However, hemorrhagic pericarditis following the use of abciximab is a rare event. This study describes a case of cardiac tamponade resulting from hemorrhagic pericarditis after the use of abciximab following PCI in a patient with STEMI.

### CASE REPORT

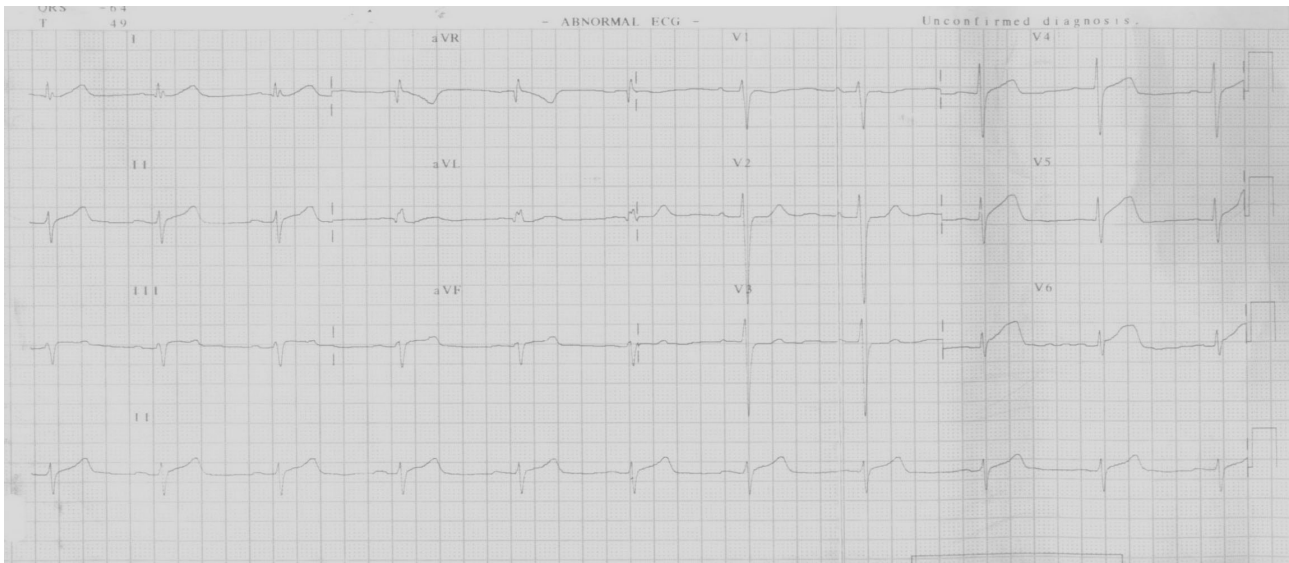
A 66-year-old male was admitted to our hospital due to ongoing and squeezing chest pain accompanied with left shoulder pain that had most recently occurred 3 days prior to admittance. His past medical history included hypertension and a smoking history of 40 pack-years. He had no familial history of coronary artery or cerebrovascular disease, and he was not on any medication at the time of admission. Upon physical examination his blood pressure was 130/90 mmHg and his heart rate was 64 beats per minute, with regular heart and normal S1 and S2 sounds. Upon auscultation, his breathing sound was clear. The initial electrocardiography indicated ST segment elevation up to 1.5 mm in lead V5 and V6 (Figure 1). Initial Echocardiography showed akinesia of the lateral wall from the mid-ventricle to the apex in the left ventricle (LV). Creatine phosphokinase (CPK), lactate dehydrogenase (LDH), CK-MB and Troponin T were 469 IU/L, 447 IU/L, 20.08 ng/mL and 0.169

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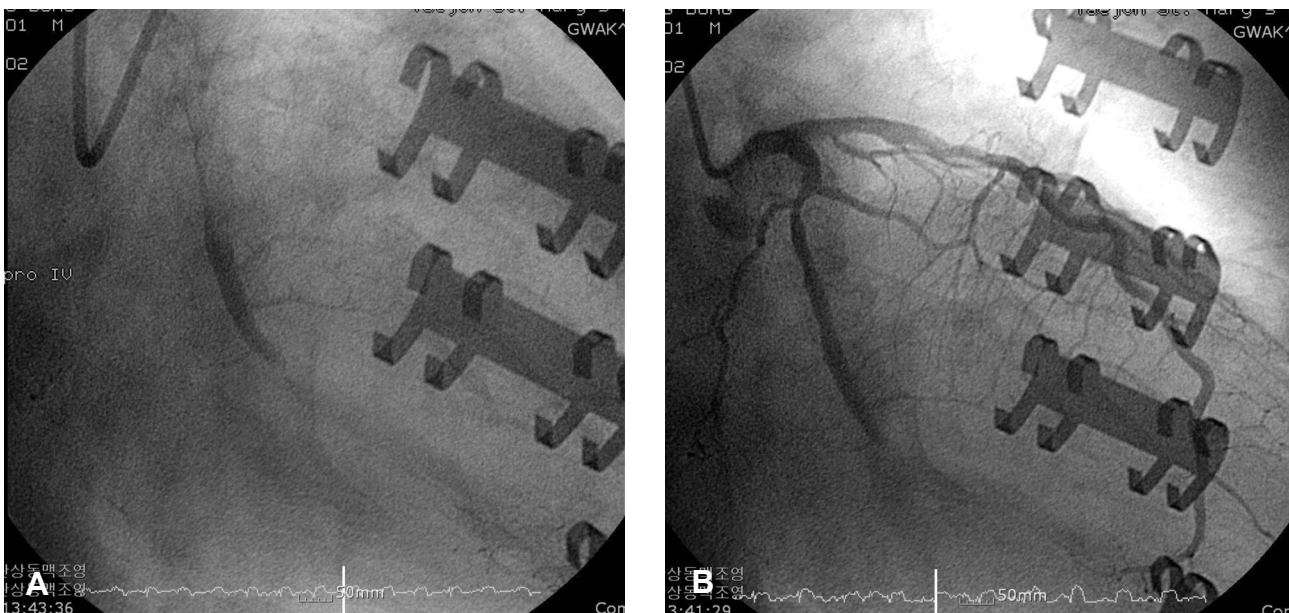
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**Figure 1.** ECG taken during the visit to the emergency room showing ST segment elevation up to 1.5 mm in lead V5 and V6. ECG indicates electrocardiography.



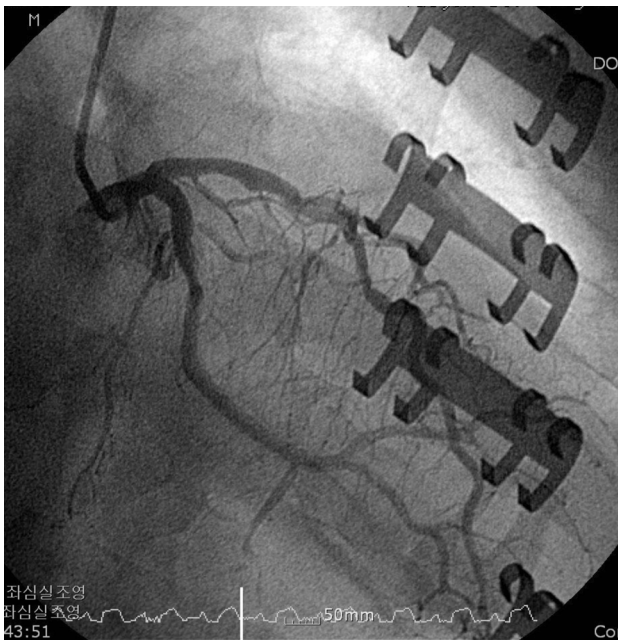
**Figure 2.** Elective PCI performed 5 days after admission showing visible thrombus at the distal part of the left circumflex artery in the RAO caudal view (A). TIMI flow of the distal left circumflex artery was zero and a visible thrombus was present in the mid portion (B). PCI indicates percutaneous coronary intervention; RAO, right anterior oblique; TIMI, Thrombolysis In Myocardial Infarction.

ng/mL, respectively. We applied conventional heparin initially (5000 unit via subcutaneous injection) followed by continuous infusion for 72 hours, subsequently targeting a prothrombin time (PT) INR from 1.5 to 2.0. Additionally, we treated the patient daily with aspirin (200 mg), clopidogrel (75 mg) and cilostazol (200 mg). After 5 days, we successfully performed elective PCI. Abciximab was applied during PCI because a visible thrombus at the left circumflex coronary artery was observed during the

coronary angiography (Figure 2). Abciximab was applied intravenously at 10 mg and was infused at 10  $\mu$ g/min for 12 hours. Vital signs were stable during and immediately following PCI (Blood pressure 120/70 mmHg; heart rate 70 bpm) and the patient did not complain of any symptoms such as chest discomfort or dyspnea. The electrocardiography (ECG) taken immediately following PCI showed no interval change compared with the previous ECG. Eleven hours after coronary intervention



**Figure 3.** ECG taken at the time of shock, which occurred 11 hours after PCI, showing ST segment elevation that progressed up to 3,0 mm in lead V5 and V6, and low voltage in limb leads. ECG indicates electrocardiography; PCI, percutaneous coronary intervention.



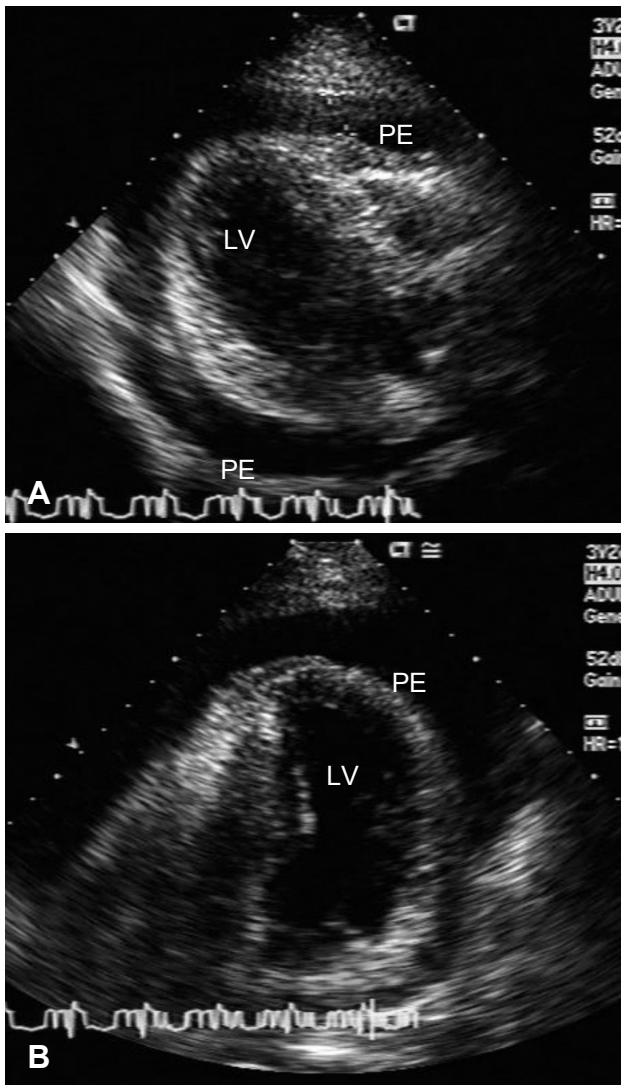
**Figure 4.** Emergent CAG performed for the purpose of ascertaining acute thrombus or coronary artery perforation, showing no evidence of these events. CAG indicates coronary angiography.

the patient complained of chest discomfort and dyspnea. Subsequently, his blood pressure decreased to 60/30 mmHg and ST elevation in lead V5 and V6 increased to 3,0 mm (Figure 3). 2nd Echocardiography after the PCI showed scanty pericardial effusion with no evidence of tamponade. We

conducted an emergent angiography to ascertain whether acute thrombus after PCI or coronary perforation had occurred, however the angiography showed no leakage of dye or thrombus in any coronary arteries (Figure 4). Vital signs had remained stable and the patient had not complained of any more chest discomfort. Three days after the PCI, the patient complained of chest discomfort and dyspnea, and shock occurred again. Echocardiography after the shock showed a large amount of pericardial effusion, which confirmed cardiac tamponade (Figure 5). Emergent pericardiocentesis was performed immediately and the blood pressure soon returned to normal. The total amount of bloody pericardial effusion was approximately 950 cc. Following the initial effusion, neither chest pain nor any sign of shock developed. Echocardiography taken 3 days after pericardiocentesis showed no evidence of pericardial effusion. The patient was discharged 6 days later and underwent follow up observation at an outpatient clinic and has remained well and free of any symptoms for more than 2 years.

## DISCUSSION

Three intravenous GP IIb/IIIa inhibitors (abciximab, eptifibatide, and tirofiban) are approved for treatment in the United States. Abciximab (Reopro, Centocor/Lilly), the first GP IIb/IIIa inhibitor approved for clinical use, is a chimeric antibody directed against the GP IIb/IIIa molecule. Abciximab acts by binding to the GP IIb/IIIa receptor, thereby preventing binding of physiologic ligands



**Figure 5.** Parasternal long axis view (A) and apical 4-chamber view (B) from when shock occurred, which indicated pericardial effusion had occurred and resulted in cardiac tamponade. PE indicates pericardial effusion; LV, left ventricle.

(e.g., fibrinogen, von Willebrand factor) and interfering with platelet aggregation. Abciximab is currently approved for prevention of ischemic complication in patients undergoing PCI and in patients with unstable angina when PCI is planned within 24 hours<sup>2</sup>. Use of GP IIb/IIIa inhibitors for treating NSTEMI patients has been approved and GP IIb/IIIa inhibitors are currently being used to reduce postprocedural ischemic complications in patients undergoing PCI.

Despite the proven effect of GP IIb/IIIa inhibitors on patients with NSTEMI who undergo PCI, their use is associated with major or minor bleeding and thrombocytopenia, especially when abciximab is used<sup>3</sup>. The risk of bleeding can be reduced by the use of low-dose adjunctive heparin, early sheath removal, and

careful postprocedural care and attention to the vascular access site. Major vascular complications after PCI when GP IIb/IIIa inhibitors are used, such as pseudoaneurysm, arteriovenous fistula, and retroperitoneal hematoma reportedly occur in 0.5~9%, 0.2~2.1% and 0.15~0.44% of cases, respectively<sup>4</sup>.

Although abciximab can increase the possibility of bleeding complications after PCI, as occurred in this case, hemorrhagic pericarditis leading to cardiac tamponade is rare. Additionally, cardiac tamponade associated with acute myocardial infarction or PCI is a rare complication. In the case of PCI, pericardial effusion that causes hemodynamic compromise is usually the result of hemorrhagic pericarditis and it is reported that anticoagulation therapy (use of heparin, aspirin, clopidogrel, etc.) clearly increases the risk for hemorrhagic pericarditis. Cardiac tamponade following PCI can occur due to a guide wire perforation of the coronary artery<sup>5</sup>, however, in our case, emergent angiography showed no evidence of coronary artery perforation. Hemorrhagic pericarditis following abciximab treatment associated with PCI was demonstrated by echocardiography, suggesting pericardial effusion and coronary angiography, which in turn confirms that coronary artery perforation did not occur. Therefore, we concluded that cardiac tamponade was caused by hemorrhagic pericarditis, which developed as a complication of abciximab treatment.

In conclusion, we reported a case of hemorrhagic pericarditis presenting with cardiac tamponade following abciximab treatment associated with PCI in a patient who recently underwent STEMI. Further clinical trials are needed to evaluate and prevent probable complications associated with the increased use of GP IIb/IIIa inhibitors. It is particularly important to study the relationship between heparin and GP IIb/IIIa inhibitors.

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