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Alveolar Hemorrhage: A Rare Complication of Eptifibatide (Glycoprotein IIb/IIIa Inhibitor)

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ABSTRACT

A 67-year-old man presented with symptoms of typical chest pain. An electrocardiogram (EKG) showed ST elevation at the inferior leads and ST depression at the anterior leads. His blood pressure was 80/50 mmHg with a pulse rate of 100 beats/min. The glycoprotein IIb/IIIa inhibitor (eptifibatide) was administrated and a primary percutaneous coronary intervention PCI was done. Despite the restoration of TIMI grade 3/3 flow, the patient's blood pressure dropped and pulmonary edema occurred. On the second day following PCI, the hemoglobin level started to drop. While working up the case, the patient suddenly complained of dyspnea and had a bloody foamy discharge from his mouth. Due to severe hypoxemia orotracheal intubation was carried out. A chest x-ray revealed an alveolar hemorrhage. Treatment with high dose hydrocortisone was started and the patient went on mechanical ventilation for seven days. Following extubation, the patient was put under close observation for five days and then discharged from hospital without any complications. During a 3 month follow-up, he has remained symptom free.

Implication for health policy/practice/research/medical education:
Primary PCI in ST elevation myocardial infarction.

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1. Introduction

Acute coronary syndromes and myocardial infarction are often caused by platelet activation. Eptifibatide (Integrilin) is an antiplatelet agent that binds to the receptor of glycoprotein (GP) IIb/IIIa and leads to platelet aggregation inhibition (1). Percutaneous coronary intervention (PCI) is often preferred as the primary therapy (2). In previously published meta-analyses, the beneficial effects of glycoprotein have supported (GP) IIb/IIIa in PCI (2). Several types of bleeding have been reported in association with eptifibatide, however, alveolar hemorrhage remains a rare side effect of eptifibatide (3). In this case report, we want to present this infrequent complication of eptifibatide.

2. Case Report

A 67-year-old man presented to the emergency department with the chief complaint of new onset chest pain, from 10 hours previously. His risk-factors for coronary artery disease were; hypertension, hyperlipidemia and smoking. An electrocardiogram (EKG) showed ST elevation at the inferior leads and ST depression at the anterior leads. His blood pressure was 80/50 mmHg, with a pulse rate of 100 beats/min, pulmonary rales were also present. The patient was referred to the catheterization laboratory for a primary PCI. A coronary angiography revealed total cut off at the mid-part of the right coronary artery (RCA) and long significant (90%) stenosis at

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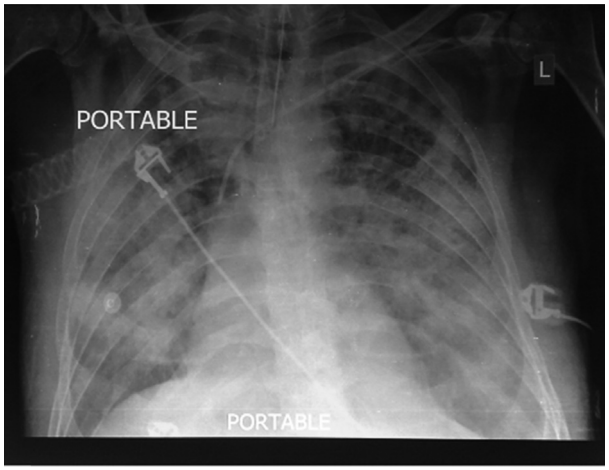

Figure 1. Chest Radiography Shows Alveolar Hemorrhage.

the proximal part of the left anterior descending artery (LAD). Glycoprotein IIb/IIIa inhibitors (eptifibatide) was administrated and a primary PCI was done on the RCA. Because of cardiogenic shock, an angioplasty was also performed on the LAD. Despite the restoration of TIMI grade 3/3 flow, the patient's blood pressure fell and pulmonary edema appeared. An inotropic agent was started, an IABP (intra-aortic balloon pump) was embedded, and orotracheal intubation completed. Mechanical ventilation was started and after 6 hours of intensive treatment, his blood pressure increased to 100/60 mmHg with a pulse rate of 80 beats/minute. On the second day post PCI, the patient was extubated, but his hemoglobin levels started to fall and exhaustive follow-up was done in order to find the source of the hemoglobin loss. Two hours after extubation, he suddenly complained of dyspnea and there was a bloody, foamy discharge from his mouth with severe hypoxemia, which led to re-intubation. After consultation with the pulmonologist, a chest X-ray was indicative of an alveolar hemorrhage. The echocardiography showed 2 + mitral regurgitation with a left ventricular ejection fraction (LVEF) of 45%. Treatment with high dose hydrocortisone was started and the patient was put under mechanical ventilation for seven days. After extubation, he was put under close observation for a further five days. He was discharged from hospital without any complications. During the 3 months follow up period, he was free of symptoms.

3.Discussion

A serious risk of glycoprotein IIb/IIIa inhibitors is bleeding, and it has been described in various cases (3). Alveolar hemorrhage leading to death has also been reported in association with glycoprotein IIb/IIIa inhibitors (4). Alveolar hemorrhage has been reported with all kinds of glycoprotein IIb/IIIa inhibitors. The incidence rate of eptifibatide associated alveolar hemorrhage has been estimated to be 0.5% (5).

The effects of interstitial-alveolar edema and the raising of pulmonary capillary wedge pressure, which happens among patients with; pulmonary edema, cardiogenic shock and other related situations, should be investigated as a possible reasons for an alveolar hemorrhage. According to our findings, we believe that GP IIb/IIIa inhibitors (Integrilin) should be considered conservatively for these patients. Clinicians should make this decision on an individual case basis, although more studies need to be done in the future.

It seems that cardiogenic shock is a risk factor for alveolar hemorrhage. Therefore, investigation of alveolar hemorrhage associated factors in the presence of pre Integrilin-infusion cardiogenic shock could be helpful. Finally, the role of increased pulmonary capillary wedge pressure (PCWP) as a risk factor for alveolar hemorrhage remains ambiguous and so further animal based studies are suggested.

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The authors declare that they have no Conflicts of Interest.

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Refrences

1. Yang EH, Perez E, Zhiroff KA, Burstein S. Left main stent thrombosis complicated by eptifibatide-induced acute thrombocytopenia. *Tex Heart Inst J*. 2011;**38**(2):174-8.
2. De Luca G, Suryapranata H, Stone GW, Antoniucci D, Tcheng JE, Neumann FJ, et al. Abciximab as adjunctive therapy to reperfusion in acute ST-segment elevation myocardial infarction: a meta-analysis of randomized trials. *JAMA*. 2005;**293**(14):1759-65.
3. Conley M, Patino G, Romick B, Almaleh M, Campbell C, Hawkins K, et al. Abciximab-induced alveolar hemorrhage after percutaneous coronary intervention. *Can J Cardiol*. 2008;**24**(2):149-51.
4. Brown DL. Deaths associated with platelet glycoprotein IIb/IIIa inhibitor treatment. *Heart*. 2003;**89**(5):535-7.
5. Ali A, Hashem M, Rosman HS, Kazmouz G, Gardin JM, Schrieber TL. Use of platelet glycoprotein IIb/IIIa inhibitors and spontaneous pulmonary hemorrhage. *J Invasive Cardiol*. 2003;**15**(4):186-8.