



Rapidly Progressive Dyspnea after Coronary Arterial Bypass Grafting with Bilateral Internal Mammary Artery: Report of a Case

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ABSTRACT

Diffuse alveolar haemorrhage (DAH) is defined as bleeding into alveolar spaces, and it is caused by the disruption of the alveolar-capillary basement membrane. While various diseases are associated with the development of DAH, we describe a rare case of alveolar haemorrhage after a full arterial revascularisation procedure via bilateral internal mammary arteries.

Key Words: Pulmonary alveolar spaces; haemorrhage; adult respiratory distress syndrome; myocardial revascularisation; low-molecular-weight-heparin; anticoagulant

Bilateral İnternal Mammarian Arter ile Koroner Arteryal Baypas Greftleme Sonrası Hızlı İlerleyen Dispne: Olgu Sunumu

ÖZET

Difüz alveolar hemoraji (DAH) alveolar-kapiller bazal membranın bozulmasına bağlı olarak gelişen alveolar boşluğa kanama olarak tanımlanır. Çeşitli hastalıklar DAH'ın gelişimi ile ilişkiliyken, bilateral internal mammarian arter ile tam arteryal revaskülarizasyon sonrası gelişen nadir bir alveolar hemoraji olgusu tanımladık.

Anahtar Kelimeler: Pulmoner alveol; kanama; erişkin solunum sıkıntısı sendromu; miyokardiyal revaskülarizasyon; düşük molekül ağırlıklı heparin; antikoagülan

INTRODUCTION

Diffuse alveolar haemorrhage (DAH), characterised by the extravasation of red blood cells into pulmonary alveolar spaces, is typically attributable to disseminated injury of pulmonary capillaries⁽¹⁾. DAH may occur in the context of various systemic disorders or may be present in isolation⁽²⁾. The presence of DAH after coronary artery bypass surgery is a rare complication.

We report the case history of a 44-year-old male who developed diffuse pulmonary haemorrhage after coronary bypass grafting with bilateral internal mammary artery (IMA), which to the best of our knowledge is the first case report in the English literature.

CASE REPORT

A 44-year-old male with a history of hypertension was referred to our hospital for three-vessel coronary artery disease. His preoperative physical examination, blood test, pulmonary function test, and chest X-Ray findings were all normal; therefore, a routine coronary artery bypass surgery was planned. There was no anti-coagulant or anti-aggregant drug history. Left internal mammary artery (LIMA), right internal mammary artery (RIMA) and left radial artery were harvested for the target coronary arteries (left anterior descending artery, first diagonal branch and posterior descending artery, respectively), and the revascularisation procedure was uneventfully accomplished with a routine on-pump technique. The patient was haemodynamically stable in the first hour of the postoperative period and was extubated in the fourth postoperative hour with a normal arterial blood gas sample (PO₂= 96, PCO₂= 37, Lac= 1.13, pH= 7.43 and SPO₂= 98). Low-molecular-weight heparin (LMWH, 0.6 mL enoxaparin SC) was administered in the sixth postoperative hour as per routine. In total, 850 cc bleeding from the bilateral chest tube drains was observed in the ninth postoperative hour, with accompanying drop of haemoglobin levels from 12.7 mg/dL to 7.3 mg/dL. His chest radiography showed new, severe, bilateral, diffuse perihilar infiltrates, with a simultaneous drop in oxygen saturation

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Submitted: 23.10.2014

Accepted: 17.11.2014

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Available on-line at
www.kosuyoluheartjournal.com

from 93% to 82%. His prothrombin time, international normalised ratio (INR) and partial thromboplastin time were 10.4, 1.3 and 25s, respectively. We immediately stopped anti-coagulation as soon as we suspected bleeding. No other possible aetiologies of alveolar haemorrhage, including any evidence of vasculitis and usage of drugs with pulmonary side effects, were found. Thoracic computed tomography (CT) scan confirmed the presence of diffuse bilateral ground glass opacities (Figure 1A,B,C).

On the first postoperative day, the arterial partial pressure oxygen/fraction of inspired oxygen ratio was 155 mmHg. Nevertheless, the patient suddenly developed acute respiratory failure, while the chest X-Ray revealed new infiltrates in the right lung fields.

His prothrombin time, INR and activated partial thromboplastin time were 13.8, 1.12 and 45.5 s, respectively, but his platelet count decreased to $63 \times 10^9/L$ from $240 \times 10^9/L$. However, his haemoglobin level remained at 7.4 g/dL. Infiltrates in his chest radiogram progressed bilaterally, and DAH was suspected for which bronchoscopic examination was not thought to be indicated because of the diffuse pattern of lesions

and because the patient still tolerated the extubation status and there was no risk of re-intubation. A methylprednisolone regimen was started immediately with a dose of 100 mg/day for 5 days, followed by gradual tapering. On the fourth postoperative day, the platelet count recovered to $149 \times 10^9/L$ and oxygen saturation began to increase to the level of 94%. Follow-up thoracic CT scans revealed gradual disappearance of the airspace-filling opacities with a significant improvement in the clinical status (Figure 1D,E,F). He was discharged from the hospital on aspirin on the 14th postoperative day. Clinical follow-up at 3 months was unremarkable.

DISCUSSION

DAH is a syndrome characterised by bleeding into alveolar spaces without inflammation or destruction of the alveolar structures, there by differing from the pulmonary capillaritis type by the lack of systemic vasculitis and connective tissue disorders within a spectrum ranging from asymptomatic radiographic abnormalities to severe life-threatening respiratory failure^(1,2).

In the present case, uneventful arterial revascularisation was accomplished and a prophylactic dose regimen of LMWH

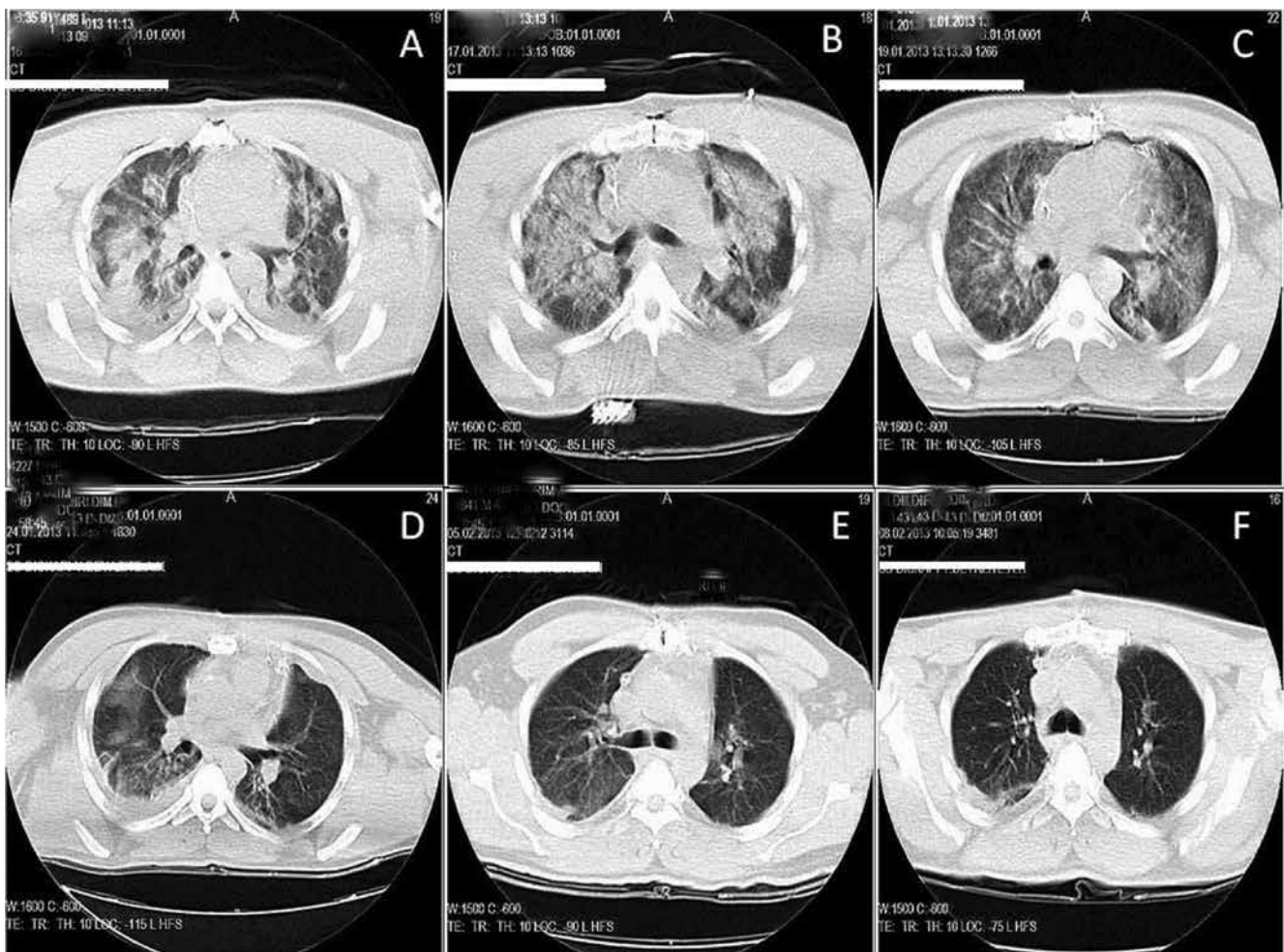


Figure 1. Radiographic findings of diffuse alveolar haemorrhage after full arterial revascularisation (A) On the first postoperative day, computed tomography revealed bilateral patchy infiltrate. (B,C) Clinical findings began to resolve after corticosteroid therapy; however, computed tomography images showed bilateral ground glass infiltrates on postoperative days 3 and 5. (D,E) Radiological findings dissolved after the sixth day of therapy. (F) Follow-up thoracic CT scan a month after the diagnosis.

was administered for routine postoperative management. Considering that the patient was bleeding and demonstrated decreasing haemoglobin levels due to anti-coagulation therapy, LMWH therapy was halted. Although bleeding into the drainage tubes disappeared, the low haematocrit levels and worsening symptoms persisted. The precise diagnosis and management of the disease are crucial for patients with DAH. While bronchoscopy with bronchoalveolar lavage (BAL) are frequently required to accurately diagnose and optimally manage patients with DAH, the haemodynamically unstable condition and the risk of re-intubation kept away us from bronchoscopic examination.

Chest radiograph after the administration of LMWH began to represent diffuse alveolar opacities. Furthermore, thoracic CT scans confirmed the presence of diffuse bilateral ground glass opacities^(3,4). On cessation of alveolar bleeding after discontinuing anti-coagulation therapy, most associated radiological abnormalities resolve within a few days to weeks. However, in our case, the radiological findings and symptoms did not resolve. Acute management of DAH involves supportive care, including ventilatory support ranging from oxygen supplementation to mechanical ventilation, and prompt initiation of high-dose methylprednisolone therapy is critical⁽⁵⁾. Because of the high mortality associated with DAH, glucocorticoids are frequently initiated while diagnostic test results are pending. Our patient rapidly responded to the corticosteroid therapy, and recovery was quickly observed from the initial hours of administration.

Meanwhile, the full arterial revascularisation technique, including bilateral IMA harvesting, may be considered as the cause of bleeding, and disrupting the thoracic wall bilaterally may trigger the cascade of DAH. In a former study

by Matsumoto, it was demonstrated that IMA harvesting may increase postoperative bleeding⁽⁵⁾. The pressure of the haematoma in the pleural space frequently facilitates atelectasis following cardiac revascularisation procedures. In our case, the cessation of anti-coagulation therapy rapidly reduced the amount of bleeding and haematoma in the pleural space; however, the clinical status of the patient only improved with corticosteroid therapy.

To the best of our knowledge, this is the first case report of DAH occurring after full arterial revascularisation. We consider that cases of alveolar haemorrhage are rare, but it may cause life-threatening respiratory failure. Accurate diagnosis and rapid management of the disease may prevent further complications.

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