Case Report

Warfarin-induced diffuse alveolar hemorrhage: Case report and a review of the literature

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ABSTRACT

Diffuse alveolar hemorrhage (DAH) refers to the intra-alveolar accumulation of blood originating from the pulmonary microvasculature. This life-threatening condition is a medical emergency as patients often develop acute respiratory failure requiring invasive mechanical ventilation. This mandates for an early diagnosis with prompt and aggressive management strategies. A host of clinical disorders are known to cause DAH; however, warfarin-induced alveolar hemorrhage is a distinct clinical rarity. A search of the literature reveals few reports documenting this entity. A 27-year-old male presented with complaints of recent-onset hemoptysis and dyspnea. One month back, he was diagnosed with lower-limb deep-venous thrombosis and pulmonary embolism. He had been taking oral anticoagulants irregularly since then without monitoring of prothrombin time. Chest radiograph, done on presentation, revealed bilateral upper-lobe infiltrates, whereas computed tomography of the chest was suggestive of bilateral upper-lobe ground-glass opacities. Serial bronchoscopic alveolar lavage yielded samples which became progressively bloodier, whereas cytological evaluation of the sample revealed numerous alveolar macrophages with intracytoplasmic hemosiderin. A diagnosis of DAH due to warfarin was made, and the patient was administered Vitamin K followed by infusion of fresh frozen plasma. There was a marked clinical recovery, and the patient has been asymptomatic since then.

KEY WORDS: Bronchoscopic alveolar lavage, computed tomography of the chest, diffuse alveolar hemorrhage, fiber-optic bronchoscopy, warfarin

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INTRODUCTION

Diffuse alveolar hemorrhage (DAH) is a rare clinical entity which refers to bleeding into the acinar portion of the lung from the pulmonary microvasculature.[1] This occurs as a result of the disruption of the alveolar–capillary basement membrane, leading to the intra-alveolar collection of red blood cells. The prognosis of patients with DAH is often dismal, and the in-hospital mortality ranges widely from 20% to 100%.[2] A strong clinical suspicion is often required for making an early diagnosis to prevent disease progression and reduce the mortality rate.[1]

A wide spectrum of clinical conditions are known to cause DAH. This includes immunological mediated conditions such as systemic vasculitis (granulomatosis with polyangitis [Wegner's granulomatosis], microscopic polyangitis), rheumatoid arthritis, systemic lupus erythematosus (SLE), antiglomerular basement membrane antibody disease (Goodpasture syndrome), antiphospholipid

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syndrome, and Bechet's disease. The nonimmunological mediated conditions include drugs, toxins, bleeding disorders, and malignancy.[1,3]

Anticoagulants are increasingly being used in a variety of clinical conditions, and bleeding forms an important adverse effect of anticoagulant use. DAH due to warfarin is an uncommon clinical entity and has been infrequently documented in the literature.[4] The paucity of the literature on the subject prompted this report of a young male who presented with hemoptysis and was subsequently diagnosed with DAH as a result of warfarin therapy.

CASE REPORT

A 27-year-old, HIV-negative, never-smoker male presented to the emergency department with complaints of hemoptysis along with shortness of breath for the past 3 days. His clinical course was characterized by multiple bouts of coughing out bright red blood around 200 ml/day for 3 days. Hemoptysis was not accompanied with malena, bleeding diathesis, and gum or nasal bleed. In addition, he also had symptoms of dyspnea at rest, which were not associated with wheezing. One month prior to presentation, he had complained of exertional dyspnea accompanied with pain and swelling in the right leg for which he underwent a computed tomography scan of the chest. At that point, a diagnosis of pulmonary embolism along with right lower-limb deep-venous thrombosis was made. He was started on oral anticoagulants (tablet warfarin: 5 mg/day) after an adequate overlap with low-molecular-weight heparin. However, the patient had been taking the drug erratically without adequate monitoring for a month prior to presentation. General physical examination revealed a young male in respiratory distress with a saturation of 90% while breathing ambient room air. There was an asymmetry in the girth of the lower limbs, with right limb girth being larger. Chest auscultation revealed vesicular breath sounds of equal intensity with bibasilar fine-end inspiratory crepitation.

Investigations

On admission, the hemoglobin levels were 14.2 gm% which subsequently fell to 10.6 gm% over the course of 3 days. Liver and renal function tests as well as the urine routine analysis were within the normal limits. The prothrombin time (PT) values done on admission were deranged with an international normalized ratio (INR) of 7.2. A review of the past records revealed PT values of 22 and an INR of 2.05 at the time of discharge. Chest radiograph done on presentation revealed bilateral upper-zone infiltrates (right > left). Contrast-enhanced computed tomography of the chest revealed bilateral eccentric filling defects in the pulmonary artery suggestive of pulmonary thromboembolism [Figure 1a]. In addition, the high-resolution computed tomography of the chest showed bilateral upper-lobe ground-glass opacities with a mosaic attenuation pattern [Figure 1b]. Compression ultrasound (CUS) along with Doppler of the right lower limb revealed partial recanalized thrombus in the right common femoral vein with streaky irregular patchy flow. In view of the presence of diffuse alveolar infiltrates, the patient's consent was taken for a fiber-optic bronchoscopy which revealed a normal tracheobronchial tree. Serial bronchoscopic alveolar lavage (BAL) was done from the right upper lobe, with each of the five sequential 20-mL aliquots of BAL being bloodier than the previous [Figure 2]. Cytological evaluation of the BAL sample revealed numerous brown intracytoplasmic hemosiderin pigment-laden macrophages on hematoxylin and eosin stain [Figure 3a] and bluish cytoplasmic granules on Pearls stain [Figure 3b]. Stains and cultures of the BAL were negative for *Mycobacterium tuberculosis* and other aerobic organisms as well as pathogenic

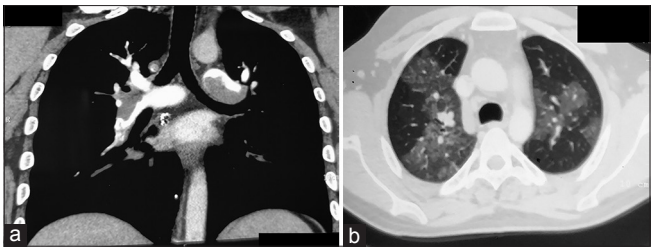

Figure 1: (a) Contrast-enhanced computed tomography of the chest (mediastinal window: coronal section) showing eccentric filling defects in the right and left main pulmonary arteries suggestive of pulmonary thromboembolism. (b) High-resolution computed tomography of the chest (lung window: axial section) showing bilateral upper-lobe ground-glass opacities with a mosaic attenuation pattern

Figure 2: Serial broncho-alveolar lavage showing progressive hemorrhagic nature of the broncho-alveolar lavage fluid

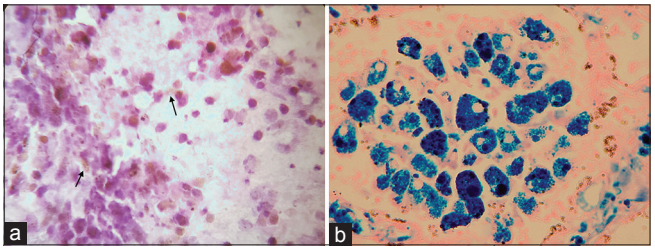

Figure 3: (a) High-power view (H and E, ×40) of the patient's broncho-alveolar lavage fluid cytology specimen showing numerous brown intracytoplasmic hemosiderin pigment-laden macrophages (black arrows). (b) High-power view (Pearls, ×40) of the patient's broncho-alveolar lavage fluid cytology specimen showing hemosiderin-laden macrophages. Hemosiderin is demonstrated as blue cytoplasmic granules

fungi. Subsequent workup included a negative test for antineutrophil cytoplasmic antibodies, antinuclear antibody, antiglomerular basement membrane, and antiphospholipid antibodies. A diagnosis of anticoagulant (warfarin)-induced DAH was made based on (a) clinical presentation; (b) radiology; and confirmed on (c) BAL as well as BAL fluid cytology.

Management

Warfarin was immediately stopped, and the patient was placed on high-flow oxygen and 10-mg intravenous Vitamin K injection was administered followed by transfusion of four units of fresh frozen plasma. In a couple of days, the patient had improvement in his symptoms, and he was weaned off the supplemental oxygen. A repeat chest radiograph done on day 4 showed clearance of the radiological opacities. As the patient was unable to afford newer oral anticoagulants, he was discharged on a lower dose of warfarin with an advice of strict INR monitoring in further follow-ups.

DISCUSSION

The clinical presentation in patients with DAH includes cough, hemoptysis, dyspnea, and chest pain.[1] Hemoptysis may be absent in one-third of patients with DAH.[5] Respiratory failure is usually the most severe form of presentation requiring ventilatory support and entailing high mortality rate. The diagnosis of DAH is mainly clinical in an appropriate setting and is usually supported by radiological and bronchoscopic findings.[3] Clinical history forms an important diagnostic armamentarium in patients with DAH, as apart from establishing a diagnosis, it often elicits the etiology. Radiological features in DAH are usually nonspecific and include patch/diffuse ground-glass opacities, "crazy-paving" pattern, or frank consolidation. These findings are often seen in acute pulmonary edema, acute respiratory distress syndrome (ARDS), congestive heart failure, and infections.[1] BAL often confirms the diagnosis based on (1) progressively bloodier return on sequential BAL samples and (2) hemosiderin-laden macrophages on BAL cytology.[1,3]

Based on the histological pattern, the causes of DAH may be grouped as: (1) those associated with pulmonary capillaritis; (2) associated with bland pulmonary hemorrhage; and (3) associated with diffuse alveolar damage.[1] Pulmonary capillaritis refers to the neutrophilic infiltration of the pulmonary capillaries leading to loss of structural integrity and bleeding into the alveolar spaces. This is usually seen in DAH associated with systemic vasculitis, Goodpasture syndrome, rheumatoid arthritis, some drugs (diphenylhydantoin, propylthiouracil, and all–trans-retinoic acid), and the rare entity of idiopathic pulmonary capillaritis. In patients with bland pulmonary hemorrhage, there occurs bleeding into the alveolar spaces without affecting the interstitial compartment. This is often seen in patients with mitral stenosis, bleeding disorders, and anticoagulant therapy. Diffuse alveolar damage is usually seen in patients with SLE, cytotoxic drug intake, and ARDS.[6]

Anticoagulant-induced DAH is a rare clinical entity and has been infrequently documented in the medical literature. Warfarin is a Vitamin K antagonist and a very potent and widely used anticoagulant. Systemic bleeding is often an important complication with warfarin therapy.[7] The risks of bleeding further increase because factors such as diet, concurrent medications, poor compliance, and alcohol consumption lead to fluctuating INR levels.[8] A search of the literature using PubMed, Cochrane, IndMed, Google, and other databases with the keywords "warfarin," "acenocoumarol," "Vitamin K antagonist," and "diffuse alveolar haemorrhage" revealed 18 studies[4,9-25] documenting 22 patients, which has been depicted in Table 1. Brown *et al*.[9] in 1965 for the first time reported DAH caused due to warfarin in a 64-year-old male who presented with hemoptysis and hematuria. He had accidentally ingested 35 mg of warfarin leading to DAH, a diagnosis of which was made clinicoradiologically. Nearly, a decade later, Finley *et al*.[10] reported three patients with warfarin-induced "occult pulmonary hemorrhage," the diagnosis of which was established on BAL. Following this, isolated reports have come up highlighting this rare clinical entity.[11-25] A recent retrospective study from Japan[24] described the clinical features and outcome of DAH caused by antithrombotic therapy. In this study, 14/39 patients had DAH due to warfarin, with eight on monotherapy, whereas the remaining six were on other antithrombotic agents too. Of the eight patients on warfarin monotherapy, only four had DAH due to the offending drug, whereas the remaining four had other causes too (vasculitis and heart failure).

Management of warfarin-induced DAH is usually supportive, with withdrawal of the offending drug being the most important treatment strategy followed by administration of Vitamin K and fresh frozen plasma which serves as an effective antidote.[1,3,4] Since DAH is often a life-threatening condition, a prompt diagnosis and early treatment is often required to decrease the mortality. Intravenous corticosteroids and ventilatory support are often required in cases with severe hypoxemia and respiratory failure. In a recent report,[20] extracorporeal membrane oxygenation has been used as a bridge to therapy in severe cases of DAH caused due to warfarin therapy with promising results.

Our case highlights two important features related to the diagnosis and management of drug-induced DAH. First, a high index of clinical suspicion is warranted for an early and prompt diagnosis as was seen in our case. BAL is often considered to be the gold standard and was used in our case to confirm the diagnosis. Second, DAH has a high mortality rate and hence, therapy should be instituted as early as possible, with reversal of anticoagulation being the cornerstone of the management strategy.

Table 1: Tabulated review of anticoagulant-induced diffuse alveolar hemorrhage

Author/years	Patients/age	Symptoms	Drug/dosage	Chest radiograph	Computed tomography chest	Fiber-optic bronchoscopy	Management/ outcome
Brown *et al*., 1965[9]	1/64 years	Hemoptysis, hematuria ×24 h	Warfarin – 35 mg	Bilateral alveolar pulmonary infiltrate	Not done	Not done	Oral/IV Vitamin K survived
Finley *et al*., 1975[10]	2 Patient 1: 69 years	Progressive malaise, chest pain, and dyspnea	Warfarin – dose NA	Bilateral lower-lobe infiltrates	Not done	Done (bloody lavage fluid)	Warfarin withdrawal/ survived
	Patient 2: 42 years	Acute onset of dyspnea	Warfarin (post-MVR)	Diffuse infiltrates	Not done	Done (bloody lavage fluid)	Warfarin stopped/ survived
Granthil *et al*., 1981 (French) (abstract in English)[11]	2 (individual detail not available)	Malena, epistaxis, dyspnea, acute respiratory distress syndrome	Oral anticoagulant (details not available)	Diffuse micronodular military pattern	Not done	Done (large number of alveolar siderophages)	No details available
Barnett *et al*., 1992[12]	1/27-year-old female	Fever. dyspnea, cough, and hemoptysis	Brodifacoum (DCon) – "superwarfarin" ingestion	Diffuse alveolar infiltrates	Not done	Not done	FFP, 60 mg of IV Vitamin K , mechanical ventilation/survived
Erdogan *et al*., 2004[13]	1/75-year-old male	Fever, severe dyspnea, dizziness, and hemoptysis	Warfarin: 5 mg/ day for AF	Bilateral alveolar infiltration	Bilateral alveolar infiltration compatible with bilateral alveolar hemorrhage	Hemosiderin-filled histiocytes	100% oxygen, 2 units of FFP, and 30-mg Vitamin K IV/survived
Thomas *et al*., 2008[14]	1/60-year-old male	Fever, cough, severe breathlessness, and hemoptysis	Warfarin 5 mg daily for MVR	Bilateral fluffy opacities	Diffuse pulmonary alveolar hemorrhage	Not done	FFP, mechanical ventilation/died within 2 h
Klenner *et al*., 2008[15]	1/84-year-old female	Hemoptysis	Phenprocoumon for AVR and AF	Diffuse alveolar infiltrates	-	Fresh blood in all segments corresponding to DAH	Blood transfusion and application of 100 IU Vitamin K-dependent blood clot factors
Mogili *et al*., 2009[16]	1/29-year-old female	Dyspnea, hypoxia	Coumadin for DVT: Dose not mentioned	Bilateral interstitial markings of the lungs, with a diffuse nodular pattern	Diffuse ill-defined interstitial opacities with nodular appearance	Bloody aliquots consistent with DAH	IV steroids/ survived
Yardan *et al*., 2012 (Turkish) (abstract in English)[17]	1/58-year-old female	Dyspnea, cough, and hemoptysis×2 days	Warfarin for AF	Done (details NA)	Done (details NA)	Done (details NA)	Done (details NA)
Waness *et al*., 2009[4]	1/62-year-old female	Hemoptysis and dyspnea×24 h	Warfarin for AF: 5 mg PO every other day (4 days/week), 2.5 mg PO every other day (3 days/week)	Alveolar opacities in both lungs, especially in perihilar and pericardiac zones	Bilateral patchy airspace disease	Done (lavage became progressively more hemorrhagic)	FFP, Vitamin K Mechanical ventilation X 14 days/survived
Itoh *et al*., 2011[18]	1/72-year-old male	Hemoptysis and dyspnea	Warfarin 2 mg/ day	Bilateral alveolar infiltration	Bilateral ground-glass opacities and multiple low-attenuation areas	BAL: Hemorrhagic Hemosiderin-filled macrophages	10-mg Vitamin K IV/survived
Baba *et al*., 2012[19]	1/85-year-old male	Dyspnea and blood-stained frothy sputum	Warfarin for AF: Alternate day dosing – 5 and 6 mg once daily	Widespread airspace shadowing in both lungs with some relative sparing of the apices and costophrenic angles	Widespread ground-glass opacity with superimposed interlobular thickening and "crazy paving" appearance	Not done	IV Vitamin K/ survived

Contd...

Table 1: Contd...

Author/years	Patients/age	Symptoms	Drug/dosage	Chest radiograph	Computed tomography chest	Fiber-optic bronchoscopy	Management/ outcome
Lee and Kim, 2013[20]	1/56-year-old male	Aggravated dyspnea×24 h	Warfarin: 2.0–2.5 mg×5 months for pulmonary vein thrombosis	Bilateral pulmonary infiltrations	Extensive ground-glass attenuations and "crazy-paving appearance"	Done (lavage became progressively more hemorrhagic)	10-mg Vitamin K, FFPs, mechanical ventilation, ECMO/ survived
Uysal *et al*., 2014[21]	1/49-year-old male	Dyspnea and hemoptysis×2 h	Warfarin: 0.5 mg/day for MVR	Diffuse alveolar infiltrates	Bilateral alveolar infiltration	Not done	Supplemental oxygen, IV Vitamin K and FFP/survived
Kaya *et al*., 2015[22]	1/59-year-old male	Hemoptysis and shortness of breath×24 h	Warfarin 2.5 mg/day for MVR	Alveolar opacities in both lungs	Bilateral patchy airspace disease	Done (no details)	FFP and RBC transfusions/ survived
Heffler *et al*., 2016[23]	1/64-year-old male	Hemoptysis, cough, and dyspnea	Warfarin for AF: 5 mg/day	Chronic signs of emphysematous Chronic obstructive pulmonary disease	Diffuse bilateral signs of alveolar hemorrhage with hydroaerial levels within emphysematous cysts	Not done	10 mg IV Fitomenadione, FFP, 1 g IV pulse dose of methylprednisolone, and supplemental oxygen/survived
Otoshi *et al*., 2016[24]	4	Clinical details NA	Clinical details NA	Clinical details NA	Clinical details NA	Clinical details NA	Clinical details NA
D'Amore *et al*., 2017[25]	1/62-year-old male	Shortness of breath and fever×2 days, massive hemoptysis	Warfarin for AF: Dose not mentioned	Left lower-lobe infiltrate	Not done	BAL: Hemosiderin-laden macrophages	Two units of fresh frozen plasma and IV Vitamin K, intubation/died after 7 days

AVR: Aortic valve replacement, AF: Atrial fibrillation, DAH: Diffuse alveolar hemorrhage, DVT: Deep-venous thrombosis, FFPs: Fresh frozen plasma, IV: Intravenous, MVR: Mitral valve replacement, NA: Not available, BAL: Bronchoscopic alveolar lavage, ECMO: Extracorporeal membrane oxygenation

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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