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A RARE CASE OF DIFFUSE ALVEOLAR HEMORRHAGE FOLLOWING ORAL AMPHETAMINE INTAKE

N.F. Peters, R. Gosselin, K.L. Verstraete[1]

Diffuse alveolar hemorrhage (DAH) is a clinical syndrome, which refers to injury to the capillaries, arterioles and venules, leading to red blood cell accumulation in the distal air spaces. It is defined by the clinical triad of hemoptysis, anemia and progressive hypoxemia. Chest radiographs reveal non-specific patchy or diffuse bilateral pulmonary consolidation. Multiple conditions are associated with DAH, of which Wegener's granulomatosis is the most frequent, and underlying disease determines the prognosis and treatment. This case describes DAH as a result of oral amphetamine abuse in a young patient of which the diagnosis was established by laboratory, clinical and radiologic findings. The patient experienced a rapid recovery without significant sequelae.

Key-word: Drugs, abuse.

Case report

A 20-year-old male patient was admitted to the emergency department because of aggressive behavior and respiratory depression with a short episode of apnea, after the administration of 20 mg of diazepam, during a Techno party. He reported the oral use of amphetamine (ecstasy). After admission there was an episode of hemoptysis and hematemesis. Arterial oxygen saturation dropped to 70%, without clinical repercussion.

Clinical findings

Lung auscultation showed discrete crepitation of the lower right lung.

Laboratory findings were a mild leukocytosis and C-reactive protein increase.

Chest radiograph (AP-view) (Fig. 1) shows a normal cardiac size and bilateral, predominantly perihilar, areas of increased opacity.

High-resolution CT examination of the lungs (Fig. 2) revealed multifocal ground glass attenuation with areas of consolidation and discrete peribronchovascular thickening, here depicted on an axial image (A). A Reformatted image in the coronal plane (B) shows the diffuse extent in both lungs. There was a normal heart size and no presence of pleural effusion.

Based on the clinical and CT findings the diagnosis of pulmonary hemorrhage was made. The presence of hemoptysis, hypoxemia, recent amphetamine use and rapid radiographic recovery supported the diagnosis.

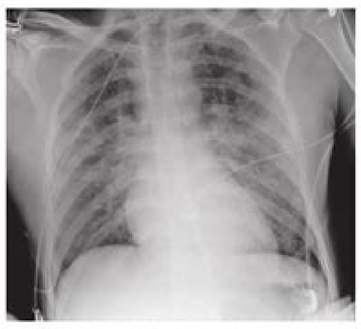


Fig. 1. — Chest radiograph (AP-view) showing normal cardiac size and bilateral, predominantly perihilar, areas of increased opacity.

The patient was admitted and treated with oxygen administration and antibiotic therapy for the risk of aspiration pneumonia. The lung opacities cleared rapidly over the course of 48 h. Laboratory results tested positive on the use of 3,4-methylenedioxymethamphetamine (MDMA, ecstasy). No other drug substances were found.

Discussion

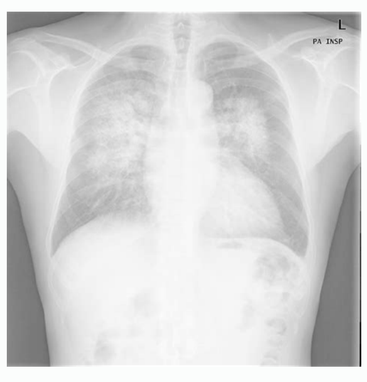
Diffuse alveolar hemorrhage (DAH) is a clinicopathological syndrome describing the accumulation of intra-alveolar red blood cells originating from the alveolar capillaries. The classical clinical triad includes hemoptysis, anemia and hypoxemia, which can be severe (1).

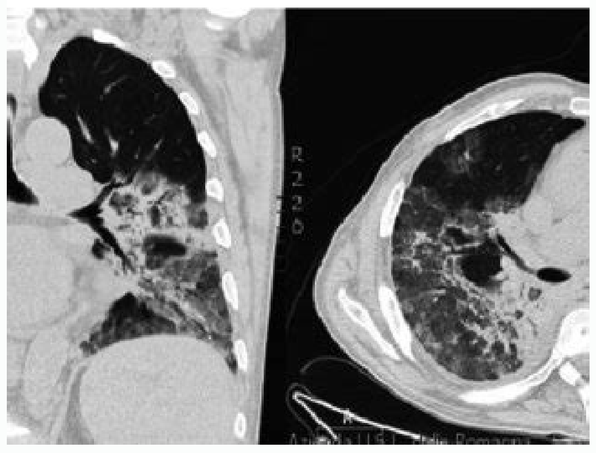
On plain radiography DAH manifests as multifocal bilateral areas of increased opacity with a normal heart size. High-resolution CT shows multifocal ground-glass attenuation, which occasionally is centrilobular in distribution and associated with interlobular septal thickening.

The differential diagnosis of these radiologic findings must be broad and include acute lung injury, diffuse infection, and noninfectious inflammatory conditions (e.g. pulmonary hemorrhage and acute hypersensitivity pneumonitis).

Inhaled and intravenously abused drugs, including opiates and cocaine

From: 1. Department of Radiology, University Hospital Ghent, Ghent, Belgium.
Address for correspondence: Dr N. Peters, M.D., Dienst Radiologie, UZ Gent, De Pintelaan 185, 9000 Gent, Belgium. E-mail : niels.peters@ugent.be





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[Google Scholar]Page 2Causes of diffuse alveolar hemorrhage View PDFVolume 25, 2018, Pages 201-203 rights and content Many disorders can cause alveolar hemorrhage; they include Coagulation disorders caused by diseases or anticoagulant drugs Drug reactions (eg, propylthiouracil, diphenylhydantoin, amiodarone, methotrexate, nitrofurantoin, bleomycin, montelukast, infliximab) Isolated pauci-immune pulmonary capillaritis Toxic exposures (eg, trimellitic anhydride, isocyanates, crack cocaine, certain pesticides) Home » Decision Support in Medicine » Pulmonary Medicine Diffuse alveolar hemorrhage (DAH) is a life-threatening disorder characterized clinically by the presence of hemoptysis, falling hematocrit, diffuse pulmonary infiltrates and hypoxemic respiratory failure. DAH should be considered a medical emergency due to the morbidity and mortality associated with failure to treat the disorder promptly. The treatment of DAH ranges from supportive care and withdrawal of offending drugs, to high-dose steroids, immunosuppressant drugs and plasmapheresis. Classification: DAH can be broadly categorized into 4 main groups Immune (vasculitis, connective tissue disease) Congestive Heart Failure (systolic/diastolic, valvular) Miscellaneous (infection, trauma, clotting disorder, cancer, drugs) Idiopathic DAH is associated with a number of clinical entities. Pulmonary renal syndromes, connective tissue disorders and drugs make up the majority of the cases of DAH. Hemorrhage originates in the pulmonary microvasculature, rather than from the bronchial circulation or parenchymal abnormalities. DAH is a clinico-pathologic syndrome characterized by intra-alveolar accumulation of red blood cells that originates from the interstitial capillaries (precapillary arterioles, alveolar capillaries, or post capillary venules). A retrospective review of 34 cases of DAH revealed nearly one-third of the cases were caused by granulomatosis with polyangiitis (previously termed Wegener granulomatosis). Are you sure your patient has Diffuse Alveolar Hemorrhage? What should you expect to find? Although hemoptysis is considered the cardinal sign of DAH, it may be absent in up to 33 percent of all patients; therefore, the absence of hemoptysis does not rule out DAH. Hemoptysis may be a dramatic event or it may evolve over days to weeks. The symptoms of DAH, other than hemoptysis, tend to be non-specific; they can include but are not limited to fever, chest pain, cough, and dyspnea. Exam findings may include rales on examination and respiratory failure. A detailed history and physical exam should be obtained, including questions pertaining to connective tissue disorders, HIV status, drug exposures, occupational exposures and a cardiac history. The cornerstone of diagnosis is a multi-faceted approached involving: History and physical Laboratories Imaging studies Non-invasive pulmonary diagnostic studies Diagnostic procedures It is important to establish the cause of DAH, since virtually all cases, except those associated with overwhelming diffuse alveolar damage, are potentially reversible. Treatment is directed at the underlying etiology and typically includes corticosteroids, total plasma exchange, and immunosuppressant therapy. Beware: there are other diseases that can mimic Diffuse Alveolar Hemorrhage: Any disorder that can lead to ground glass changes on imaging and hypoxia can appear similar to alveolar haemorrhage. These include inflammatory disorders, ILD, congestive heart failure, pneumonia, vasculitis, eosinophilic syndromes, infectious diseases. How and/or why did the patient develop Diffuse Alveolar Hemorrhage? Common to all causes of DAH is injury to the basement membrane of the alveolar capillary bed, which injury allows for accumulation of red blood cells into the alveolar space. However, the injury to the basement membrane is unique to the specific systemic disease that underlies the DAH, and in some cases the mechanism of injury remains unknown (see Table I). Mechanism of Injury in DAH General treatment strategies based on etiology of DAH Steroid-sparing immunosuppressive regimens for DAH secondary to pulmonary capillaritis Chest X-ray and CT scan illustrating the alveolar filling process of DAH Which individuals are of greatest risk of developing Diffuse Alveolar Hemorrhage? By far the greatest risk factor for the development of DAH is an established diagnosis of a systemic vasculitis (ANCA-associated granulomatous vasculitis, anti-GBM disease, SLE, etc.) Alveolar hemorrhage may be the presenting manifestation of granulomatosis with polyangiitis (GPA) in approximately 8% of patients with the disease. It is diagnosed in less than 2–4% of SLE patients; however, the mortality rate approaches 50%. DAH is found in up to 66% of patients in autopsy series. Only 5–10% of cases of Goodpasture syndrome (anti-GBM disease) will have presented with DAH alone. Although the literature about HIV and DAH is limited, a case series of HIV patients with Kaposi sarcoma (KS) found that 75% of these patients had alveolar hemorrhage. There was no effect on 12-month mortality. Other important clinical causes of DAH that are not vasculitic in nature are infection, ARDS, the use of anticoagulants with supra-therapeutic bleeding times, and the use of IIB/IIIA medications following percutaneous interventions. DAH after hematopoietic stem cell transplant (HSCT) is a devastating complication that carries an overall mortality of 70–100%. Median time to onset after transplant was between 21 and 23.5 days according to two reviews. There are no prospective studies that estimate the relative frequency of DAH. Epidemiologic studies show that the prevalence and incidence of the autoimmune disorders is increasing, with the overall prevalence of the primary systemic vasculitis’ of 90 to 257 per million and the incidence is estimated at 10 to 20 per million. What laboratory studies should you order to help make the diagnosis, and how should you interpret the results? An unexplained drop in hemoglobin in the setting of respiratory failure with diffuse infiltrates should prompt a suspicion for DAH. Further work up should be directed at the etiology of DAH: autoimmune serologies (e.g., ANA, dsDNA, ANCA, anti-GBM), coagulation studies, and peripheral blood smear should routinely be sent. Urinalysis with microscopic evaluation should be performed to evaluate for possible pulmonary-renal syndrome. There are no genetic tests available to confirm the diagnosis of DAH. What imaging studies will be helpful in making or excluding the diagnosis of Diffuse Alveolar Hemorrhage? Chest radiograph is nonspecific and reveals an alveolar filling process that can be patchy, focal, or diffuse in nature. Chest radiograph can also be used to follow the clinical course of disease. Chest CT will typically show diffuse and frequently bilateral ground glass opacities. CT can also be used to further define the extent of the disease and help to localize the segment where bronchoalveolar lavage (BAL) should be performed. What non-invasive pulmonary diagnostic studies will be helpful in making or excluding the diagnosis of Diffuse Alveolar Hemorrhage? Pulmonary function tests (PFT) are rarely indicated in the acute setting. If obtained, an increased diffuse capacity of carbon dioxide (DLCO) should alert the physician to the possibility of alveolar hemorrhage. Pulmonary function testing should be performed following resolution of DAH, particularly in those patients who are at risk of recurrence of DAH (i.e. systemic vasculitis). Transthoracic echocardiography (TTE) should be performed to rule out the presence of valvular disease or myocardial dysfunction as a cause of DAH. What diagnostic procedures will be helpful in making or excluding the diagnosis of Diffuse Alveolar Hemorrhage? The diagnosis is established after bronchoscopy with serial bronchoalveolar lavage (BAL) revealing progressively bloodier fluid or increasing RBC counts. The bronchoscope is wedged in a sub-segmental bronchus proximal to the region of involvement, and three serial aliquots of 30 to 60 mL of saline are instilled and withdrawn. If the BAL becomes progressively more hemorrhagic, suggesting alveolar or capillary origin, the diagnosis of DAH is confirmed. Alternatively, if the returned BAL aspirate clears with each aliquot, the bleeding is not consistent with DAH. The presence of hemosideren-laden macrophages on the BAL (>20%) is helpful in detecting DAH as well. Bronchoalveolar lavage fluid should be sent for bacterial cultures and viral PCR to rule out infectious causes of diffuse alveolar damage and to ensure no infection is present prior to high dose immunosuppression, if needed. Surgical lung biopsy should be considered if the clinical history or serologic testing is unrevealing or the disease is refractory to treatment. Transbronchial biopsies are insufficient for histopathologic diagnosis. If there is a suspicion for a pulmonary-renal syndrome, a renal biopsy with direct immunofluorescence should be pursued, as this procedure has a low morbidity rate. What pathology/cytology/genetic studies will be helpful in making or excluding the diagnosis of Diffuse Alveolar Hemorrhage? DAH has three histopathologic patterns on open lung biopsy: Pulmonary capillaritis: Neutrophilic infiltration of the alveolar wall and destruction of the capillary leading to hemorrhage into the alveolar sacks. Bland hemorrhage: Hemorrhage without alveolar destruction or inflammation. Diffuse alveolar damage: Edematous septa but no inflammation. The most common histopathologic pattern found to cause DAH is pulmonary capillaritis, typically in the setting of a rheumatologic disease or systemic vasculitis. Renal biopsy to assess pulmonary renal syndromes, if indicated and to rule out other disorders that can present with pulmonary haemorrhage: Immunofluorescence can show: SLE: “clusters” immune complex Anti-GBM: linear deposition in the basement membranes ANCA-associated granulomatous vasculitis and Microscopic Polyangiitis: absence of immune complex deposition If you decide the patient has Diffuse Alveolar Hemorrhage, how should the patient be managed? The goal of management is to stabilize the patient, halt the progression of the disease process, and limit end organ damage. Treatment should be directed at the underlying cause of DAH (see Table 2), most common being systemic vasculitis. In rapidly progressive or fulminant cases, institution of empiric treatment is appropriate. Pulmonary Capillaritis Bland Hemorrhage Diffuse Alveolar Damage Connective Tissue Diseases: Mixed connective tissue disease Antiglomerular basement membrane antibody disease (Goodpasture’s) Polymyositis Primary antiphospholipid antibody syndrome Rheumatoid arthritis Systemic lupus erythematosis (SLE) Systemic scleroderma Connective Tissue Diseases: Antiglomerular basement membrane antibody disease (Goodpasture’s) Systemic lupus erythematosis Infection: Any associated with ARDS Viral Systemic Vasculitides: ANCA-associated granulomatous vasculitis (Wegener’s) Microscopic polyangitis Behcet’s syndrome Cryoglobulinemia Pauci-immune glomerulonephritis Henoch-Schoenlein purpura Isolated pulmonary capillaritis (ANCA-positive or -negative) Idiopathic glomerulonephritis Drugs*: Anticoagulant therapy Platelet glycoprotein IIA/IIIB Drugs*: Amiodarone Cytotoxic agents Nitrofurantoin Penicillamine Propylthiouracil Sirolimus Drugs*: Diphenylhydantoin Propylthiouracil Retinoic acid syndrome Other: Hematopoietic Stem Cell Transplant Acute lung transplant rejection Ulcerative Colitis Myasthenia gravis Leptosporosis Other: Idiopathic pulmonary hemosiderosis Mitral stenosis Pulmonary veno-occlusive disease Subacute bacterial endocarditis Leptosporosis Obstructive sleep apnea Connective Tissue Disease: Polymyositis Systemic lupus erthematosis Other: Crack cocaine inhalation Hematopoietic stem cell transplant Radiation therapy ARDS (any cause) Miscellaneous histologies Pulmonary veno-occlusive disease Lymphangioleiomyomatosis Pulmonary capillary hemangiomatosis Fibrillary glomerulonephritis Metastatic renal cell carcinoma Epithelioid hemangioepithelioma Angiocarcoma Choriocarcinoma syndrome * Drugs are a frequent cause of DAH. pneumotox.com is a search engine that allows a clinician to determine quickly whether a drug has been reported to cause pulmonary disease. Acute Exacerbation of Interstitial Lung Disease Diffuse alveolar damage on backdrop of the underlying ILD Idiopathic pulmonary fibrosis, Connective-tissue-associated ILD Acute Interstitial Pneumonitis Organizing diffuse alveolar damage Idiopathic (Hamman-Rich syndrome), collagen vascular disease, cytotoxic drugs, infections Acute Eosinophilic Pneumonia Eosinophilic infiltration and diffuse alveolar damage Idiopathic, drugs Acute Cryptogenic Organizing Pneumonia Organizing pneumonia Idiopathic, collagen vascular disease, drugs, radiation, infections Acute Hypersensitivity Pneumonitis Granulomatous and cellular pneumonitis with diffuse alveolar damage Inhaled antigens Glucocorticoids are the mainstay of therapy if pulmonary capillaritis is the etiology of DAH. Dose: Methylprednisolone 500 mg to 1g daily or in divided doses over three days then 0.5 mg/kg/day Duration: typically prolonged taper over months as steroid-sparing immunosuppressive agents are initiated and take effect Other immunosuppressive medications are directed at the underlying etiology (see Table 3) and are given in conjunction with or just following glucocorticoid pulse. Serologic Non-serologic Complete blood cell counts, Comprehensive metabolic panel, Coagulation studies, Blood smear Urinalysis with microscopic evaluation ANA, RF, anti-CCP, P-ANCA, C-ANCA (MPO, PR3), Anti-GBM antibodies, Anti-ds DNA, Antiphospholipid antibodies, Anti-Smith Transthoracic echocardiogram Complements, Cryoglobulins CT chest Serial chest X-rays Cyclophosphamide can be given more easily in patients with a pre-existing diagnosis of systemic vasculitis and should be considered in appropriate patients with severe vasculitis. Careful consideration should be given to its empiric use because of the potential for prolonged toxicity and its delay in therapeutic effects, which may be up to three weeks following administration. Cyclophosphamide has a substantial side effect profile, including bone marrow suppression–which may prohibit its use in the intensive care unit–and hemorrhagic cystitis. Despite these reservations, the combination of pulse corticosteroids and cyclophosphamide has dramatically decreased mortality in patients with systemic vasculitis. If the decision is made to begin cyclophosphamide, intravenous therapy has advantages over oral administration, particularly in the critically ill patient. Intravenous versus oral administration is equally effective for induction of remission in the ANCA-associated vasculitis’ and has less risk of neutropenia. Adequate hydration and pre-treatment with Mesna should be utilized to help decrease the risk of hemorrhage cystitis. Careful monitoring of complete blood counts and renal function should be performed. Appropriate dose adjustments should be made if there is evidence of neutropenia or renal injury. Rituximab is an anti-CD20 monoclonal antibody that targets B-cells. Pulse dose corticosteroids and IV cyclophosphamide are considered the current standard of treatment for DAH that is due to systemic vasculitis; however, the side effect profile and the potential for severe immunosuppressive may limit the use of cyclophosphamide. According to RAVE-ITN, a randomized, double-blind, double-dummy, non-inferiority trial, the use of rituximab in patients with ANCA-associated vasculitis was as effective in inducing remission as cyclophosphamide. Other therapies Supportive measures should be undertaken to prevent morbidity. In the critically ill patient, the use of lung-protective strategy to ventilate and correct an underlying coagulopathy should be performed. Infection is an important cause of mortality in patients who are immunosupressed, so care should be given to prevent iatrogenic infections. Salvage therapies are used in to prevent death in critically ill patients for whom rapid stabilization is required, and to allow established therapies time to have effect: ECMO: Extracorporal membrane oxygenation (ECMO) is a life-saving therapy in neonates and children with severe respiratory failure. It requires access to a skilled perfusionist and a tertiary care center with experienced surgeons. ECMO has a relative contraindication in patients with systemic disease; however, several case reports describe the use of ECMO in cases of severe respiratory failure that was due to DAH in systemic vasculitis. Although these reports suggest ECMO can be used, care should be given to choosing the appropriate patient for this therapy, which should be considered a last-line therapy. Human recombinant factor VIIa: The process of hemostasis requires an intact coagulation factor and normal vascular endothelium. The process of clot formation begins following injury to the vascular endothelium which releases factor VII. The circulating factor VII binds to tissue factor found on stromal cells and fibroblasts creating the VIIa/TF complex, thus initiating the extrinsic coagulation cascade. Several case reports have shown the effective return of hemostasis in patients with DAH when Human rf VIIa is given via bronchoscopic administration or nebulized treatment. 1 Transexcamic acid (TXA): An inexpensive, synthetic anti-fibrinolytic agent that has been useful in controlling bleeding from oral and cardiac surgery. Researchers successfully used both aerosolized and intrapulmonary injections of transexamic acid to control six cases of DAH of different etiologies. Several sources cite increased risk of post-operative seizures in patients who received TXA. A large study of patients given TXA during cardiac surgery found TXA had higher rates of seizures, need for transfusions and mortality. Withdrawal of the suspected drug or toxin and supportive care are crucial in DAH cases caused by drugs or exposures. What is the prognosis for patients managed in the recommended ways? The natural history and prognosis is dependent upon the underlying cause of DAH. In those patients who are aggressively treated with appropriate diagnosis directed treatment, the mortality rate remains high, ranging from 13 percent to 50 percent, depending on the underlying diagnosis and side effects of treatment. If DAH goes untreated, patients die of progressive respiratory failure. Despite best practices, patients may develop repeated episodes of DAH, with which long-term pulmonary sequela may occur, such as evidence of pulmonary fibrosis and emphysema. What other considerations exist for patients with Diffuse Alveolar Hemorrhage? 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