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DIFFUSE ALVEOLAR HEMORRAGE AS A PRESENTATION OF ACUTE PROMYELOCYTIC LEUKAMIA: A RARE CASE REPORT

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ABSTRACT
Diffuse alveolar hemorrhage is a potentially fatal pulmonary disease syndrome that affects individuals with hematological and non hematological malignancies. The range of inciting factors is wide for this syndrome and includes thrombocytopenia, underlying infection, coagulopathy, and the frequent use of anticoagulants, given the high incidence of venous thrombosis in this population.We present a case of a 50 years old female with newly diagnosed acute myeloid leukemia presenting with diffuse alveolar hemorrhage.

Keywords: Acute myeloid leukamia, Acute promyelocytic leukamia, Diffuse alveolar hemorrage, Thrombocytopenia

INTRODUCTION
Acute myeloid leukemia (AML) is a hematopoietic neoplasm of the myeloid line of blood cells. Acute Promyelocytic Leukemia (APL) is a biologically and clinically distinct form of AML. APL is rarely seen in the first decade but increases with the second decade and into early adulthood[1].APL patients present with symptoms such as pancytopenia, fatigue, infection, bleeding gums, bleeding, nosebleeds and disseminated intravascular coagulation[2].Among patients with leukemia, DAH is one of the most common, noninfectious complications related to thrombocytopenia[3].DAH is a clinical syndrome defined by a disturbance of the alveolar-capillary basement membrane that causes bleeding into the pulmonary alveoli[4].The onset of symptoms is typically acute and nonspecific;fever, cough, dyspnea, and chest pain are among the most commonly presenting symptoms[4].Acute respiratory failure with hypoxia and an increasing oxygen requirement is also often seen as a presenting symptom[3].With the rapid progression and severity of DAH, patients are often unable to complete pulmonary function testing at the time of symptoms and diagnosis[5].Abnormal findings in chest radiography or computerized tomography are very common in DAH[6]. The typical findings on a chest radiograph are

interstitial infiltrates (i.e., septal lines and peribronchial cuffing, ground glass opacity) and pleural effusion. An increased cardio thoracic ratio (up to 87%) and parenchymal consolidation are also frequently encountered (47%), with or without air bronchogram[7].CT of the chest will characteristically show diffuse or patchy, bilateral ground-glass opacities.These opacities are often more centrally located and at the bases; they typically spare the periphery[8].Bronchoscopy with BAL showing progressively bloody effluent is usually diagnostic[9] .Here we present a case of newly diagnosed APL presenting with progressive dypnoea,rapidly deteriorating requiring ventilatory support due to diffuse alveolar hemorrhage.

CASE STUDY

A 50 year old female presented to emergency department with complain of sudden onset shortness of breath, hemoptysis, epistaxis since 1 hour.At the time of presentation she was in respiratory distress with poor oxygenation. She was given a trial of NIV,in view of her increasing oxygen requirement and falling saturation, she was intubated and connected to mechanical ventilator.

LAB INVESTIGATIONS

HB-5gm/dl, WBC-5000/cmm, Platelets-24000/cmm, Polymorphs-5%, Lymphocytes-24%, Eosinophils 0%, Monocytes-9%, Blasts-62%, Peripheral smear -normocytic normochromic anemia with visible blast cells, Serum ANA-negative, Serum LDH-510U/L, Bone marrow aspiration and biopsy suggestive of Acute myeloid leukamia, Immunophenotyping-CD34.33,13,117 positive suggesting acute promyelocytic leukamia, Cytogenetic study- PML:RARa and BCR1 detected, Chest radiograph-diffuse patchy opacities, HR CT- Diffuse alveolar hemorrage.

MANAGEMENT

Patient was kept on mechanical ventilation due to progressive respiratory distress on NIV.Chest CT showed significant worsening in bilateral ground glass opacity and septal thickening. She was treated with platelet transfusion, nebulized tranexamic acid, high dose corticosteroids, and empiric antibiotic therapy. Emergent induction chemotherapy was started.Her condition kept on deteriorating rapidly and she expired on day 4 of her hospital stay.

DSICUSSION

APL presenting as Diffuse alveolar haemorrhage and progressing at such a rapid pace is very rare.Our patient exhibited hemoptysis as early signs of diffuse alveolar hemorrage.Clinical presentation and radiological imaging helped us to diagnosis DAH. Management of DAH is challenging we had used ventilatory support and supportive care which include with platelet transfusion, nebulized tranexamic acid, steroids, and antibiotic therapy. Standard treatment strategies for DAH are typically directed toward the underlying etiology. Supportive-care measures, including hemodynamic support and invasive or noninvasive oxygen supplementation, discontinuation of any offending agents, and reversal of coagulopathy are basic first-line therapies.Systemic high-dose steroids are typically recommended for noninfectious cases of DAH to combat the inflammation precipitated by the underlying disease process. However, research has not shown significant differences in rates of mortality between patients with DAH receiving low, medium, or high-dose steroids[10].Use of platelet transfusions to treat thrombocytopenia in the setting of hemorrhage is widely accepted..In cases of DAH with an etiology suspected to be attributable to autoimmune vasculitides, immunosuppressive therapies and plasmapheresis have been used. Early diagnosis of cause and aggressive management may help in better outcome.

CONCLUSION

Initial presentation of APL as DAH is unusual and a high index of suspicion should be maintained in patients who does not fit into infectious and immune causes which we generally encountered. Early recognition of acute respiratory failure etiology and aggressive management is essential. DAH should always be on the differential as cause of respiratory failure, early identification and treatment can likely improve outcomes.

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