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A Case of Diffuse Alveolar Hemorrhage

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Not Your Typical Pneumonia

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Introduction

Diffuse alveolar hemorrhage (DAH) is the process of bleeding into the alveolar spaces of the lungs due to disruption of the alveolar-capillary basement membrane. It can be caused by many disease processes, including rheumatic diseases, vasculitis, infection, drugs, etc[4]. DAH is a rare, but serious pulmonary complication of systematic lupus erythematosus[1]. Cough, hemoptysis, fever, and dyspnea are common initial symptoms, however, hemoptysis can be absent in up to one third of patients[3]. In the absence of hemoptysis, new radiographic opacities, decreasing hemoglobin level, and the finding of hemorrhagic fluid on bronchoalveolar lavage favor the diagnosis[5].

Case Presentation

CC: "Persistent fever, rash, cough"
HPI: 22 year old female G1P0 14 weeks pregnant is presenting to the ED with chief complaint of fevers, body aches, lesions on the hand and in the mouth x2 weeks. Patient states that she was diagnosed with hand-foot-mouth disease, 12 days ago, states that since then has been having high fevers. Patient denies any severe headaches, neck pain, chest pain, shortness of breath. Patient is a current tobacco abuser. Patient denies any drug use or alcohol abuse. Patient has no other medical problems. Followed up with her Ob two days ago, US of baby wnl per patient. Patient denies having any abdominal cramping or vaginal bleeding or discharge.

ROS: Positive for fatigue, myalgia, fevers, rash, cough

Vitals: BP 112/62 mmHg | Pulse 142 | 39.9ºC | RR 20 | SpO2 100%

Physical Examination:
Constitutional: Well-developed and well-nourished. Alert.
Eyes: Pupils are equal, round, and reactive to light. EOM are normal.
Throat: Pustules noted over roof of mouth
Neck: Normal range of motion and full passive range of motion without pain. No neck rigidity.
Cardiovascular: Tachycardia, no murmurs
Pulmonary/Chest: Effort normal and breath sounds normal. No respiratory distress.
Abdominal: Soft. Bowel sounds are normal. She exhibits no distension. There is no tenderness. There is no rebound and no guarding.
Musculoskeletal: Normal range of motion.
Neurological: AAOX3. 5/5 strength B/L UE and LE. No cranial nerve deficit. Negative finger to nose test
Skin: Pustules/erythematous lesion noted on palms of bilateral hands, elbows, and lesions surrounding chin, on face, no evidence of sloughing of skin, no signs of burns noted

Hospital Course

Henry Ford Macomb ED Course 10/13/2018:

On initial presentation, a code sepsis was called based on presenting vitals – blood cultures, UA, CBC, BMP, Coags, CXR, lactic acid, influenza, monospot, and strep culture were ordered. 30 cc/kg fluid bolus and Tylenol was ordered.

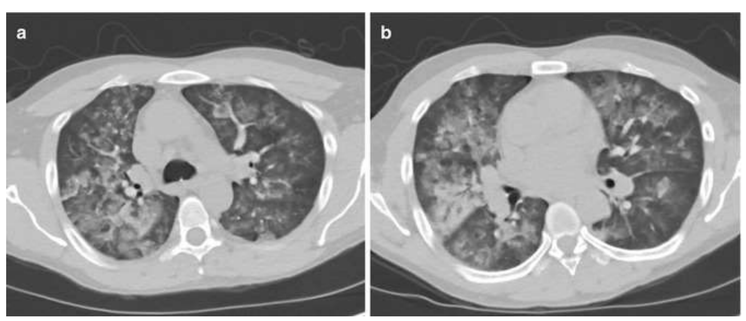
Labs significant for:
WBC: 3.3 Hgb: 8.3 Lactic Acid: 2.7 Na:129 K: 3.1

Patient initially improved clinically, lactic acid cleared after fluid bolus, however during the ED course, patient became hypoxic on room air at 88%, improved with 2 L of oxygen when sitting upright, patient felt short of breath lying flat, patient had bilateral coarse breath sounds, worse on the right, chest x-ray was repeated, BNP and troponin obtained. Patient's repeat chest x-ray showed worsening right lung interstitial pattern, concerns for pneumonia, at this time sepsis identified, IV Rocephin and IV azithromycin were started and patient was admitted for further evaluation.

Henry Ford Macomb Hospital Course 10/14/18-10/15/2018:
Patient continued to have recurrent fevers and tachycardia, repeat troponin was ordered, elevated at 110, peaked at 250 then trended down, likely secondary to NSTEMI type 2. ECHO 10/14/18 : EF: 57%. No evidence of ischemic, valvular, hypertrophic, or pulmonary heart disease.

17:48
Rapid response is called for tachypnea and tachycardia, patient was febrile 102.8F, HR 130s-140, blood pressure stable, sinus tachycardia on EKG, patient complained of positional chest pain at that time, patient was transferred to the ICU for further monitoring.

10/15/18
Per ID notes, there was concern for disseminated zoster as patient was not improving on antibiotics, started on acyclovir. Due to patient being a high risk pregnancy, with worsening respiratory status, requiring HFNC – decision was made to transfer to Henry Ford Main



Diffuse Alveolar Hemorrhage

Henry Ford Main Hospital Course:
Patient was transferred to HF Main MICU for escalation of care on 10/15/18. A multidisciplinary approach was taken with ID, Rheumatology, Dermatology, Nephrology, Hematology, and Maternal Fetal Medicine. Skin biopsies were collected, with samples positive for cutaneous lupus, patient was started on Methylprednisone 1g x 3 days and then Prednisone 70mg. Patient's respiratory status deteriorated, she was intubated on 10/17/18. Bronchoscopy on 10/18/18 demonstrated diffuse alveolar hemorrhage – six rounds of plasmapheresis were completed. Given the patient's severe symptoms secondary to SLE, Cytoxan therapy was initiated along with prophylactic Bactrim. Patient was cleared for discharge home with close follow up with her OB/Gyn and Rheumatology team.

Throughout the hospital course, patient had an extensive immunological, hematological, and infectious work up, which was all negative except the following: ANA 1:640 speckled, low complements C3 and C4, RNP +, Smith+, DAT+ Mycoplasma IgG

Discussion

DAH is a difficult diagnosis; clinical suspicion needs to be high. DAH accounts for only for 1.5 to 3.7% of hospital admission due to SLE[2]. In this particular case, when you look at the presentation of this patient, it classically fits DAH. The patient appeared to have pneumonia, however, clinical symptoms were not improving with antibiotics and continued to have infiltrates on imaging, her skin biopsies and lab work was consistent with SLE, and her BAL revealed hemorrhage fluid. This case was particularly difficult, as patient did not present with hemoptysis, she did not have a known diagnosis of SLE, she was pregnant, and had a complicated clinical course. DAH has a high mortality rate, 25-50%, therefore, it is imperative to diagnosis and treat early[4]. Treatment for DAH related to SLE includes a combination of systemic glucocorticoids and immunosuppressive therapy, rituximab, or plasmapheresis, and support care[3].

Resources

1. Kazzaz, N, et al. Systemic lupus erythematosus complicated by diffuse alveolar haemorrhage: risk factors, therapy and survival. Lupus science & medicine. 2015 Sept. Vol 2(1) e000117.

2. Mahmoud, G. Gheith, R. Kamel, M., Soliman, R. Alveolar hemorrhage in systemic lupus erythematosus: an overview. Egypt Rheumatology. 2011 Jan. 33 (1): 1-11

3. Nasser, M. and Cottin, V. Alveolar Hemorrhage in Vasculitis (Primary and Secondary). Semin Respir Crit Care Med. 2018 Aug. 39(4):482-493.

4. Schwarz, M. Diffuse Alveolar Hemorrhage. In Uptodate, Post, TW (Ed), UpToDate, Waltham, MA, 2018

5. Zamora, M, Warner, M., Tuder, R., et al. Diffuse alveolar hemorrhage and systemic lupus erythematosus. Clinical presentation, histology, survival, and outcome. Medicine (Baltimore) 1997 May.76:192–202