Postpartum Shortness of Breath: A challenging case to manage and diagnose in era of COVID 19 pandemic.

Abstract:

Shortness of breath after childbirth is not an uncommon presentation to Emergency Department. This presentation has a wide range of differential diagnosis. The differentials can be divided broadly into cardiac and non-cardiac causes. The main objective of this presentation is to stabilize the patient first and then look for the causes of shortness of breath. We encountered a similar case with postpartum shortness of breath, she was managed as a case of iatrogenic pulmonary edema with diuretics, and the patient remained well on follow up.

Introduction:

Postpartum shortness of breath has a wide range of differential diagnoses, including pulmonary embolism, amniotic fluid embolism, pneumonia, and pulmonary edema. Pulmonary edema is an emergency condition that accounts for 0.08% of pregnancies. (1)

Pulmonary edema is generally divided into broader categories, cardiogenic pulmonary edema and non-cardiogenic pulmonary edema (2). Non-cardiogenic pulmonary edema causes include acute respiratory distress syndrome (ARDS), high altitude and neurogenic pulmonary edema, high dose opioid use, pulmonary emboli, eclampsia-associated pulmonary edema, and acute pulmonary damage in transfusion (3-5). Cardiogenic and non-cardiogenic pulmonary edemas have a similar presentation and radiological appearance. Both clinical conditions cannot always be distinguished clearly, and even both may appear simultaneously. No matter what the underlying pathology is, appropriate resuscitation is the top priority. Once the patient is stable, the focus can be turned to diagnosing and treating the underlying cause. We encounter a case with shortness of breath after delivery of healthy baby by cesarean section managed with diuretics, we aim to highlight the possible differential diagnosis, work up and management of such cases.

Case presentation:

A thirty-seven-year-old female who recently had an elective cesarean section six days ago, presented to the emergency department with a complaint of progressive shortness of breath since hospital discharge (from last four days). Initially, the shortness of breath was on exertion later, she started to have shortness of breath at rest and she started to have orthopnea. She did not have any co-morbidities, and she works as an accountant in a bank.

On examination, the patient was dyspneic. She required 4-5 liters of oxygen to keep saturation above 94%, Respiratory rate was 30breaths/minute, Blood pressure was 130/80, and heart rate was 96/min.

Jugular veins were not distended (JVP was not raised). There was no lymphadenopathy, cyanosis, or clubbing. On chest examination, bilateral coarse crackles were heard from the mid

to lower zones of lungs, and bilateral leg pitting edema was present. The rest of the examination was unremarkable.

Her ECG did not show any acute changes. Her blood investigations showed microcytic hypochromic anemia (hemoglobin 10.4 gm/dl). Kidney and liver functions and cardiac enzymes were within the normal range. However, pro BNP was 1,590 pg/mL (normal is <125 pg/m), and D-dimers were 2.58 mg/L(0-0.44mg/L), urine protein/creatinine ration was 39.68 mg/mmol(normal value 0-22.6 pg/mmol). Her Covid 19 PCR was negative, and chest x-ray (Figure:1)showed bilateral fluffy infiltrates with increased broncho vascular markings, and there was blunting of right costophrenic angle.

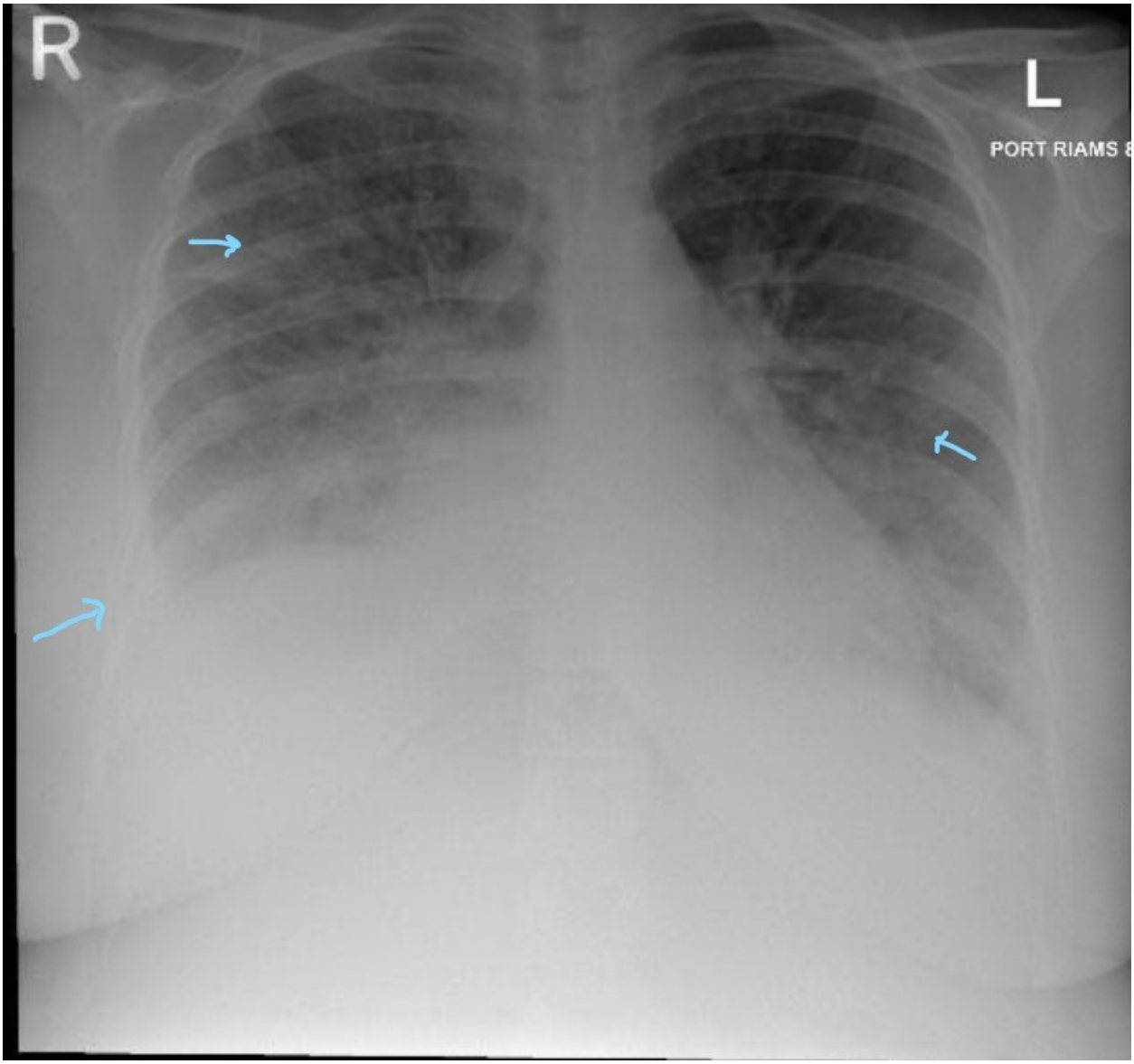

Figure:1 X ray showing bilateral fluffy infiltrates and blunting of right costophrenic angle.

A stat short of 40mg furosemide and ceftriaxone 2gm was given intravenously. CT pulmonary angiogram also came out negative for pulmonary embolism. However, the patient was kept in isolation due to the high possibility of a COVID 19 infection. She was started on intravenous furosemide 40 mg twice a day with continuation of Ceftriaxone, Azithromycin, and DVT prophylaxis while waiting for the second set of Covid 19 PCR and culture results.

The second set Covid 19 came negative, and the cultures did not grow any bacteria. There was no spike of fever and no rise inflammatory markers, so her antibiotics were stopped, but she was kept on diuretics. Echocardiography was done on the third day of admission, showed no regional wall motion abnormality, no valvular abnormality, and normal ejection fraction (65%).

The patient responded to diuretics. She started to maintain oxygen saturation>94% on room air. On the fourth day of admission, her chest x-ray(figure:2) showed an improvement. Her intravenous furosemide was changed to oral furosemide 40mg bid. The next day, the patient was discharged home with follow-up in outpatient after seven days.

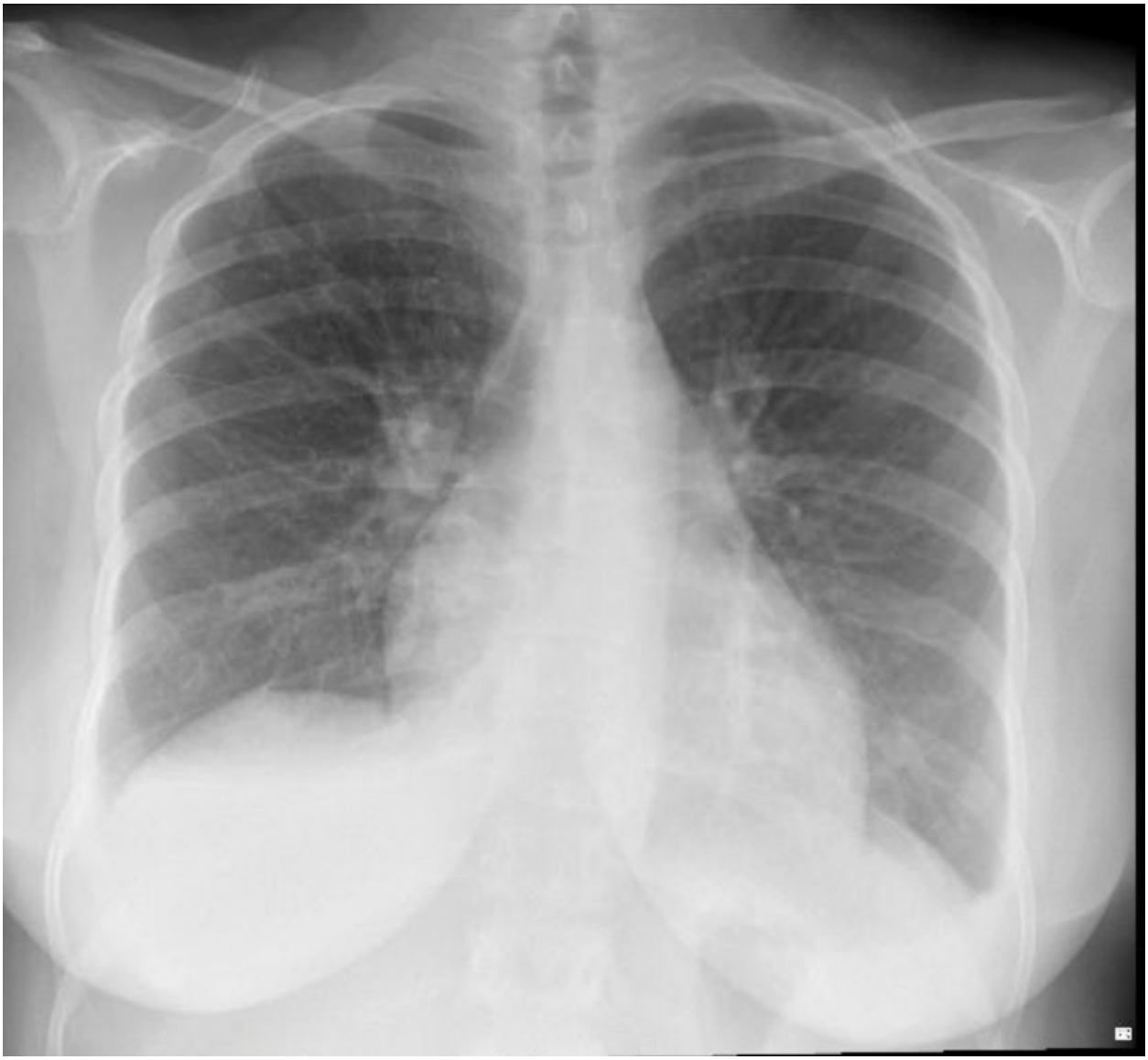

Figure:2 showed improvement in chest Xray after giving diuretics.

The patient was followed in an outpatient clinic after seven days. The patient was back to her baseline, and she had stopped taking furosemide for three days before the appointment. Her chest was clear, no new prescription for furosemide was given, the patient was advised to come to the emergency department in case of shortness of breath. The patient was followed for one month patient remained asymptomatic off diuretics.

Discussion:

There is a broad differential of dyspnea after delivery, including pathologies associated with pulmonary edema and pathologies not associated with pulmonary edema.

Pathologies not associated with pulmonary edema include pulmonary/amniotic fluid embolism, pneumonia, and sepsis (6). In this case, D-dimers were high, but CTPA ruled out pulmonary embolism, and the overall picture was not going with pulmonary embolism/amniotic fluid embolism as the patient had progressive shortness of breath with orthopnea and the patient responded well to diuretics. The patient did not spike a fever, and her inflammatory markers were in the normal range, so the possibility of infection was ruled out.

Pathologies associated with pulmonary edema are further divided into cardiac and no cardiac pulmonary edema. Cardiac causes of pulmonary edema in pregnancy include preexisting cardiac disease, peripartum cardiomyopathy, and myocardial infarction (7). In this case, the patient did not have any acute changes on an Electrocardiogram (ECG), and cardiac enzymes were in the normal range (Troponin T 12 ng/L). Echocardiography did not show any regional wall motion abnormality, valvular abnormality, and the ejection fraction was 65%(normal).

Noncardiac causes include Iatrogenic fluid overload, Tocolytic-induced pulmonary edema, Preeclampsia/eclampsia-related pulmonary edema (8). Our patient did not have taken any tocolytic agent/oxytocin. She did not have any history of preeclampsia/Eclampsia, and her BP was in the normal range throughout the pregnancy, and the protein creatinine ratio was not suggestive of proteinuria.

The patient was managed as a case iatrogenic pulmonary edema. The patient's response to diuretics in the absence of other causes makes it a more probable diagnosis.

Iatrogenic pulmonary edema is mostly caused by inappropriate usage/side effects of drugs, Usually, there is no single identifiable cause of iatrogenic pulmonary edema rather a single factor precipitate group of factors resulting in pulmonary edema. Iatrogenic pulmonary edema can be precipitated by drugs, negative pressure on the airway (obstruction, bronchospasm), circulatory overload (blood, fluids, absorption of irrigating fluids); and injury to alveolar membranes by noxious inhalants, excretion of poisons (kerosene). (9)

We believe our patient’s presentation was precipitated by circulatory overload due to continuous Intravenous fluid which she received during hospital stay at the time of delivery.

The patient was followed in the clinic after seven days. The patient took furosemide for four days, and then she stopped it herself. The patient did not have shortness of breath or orthopnea. Again, patient was followed one month off diuretics. She remained asymptomatic.

Peripartum acute pulmonary edema can lead to detrimental consequences. It is vital to identify the at-risk patient and provide them with appropriate care. After stabilization of the patient, look and address the underlying cause.

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