



# Acute Respiratory Distress Syndrome due to Sepsis in Pregnancy

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## Introduction

Acute respiratory distress syndrome (ARDS) is an uncommon condition in pregnant patients. The causes of ARDS are associated with obstetric causes such as amniotic fluid embolism, preeclampsia, septic abortion, and retained products of conception or nonobstetric causes that include sepsis, aspiration pneumonitis, influenza pneumonia, blood transfusions, and trauma [1-3]. The incidence of ARDS in pregnancy has been reported at single institutions and one report found the incidence of ARDS to be 1 in 6229 deliveries for an incidence of 17 patients per 100000 [4]. The mortality associated with ARDS is elevated and has been reported to range from 24% to 39% in pregnant patients [5,6]. An essential component in management of ARDS involves good communication between the obstetrics team and critical care specialist and a fundamental understanding of mechanical ventilatory support.

In this case report, a pregnant patient with ARDS due to sepsis was admitted to the intensive care unit, the potential causes for this admission and, the treatment offered to such patients has been discussed.

## Case

A twenty years old prima-gravida, in the 21 weeks of pregnancy, complained of headache and abdominal pain developed after a one-hour plane ride and was admitted to an emergency department of another hospital. There was no history of dysuria, vaginal discharge, pervaginal bleeding or fluid, cough, sputum, or recent upper respiratory tract infection.

On presentation, the blood pressure was low (BP): 80/40 mmHg, temperature: 38°C, heart rate (HR): 110/min, respiratory rate (RR): 28 b/min. She was conscious, oriented and cooperative, respectively. When admitted to ICU her observation was as follows: HR: 120/min, RR: 98/36 mmHg, temperature: 37°C, SS: 33/min, SpO<sub>2</sub>: 82%. Uterus was the level of the umbilicus and fetal movements were observed. Right costovertebral angle tenderness was present. The cardiorespiratory examination was otherwise unremarkable. On gynaecological examination there was no foul-smelling discharge or bleeding or pelvic tenderness.

The initial investigations revealed on urinary analysis of  $8 \times 10^6$  leukocytes, Platelet value 50.000/mm<sup>3</sup> and liver function tests (LFT) abnormalities was detected (Table 1). Due to the pregnancy, a chest X-ray was not performed on admission.

The patient was initially offered fluid resuscitation, low flow oxygen 3 l/m and given a broad spectrum antibiotic intravenous sulbactam-cefoperazone and admitted to the ICU for monitoring and management. Further blood, urine and vaginal swab cultures were sent. Repeated blood tests revealed mild anaemia (Hb: 9.5 g/dl), thrombocytopenia, leukopenia, hyponatremia, hypoalbuminemia, mild LFT elevation was present. C-reactive protein (CRP) 123 mg/dL (n: 0.5 to 6 mg/dL) and procalcitonin (PCT) 7.2 ng/ml (n<0.05 ng/mL) was determined (Table 1).

An obstetric ultrasound showed a positive fetal heart rate and, the fetus size was consistent with expected gestational age. Given the costovertebral tenderness and raised leucocytosis on repeat

uninanalysis of leucocytes  $40 \times 10^6$  and raised initial PCT (7.2 ng/mL) the patient was treated as a suspected urinary tract with cefotaxime.

During follow-up in ICU the patient remained hypotensive, with tachypnea and hypoxemia, eventually requiring non-invasive mechanical ventilation support. At the 16<sup>th</sup> hour of ICU admission a Chest X-ray with fetal protection was performed which was revealed extensive bilateral central to peripheral areas of coalescing consolidation consistent with ARDS. Patient was electively intubated due to deepening hypoxemia (Table 2). Lung protecting ventilation strategy with low tidal volume (6 ml/kg) and high PEEP was started. PEEP level was titrated according to plateau pressure.

Cardiology review and a transthoracic echocardiogram showed a normal left ventricular function, with an ejection fraction of 65%. There were no findings suggestive of valvular disease or cardiomyopathy. However a slightly increased right ventricular dimensions were observed. And accordingly consideration was given for an amniotic fluid embolism or pulmonary embolism. Despite the fact sent D-dimer 4990 ng/mL (N <500 ng/ml), pulmonary embolism was not considered clinically. Pulmonology consultation was taken at this time and a thromboembolic event was not considered likely.

For a patient with sepsis without clear microbiological cultures and an abdominal-pelvic ultrasonography was undertaken looking for a source of sepsis but this was reported as «revealed no pathological findings».

Unfortunately whilst intubated the patient had vaginal bleeding and amniotic fluid discharge leading to spontaneously abortion of a stillborn fetus. The fetus was sent to pathology for examination.

A computed tomography pulmonary angiogram, was negative for a pulmonary embolism however there was extensive parenchymal consolidation areas.

Given the raised PCT value of 7.2 ng/mL, the main diagnostic consideration was for a sepsis-associated ARDS. The antibiotic treatment was broadened to include meropenem, anidulofungin, teicoplanine, and oseltamivir. The blood, urine, and endotracheal aspirates did not isolate any positive culture. Furthermore H1N1 and H3N2 were negative antigen tests for respiratory secretions, and serum

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Laboratory Findings	Admission	1 <sup>st</sup> day	3 <sup>rd</sup> day	5 <sup>th</sup> day	Discharge
Hg (gr/dL)	9.5	10.4	9.2	10.9	11.9
Hct (%)	28.8	30.5	27.0	33.7	36.1
WBC	3990	11210	7740	15850	12060
Neutrophil (%)	86	79	68	79	64
Neutrophil count	3440	8840	5300	12520	7730
Lymphocyte (%)	8.5	18.1	27.1	16.7	26.5
Lymphocyte count	340	2030	2090	2660	3200
Platelet count	58700	57700	79100	244800	255200
Sodium (mmol/L)	132	134	135	138	137
Potassium (mmol/L)	3.5	3.4	3.3	2.9	3.4
Calcium (mg/dL)	7.0	6.9	7.5	6.9	7.6
Albumine (gr/dl)	2.4	2.2	2.4	2.5	2.9
AST (U/L)	168	115	58	93	37
ALT (U/L)	65	57	41	82	56
ALP (U/L)	61	79	87	88	82
GGT (U/L)	31	33	44	116	99
LDH (U/L)	556	689	505	427	361
BUN (mg/dL)	4.4	6.8	16.8	12.9	17
Creatinine (mg/dL)	0.4	0.3	0.3	0.3	0.4
C-reactive protein (mg/dL)	123	-	102	4	3
Procalcitonin (ng/mL)	7.2	11.7	4.1	0.09	0.05

Table 1: Patient's laboratory findings.

Parameter	Admission (under 3 ltr/m nasal O2)	16 <sup>th</sup> hour (under NIMV*)	3 <sup>rd</sup> day (intubated)	Discharge (room air)
Ph	7.41	7.43	7.49	7.43
PaO2 (mmHg)	78.9	49.3	79.0	80.0
PaCO2 (mmHg)	26.3	25.5	35.4	32.7
HCO3 (mmol/L)	18.3	16.0	28.2	23.0
SpO2 (%)	96.6	88.2	96.4	96.6
BE (mmol/L)	-6.8	-7.7	3.1	-2.2

Table 2: Patient's blood gas values.

Candida antigen, serum CMV PCR, Panfungal antigen tests were negative. Vaginal swabs were normal.

The follow up resulted in progressive improvement in the patient's lungs films (Figure 1), that correlated with a decrease in oxygen demand, and improved hemodynamic and respiratory parameters. Given no clear identification of septic source the the anidulofungin, teicoplanin and oseltamivir treatment was ceased on day 5, while meropenem therapy was discontinued on day 7.

The blood parameter for liver function tests and platelet count turned to normal and CRP (3 mg/dl), PCT (0.05 ng/ml) all decreased (Table 1). The patient was discharged to ward on room air with normal oxygen saturation.

## Discussion

Life-threatening ARDS during pregnancy, is a condition that can cause maternal and fetal death. Intensive care management of these patients may require a multidisciplinary approach involving obstetrics and gynecology, cardiology, infectious diseases, pulmonology and critical care team collaboration. Our case demonstrates such a case with multidisciplinary consultative approach. Although sadly the fetus was lost, but the maternal survival was achieved.

During pregnancy, nonobstetric causes for ARDS include sepsis due to pyelonephritis, pneumonia, intracerebral hemorrhage, blood transfusion, and trauma. Pregnant patients are vulnerable to develop aspiration pneumonitis during lobar termed Mendelson syndrome [7].

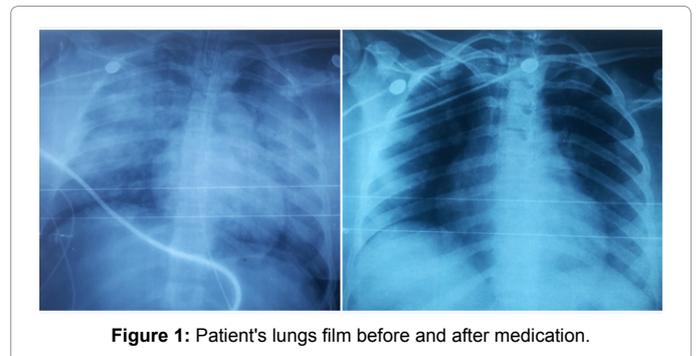


Figure 1: Patient's lungs film before and after medication.

The incidence of sepsis in pregnancy varies between 0.1-0.3% [(8,9)]. The most common cause of bacteremia in pregnancy is chorioamnionitis, genital infections, and urinary tract infection [10]. In our case, the placenta and attachments sent to pathology examination but no evidence of chorioamnionitis was detected. Furthermore all cultures were negative. Bacteremia is associated with an 10-28% fetal mortality during pregnancy. The agents most frequently isolated are *E. coli*, group B streptococci, *S. aureus*, *L. monocytogenes* and anaerobic bacteria [9,11]. Group B streptococci related sepsis are responsible for 50% of maternal mortality [12]. In our case, none of the cultures were positive and no cause of sepsis could be determined. There was broad spectrum antibiotic instituted in emergency department prior to ICU admission may have affected our subsequent microbiological sampling and cultures accuracy.

In pregnancy, advanced maternal age, obesity, insulin-dependent diabetes mellitus, multiple pregnancy, invasive diagnostic or therapeutic interventions are risk factors for sepsis [12,13]. Our case is a young (20 years old woman), lean body habitus (BMI: 22 kg/m<sup>2</sup>) and was a singleton pregnancy without an underlying disease or previous invasive procedures.

For our case, ARDS due to sepsis syndrome was considered. However clinical picture and treatment response was in keeping with non-cardiogenic pulmonary edema. Our case does demonstrate a life-threatening ARDS of pregnancy is a condition that may require multidisciplinary approach and intensive care treatment.

In conclusion, sepsis management in pregnancy may be challenging due to hemodynamic changes associated with pregnancy as well as the myocardial suppression due to sepsis.

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