

Adult Respiratory Distress Syndrome Complicating Recurrent Antepartum Pyelonephritis

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Acute pyelonephritis is estimated to occur in 1% to 2% of all pregnancies.¹ Prompt antimicrobial therapy is essential to prevent the well-described hemodynamic, hematologic, and renal complications of urosepsis. Recently, a series of case reports have shown adult respiratory distress syndrome (ARDS) to be an additional potential complication of antepartum pyelonephritis. The background to this complication is presented along with the first reported case of ARDS associated with recurrent pyelonephritis.

CASE REPORT

A 16-year-old primigravida presented to the University of Arizona College of Medicine Family Practice Office at 31 weeks' gestation with flank pain, pyuria, and a temperature of 40.1°C (104°F). Her pregnancy had been complicated only by a prior admission at 22 weeks' gestation for pyelonephritis caused by infection with *Escherichia coli*. She was successfully treated with cefazolin on that occasion and had normal findings on follow-up urine studies. Her managing physicians had decided against ongoing suppression therapy with antibiotics.

The patient was admitted and begun on intravenous cefazolin and maintenance fluids. She was assessed not to be significantly dehydrated. Her renal function tests at admission were normal, and her white cell count was $16.4 \times 10^9/L$.

Approximately 39 hours after admission, she reported the rapid onset of dyspnea. Her hematocrit had fallen to

0.29 from an admission value of 0.36, and an arterial blood gas on room air revealed a PO_2 of 7.1 kPa (53 mm Hg), PCO_2 of 2.7 kPa (20 mm Hg), oxygen saturation of 0.90, bicarbonate of 2.7 kPa (18 mm Hg), and a pH of 7.42. A chest radiograph showed findings consistent with pulmonary edema (Figure 1). She was transferred to the intensive care unit (ICU), where hemodynamic monitoring revealed a pulmonary capillary wedge pressure of 1.6 kPa (12 mm Hg). A fractional inspired flow of oxygen of 60% was required for the first 24 hours; however, she was subsequently weaned to room air and transferred out of the ICU after 48 hours. Her urine grew *E coli* sensitive to cefazolin, but blood cultures were negative. She was discharged home on the 8th day of her hospitalization.

Suppression therapy with nitrofurantoin was begun at discharge, and she was followed for the remainder of her pregnancy without further problems. She subsequently had an uncomplicated vaginal delivery of a normal full-term infant.

DISCUSSION

Cunningham et al² initially reported respiratory insufficiency complicating antepartum pyelonephritis in 1984. In a 1987 review of 75,000 deliveries over a 7-year period,³ they noted respiratory distress to affect approximately one in every 50 pregnant women admitted for pyelonephritis. Fifteen ARDS patients were presented in this latter review, but unlike the case presented here, none was stated to have had more than one episode of pyelonephritis.

Several similarities are noted between this case and those reported by Cunningham and others.²⁻⁵ Respiratory distress complicating pyelonephritis often occurs around 30 weeks' gestation, with the mean gestational age in the largest series being 28.7 weeks (± 5.6 weeks).³ Onset of dyspnea in all cases was seen within 48 hours after admis-

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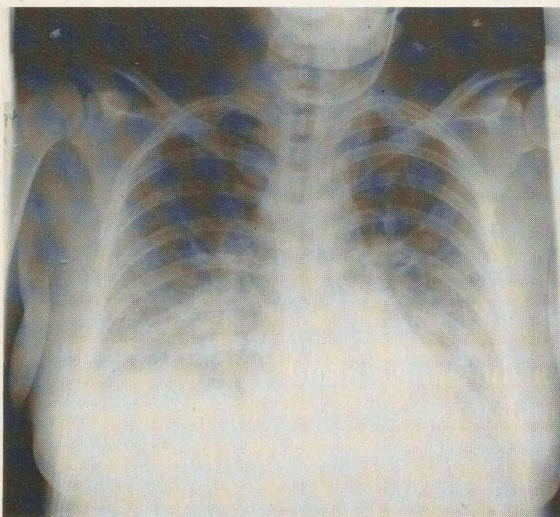


Figure 1. Chest radiograph demonstrating pulmonary edema.

sion, with a mean of approximately 30 hours. Eighty-five percent of reported cases have had parities of one or none, with the majority being nulliparous. *E coli* has been the primary bacterial organism in most cases; however, 25% of these patients grew *Klebsiella pneumoniae*. Additionally, the hematocrit levels of all but one patient were less than 0.30 when respiratory distress was apparent, and evidence of hemolysis was commonly seen. Unlike this case, severe morbidity has been reported, with three women requiring mechanical ventilation, and one death from sepsis recorded.³

Iatrogenic causes of pulmonary edema, such as fluid overload, have been ruled out in this and other cases. Proposed mechanisms of pathogenesis in these patients have centered on the role of endotoxins. Nonpulmonary evidence of endotoxin-induced organ dysfunction has been manifested in these patients in the form of thrombocytopenia, hemolytic anemia, and transient renal dysfunction.³ The case illustrated here may also provide evidence of a progressive, pregnancy-enhanced susceptibility to endotoxins, as this patient endured a second-trimester episode of pyelonephritis without difficulty, then later experienced ARDS with a third-trimester infection.

CONCLUSIONS

Since pyelonephritis is so common during pregnancy, it is important that family physicians providing prenatal care be aware of this serious complication. Prompt recognition and aggressive therapy of ARDS are essential to avoiding further morbidity or mortality.

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