OBSTETRICS

Acute pyelonephritis in pregnancy: an 18-year retrospective analysis

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OBJECTIVE: We sought to describe the incidence of acute pyelonephritis in pregnancy, and to assess its association with perinatal outcomes in an integrated health care system.

STUDY DESIGN: A retrospective cohort study was performed using medical records on 546,092 singleton pregnancies delivered in all Kaiser Permanente Southern California hospitals from 1993 through 2010. These medical records include the perinatal service system along with inpatient and outpatient encounter files. Adjusted odd ratios (ORs) and 95% confidence intervals (CIs) were used to estimate associations.

RESULTS: The incidence of acute antepartum pyelonephritis was 0.5% (2894/543,430). Women with pyelonephritis in pregnancy were more likely to be black or Hispanic, young, less educated, nulliparous, initiate prenatal care late, and smoke during pregnancy. Pregnancies of women with pyelonephritis compared to those without were more likely to be complicated by anemia (26.3% vs 11.4%; OR, 2.6; 95% CI, 2.4—2.9), septicemia (1.9% vs 0.03%; OR, 56.5; 95% CI, 41.3—77.4), acute pulmonary insufficiency (0.5% vs 0.04%; OR, 12.5; 95% CI, 7.2—21.6), acute renal dysfunction (0.4% vs 0.03%; OR, 16.5; 95% CI, 8.8—30.7), and spontaneous preterm birth (10.3% vs 7.9%; OR, 1.3; 95% CI, 1.2—1.5). Most of the preterm births occurred between 33-36 weeks (9.1%).

CONCLUSION: We characterize the incidence of pyelonephritis in an integrated health care system where routine prenatal screening for asymptomatic bacteriuria is employed. Maternal complications are commonly encountered and the risk of preterm birth is higher than the baseline obstetric population.

Key words: perinatal outcomes, pregnancy, preterm labor, pyelonephritis

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Acute pyelonephritis is one of the most common nonobstetric indications for antepartum hospitalization and is estimated to complicate up to 2% of all pregnancies in the United States.[1] While still common in developing countries, during the past few decades, the incidence of acute pyelonephritis during pregnancy has decreased substantially in developed countries. Pyelonephritis in pregnancy occurs mostly before delivery, with all but 10-20% of cases diagnosed in the second and third trimesters.[2,3] Nearly one quarter of affected women will have ≥1 recurrences during the same pregnancy.[4] Women with asymptomatic bacteriuria, defined as a urine culture from midstream collection with a single isolate of >100,000 colony-forming units of a uropathogen[2] are at increased risk of developing pyelonephritis in pregnancy compared to women without bacteriuria. Screening for and treatment of asymptomatic bacteriuria in pregnancy reduces the risk of subsequent pyelonephritis from approximately 20-35% to 1-4%.[3] Other potential risk factors that have been identified include multiparity, diabetes mellitus, urinary tract stones or malformations, and low socioeconomic status.[4,5] Untreated pyelonephritis can lead to increased risk of maternal and fetal morbidity and mortality including maternal fever, acute respiratory distress, acute renal failure, stillbirth, and preterm birth.[5]

The purpose of this study is to describe the recent trends in acute pyelonephritis among pregnant women, to accurately characterize at-risk mothers, and to examine whether acute pyelonephritis is associated with increased risk of perinatal outcomes in an integrated health care system. Previous reports in the literature describing pyelonephritis in pregnancy have been set in urban university environments.[6-8]

Materials and Methods

This was a retrospective cohort study of pregnant women delivering singletons in Kaiser Permanente Southern California (KPSC) hospitals from 1993 through 2010 (n = 546,092). KPSC is one of the largest integrated health care systems in the United States with approximately 31,000 annual deliveries, and providing comprehensive care to >3.4 million residents of Southern California.

We extracted medical records compiled electronically from all KPSC facilities. To ascertain exposures and outcomes, we used a unique maternal medical record number to link 4 different electronic medical records: the perinatal

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services system, maternal and infant hospitalization records, maternal outpatient health care encounters records, and laboratory records. The perinatal services system records contain maternal sociodemographic (age, race/ethnicity, education) and behavioral (smoking during pregnancy, timing of prenatal care initiation) characteristics as well as birthweight and gestational age at delivery from the infants' birth certificate. The maternal and infant hospitalization and outpatient physician encounter records include *International Classification of Diseases, Ninth Revision* (*ICD-9*), *Clinical Modification* (*ICD-9-CM*) codes from which we derived maternal medical history, obstetrical history, and procedures for services throughout KPSC. Microbiology laboratory records of each acute pyelonephritis patient were reviewed for the presence of uropathogens in the culture results. The study was approved by the KPSC Institutional Review Board.

Variables that were evaluated as potential confounders or mediators included maternal age (<20, 20-29, 30-34, ≥35 years); race/ethnicity categorized as non-Hispanic white (white), non-Hispanic black (black), Hispanic, Asian/Pacific Islander, and other race/ethnicity; maternal education (<12, 12, and ≥13 years of completed schooling); maternal smoking during pregnancy (yes/no); timing of initial prenatal care (first trimester, later or none); parity (0, 1, ≥2); medical conditions (chronic hypertension and pregestational diabetes); gestational diabetes; and birth year (1993 through 1995, 1996 through 1998, 1999 through 2001, 2002 through 2004, 2005 through 2007, and 2008 through 2010). Since the annual incidence rates for acute pyelonephritis were low, we chose to combine 3 years of records for statistical stability.

We used (*ICD-9*) codes 590.x to identify acute pyelonephritis. This and all subsequent diagnoses were made clinically and confirmed by laboratory tests. The outcome of interest examined in this study were: anemia (anemia complicating pregnancy childbirth or the puerperium; *ICD-9* codes 648.2x), septicemia (systemic inflammatory response syndrome; *ICD-9* codes 995.9x), acute renal failure (an abrupt or rapid decline in renal filtration function; *ICD-9* codes 584.x and 669.x), respiratory distress (*ICD-9* codes 518.8x), spontaneous preterm birth (a premature labor and delivery occurring at 20-36 completed weeks of gestation [grouped into blocks of 20-28, 29-33, and 34-36 weeks of gestation]), stillbirth (the intrauterine death of an infant >20 completed weeks of gestation), chorioamnionitis (an inflammation at the maternal-fetal interface; *ICD-9* codes 762.7x and 658.4x), preeclampsia (hypertensive disorder >20 weeks of pregnancy, combined with proteinuria and/or edema; *ICD-9* codes 642.4 and 642.5), and neonatal death (the death of a live born infant within 28 days of life). The exposure variable of interest was acute antepartum pyelonephritis (*ICD-9-CM* codes 590.1x).

We validated the accuracy of the *ICD-9-CM* coding by abstracting a random sample of 400 medical records. For this validation study, pregnancies resulting in low birthweight or premature births were oversampled to ensure adequate number of these risk factors to be reviewed. Because we applied a stratified sampling approach, the accuracy measures were estimated using weighted analyses. Abstracted records were compared with diagnosis codes collected electronically. After adjusting for sampling fractions, the estimated sensitivity, specificity, and positive and negative predictive values were 97%, 99%, 97%, and 99% for anemia; 100%, 99%, 92%, and 100% for chorioamnionitis; 82%, 95%, 74%, and 97% for group B streptoccus infection; 92%, 98%, 80%, and 99% for gestational fever; and 97%, 97%, 68%, and 100% for preeclampsia, respectively. These findings support the validity of the diagnosis codes in our study.

Gestational age data are based on a clinical estimate of gestational age and were categorized into 3 groups: 20-28, 29-33, and 34-36 weeks as well as term birth (37-42 weeks).

From 1993 through 2010, there were 546,092 singleton live births and fetal deaths recorded in all KPSC hospitals. We sequentially excluded births at <20 weeks' gestation (n = 1707), and early termination of pregnancy (n = 922). After exclusions, a total of 543,463 singleton pregnancies at ≥20 weeks of gestation remained for analysis.

Statistical analyses

We estimated the incidence of acute pyelonephritis diagnosis among singleton pregnant women delivered in all KPSC hospitals. Second, we compared the maternal demographic and behavioral characteristics between women with and without acute pyelonephritis using the χ^2 test. Differences with $P < .05$ were considered statistically significant. Third, logistic regression models were applied to examine the associations between maternal characteristics and acute pyelonephritis before and after adjusting for several potential confounding factors. Fourth, we further examined the association between acute pyelonephritis and perinatal outcomes after accounting for the effects of potential confounding factors listed in Table 1. The analyses were also stratified by spontaneous preterm birth into 3 groups defined above and by low birthweight (<1500 g and 1500-2499 g) categories to determine if associations were modified by these factors. The strength of the associations was explored based on the odds ratios (ORs) and their 95% confidence intervals (CIs). All analyses were performed using software (SAS, version 9.2; SAS Institute, Cary, NC).

RESULTS

During the 18-year study period, 2894 cases of acute antepartum pyelonephritis were identified, for an incidence level of 5.3 per 1000 births. Rates gradually increase from 4.6 per 1000 births in 1993 to 5.9 per 1000 births in 2010, reflecting a relative increase over the time period of 29%; P value for linear trends < .001. Most cases of acute pyelonephritis were diagnosed in the second and third trimester of pregnancy, accounting for 90.8% of cases in this analysis. Women were hospitalized for a mean of 2.8 days (SD 1.7). Women who were diagnosed with acute antepartum pyelonephritis were more likely to be younger, have fewer years of education, be of black or Hispanic ethnicity, smoke during pregnancy, initiate prenatal care late in

their pregnancy, and be nulliparous compared to women who were not diagnosed with acute pyelonephritis in pregnancy (Table 1).

The association between maternal characteristics and acute pyelonephritis during pregnancy is shown in Table 2. Compared with women 20-29 years old, women who were <20 years old were more likely to have a pregnancy complicated by acute pyelonephritis (OR, 2.0; 95% CI, 1.8−2.3). Compared with white women, Hispanic and black women were at significantly increased risk of acute pyelonephritis during their pregnancy, however, the risk for black women disappeared after adjusting for multiple confounders. Compared with women who had ≥13 years of formal education, women who completed 12 years of education had significantly increased risk of acute pyelonephritis (OR, 1.3; 95% CI, 1.2−1.5) as were women who had <12 years of formal education (OR, 1.5; 95% CI, 1.4−1.7). Late initiation of prenatal care and smoking during pregnancy were each associated with 1.1-fold increased risk of acute pyelonephritis. Pregnancy complicated by pregestational diabetes was associated with 1.7-fold (95% CI, 1.3−2.1) increased risk of acute pyelonephritis.

We did not find significant differences in the frequencies of medical complications such as chronic hypertension and gestational diabetes between those women with and without the diagnosis of pyelonephritis. Table 3 shows findings on the association between acute pyelonephritis during pregnancy and adverse perinatal outcomes. Women with acute pyelonephritis during pregnancy were at significantly increased risk of anemia (OR, 2.6; 95% CI, 2.4−2.9), septicemia (OR, 56.5; 95% CI, 41.3−77.4), acute renal failure (OR, 16.5; 95% CI, 8.8−30.7), respiratory distress (OR, 12.5; 95% CI, 7.2−21.6), spontaneous preterm birth (OR, 1.3; 95% CI, 1.2−1.5), and low birthweight birth (OR, 1.3; 95% CI, 1.1−1.5) compared to women without. Nearly 10% of women diagnosed with acute pyelonephritis delivered prematurely, compared to 7.9% without the condition. Most of these spontaneous preterm births occurred between 33-36 weeks' gestational age. Acute pyelonephritis was also associated with increased risk of chorioamnionitis (OR, 1.3; 95% CI, 1.1−1.5) and primary cesarean delivery (OR, 1.2; 95% CI, 1.1−1.3). However, acute pyelonephritis

TABLE 1
Distribution of maternal and infant characteristics based on pyelonephritis status

Characteristics	No pyelonephritis n = 540,536 (%)	Pyelonephritis n = 2894 (%)
Maternal age, y[a]		
<20	7.7	21.1
20-29	47.9	52.9
30-34	27.0	16.5
≥35	17.4	9.5
Maternal race[a]		
Non-Hispanic white	27.0	21.9
Non-Hispanic black	10.5	11.1
Hispanics	49.2	58.4
Asian/Pacific Islanders	10.9	6.2
Others/unknown	2.4	2.4
Maternal education, y[a]		
<12	13.7	22.2
12	30.8	37.3
≥13	50.8	36.4
Late initiation of prenatal care[a]	15.0	20.1
Smoking during pregnancy[a]	9.9	11.9
Gravida[a]		
Primigravida	29.2	37.5
Multigravida	70.8	62.5
Parity[a]		
Nullipara	39.9	48.6
Multipara	60.1	51.4
Chronic hypertension	3.3	3.1
Pregestational diabetes	2.2	2.9
Gestational diabetes	9.5	8.2
Year of delivery[a]		
1993 through 1995	15.0	15.4
1996 through 1998	17.2	16.0
1999 through 2001	17.6	14.7
2002 through 2004	16.4	14.4
2005 through 2007	16.6	16.6
2008 through 2010	17.2	22.9

[a] Differences between pyelonephritis and no pyelonephritis by maternal and infant characteristics were statistically significant ($P < .001$).

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TABLE 2
Association between maternal characteristics and acute pyelonephritis

Characteristics	OR (95% CI) Crude	Adjusted[a]
Maternal age, y[a]		
<20	2.5 (2.3–2.7)	2.0 (1.8–2.3)
20-29	1.0 (Ref)	1.0 (Ref)
30-34	0.6 (0.5–0.7)	0.6 (0.5–0.7)
≥35	0.5 (0.4–0.6)	0.5 (0.4–0.6)
Maternal race[a]		
Non-Hispanic white	1.0 (Ref)	1.0 (Ref)
Non-Hispanic black	1.3 (1.1–1.5)	1.1 (0.9–1.2)
Hispanics	1.5 (1.3–1.6)	1.2 (1.1–1.3)
Asian/Pacific Islanders	0.7 (0.6–0.8)	0.8 (0.7–0.9)
Others/unknown	1.2 (1.0–1.6)	1.1 (0.8–1.4)
Maternal education, y[a]		
<12	2.3 (2.0–2.5)	1.5 (1.4–1.7)
12	1.7 (1.6–1.8)	1.3 (1.2–1.5)
≥13	1.0 (Ref)	1.0 (Ref)
Late initiation of prenatal care[a]	1.4 (1.3–1.5)	1.1 (1.0–1.2)
Smoking during pregnancy[a]	1.2 (1.1–1.4)	1.1 (1.0–1.3)
Gravida[a]		
Primigravida	1.0 (Ref)	1.0 (Ref)
Multigravida	0.7 (0.6–0.8)	1.1 (0.9–1.2)
Parity[a]		
Nullipara	1.0 (Ref)	1.0 (Ref)
Multipara	0.7 (0.6–0.8)	0.9 (0.8–1.0)
Chronic hypertension	0.9 (0.8–1.4)	1.0 (0.8–1.3)
Pregestational diabetes	1.3 (1.1–1.6)	1.7 (1.3–2.1)
Gestational diabetes	0.9 (0.7–1.0)	1.0 (0.9–1.1)
Year of delivery[a]		
1993 through 1995	1.0 (Ref)	1.0 (Ref)
1996 through 1998	0.9 (0.8–1.0)	0.9 (0.8–1.1)
1999 through 2001	0.8 (0.7–0.9)	0.8 (0.7–1.0)
2002 through 2004	0.9 (0.7–1.0)	0.9 (0.8–1.1)
2005 through 2007	1.0 (0.9–1.1)	1.1 (0.9–1.2)
2008 through 2010	1.3 (1.2–1.5)	1.5 (1.3–1.7)

CI, confidence interval; *OR*, odds ratio; *Ref*, reference.

[a] Adjustments were made for other variables in table.

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was not associated with increased risk of very low birthweight, preeclampsia, stillbirth, or neonatal mortality.

In those pregnant women with acute pyelonephritis, urine cultures identified pathogens in 1887 (65.2%) subjects. The frequencies of uropathogenic bacteria isolated included: *Escherichia coli* (82.5%), *Streptococcus* species (21.4%), *Klebsiella pneumoniae* (7.6%), *Staphylococcus* species (6.5%), *Proteus mirabilis* (4.9%), and *Enterococcus* species (5.7%). Of those women who developed adult respiratory distress syndrome (ARDS) in association with pyelonephritis, the causative microorganisms were: *Escherichia coli* (50%), *Staphylococcus* species (14.3%), *P mirabilis* (7.1%), *Enterococcus* species (7.1%), and *Streptococcus* species (7.1%).

Comment

Overall, 0.5% of the pregnant women included in this study were hospitalized for acute pyelonephritis, which is less than historical reports.[9] We speculate that the implementation of the guidelines for screening for asymptomatic bacteriuria in pregnancy by the US Preventative Health Task Force[10] and the American College of Obstetricians and Gynecologists[11] may be partly responsible for this, leading to greater detection and treatment of this predecessor to many cases of pyelonephritis. It is also possible the high rates of early entry into prenatal care and the population characteristics might have contributed to the observed low rate.

Compared with women without a diagnosis of acute pyelonephritis, women who were diagnosed with acute pyelonephritis in pregnancy in this managed care organization were more likely to be <29 years of age, less educated, Hispanic or black, and nulliparous. As with previous investigations we found associations with lower educational achievement levels and late entry to prenatal care. These may be markers of lower socioeconomic status, a known association with the development of acute pyelonephritis in pregnancy.[4,7,8,12,13] We did not find associations with chronic hypertension, nor did we find associations with gestational diabetes.

We also demonstrated increasing frequencies of pyelonephritis when analyzing our data in 3-year epochs. We speculate that despite increased adherence to guidelines recommending routine screening for asymptomatic bacteruria in

TABLE 3
Associations between pyelonephritis and perinatal outcome measures

Outcomes	No pyelonephritis n = 540,536 (%)	Pyelonephritis n = 2894 (%)	Unadjusted OR (95% CI)	Adjusted[a] OR (95% CI)
Anemia	11.4	26.3	2.8 (2.6–3.0)	2.6 (2.4–2.9)
Septicemia	0.03	1.9	59.9 (44.3–81.1)	56.5 (41.3–77.4)
Acute renal failure	0.03	0.4	14.8 (8.0–27.4)	16.5 (8.8–30.7)
Respiratory distress/ARDS	0.04	0.5	12.9 (7.5–22.3)	12.5 (7.2–21.6)
Spontaneous preterm birth, wk				
<37	7.9	10.3	1.3 (1.2–1.5)	1.3 (1.2–1.5)
20-27	0.6	0.7	1.1 (0.7–1.7)	1.1 (0.7–1.8)
28-32	1.1	1.2	1.2 (0.8–1.6)	1.2 (0.8–1.6)
33-36	6.2	9.1	1.4 (1.2–1.6)	1.4 (1.2–1.6)
Low birthweight, g				
<1500	1.2	1.4	1.2 (0.9–1.7)	1.2 (0.9–1.7)
1500-2499	4.2	5.5	1.3 (1.1–1.6)	1.3 (1.1–1.5)
Chorioamnionitis	3.4	4.5	1.3 (1.1–1.6.)	1.3 (1.1–1.5)
Preeclampsia	5.1	5.5	1.1 (.0.9–1.3)	1.0 (0.9–1.2)
Primary cesarean	13.5	14.9	1.1 (1.0–1.3)	1.2 (1.1–1.3)
Stillbirth	0.4	0.3	0.8 (0.4–1.5)	0.9 (0.4–1.8)
Neonatal death	0.3	0.2	0.8 (0.4–1.7)	0.8 (0.4–1.7)

ARDS, adult respiratory distress syndrome; *CI*, confidence interval; *OR*, odds ratio.

[a] Analyses were adjusted for maternal age, race/ethnicity, education, prenatal care, gravida, chronic hypertension, pregestational and gestational diabetes, smoking during pregnancy, and year of delivery.

Wing. Acute pyelonephritis in pregnancy. Am J Obstet Gynecol 2014.

pregnancy, other age-related comorbidities may place the population at increased risk for pyelonephritis.

The uropathogens identified in this investigation were consistent with previous reports.[3,8,14,15] Because of the nature of the investigation, we could not profile chronologic differences in the appearance of some of these uropathogens as has been done by others,[8] although the appearance in later gestations concurs with findings of others.[16]

Anemia, the most common complication associated with pyelonephritis, occurs historically in approximately 25% of patients; our findings are similar.[8] The low frequency of ARDS in our investigation (0.5%) was considerably less than historic reports that reflect a range from 1-8%.[7,17] Our result may reflect improvements in treatment for acute antepartum pyelonephritis with judicious intravenous fluid management and rapid initiation of antimicrobial therapy. It is also possible that underreporting is responsible, a limitation imposed by retrospective analysis. Contrary to previous reports however,[18] the most common uropathogen identified in women with ARDS and pyelonephritis was *Escherichia coli,* not *K pneumoniae.* In fact, none of the cases of ARDS were due to *K pneumoniae.*

Approximately 15-20% of women with pyelonephritis will have bacteremia.[8,14,15] Gram-negative bacteria possess endotoxin that, when released into the maternal circulation, can lead to a cascade response of cytokines, histamine, and bradykinin. The resulting capillary endothelial damage, diminished vascular resistance, and alterations in cardiac output may lead to serious complications such as septic shock, disseminated intravascular coagulation, respiratory insufficiency, or ARDS. In a review of admissions for antepartum pyelonephritis in a major metropolitan center over 2 years, 17% of patients were also septicemic.[8] A separate analysis noted that 12% of antepartum admissions to the obstetric intensive care unit at the same institution were for sepsis caused by pyelonephritis.[19,20] Our results indicated a nearly 2% frequency of septicemia in association with acute pyelonephritis, nearly 50 times that of women without. This may reflect differences in socioeconomic status of our subjects diagnosed with gestational pyelonephritis and septicemia that permit earlier access to medical care and/or adherence to standardized protocols for treatment of this infectious complication of pregnancy that lead to improvements and perhaps prevention of this added complication.

Because blood cultures were not universally obtained, we could not assess the

frequency of bacteremia in our cohort. In fact, in only 841 of the 2949 cases of acute pyelonephritis cases in pregnancy were blood cultures performed. The limited utility of blood cultures in the management of pyelonephritis in pregnancy has been commented on previously.[21]

The exact risk of preterm labor and delivery directly attributable to pyelonephritis in pregnancy is difficult to estimate, particularly because delivery may not occur during the admission for the acute disease, and the risk factors for both pyelonephritis and preterm delivery overlap.[22] Nonetheless, our results indicate that women with a diagnosis of acute pyelonephritis in pregnancy have a modestly higher likelihood of preterm delivery compared to women without (10.3% vs 7.9%, $P < .001$). Our results are substantially higher than those of 368 women delivered at Parkland Hospital (Dallas, TX) with a history of pyelonephritis during the pregnancy. Of them, 19 of 368 (5%) delivered at <37 weeks, and only 4 of 368 (1%) delivered preterm during their admission for acute pyelonephritis.[8]

The strengths of this study include its population-based nature that includes a racially and ethnically diverse population of pregnant women and taking into account several potential confounding factors. The large number of cases and standardized approaches to medical care with basis in a large integrated health care system are added benefits. Another strength of the study was repeated validation of the clinical diagnosis codes using medical record gold standards. Limitations of this study include its retrospective nature, and the possibility of potential confounders that were not accounted for (residual confounding) in this study. By the nature of this examination, we were unable to evaluate the temporality of the appearance of these risk factors or complications related to the diagnosis of acute pyelonephritis in pregnancy. Therefore, the associations found between acute pyelonephritis and the various perinatal outcome measures in this study do not imply causality. Lastly, the small number of patients with blood culture tests precluded assessment of bacteremia in our cohort.

The findings of this study confirm that the diagnosis of acute antepartum pyelonephritis increased during the study period. It further underscores that acute pyelonephritis is an important complication of pregnancy, and a major cause of perinatal morbidity. ■

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