Background. Congenital infections cause significant morbidity globally. In the United States, population studies have indicated that congenital infections disproportionately affect minorities and the economically disadvantaged. Through their chronic and disabling effects these infections perpetuate generational poverty among these groups. The objectives of this study were to (i) provide a national prevalence estimate of congenital infections in children 0–2 weeks using discharge diagnosis codes; (ii) compare risk of congenital infection between white and non-White children; and (iii) investigate the relationship between socioeconomic status and risk of congenital infection in the United States.

Methods. The 2012 HCUP Kids’ Inpatient Database was used to identify discharges of children 0–28 days with an ICD-9 diagnosis code for congenital CMV (771.1), congenital syphilis (090.0–9), or congenital infection other (771.2). Univariate and multivariate logistic regression was used to estimate prevalence rates and potential risk factors for these infections.

Results. Prevalence of any congenital infection in children 0–2 years is 0.048%. Risk factor analyses found that African-American children are 1.85 times more likely to have any congenital infection compared with Caucasians (95% CI: 1.56–2.20), 1.49 times more likely to have congenital CMV (95% CI: 1.10–2.02), and 1.59 times more likely to have congenital syphilis (95% CI: 1.35–1.87). Children with private insurance are less likely than those with Medicaid to have any congenital infection (RR = 0.54, 95% CI: 0.43–0.66), congenital CMV (RR = 0.49, 95% CI: 0.37–0.65), or congenital syphilis (RR = 0.29, 95% CI: 0.19–0.43). Finally, children from higher income households are twice as likely to have any congenital infection (RR = 0.87, 95% CI: 0.80–0.94).

Conclusion. Risk for congenital infections in children 0–2 years in the United States is substantially higher for non-Whites, those with Medicaid insurance, and those in lower income households. Supporting previous literature suggesting that inequities disproportionately affect socially and economically disadvantaged groups. Further research is needed to define optimal cost-effective screening and prevention strategies.

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607. Group B Streptococcus Resistance to Clindamycin: Regional Antibiogram Surveillance in Los Angeles County
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Background. Intrapartum antibiotic prophylaxis (IAP) is used to prevent perinatal infection. The most effective way to prevent perinatal chlamydial infection is prenatal screening and treatment of pregnant women. These data have important implications for maternal and infant health globally.

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609. Acute Kidney Injury During Treatment with Intravenous Acyclovir (AKITA) for Suspected Neonatal Herpes Simplex Virus Infection
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Background. Intravenous (IV) acyclovir is often administered empirically in neonates with suspected herpes simplex virus (HSV) disease. Acute kidney injury (AKI) can develop within 1–2 days after starting acyclovir due to crystal nephropathy, but the epidemiology of acyclovir-associated AKI in infants is not well described. Our objective was to detail the incidence and timing of AKI among acyclovir-exposed infants.

Methods. We identified all hospitalized infants age <60 days treated with ≥1 dose of IV acyclovir for suspected or confirmed neonatal HSV disease from January 2013 to December 2015 at four US hospitals. Subjects were included if they had both a baseline (lowest value obtained before initiation of acyclovir) and follow-up serum creatinine (SCr); obtained after at least one dose of acyclovir (Day 0) through 48 hours after completion (Day 7) recorded. Infants with congenital kidney disease were excluded. We defined AKI using Kidney Disease: Improving Global Outcomes (KDIGO) criteria: ≥25% increase from baseline, or ≥0.3 mg/dL change within any 48-hour period.

Results. We identified 3,374 infants who received IV acyclovir, 1,535 of whom (45.5%) had SCr as defined for inclusion in our analyses (range 52–898 infants per hospital); 50% were white, 44% were female, and the median gestational age was 37 weeks (IQR 35 – 39). On acyclovir Day 0, the median age was 6 days (IQR 2–18), and 50.0% (n = 768) were admitted to the NICU. The median acyclovir dose was 59.5 mg/kg/12h (IQR 48.8-61.2) and the median duration of treatment was 3 days (IQR: 3–6). Thirty-two infants had confirmed HSV disease (10 CNS, 14 disseminated, and eight skin, eye, and mucous membrane disease). In all, 96 infants (6.3%) had AKI detected after acyclovir initiation including 62 (64.5%) on Day 0, 20 (20.8%) on Day 1 or 2, and 14 (14.6%) on/after Day 3. Of those with AKI on Day 1 or later, 41% (n = 14) had Stage 1 AKI (doubling of SCr or more from baseline), Seven of 32 (21.8%) infants with confirmed HSV had AKI including 4 on Day 0, 2 on Days 1–2, and 1 on Day 12.

Conclusion. The incidence of AKI among infants treated with IV acyclovir in our study was low. Most AKI was detected soon after acyclovir initiation, potentially owing to more severe illness at the start of treatment and/or drug toxicity, but AKI also developed later. SCR monitoring should be considered throughout acyclovir treatment in infants.

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