Session: 63. Maternal-Child Infections
Thursday, October 4, 2018: 12:30 PM

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 years 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 0–2 years 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 ages 0–2 years is .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 5.97 times more likely to have congenital syphilis (95% CI: 4.36–8.17). 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 less likely than those of lower income 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 these infections disproportionately affect socially and economically disadvantaged groups. Further research is needed to define optimal cost-effective screening and prevention strategies.

Disclosures. **All authors:** No reported disclosures.

607. Group B *Streptococcus* Resistance to Clindamycin: Regional Antibiogram Surveillance in Los Angeles County

Cary Krug, MPH[1]; Dawn Terashita, MD, MPH[1]; Wendy Manuel Knight, MPH[1]; Sandeep Bhaurla, MPH, CIC[1]; Joanna Felix-Mendez, HSDG[2]; Aaron Miner, BA[2]; Leah Bloomfield, BS[2]; David Ha, PharmD[3,4]; Susan Butler-Wu, PhD[5]; Priyanka Fernandes, MBBS, MPH[6]; Omai Garner, PhD, D (ABMM)[6] and James McKinnell, MD[1,2,6]; [1]Department of Public Health, Los Angeles County, Los Angeles, California, [2]Infectious Disease, LA BioMed Research Institute at Harbor-UCLA, Torrance, California, [3]Keck Graduate Institute, Claremont, California, [4]Pomona Valley Hospital Medical Center, Pomona, California, [5]Keck School of Medicine, Los Angeles, California, [6]University of California Los Angeles, Westwood, California

Session: 63. Maternal-Child Infections
Thursday, October 4, 2018: 12:30 PM

Background. Intrapartum antibiotic prophylaxis (IAP) prevents neonatal mortality from Group B *Streptococcus* (GBS). Clindamycin resistance among GBS isolates complicates IAP for GBS-positive women allergic to penicillin and cephalosporins. GBS screening by nucleic acid amplification tests (NAATs) provides rapid results, but no susceptibility data to inform IAP. We sought to estimate burden of clindamycin resistance among GBS in Los Angeles County (LAC).

Methods. Hospital antibiogram data were gathered from all LAC acute care hospitals from 2015 to 2016. Weighted averages for GBS resistance to clindamycin, erythromycin, penicillin, and TMP/SMX were calculated. Facilities which reported clindamycin susceptibilities were interviewed regarding antimicrobial susceptibility testing methods.

Results. A total of 2,339 GBS isolates from 22 hospitals were reported between 2015 and 2016. Thirteen hospitals tested GBS for clindamycin (nine reported in 2015 and 2016, four hospitals reported in 2016 only). Clindamycin resistance was found in 61.7% of 1,794 GBS isolates (79.3% of 891 in 2015, 44.3% of 903 in 2016). Erythromycin resistance was 42% in 735 isolates reported, 0.1% penicillin of 1,916 isolates reported, and 1.5% TMP/SMX of = 135 isolates reported. Facilities tested GBS by manual minimum inhibitory concentration (MIC) broth dilution (n = 1), automated MIC dilution (n = 4), agar plate diffusion (n = 1), and MIC dilution followed by agar plate diffusion (n = 1). Two hospitals did not perform testing on-site.

Conclusion. The 62% prevalence of clindamycin-resistant GBS in LAC is threefold higher than national CDC estimates and complicates IAP for GBS-positive women allergic to penicillin and cephalosporins. These data support CDC recommendations for susceptibility testing in addition to NAAT screening which does not include assays for common determinants of clindamycin resistance, *erm*-methylase, *mef*, and *isa*. There is an opportunity for diagnostic manufacturers and clinical labs to help clinicians choose appropriate IAP and prevent neonatal mortality. The CDC and public health should be aware of regional variations in clindamycin resistance. Clinicians should be aware of local resistance to inform IAP stewardship recommendations.

Disclosures. **S. Butler-Wu**, BioFire (bioMerieux): Investigator, Research support.

608. The Effect of Prenatal Screening for *Chlamydia trachomatis* (CT) on Chlamydial Conjunctivitis in Infants

Natalie Banniettis, MD[1]; Sheinese Clement, HS[2]; Margaret Hammerschlag, MD[1] and Stephan Kohlhoff, MD[1]; [1]Pediatrics, The State University of New York, Downstate Medical Center, Brooklyn, New York, [2]The City University of New York, Medgar Evers College, Brooklyn, New York

Session: 63. Maternal-Child Infections
Thursday, October 4, 2018: 12:30 PM

Background. CT was the most common identifiable infectious cause of neonatal conjunctivitis in the USA during the 20th century, accounting for 20–40% of cases. Infection is transmitted to newborns via exposure to infected mothers during passage through the birth canal. The transmission risk for an infant born vaginally to a woman with CT has been reported to be as high as 70%, including newborns with asymptomatic respiratory infection; 8–44% will develop conjunctivitis. The CDC recommended routine screening and treatment of pregnant women for CT infection in the United States in 1993. The aim of this study was to determine the impact of screening and treatment during pregnancy on chlamydial conjunctivitis in infants in our population.

Methods. A retrospective, observational study of all infant eye samples submitted to the Chlamydia Research Laboratory at SUNY Downstate Medical Center for CT culture from 1986 to 2002. Culture results were divided into two groups by time period: pre-screening (1986–1993) and post-screening (1994–2002).

Results. A total of 880 samples obtained from infants with signs and symptoms of conjunctivitis were submitted for CT culture, 103 (11.7%) were positive. The number of submitted samples and positive cultures both declined over time. The positivity rate for eye cultures was 15.6% during the pre-screening period (1986–1993) and was 1.8% during post-screening period (1994–2002) ($P < 0.0001$). A separate hospital audit confirmed >95% of pregnant women were screened during the post-screening period.

Conclusion. The prevalence of neonatal chlamydial conjunctivitis decreased significantly in our population after the implementation of routine screening and treatment of pregnant women in the United States in 1993.These results also confirm that 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.

Disclosures. **All authors:** No reported disclosures.

609. Acute Kidney Injury During Treatment with Intravenous Acyclovir (AKITA) for Suspected Neonatal Herpes Simplex Virus Infection

Brittany L. Haltzman, MS, MPH, CCRC[1]; Kevin J. Downes, MD[1]; Susan E. Coffin, MD, MPH, FSHEA, FPIDS[1]; Kellie M. Liston, MSc[1]; Hannah M. Emerson, MPH[1]; Edwin Doe, MD[2]; Rosanna Fulchiero, DO[2]; Van Tran, PharmD, BCPPS, BCPS, MBA[2]; Phuong Lieu, PharmD[3]; Lilly Yen, PharmD[3]; Sara L. Van Driest, MD, PhD[4]; Alison G. Grisso, PharmD, BCPPS[5]; Ida Aka, MPH[4]; Jennifer Hale, PharmD, MPA, BCPPS[5]; Jessica Gillon, PharmD, BCPS[5]; Julie Pingel, PharmD, BCPPS[5]; Diqiong Xie, PhD[6]; Gerold Wharton, MS[7] and Ann McMahon, MD, MPH[8]; [1]Division of Infectious Disease, Children's Hospital of Philadelphia, Philadelphia, Pennsylvania, [2]Neonatal Intensive Care, Inova Children's Hospital, Falls Church, Virginia, [3]Children's Hospital Los Angeles, Los Angeles, California, [4]Department of Pediatrics, Vanderbilt University Medical Center, Nashville, Tennessee, [5]Pharmaceutical Services, Vanderbilt University Medical Center, Nashville, Tennessee, [6]Food and Drug Administration, Silver Spring, Maryland, [7]Office of Pediatric Therapeutics, Food and Drug Administration, Silver Spring, Maryland, [8]Food and Drug Administration, Rockville, Maryland

Session: 63. Maternal-Child Infections
Thursday, October 4, 2018: 12:30 PM

Background. Intravenous (IV) acyclovir is often administered empirically in neonates with suspected herpes simplex virus (HSV) disease. Acute kidney injury (AKI) can occur 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 2011 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) recorded. Infants with congenital kidney disease were excluded. We defined AKI using Kidney Disease: Improving Global Outcomes SCr criteria: ≥50% 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/day (IQR: 55.8–61.2) 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 2 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.

Disclosures. **B. L. Haltzman**, Pfizer, Inc.: Study Coordinator, Salary. **K. J. Downes**, Pfizer, Inc.: Investigator, Research support. Merck, Inc.: Investigator, Research support. **S. E. Coffin**, Merck, Inc.: Investigator, Research support.