Abstracts of current literature on epidemiology, diagnosis, and treatment

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INFECTION AND SUDDEN UNEXPECTED DEATH IN INFANCY

In order to determine whether infection causes sudden unexpected death in infancy (SUDI), the authors performed a systematic retrospective review of SUDI autopsies completed at a specialist center (London, UK) between 1996 and 2005. Microbial isolates gathered at autopsy were classified as nonpathogens, group 1 pathogens (organisms usually associated with an identifiable focus of infection), or group 2 pathogens (organisms known to cause septicemia without an obvious focus of infection). Of 546 infants (aged 7–365 days) who died suddenly and unexpectedly, 39 autopsies were excluded because a viral or pneumocystis infection or secondary bacterial infection developed after initial collapse and resuscitation. Bacteriologic samples were available from 470 (93%) of the remaining 507 autopsies. Of the 2079 samples taken, 571 (27%) were sterile. Positive cultures yielded 2871 isolates, of which 484 (32%) were pure growth and 1024 (68%) were polymicrobial. Significantly more isolates from infants whose deaths were caused by bacterial infection (78/322 [24%]) and from those whose death was unexplained (440/2306 [19%]) contained group 2 pathogens than did those from infants whose death was due to a noninfective cause (27/243 [11%]), with a difference of 13.1% for bacterial infection (95% confidence interval [CI], 6.9–19.2; $P < 0.0001$) and 8.0% for unexplained (95% CI, 3.2–11.8; $P = 0.001$). Most organisms obtained from postmortem bacteriologic cultures in SUDI appear to be unrelated to the cause of death. However, the high rate of detection of group 2 pathogens in otherwise unexplained cases of SUDI suggests that these bacteria could be associated with this condition.

Weber MA, Klein NJ, Hartley JC, et al. Infection and sudden unexpected death in infancy: a systematic retrospective case review. Lancet 2008; 371:1848–53.

PERIOPERATIVE ANTIBACTERIAL PROPHYLAXIS AND RISK OF CLOSTRIDIUM DIFFICILE INFECTION

Researchers performed a retrospective cohort study in order to determine the risk for perioperative antibacterial prophylaxis (PAP)–induced *Clostridium difficile* infection (CDI) after selected surgical procedures (ie, abdominal hysterectomy, hip arthroplasty, craniotomy, or colon, cardiac, or vascular surgery) and to compare the risk of obtaining CDI before and after the emergence of the hypervirulent strain of *C. difficile*. All adult patients (aged ≥ 18 yr) who underwent at least 1 of the selected surgeries from August 1999 through May 2005 in a tertiary care hospital (Quebec, CA) were included. PAP was used in 7600 of the 8373 surgical procedures performed. Of 98 CDI episodes identified, 40 occurred after patients received PAP only. The risk of CDI was 14.9 cases per 1000 surgical procedures among patients who received PAP only during the period 2003 to 2005, as compared with 0.7 cases per 1000 surgical procedures during the period 1999 to 2002 ($P < 0.001$). Older age (≥ 65 yr), administration of cefoxitin (instead of cefazolin) alone or in combination with another drug, and year of surgery were independent risk factors for acquiring CDI in patients given PAP only. In a large epidemic of CDI associated with the emergence of a novel strain, 1.5% of patients who received PAP as their sole antibiotic treatment developed CDI. If PAP is being administered solely to prevent infrequent and relatively benign infections, the risks of its use may outweigh its benefits in some elderly patients.

Carignan A, Allard C, Pépin J, et al. Risk of Clostridium difficile *infection after perioperative antibacterial prophylaxis before and during an outbreak of infection due to a hypervirulent strain. Clin Infect Dis 2008; 46:1838–43.*

RACE, CKD INCIDENCE, AND PROGRESSION TO ESRD IN HIV-INFECTED PERSONS

Investigators measured chronic kidney disease (CKD) incidence, glomerular filtration rate (GFR) slope, and progression to end-stage renal disease (ESRD) in a cohort of 3332 African American and 927 white HIV-infected patients in order to determine causes of racial differences in disease. Of the 284 patients who developed CKD, 100 (35%) subsequently progressed to ESRD. African American patients were at a slightly increased risk for developing incident CKD when compared with white patients (hazard ratio [HR], 1.9 [95% CI, 1.2–2.8]). After CKD had developed, African American patients progressed more rapidly to ESRD than did white patients (HR, 17.7 [95% CI, 2.5–127.0]). Likewise, the GFR declined 6 times more rapidly in African American patients than it did in white patients ($P < 0.001$). In patients with kidney biopsy data available, African American patients were more likely to progress to ESRD than white patients, regardless of the presence of HIV-associated nephropathy (HR, 45 [95% CI, 6–343]; $P < 0.001$). Disparities between African American and white patients in HIV-related ESRD are explained by a more aggressive natural history in African American patients and less by racial differences in CKD incidence.

Lucas GM, Lau B, Atta MG, et al. Chronic kidney disease incidence, and progression to end-stage renal disease, in HIV-infected individuals: a tale of two races. J Infect Dis 2008;197:1548–57.

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