Abstracts of current literature on epidemiology, diagnosis, and treatment

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TRANSFUSION-TRANSMITTED BABESIOSIS TRENDS, 1997–2007

In order to assess trends in transfusion-transmitted babesiosis reporting since 1997 (fiscal years, 1997–2007), the authors reviewed data from 3 US Food and Drug Administration safety surveillance systems for suspected blood collection and transfusion complications (fatality reports for blood donors and transfusion recipients, the Adverse Event Reporting System, and the Biological Product Deviations Reporting System [BPDRS]). Fatality reports were analyzed for time frames, clinical presentations, and demographic characteristics of patients and donors. Reports of infected donors detected by prospective research were excluded. Since 1997, 9 deaths attributed to transfusion-transmitted babesiosis were reported; 8 of these deaths occurred within a 3-year period (2005–2007) and 1 in 1998. During this 10-year period, the BPDRS has received increasing numbers of reports of potential *Babesia* infection (with 25 reports submitted in 2007), whereas the Adverse Event Reporting System received no reports during the same period. Four implicated donors and 5 patients lived in areas where *Babesia* infection is not endemic. Both the increased number of reported deaths attributable to babesiosis and increased number of reports of possible *Babesia* infection submitted to BPDRS suggest an increasing incidence of transfusion-transmitted babesiosis. Babesiosis should be included in the differential diagnosis in immunocompromised, febrile patients with a history of recent transfusion, even in areas where *Babesia* infection is not endemic.

Gubernot DM, Lucey CT, Lee KC, et al. Babesia *infection through blood transfusions: reports received by the US Food and Drug Administration, 1997–2007. Clin Infect Dis 2009;48:25–30.*

MYCOPLASMA GENITALIUM INFECTION IN WOMEN WITH PELVIC INFLAMMATORY DISEASE

In order to examine the characteristics of *Mycoplasma genitalium* infection among women with clinically suspected pelvic inflammatory disease (PID), researchers recruited 722 women who were enrolled in the PID Evaluation and Clinical Health multicenter study and compared women who had *M. genitalium* monoinfection (n = 22) with women who had either *Neisseria gonorrhoeae* monoinfection (n = 74) or *Chlamydia trachomatis* monoinfection (n = 45). As compared with women with gonococcal PID, women with *M. genitalium* infection had fewer signs of systemic inflammation such as erythrocyte sedimentation rate exceeding 15 mm/hr (5 [22.7%] of 22 *M. genitalium* patients versus 45 [60.8%] of 74 gonococcal patients; $P = 0.002$); white blood cell count exceeding 10,000 cells/mL (4 [28.6%] of 14 *M. genitalium* patients versus 42 [64.6%] of 65 gonococcal patients; $P = 0.018$); and an oral temperature of at least 38.3°C (0 [0.0%] of 22 *M. genitalium* patients versus 10 [13.9%] of 72 gonococcal patients; $P = 0.085$). Mucopurulent cervicitis (9 [47.4%] of 19 *M. genitalium* patients versus 60 [83.3%] of 72 gonococcal patients; $P = 0.001$) and high pelvic pain score ($P = 0.014$) were less commonly seen in women with *M. genitalium* infection as compared to women with gonococcal infection. In contrast, women with chlamydial PID had signs and symptoms that were similar to those seen in women with *M. genitalium* infection. Because symptoms might be mild, women with *M. genitalium* infection might not seek treatment for PID.

Short VL, Totten PA, Ness RB, et al. Clinical presentation of Mycoplasma genitalium *infection versus* Neisseria gonorrhoeae *infection among women with pelvic inflammatory disease. Clin Infect Dis 2009;48:41–7.*

AZITHROMYCIN TREATMENT FAILURE IN NONGONOCOCCAL URETHRITIS

In order to determine why azithromycin treatment failure occurred in cases of nongonococcal urethritis caused by *M. genitalium* infection, investigators tested 7 *M. genitalium* strains isolated from men (from Australia, Norway, and Sweden; n = 12) who experienced azithromycin treatment failure for in vitro susceptibility to macrolides using a cell culture–based method. Sequencing parts of the 23S ribosomal RNA (rRNA)–gene and the genes encoding the L4 and L22 proteins was performed to determine the genetic basis of resistance. Specimens obtained before and after azithromycin treatment failure were examined using polymerase chain reaction. Minimum inhibitory concentrations exceeding 8 μg/mL for azithromycin and erythromycin were observed in the 7 isolated strains of *M. genitalium*. Three different mutations at positions 2058 and 2059 (*Escherichia coli* numbering) in region V of the 23S rRNA gene were found. Only 2 of the 9 patients with specimens obtained before and after treatment had an initial specimen in which the mutation was present, which indicates that macrolide resistance was induced by an inappropriate dosage of azithromycin. Development of macrolide resistance correlated with subsequent azithromycin treatment failure. Drug resistance was caused by mutations in region V of the 23S rRNA gene. These findings raise concern about the use of single-dose azithromycin for treatment of nongonococcal urethritis of unknown etiology.

Jensen JS, Bradshaw CS, Tabrizi SN, et al. Azithromycin treatment failure in Mycoplasma genitalium*–positive patients with nongonococcal urethritis is associated with induced macrolide resistance. Clin Infect Dis 2008;47:1546–53.*

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