when they arrived in Brazil is in agreement with previous findings of the differential distribution of TB and with a tuberculin survey on the African continent, supporting the hypothesis of native African TB (7,8). Therefore, the hypothesis of Africa as virgin soil for TB (1,9) cannot be easily supported. The incidence of TB among the slaves/Blacks in Rio de Janeiro was less than expected given their social and sanitary conditions (10), especially in a TB-endemic situation (4). Previous exposure to MTC might explain their apparent relative resistance.

Other evidence showing African contact with Europeans before the sixteenth century, supports the existence of TB in Africa (8), and TB was prevalent in urbanized centers along coastal areas of western Africa (7,8). Although some of those cases were probably the result of European contact, it is not possible to exclude that some were caused by TB native to Africa. We can affirm that persons buried in PNC, who were transported to Brazil as slaves from Africa, brought TB infection with them; whether the infection was caused by European TB endemic to Africa or by TB native to Africa is not known.

Acknowledgments

We thank the Laboratory of Molecular Biology Applied to Mycobacteria, Instituto Oswaldo Cruz (IOC)/FIOCRUZ for providing samples of Mycobacterium tuberculosis complex DNA for positive control assays. We also thank the genomics platform of Programa de Desenvolvimento Tecnológico em Insumos para Saúde/ FIOCRUZ for technical assistance.

Support for this study was provided by IOC/FIOCRUZ; a grant from Fundação de Amparo à Pesquisa do Estado do Rio de Janeiro (grants E-26/110.189/2008 and E-26/111.637/2010); and Conselho Nacional de Desenvolvimento Científico e Tecnológico fellowships (to L.H.J., grant 142260/2010-0, and to A.M.I., grant 300484/2008-9).

Lauren H. Jaeger, Sheila M.F.M. de Souza, Ondemar F. Dias, and Alena M. Iñiguez

Author affiliations: Fundação Oswaldo Cruz, Rio de Janeiro, Brazil (L.H. Jaeger, S.M.F.M. de Souza, A.M. Iñiguez); and Instituto de Arqueologia Brasileira, Belford Roxo, Brazil (O.F. Dias).

DOl: http://dx.doi.org/10.3201/eid1905.120193

References


Address for correspondence: Alena M. Iñiguez, Laboratório de Biologia de Tripanosomatídeos, Pavilhão Rocha Lima, Instituto Oswaldo Cruz, Fundação Oswaldo Cruz, Avenida Brasil 4365, Manguinhos 21045-900 Rio de Janeiro, RJ, Brazil; email: alena@ioc.fiocruz.br

Treatment of Listeriosis in First Trimester of Pregnancy

To the Editor: Foodborne infections with Listeria monocytogenes continue to be dangerous and disruptive. A 2011 outbreak in the United States, linked to cantaloupes, affected 147 persons; 33 persons died, and 1 pregnant woman experienced a miscarriage (1). Moreover, the incidence of listeriosis has been rising in several European countries (2). Compared with the general population, pregnant women are at markedly increased risk of acquiring listeriosis (3). Women who are infected with L. monocytogenes in the third trimester of pregnancy are typically treated with antimicrobial drugs until the child’s delivery (3). However, the optimal treatment regimen for listeriosis early in pregnancy is unknown.

We cared for a 28-year-old, previously healthy woman who sought treatment at 12 weeks’ gestational age with fever, headache, and neck stiffness; blood cultures were positive for L. monocytogenes. Lumbar puncture on admission to our hospital in Boston, Massachusetts, in December 2011, revealed clear fluid and an opening pressure of 15 mm Hg; 1 leukocyte was observed under high-powered field, and cultures of the cerebrospinal fluid were sterile. Pelvic ultrasound showed no abnormalities of the fetus, gestational sac, or uterus.

We treated the patient’s condition with intravenous ampicillin...
for 2 weeks, 2 g every 4 hours, and gentamicin, 100 mg every 8 hours, followed by ampicillin alone for 2 weeks. Shortly after the antimicrobial drugs were initiated, the patient defervesced and her blood cultures cleared. Her hospital course was complicated by spinal headache and transient acetaminophen-induced liver injury, but she was eventually discharged to her home in good condition. Blood cultures taken after discontinuation of antimicrobial agents were sterile, and the remainder of her pregnancy was unremarkable.

She ultimately gave birth to a healthy 2,405-g boy with Apgar scores of 4 and 7 (at 1 and 5 min, respectively) at 35.1 weeks’ gestation by spontaneous vaginal delivery. Pathologic examination of the placenta showed no evidence of cho
trioamnionitis, villitis, or parenchymal abscesses, and placental cultures were sterile. The patient and her child are currently doing well without obvious sequelae of infection.

Listeriosis in early pregnancy presents a unique challenge for the infectious diseases clinician. Up to 30% of L. monocytogenes infections in pregnancy result in stillbirth, miscarriage, or preterm labor, and approximately two thirds of surviving neonates are infected (4). L. monocytogenes uses 2 surface proteins, InlA and InlB, to invade host cells, including the placenta (5). Once established within the placenta, L. monocytogenes forms microabscesses, which can lead to recurrence of infection (6). A recent study in which researchers used a guinea pig model suggests that eradication of microabscesses from the placenta may be critical to achieving the cure of the mother and the prevention of fetal illness and death (7).

What, then, is the optimal treatment strategy to cure the mother and sterilize the placenta? In a large case series of pregnant women with listeriosis, most patients were given a b-lactam antimicrobial drug, with or without gentamicin (6). However, most women in this case series were in their third trimester of pregnancy and received treatment until delivery. In women who are infected in the first or second trimester, continuing intravenous antimicrobial drugs until delivery is impractical, and the efficacy of oral antimicrobial agents in preventing recurrence of infection is unknown.

Our case demonstrates that 4 weeks of intravenous therapy can sterilize the placenta and enable good maternal and fetal outcomes in a woman infected with listeriosis in the first trimester. We also identified 13 case reports of women in whom listeriosis developed in the first or second trimester of pregnancy (online Technical Appendix, wwwnc.cdc.gov/EID/article/19/5/12-1397-Techapp1.pdf). Among these 13 case-patients, 8 instances occurred in which both mother and neonate survived without sequelae; all 8 patients had received ampicillin/penicillin with or without gentamicin.

The role of gentamicin in treatment of listeriosis in pregnancy is controversial. The combination of ampicillin and gentamicin has been thought to be synergistic, although in vivo evidence of clinical benefit, compared to that of treatment with ampicillin alone, is lacking (3,6). A particular concern in pregnancy is gentamicin’s poor penetration into the intracellular space, where L. monocytogenes is likely to reside, in the placenta (8). Furthermore, some concern exists that gentamicin use in pregnancy could cause fetal ototoxicity, although few such cases have been reported, and several small cohort studies have not shown this association (9,10). Our patient’s child had a normal result when standard audiology testing was performed several days after delivery.

Infectious diseases clinicians will likely see patients with listeriosis in early pregnancy, given the increasing incidence of this infection in many countries and the ongoing threat of food-borne outbreaks. The collected experience from the cases reported here may be useful, particularly given the absence of high quality clinical data that support treatment recommendations for this population. Intravenous ampicillin, with or without gentamicin, effectively sterilizes the placenta and prevents maternal and fetal illness and death in cases of listeriosis in early pregnancy.

This work was supported by National Institutes of Health awards T32 AI007433 (to B.T.C.) and K08 AI081747 (to R.P.W.) and by the Cancer Research Institute-Irvington Institute Fellowship Program (to R.P.W.).

Brian T. Chan, Elizabeth Hohmann, Miriam Baron Barshak, and Read Pukkila-Worley

Author affiliations: Massachusetts General Hospital, Boston, Massachusetts, USA (B.T. Chan, E. Hohmann, M.B. Barshak, R. Pukkila-Worley); and Brigham and Women’s Hospital, Boston (B.T. Chan)

DOI: http://dx.doi.org/10.3201/eid1905.121397

References


4. Smith B, Kemp M, Ethelberg S, Schi

etymologia

Acinetobacter [asë-net’o-bak’tar]

From the Greek akeneto (immobile), a genus of gram-negative paired coccocacilli that are widely distributed in nature and can cause severe primary infections in compromised hosts. *Acinetobacter* was most likely first described as *Diplococcus mucosus* in 1908. In 1954, Briosou and Prévot proposed the genus *Acinetobacter* to indicate that the bacteria were nonmotile because they lacked flagella. *Acinetobacter* are still generally described as nonmotile, but most isolates exhibit “twitching” motility. *Acinetobacter baumannii*—named in honor of American bacteriologists Paul and Linda Baumann—is a nosocomial pathogen with acquired multidrug resistance that is emerging as a major concern worldwide. Motility is linked to increased virulence in bacteria such as *Pseudomonas aeruginosa* and *Dichelobacter nodosus*; however, whether motility plays a role in the virulence of *A. baumannii* remains unclear.

Sources

2. Bouvet PJ, Grimont PA. Taxonomy of the genus *Acinetobacter* with the recognition of *Acinetobacter baumannii* sp. nov., *Acinetobacter haemolyticus* sp. nov., *Acinetobacter johnsonii* sp. nov., and *Acinetobacter junii* sp. nov. and emended descriptions of *Acinetobacter calcoaceticus* and *Acinetobacter lwoffi*. Int J Syst Bacteriol. 1986;36:228–40. http://dx.doi.org/10.1099/00207713-36-2-228

Address for correspondence: Ronnie Henry, Centers for Disease Control and Prevention, 1600 Clifton Rd NE, Mailstop E03, Atlanta, GA 30333, USA; email: boq3@cdc.gov

DOI: http://dx.doi.org/10.3201/eid1905.ET1905
Treatment of Listeriosis in First Trimester of Pregnancy

Technical Appendix

Table. Studies describing treatment of listeriosis in pregnant women in first and second trimesters, 1961–2002*

<table>
<thead>
<tr>
<th>GA of fetus</th>
<th>Treatment</th>
<th>Outcome</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bacteremic/6 mo</td>
<td>OXT × 1 wk</td>
<td>Preterm delivery; neonate with Listeria meningitis</td>
<td>Hood, 1961</td>
</tr>
<tr>
<td>Bacteremic/26 wk</td>
<td>AMP × 2 wk</td>
<td>Uncomplicated delivery (induced, 41.5 wk); neonate unaffected</td>
<td>Fleming et al., 1985</td>
</tr>
<tr>
<td>Bacteremic/20 wk</td>
<td>PEN × 9 d</td>
<td>Uncomplicated term delivery; neonate unaffected</td>
<td>Zervoudakis and Cederqvist, 1977</td>
</tr>
<tr>
<td>Bacteremic/19 wk</td>
<td>AMP × 3 wk</td>
<td>Uncomplicated term delivery; neonate unaffected</td>
<td>Hume, 1976</td>
</tr>
<tr>
<td>Bacteremic/18 wk</td>
<td>AMP × 2 wk; then PO AMX</td>
<td>Uncomplicated delivery at 39 wk; neonate unaffected</td>
<td>Benshushan et al., 2002</td>
</tr>
<tr>
<td>Bacteremic/18 wk</td>
<td>AMP × 7 d, GEN × 2 d; then PO AMX</td>
<td>Diagnosed postpartum; preterm delivery with fetal demise; mother recovered</td>
<td>Mylonakis et al., 2002</td>
</tr>
<tr>
<td>Bacteremic/15 wk</td>
<td>AMP × 3 wk</td>
<td>Uncomplicated delivery at 39 wk by C-section; neonate unaffected</td>
<td>Benshushan et al., 2002</td>
</tr>
<tr>
<td>Bacteremic/13 wk</td>
<td>AMP, and GEN × 3 wk</td>
<td>Uncomplicated delivery at 36 wk; neonate unaffected</td>
<td>Fuchs et al., 1994</td>
</tr>
<tr>
<td>Bacteremic/13 wk</td>
<td>AMP × 10 d, GEN × 6 d, then TMP/SXT × 10 d</td>
<td>Uncomplicated term delivery; neonate unaffected</td>
<td>Cruikshank and Warenksi, 1989</td>
</tr>
<tr>
<td>Bacteremic/12 wk</td>
<td>AMP and GEN × 2 wk, then AMP × 2 wk</td>
<td>Uncomplicated delivery at 35 wk; neonate unaffected</td>
<td>This study</td>
</tr>
<tr>
<td>+ Uterine culture/27 wk</td>
<td>AMP and GEN × 2 d, then ERY × 1 wk</td>
<td>Preterm delivery; neonate unaffected</td>
<td>Mylonakis et al., 2002</td>
</tr>
<tr>
<td>+ Uterine culture/18 wk</td>
<td>CLI and GEN</td>
<td>Sought treatment with contractions and fever; spontaneous abortion resulted; acute chorioamnionitis noted</td>
<td>Benshushan et al., 2002</td>
</tr>
<tr>
<td>+ Uterine culture/17 wk</td>
<td>AMP and GEN × 2 d, then PO AMP × 14 d</td>
<td>Sought treatment with spontaneous abortion and fetal demise; mother recovered</td>
<td>Mylonakis et al., 2002</td>
</tr>
<tr>
<td>+ D&amp;C culture/17 wk</td>
<td>PEN and CVA, followed by AMP, CLI, and GEN</td>
<td>Sought treatment with fever and missed abortion; D&amp;C performed</td>
<td>Benshushan et al., 2002</td>
</tr>
</tbody>
</table>

*GA, gestational age; OXT, oxytetracycline; AMP, ampicillin; C-section, cesarean section; PEN, penicillin; AMX, amoxicillin; PO, per os (by mouth); GEN, gentamicin; TMP/SXT, trimethoprim/sulfamethoxazole; ERY, erythromycin; CLI, clindamycin; D&C, dilatation and curettage; CVA, clavulanic acid.

References


Fleming AD, Ehrlich DW, Miller NA, Monif GR. Successful treatment of maternal septicemia due to 


