COMMENTARY

To Screen or Not to Screen?
Bacterial Vaginosis in Pregnancy

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Bacterial vaginosis (BV) is a common condition in which the normal vaginal microflora are replaced by a polymicrobial overgrowth that includes *Gardnerella vaginalis*, *Mycoplasma hominis*, and anaerobes such as nonfragilis bacteroides, Mobiluncus, and peptococcus.[1] It is now clear that bacterial vaginosis is associated with an increased risk of preterm delivery. The meta-analysis by Flynn and colleagues[2] shows that the odds ratio of preterm delivery for patients with bacterial vaginosis during pregnancy is approximately 1.6. Although this is not an enormous increase in risk, it is both statistically and clinically significant. However, the association between BV and preterm delivery does not prove causation or that prenatal screening and treatment will improve perinatal outcomes.

Flynn and coworkers have carried out a detailed and thoughtful meta-analysis, but even a meta-analysis cannot overcome the inherent biases of the underlying case control and cohort studies which can not prove cause and effect. The Heart and Estrogen/Progestin Replacement Study[3] demonstrated this phenomenon. That randomized placebo-controlled trial of postmenopausal hormone replacement therapy did not show the reduction in cardiac events that had been expected on the basis of previous observational case control and cohort data.

In the context of preterm delivery, BV is undoubtedly only a marker for something else — presumably subclinical infection of the upper genital tract — which then leads to preterm delivery. The combination of microbes that causes the upper tract infection is not known for certain. Studies in which the authors attempted to treat BV during pregnancy have shown no improvement in perinatal outcomes with oral amoxicillin[4] or topical vaginal clindamycin,[5,6] supporting the theory that anaerobic infection of the upper genital tract is the link between BV and preterm delivery. Spontaneous regression of BV during pregnancy assessed by vaginal gram stain also does not appear to improve perinatal outcomes,[7] which again supports the idea that BV is not the direct cause of preterm delivery.

Three studies have shown that prenatal screening and treatment with oral metronidazole or with metronidazole and erythromycin seems to reduce the incidence of preterm delivery in patients with BV who also have other risk factors, such as a history of a previous preterm delivery or a low prepregnancy weight.[8-10] However, 2 of these studies were weakened by methodologic problems[8,10] or small sample numbers.[10] These results need to be replicated in larger studies of high-risk women before they can be considered reliable. The case for screening and treating low-risk women is even less clear.

If BV is a marker for something else, would treating all high-risk women, even those without BV, also reduce the number of preterm deliveries? Hauth and colleagues[9] included an active control group of 358 high-risk women without BV and found that antibiotics did not improve perinatal outcomes in those patients.

As Flynn and colleagues point out, their result is disease-oriented evidence. Even those studies showing that antibiotic treatment reduces preterm deliveries do not address the ultimate patient-oriented outcome: the conditions of the neonate and the mother after delivery. Animal research has shown that using antibiotics to delay delivery in the presence of chorioamnionitis leads to fetal brain damage.[11] Although women with BV do not have clinical chorioamnionitis, this data strikes a cautionary chord, suggesting that prolonging pregnancy in the setting of infection may not be the wisest course. Future studies must include data on the neonatal and postpartum sequelae of antibiotic treatment before we can be sure that we understand what the risks of treatment are and that they are outweighed by proven benefits.

CURRENT GUIDELINES

The American College of Obstetricians and Gynecologists suggests that screening for BV may be appropriate in women with a history of preterm delivery or who weigh less than 50 kg before pregnancy.[12,13] Screening of other patients is not warranted on the basis of current evidence. BV can be treated after the first trimester with metronidazole 250 mg orally 3 times daily for 7 days or 500 mg orally twice daily for 7 days. Alternatives include metronidazole 2 g orally in a single dose or clindamycin 300 mg orally twice daily for 7 days. During the first trimester, symptomatic BV should be treated with clindamycin cream 2%, one applicatorful (5 g) intravaginally at bedtime for 7 days. Clindamycin can also be used orally during the first trimester, but the cream is preferred to minimize fetal exposure. However, vaginal therapy is not effective in preventing preterm delivery. There is no data regarding repeated screening or retreatment of persistent or recurrent BV.[12-14]

For now, physicians should consider prenatal screening and treatment for BV at the end of the second or the beginning of the third trimester in women with a history of preterm delivery (before 37 weeks' gestation) or who weighed less than 50 kg before pregnancy. For all other patients, until larger, better-randomized studies of antibiotic treatment of BV during pregnancy become available, the answer to the question of screening remains "No."

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REFERENCES

1. Gilbert D, Moellering R, Sande M. The Sanford guide to antimicrobial therapy. Vienna, Va: Antimicrobial Therapy, Inc; 1998.
2. Flynn CA, Helwig AL, Meurer LN. Bacterial vaginosis in pregnancy and the risk of prematurity: a meta-analysis. J Fam Pract 1999; 48:885-892.
3. Hulley S, Grady D, Bush T, et al. Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women: Heart and Estrogen/progestin Replacement Study (HERS). JAMA 1998; 280:605-13.
4. Duff P, Lee M, Hillier S, Herd L, Krohn M, Eschenbach D. Amoxicillin treatment of bacterial vaginosis during pregnancy. Obstet Gynecol 1991; 77:431-5.
5. McGregor J, French J, Jones W, et al. Bacterial vaginosis is associated with prematurity and vaginal fluid mucinase and sialidase: results of a controlled trial of topical clindamycin cream. Am J Obstet Gynecol 1994; 170:1048-60.
6. Joesof M, Hillier S, Wiknjosastro G, et al. Intravaginal clindamycin treatment for bacterial vaginosis: effects on preterm delivery and low birth weight. Am J Obstet Gynecol 1995; 173:1527-31.
7. Gratacos E, Figueras F, Barranco M, et al. Spontaneous recovery of bacterial vaginosis during pregnancy is not associated with an improved perinatal outcome. Acta Obstet Gynecol Scand 1998; 77:37-40.
8. Morales W, Schorr S, Albritton J. Effect of metronidazole in patients with preterm birth in preceding pregnancy and bacterial vaginosis: a placebo controlled double-blind study. Am J Obstet Gynecol 1994; 171:345-9.
9. Hauth J, Goldenberg R, Andrews W, DuBard M, Copper R. Reduced incidence of preterm delivery with metronidazole and erythromycin in women with bacterial vaginosis. N Engl J Med 1995; 333:1732-6.
10. McDonald H, O'Loughlin J, Vigneswaran R, et al. Impact of metronidazole therapy on preterm birth in women with bacterial vaginosis flora (Gardnerella vaginalis): a randomised, placebo controlled trial. Brit J Obstet Gynecol 1997; 104:1391-7.
11. Brocklehurst P. Infection and preterm delivery. BMJ 1999; 318:548-9.
12. Committee on Obstetric Practice. Bacterial vaginosis screening for prevention of preterm delivery: ACOG committee opinion. Washington, DC: American College of Obstetricians and Gynecologists; 1998.
13. Committee on Educational Bulletins. Antimicrobial therapy for obstetric patients: ACOG educational bulletin. Washington, DC: American College of Obstetricians and Gynecologists; 1998.
14. Centers for Disease Control and Prevention. 1998 guidelines for treatment of sexually transmitted diseases. MMWR 1998; 47:1-116.