A woman with a postpartum genital tract infection

Commentary by
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A woman presenting nine weeks after a caesarean section is afrebile but has a tender uterus on examination. A vaginal swab is positive for *Haemophilus influenzae*. How should she be managed?

Case scenario

Rebecca is 33 years of age and presents nine weeks postpartum with her third child, who was born, as were her other two babies, by caesarean section. She is breastfeeding successfully, and her vaginal bleeding and discharge had ceased by five weeks after the birth. Rebecca has not yet resumed sexual activity. Over the few days before her consultation she had a recurrence of blood-tinged vaginal discharge associated with what she described as a 'niggling and intermittent discomfort' in her lower abdomen. Rebecca feels tired, but is afebrile. Bimanual palpation reveals a tender uterus, and a swab of her vaginal discharge produces a heavy and unexpected growth of *Haemophilus influenzae*.

Is Rebecca likely to have acquired this infection at the time of the caesarean section? What is the best way to manage her infection?

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Commentary

Haemophilus influenzae is a Gram-negative coccobacillus, some strains of which have a polysaccharide capsule, which is an important virulence factor. *H. influenzae* type b (Hib) is a capsulated type that can cause severe infections such as bacterial meningitis, particularly in young children. With the availability of Hib vaccination, Hib infections have become rare; however, colonisation and infection (mainly respiratory) with noncapsulated *H. influenzae* are not prevented by the vaccine.[1,2]

H. influenzae in the female genital tract

Colonisation of the vaginal mucous membranes by noncapsulated *H. influenzae* is not uncommon in prepubertal girls and postmenopausal women,[3] but it is uncommon in women of reproductive age.

The normal female genital tract is endowed with many commensal organisms – most importantly, lactobacilli. Lactobacilli produce hydrogen peroxide, which renders the vaginal environment acidic and thereby protects against the growth of many pathogens.[4]

Breastfeeding women such as Rebecca have low endogenous oestrogen levels, similar to those of prepubertal girls and postmenopausal women. The vaginal environment becomes atrophic and lactobacillus numbers decrease so the production of hydrogen peroxide is reduced; thus, the vaginal environment becomes more alkaline and therefore at risk of 'replicative dominance' by opportunistic pathogens such as noncapsulated *H. influenzae*.[4]

H. influenzae infection of the female genital tract is uncommon, but the consequences can be serious when it happens. *H. influenzae*

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has been reported as the causative organism in Bartholin's abscess, vulvovaginitis, endometritis, salpingitis and tubo-ovarian abscess. In pregnancy, it has been linked with severe infections causing septic miscarriages, preterm delivery, neonatal death and neonatal infections.

*H. influenza*e infection in pregnancy

Most laboratories do not routinely report the presence of *Haemophilus* spp. in vaginal swabs unless it is a heavy pure growth of the organism, as in Rebecca's case. The prevalence of colonisation by vaginal *H. influenzae* is therefore difficult to assess. Figures ranging from 1–2 per 1000 to 9% have been reported among pregnant women.[1,2]

In a very large population-based study, the incidence of laboratory-confirmed *H. influenzae* infection (including bacteraemia, pneumonia and pelvic infections) was found to be low, at 0.50 per 100,000 women of reproductive age (15 to 44 years) in England and Wales.[2] Most *H. influenzae* infections occurred in women with pre-existing chronic medical conditions, such as chronic respiratory diseases, malignancy or immunosuppression. However, in healthy pregnant women there was a sixfold higher incidence than among the nonpregnant population.[2] Patients in whom infection was identified during pregnancy experienced pregnancy loss or extreme preterm delivery in 100% of cases if infection occurred during the first 24 weeks of gestation.[2] Overall, the fetal and neonatal fatality rate was 62.3%.[2] Among babies born alive, 80.8% had respiratory distress, sepsis or both.[2] No maternal mortality was reported in this group of patients, but infection was obviously associated with significant morbidity.

Thus, *H. influenzae* is thought to be present uncommonly in the reproductive tract of pregnant women, yet when it is present it often causes significant infections in both the mother and the fetus. This is in contrast to group B streptococcus, which often colonises the reproductive tract during pregnancy but only occasionally causes invasive disease in newborn infants.

Caesarean section-related infection

Most caesarean section-related infections occur within six weeks of the operation. The causative organisms are mostly commensal organisms of the skin, bowel or genitourinary tract. The most common type of infection is urinary tract infection, followed by superficial cellulitis. More seriously, a pelvic collection can occur or a rectus sheath haematoma may become infected. These infections usually present early, classically in the first week after the operation, so are usually managed while the woman is still in hospital.

Endometritis, on the other hand, is usually a low-grade grumbling infection with subtle symptoms and signs that can be overlooked initially. Even so, patients usually present within six weeks after the birth. Had Rebecca had a low-grade infection, since the operation she would most likely have had persistent vaginal bleeding that became heavier. It may have been associated with lower abdominal discomfort over the uterus, and possibly backache. This scenario is not consistent with Rebecca's history, however.

Localised endometritis-related tenderness can sometimes be difficult to distinguish from post-caesarean abdominal tenderness. However, postoperative tenderness tends to be more marked over the wound; endometritis-related tenderness is more localised to the uterus, which is often exquisitely tender on palpation. Women sometimes report smelly bleeding or discharge. As these women tend to be young and healthy, they can look reasonably well despite significant endometritis and have only subtle systemic symptoms until sepsis sets in. Hence, it is important to recognise the mild symptoms and to act promptly.

Management

Rebecca could have been asymptomatically colonised with *H. influenzae* during pregnancy or have become colonised postpartum. It is possible, but unlikely, that she became colonised during the caesarean section. Rebecca was quite well; her bleeding had settled by five weeks postpartum only to recur a few days before presentation at nine weeks postpartum. It is most likely that pre-existing vaginal colonisation developed into overt infection because of her hypo-oestrogenic state due to breastfeeding.

Endometritis can cause serious damage (such as salpingitis, tubo-ovarian abscess or septicaemia), and *H. influenzae* is a particularly virulent infection. Although the organism was only isolated from the vagina, the presence of recurrent vaginal bleeding and lower abdominal discomfort is enough to allow the assumption of *H. influenzae* endometritis.

Rebecca needs further assessment to clarify the optimal treatment regimen. The antibiotic susceptibility of the causative organism is as critical to treatment success as is the severity of infection, and Rebecca should be asked specifically whether she has experienced fever and rigors. A white-cell count and C-reactive protein (CRP) estimation are needed. Pelvic ultrasound is also advisable.

The presence of fever and rigor or significant white-cell elevation warrants referral of the patient to a specialist and hospitalisation for treatment with intravenous antibiotics. CRP estimation is a useful tool for monitoring treatment response.

Pelvic ultrasound is useful for excluding pelvic collections. It should be stressed that the ultrasound is likely to show the endometrial cavity filled with blood and inflammatory exudates due to endometritis. This can be reported as 'possible retained products'; however, these are unlikely to be retained products because Rebecca has had a caesarean section, and proper technique calls for clearing all visible placental tissue and membranes at the operation. Therefore, a recommendation for dilatation and curettage should not be rushed into on the basis of such a finding.

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Additionally, from the case report we can probably assume that the baby was well. Considering the high likelihood of vertical transmission of *H. influenzae* in pregnancy, the fact the baby was well strengthens the assumption that the infection developed postpartum, with or without pre-existing colonisation.

Treatment

Most *H. influenzae* strains are sensitive to antibiotics such as erythromycin, azithromycin, ampicillin, amoxicillin–clavulanate, trimethoprim and third-generation cephalosporins. Rebecca should be treated in line with the bacterial sensitivity report from the diagnostic laboratory.

If outpatient treatment with oral antibiotics is appropriate, Rebecca must be treated with an appropriate dose for at least 10 to 14 days, rather than just one course of five to seven days.

In addition to symptomatic improvement within 48 hours, daily measurement of white-cell counts and CRP levels can be used initially to ensure Rebecca is responding to treatment.

Conclusion

H. influenzae is an unusual pathogen of the female genital tract. In Rebecca's case, it is unlikely that colonisation was acquired during the caesarean section, but more likely that the onset of symptomatic infection with *H. influenzae* was related to pre-existing colonisation and her postpartum hypo-oestrogenic breastfeeding state.

Women with postpartum endometritis can appear deceptively well until systemic sepsis sets in – and *H. influenzae* is a virulent infection. A high index of suspicion, prompt treatment and timely follow up are required to avoid serious sequelae. MT

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