Uterine Anomalies, Induction of Labor, and Uterine Rupture

In this issue, Drs. Samuels and Awonuga examine a case of uterine rupture associated with a second-trimester pregnancy within a congenital rudimentary uterine horn after administration of misoprostol. It is important because it emphasizes 1) the increased dangers associated with pregnancies that reside within congenital rudimentary uterine horns, 2) the importance of identifying uterine anomalies in gravid women, and 3) the potentially lethal dangers of misoprostol use during pregnancy.

Uterine rupture in pregnancy is associated with a high rate of fetal and maternal morbidity and mortality. Fortunately, the normal, unscarred uterus ruptures infrequently. In a study by Gardeil et al, the overall rate of unscarred uterine rupture during pregnancy was 1 in 30,764 (0.0033%). No case of uterine rupture occurred among 21,998 primigravidas, and only 2 occurred among 39,529 multigravidas without a prior uterine scar (0.0051%).

Several factors increase the risk of uterine rupture during pregnancy. Aside from the well-known risk attributable to preexisting uterine scars, the following factors are also associated with a higher rate of uterine rupture: grand multiparity, malpresentation, breech extraction, uterine instrumentation, uterine trauma, dystocia, fetal macrosomia, labor induction, and the presence of a uterine anomaly.

The first issue pertains to the risk of uterine rupture associated with pregnancies occupying anomalous uteri. Congenital uterine malformations complicate 1:594 pregnancies. Importantly, the walls of congenitally abnormal uteri are thinner than for normal uteri. Moreover, their myometrium tends to diminish in thickness as gestation advances and can be inconsistent over different aspects of the uterus. Furthermore, additional wall thinning can occur as a result of uterine contractions.

Unicornuate uteri constitute 5% of uterine malformations. However, most of these so-called “unicornuate” uteri possess a rudimentary horn of contralateral müllerian origin (74%). More than 50% of these horns are cavitary, and the majority are noncommunicating (72%–85%), meaning that they contain an endometrial cavity with no communication to the cervix, vagina, or the contralateral hemiuterus (Fig. 1). All pregnancies occupying such “blind” rudimentary uterine horns result from transperitoneal migration of sperm or fertilized ova, which typically occurs in 50% of human pregnancies. Because these horns have no cervical or vaginal outlet, they have an unusual propensity to rupture.

Rudimentary uterine horn pregnancies occur in 1:76,000 gestations. Of these, 85% occupy noncommunicating horns. Even for the 15% with connection to the cervix, there is an unacceptably high rate of uterine rupture. During the 20th century, 47% of reported noncommunicating uterine horn pregnancies ruptured (358 of 419 cases), as did 52% of communicating rudimentary horns (29 of 56 cases) (no significant differ-
The relationship of prostaglandin use to uterine rupture has been studied primarily in women with prior cesarean deliveries. In this population, Lydon-Rochelle et al. reported a 15.6-fold increased risk of uterine rupture when prostaglandins were used for labor induction compared with controls (95% confidence interval 8.1–30.0). Taylor et al. found a uterine rupture rate of 5.2% in women with a single prior cesarean delivery who received PGE2 alone for labor induction, compared with 1.1% in women who did not. Ravasia et al. reported a 1.7% uterine rupture rate for women who had their labor induced with prostaglandin E2 (PGE2) alone, compared with 0.45% in those having spontaneous labor. In contrast, Flamm et al. found that women treated with PGE2 in combination with oxytocin had a uterine rupture rate of 1.3%, which was not significantly higher than for controls (0.7%).

For pregnancies within noncommunicating rudimentary uterine horns, there is no possibility of vaginal delivery. Under these circumstances, the induction or augmentation of uterine contractions has no potential benefit, and uterine activity may precipitate uterine wall strain that can cause accelerated myometrial thinning and potential rupture. Because of this, when a fetus attains prospective extrauterine viability, previous management recommendations have included the administration of tocolytic agents. This strategy, which is intended to minimize uterine contractility, has precisely the opposite effect as agents such as prostaglandins and oxytocin. In many instances, reports of uterine rupture involving congenitally abnormal uteri have occurred after pharmacologic interventions that were meant to either induce or augment uterine contractions. Regardless of the extent of uterine activity, a sonographically estimated uterine wall thickness of less than 5 mm in any aspect during pregnancy should be cause for immediate concern, because this may portend impending uterine rupture. Because of these considerations, elective hysterotomy is the preferred mode of delivery for all types of rudimentary horn pregnancies, in addition to those that occupy other types of major uterine malformations.

In summary, for women who have major uterine anomalies, there is an increased risk of uterine rupture during pregnancy and this risk is typically aggravated by uterine contractions. Therefore, it is incumbent upon obstetric practitioners to make an informed determination that a major uterine anomaly does not exist before inducing or augmenting labor. This recommendation applies regardless of whether a fetus is viable and regardless of whether a live birth or a termination of pregnancy is the goal. This situation is maximally exacerbated in the case of rudimentary horn pregnancies, where the uterine rupture rate is 50%.
Given these considerations, a set of practical guidelines for managing women with congenitally abnormal uteri can be suggested:

If a major asymmetrical uterine malformation is identified in a nongravid patient, either elective surgical removal of the minor horn or metroplasty is indicated before a pregnancy occurs.

If a pregnancy is identified within a rudimentary uterine horn before the mid second-trimester, it should be terminated upon recognition (Nahum GG. Medical termination of early rudimentary horn pregnancy [letter-reply]. J Reprod Med 2002;47:878–80).

If a desired pregnancy is advanced beyond the mid second-trimester within a rudimentary uterine horn, the clinical strategy in nonemergency situations should be

To minimize uterine activity, with the administration of tocolytic agents as appropriate,
To avoid the use of prostaglandins, oxytocin, and other uterine stimulants, and
To deliver by elective hysterotomy as soon as sufficient fetal maturity is attained or when the uterine wall thickness decreases to less than 5 mm on pelvic imaging studies, whichever occurs first.

If a desired pregnancy occupies an incompletely characterized anomalous uterus, the clinical strategy in nonemergency situations should be

To define the type of uterine anomaly to the best extent possible by reviewing all medical records and appropriate diagnostic studies and
To assume that the risk of uterine rupture is high unless the anomaly can be definitively identified as a minor malformation of low-risk type (e.g., a subseptate uterus), and employ the same management strategy as outlined above.

In cases of pregnancies that reside within major uterine malformations, proceeding otherwise invites an increased risk of catastrophic uterine rupture together with all of its major morbidities and an increased risk of maternal and fetal death.

REFERENCES