

# Unscarred uterine rupture after induction of labor with misoprostol: A case report

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## Summary

The rupture of an unscarred uterus is very rare and presents an emergency situation that threatens the life of the fetus and mother. The agents used for induction of labor, like oxytocin and/or prostaglandins, can be responsible for this catastrophic event.

We report a case of intrapartum rupture of an intact uterus after using intravaginal misoprostol for cervical ripening and labor induction in a term pregnancy and we discuss the other cases reported in the literature.

*Key words:* Misoprostol; Rupture of unscarred uterus.

## Introduction

Prostaglandins have been shown to induce cervical ripening and stimulate uterine contractions. It has been shown in numerous clinical trials that they are effective at a variety of doses regardless of the route of administration. Misoprostol, prostaglandin E<sub>1</sub>, is much less expensive than dinoprostone although both are synthetic prostaglandin analogues. The administration of misoprostol is also very easy. Although not licensed, the use of misoprostol for cervical ripening and induction of labor has been increasing during the last decade. Recent studies have demonstrated that misoprostol shortens the time from induction-to-delivery and reduces the need for oxytocin augmentation [1]. Although disclaimed by some authors, increased incidence of dose-dependent tachysystole and a nonreassuring fetal heart rate (FHR) are the most important side-effects of misoprostol when its side-effect profile is compared to other prostaglandin analogues [2, 3]. The effects of misoprostol in patients with previous caesarean section were studied and the results showed that misoprostol increases the possibility of uterine rupture when compared to other induction agents like other prostaglandins and oxytocin [4].

Uterine rupture is an emergency situation that threatens the life of the fetus and mother. The reported incidence of this catastrophic event is 0.03-0.08% among all pregnant women [5]. A history of myomectomy, caesarean section, uterine perforation or any previous trauma to the uterus poses an important risk for uterine rupture during induction of labor regardless of the agent used. However, the rupture of an unscarred uterus is a very rare condition and its incidence has been declining [6]. Modifications in obstetric management of patients with fetal macrosomia and malpresentations have yielded better outcomes throughout the years. In the literature, there are only three cases of uterine ruptures caused by misoprostol during induction of labor. In this article we present a case of intrapartum rupture of an intact uterus after using intravaginal misoprostol for cervical ripening and labor induction in a term pregnancy.

## Case

A 30-year-old healthy women at 42 weeks of gestation was admitted to the hospital for induction of labor because of a low amniotic fluid index (4.3 cm) and postmaturity. Her obstetric history, apart from two previous term pregnancies, was unremarkable. The initial non-stress test (NST) was reactive.

The patient was included in a randomized study evaluating the effect and dose regimen of misoprostol. The initial Bishop's score was two (1-2 cm dilated, uneffaced, centralized cervix and an unengaged fetal head) at vaginal examination on admission. The first 25 µg misoprostol tablet was applied to the posterior vaginal fornix and tablets were to be repeated at 4-hour intervals for a maximum of three times according to the study protocol. The first dose of misoprostol stimulated only mild irregular uterine contractions. Three hours and forty-five minutes after the second dose of misoprostol, hyperstimulation was noted. Fetal monitoring revealed severe and moderately variable decelerations with normal variability. The cervix was 2 cm dilated and 40% effaced, and the vertex was at -2 station. Misoprostol was withheld because of tachysystole and variable decelerations; the patient was turned on her left side and oxygen was administered. Tachysystole persisted in spite of this intervention, so a tocolytic agent, intravenous ritodrin, was administered (terbutalin is not commercially available in our country). Tocolysis was withdrawn after 45 minutes when atypical variable decelerations were decreased. However, tachysystole persisted for the next hour. The fetal heart rate remained stable during this period of time. Meanwhile, the cervix was 4 cm dilated and 50% effaced and the vertex was at -2 station. Augmentation was begun with oxytocin (2mU/min) since irregular and rare contractions were observed. Oxytocin was increased at doses of 2 mU/min. until effective contractions were achieved and the maximum dose of oxytocin was to be below 8 mU/min. When the cervix was 5 cm dilated, 60% effaced and the vertex was at -2 station, bright red vaginal bleeding of about 80 ml occurred, lasting for 40 minutes. Neither were there any tetanic contractions of the uterus which could have indicated placental abruption nor any problems concerning intrapartum monitoring, so the bleeding was determined to be the result of dilation. Manual hematocrit was measured as 32%. When the cervix was 7 cm dilated, 60-70% effaced and the vertex was at -2 station, vaginal bleeding restarted and lasted for 20 minutes. During bleeding, FHR remained stable and the patient was followed-up closely. The cervix was 10 cm dilated and the vertex at +1 station 19 hours after the first misoprostol administration. The

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fetus developed bradycardia and vacuum extraction was performed. A 3,630 g infant was delivered with Apgar scores of 4 and 7 for 1 and 5 minutes, respectively. Arterial cord blood pH was 6.926. After delivery of the placenta, the patient was taken to the recovery room for postpartum monitoring where she developed tachycardia and cyanosis after just 30 minutes. Speculum and bimanual examination revealed no lacerations in the vagina and/or bleeding; the uterus was contracted but very mobile, and palpated above its normal localization. However, transabdominal ultrasonography exposed echodense images in the retroperitoneal area suggesting a hematoma. When the patient was taken into the emergency operating room, she developed cardiac arrest. After resuscitation for a couple of minutes, the patient recovered and laparotomy was performed just 40 minutes after delivery. A hematoma of about 2,500-3,000 cc localized in the retroperitoneal area was encountered. A 9 cm linear defect extending from the right isthmus of the cervix to the fundus of the uterus was noted (Figure 1). The right uterine artery was involved within this defect, but the serosa was intact. Hysterectomy was performed, and the right hypogastric artery was ligated. Nine units of whole blood and 8 units of fresh frozen plasma were transfused. Eight days after the operation the patient and her infant were discharged from the hospital in good condition.



Figure 1. — Right lateral view of the hysterectomy specimen showing the rupture site.

## Discussion

The rupture of an intact uterus is a rare condition that can be seen any time during pregnancy or during spontaneous and induced labor. It is an obstetric emergency that threatens the life of the mother and fetus. The reported

incidence of this catastrophic event is 0.03-0.08% among all pregnant women [5]. Research has revealed the risk factors as grand multiparity, fetal macrosomia, fetopelvic disproportion, induced labor with oxytocin or prostaglandin (PG), difficult forceps applications, and intrauterine obstetric manipulation. However, these studies are retrospective, and do not reflect the conclusive role of the studied conditions as a cause of uterine rupture.

The resultant data revealed that the agents used for induction of labor like oxytocin and/or prostaglandins may be responsible for this catastrophic condition. Therefore, currently the induction of labor with these agents is accepted as a risk factor of uterine rupture. A prostaglandin E<sub>1</sub> analogue, misoprostol, initially developed and licensed for other medical indications, became a very popular and effective drug for induction of labor during the last decade. However, it should be accepted as a risk factor contributing to uterine rupture under the influence of the data discussed above. Articles about the relationship between misoprostol use and uterine rupture have been published in the last two years. Most of them indicated the higher risk of uterine rupture development in patients with a history of previous caesarean section. However, there are three cases of uterine rupture without a history of previous caesarean section in the literature.

The first case, a 34-year-old woman, was published by Bennett in 1997. Her obstetric history included three term vaginal deliveries followed by a D&C for spontaneous first trimester abortion. She was induced with 25 µg misoprostol and uterine rupture developed. It was indicated that the fetus had developed fetal bradycardia after the second dose of misoprostol thus caesarean section was performed. A 15 cm linear rupture of the left posterior uterine wall was observed. A third dose of misoprostol was withdrawn because of tachysystole. Bennett discussed the possibility of some unknown myometrial damage or perforation during the curettage of previous abortion material [7].

The second case was discussed by Blanchette *et al.* in an article [8]. The aim of the study was to compare the safety of misoprostol to dinoprostone. The authors indicated that misoprostol could have been responsible for uterine rupture in one of their cases. With this case, three other cases were presented in the same article. In these latter three cases previous caesarean section was suggested as a reason for uterine rupture. The presented case was a 39-year-old woman who had had three previous normal deliveries. Misoprostol was administered at a dose of 25 µg twice, and the third dose was 50 µg administered 7 1/2 h before the uterine rupture developed. A macrosomic infant (9 lb, 14 oz) was delivered in spite of shoulder dystocia. The placenta was not delivered spontaneously and the retained placenta was manually recovered. During this maneuver it was noted that the uterus was ruptured. A round table discussion concluded the article, and it was stated that uterine rupture could have developed because of the shoulder dystocia. We suggest that it also could have developed during the maneuver to deliver the placenta manually. The length of the rupture and whether it included peritoneum was not described.

The third case report [9] was published by Mathews and colleagues. Uterine rupture had occurred in a multiparous patient who had a history of a post-abortual curettage after a precipitated labor.

Our case did not have any of the risk factors discussed above. There was not any history of previous surgical intervention that could be responsible for myometrial damage or perforation (eg. D&C or hysteroscopy, etc.). Use of oxytocin and vacuum extraction are among the factors that may have caused uterine rupture in our case. Since the dose of oxytocin used during augmentation is limited to 8 mU/min, it seems unlikely that this factor alone would have played an important role during events leading to the rupture. Whether vacuum extraction causes uterine rupture is still debated. We performed vacuum extraction because of fetal bradycardia when the vertex was at +1 - +2 station without encountering any complications or difficulties such as shoulder dystocia. However, the possibility of uterine rupture was ruled out by both a normal FHR pattern and absence of any uterine tetanic contractions despite the presence of mild vaginal bleeding. It is possible that the myometrium started to rupture during this time. Miller *et al.* discussed ten cases with ruptured intact uteri [6]. They noticed vaginal bleeding during the course of delivery in only one case. Vacuum extraction was performed in two of the cases when the vertex was at +2 station. PG E<sub>2</sub> was used for the induction of labor in three cases.

The time interval between the delivery and laparotomy is a very important point that should be discussed. In our case the time between delivery and laparotomy was 40 minutes. Miller *et al.* reported that the interval between the two events was a maximum 25 minutes [6]. Cardiac arrest secondary to hypovolemia could have been prevented if laparotomy had been performed earlier. Except for this situation, there are similarities between the cases of Miller *et al.* and our case. However, there is not any clear explanation on how the different risk factors are effective in the development of rupture in an unscarred uterus. The physiopathology of the condition, factors that can affect the myometrium and the course of rupture development are the major questions to be answered. Therefore, we propose that misoprostol alone can not be a cause of unscarred uterine rupture.

Misoprostol is easy to use and inexpensive. There are

many prospective randomized publications indicating the efficacy and safety of misoprostol for cervical ripening and induction of labor. Developed tachysystole can be pointed to as the reason for a ruptured uterus. However, there is not enough data available to support this hypothesis, thus it remains speculative without any clear explanation of the physiopathology of the uterine rupture. We believe that misoprostol does not increase the prevalence of rupture of an unscarred uterus compared to oxytocin and other prostaglandins used for induction of labor or cervical ripening.

## References

- [1] Sanchez-Ramos L., Kaunitz A. M., Wears R. L., Delke I., Gaudier F. L.: "Misoprostol for cervical ripening and labor induction: A meta-analysis". *Obstet. Gynecol.*, 1997, 89, 633.
- [2] Kramer R. L., Gilson G. J., Morrison D. S., Martin D., Gonzales J., Qualls C.: "A randomized trial of misoprostol and oxytocin for induction of labor: Safety and efficacy". *Obstet. Gynecol.*, 1997, 387.
- [3] Wing D. A., Paul R. H.: "A comparison of differing dosing regimens of vaginally administered misoprostol for preinduction cervical ripening and labor induction". *Am. J. Obstet. Gynecol.*, 1996, 175, 158.
- [4] Wing D. A., Lowett K., Paul R. H.: "Disruption of prior uterine incision following misoprostol for labor induction in women with previous cesarean delivery". *Obstet. Gynecol.*, 1998, 91, 823.
- [5] Phelan J. P., Korst L. M., Settles D. K.: "Uterine activity patterns in uterine rupture: A case-control study". *Obstet. Gynecol.*, 1998, 92, 394.
- [6] Miller D. A., Goodwin T. M., Gherman R. B., Paul R. H.: "Intrapartum rupture of the unscarred uterus". *Obstet. Gynecol.*, 1997, 89, 671.
- [7] Bennett B. B.: "Uterine rupture during induction of labor at term with intravaginal misoprostol. *Obstet. Gynecol.* 1997; 89:832.
- [8] Blanchette H. A., Nayak S., Erasmus S.: "Comparison of the safety and efficacy of intravaginal misoprostol (prostaglandin E<sub>1</sub>) with those of dinoprostone (prostaglandin E<sub>2</sub>) for cervical ripening and induction of labor in a community hospital". *Am. J. Obstet. Gynecol.*, 1999, 180, 1551.
- [9] Mathews J. E., Mathai M., George A.: "Uterine rupture in a multiparous woman during labor induction with oral misoprostol". *Int. J. Gynecol. and Obstet.*, 2000, 68, 43.

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