Spontaneous delivery through a cervical tear without cervical os dilatation

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Key Clinical Message
Spontaneous delivery through a cervical tear, provoked by prostaglandin-induced uterine contractions, was described in a G2P0 woman with a history of cervical dilatation and uterine curettage. This rare complication with potentially serious maternal–fetal consequences can be predicted by an aberrant cervical response to prostaglandins in parturients with previous cervical interventions.

Keywords
Delivery through a cervical tear, intrapartum cervical lesions, labor induction, postpartum hemorrhage.

Introduction
Cervical injuries represent frequent morbidities associated with vaginal deliveries [1]. The rates of intrapartum cervical lesions increase in the cases of nulliparity, precipitous labor, operative vaginal delivery, and cervical surgical interventions such as cervical cerclage during pregnancy [2]. Induction of labor also increases the probability of cervical damage [3].

Spontaneous deliveries through intrapartum cervical lesions have been documented in women with medical history of cervical interventions. These include loop electrosurgical excision procedure due to cervical intraepithelial neoplasia [4] and cervical cerclage in previous pregnancy [5, 6]. We report a case of cervical tearing without external os dilatation that occurred upon prostaglandin induction of labor in a woman with a history of voluntary interruption of pregnancy by cervical dilatation and uterine curettage (CD&C). The tear resulted in a spontaneous delivery of the fetus through the lesion and postpartum hemorrhage successfully treated by the lesion suture.

Case Presentation
A 39 year-old G2P0 woman, with a spontaneously conceived, singleton and uneventful pregnancy was admitted to our institution at 41 weeks of gestation for labor induction. Her past obstetric history revealed a voluntary interruption of pregnancy 13 years before, at 6 weeks, performed by CD&C without complications. There was no suggestive symptom or clinical evidence of any cervical lesion, consequent fibrosis, and stenosis. The patient menstrual history after the intervention did not reveal any deviation from normality. The speculum and digital examinations during the current pregnancy did not indicate any pathology. Neither relevant medical conditions nor abdominopelvic surgical interventions were reported.

The digital examination on admission revealed a 15 mm long, posteriorly oriented cervix of rigid consistency with a 5 mm wide external os and cephalic presentation. To promote cervical ripening, 2.5 mg of dinoprostone (i.e., prostaglandin E2 [PGE2]) were vaginally applied. However, no significant cervical changes
occurred during the induction day 1. Observing normal CTG records without significant uterine contractility, 50 μg of misoprostol were vaginally given the next day (induction day 2). As presented in Figure 1, the regular uterine contractility was initiated nearly 4 h after the application of this prostaglandin E1 (PGE1) analog. The patient required analgesia and received loco-regional anesthesia. The establishment of regular uterus contractions coincided with spontaneous rupture of the membranes and resulted in cervical effacement. In contrast, no change of the external os occurred. It remained rigid and 5 mm dilated, as evidenced by serial digital assessments 0, 2, 4, and 5 h after the misoprostol application (Fig. 1, red dots). The next digital evaluation, performed ~45 min later, surprisingly indicated “a complete dilatation” with the presentation at the level of ischial spines (De Lee station 0). A healthy male newborn was vaginally delivered weighing 3355 g, with Apgar score 10/10. After the spontaneous placental removal, an intense vaginal bleeding drew attention. The uterus was well contracted while no ultrasonography signs of placental or fetal membrane retention were visualized. The examination with a speculum revealed that original cervical os remained closed. In parallel, a 4 cm wide, posterior cervical tear was observed from 4 to 9 o’clock without extension to the vaginal fornix, as schematically presented in Figure 2. The lesion had provided a gate out to the fetus and caused the hemorrhage. The suspicion of a significant lesion of the uterine corpus was rejected by the identification of the tear borders. Additionally, there were no clinical signs of hemoperitoneum while ultrasonography confirmed the absence of free fluid in the abdominopelvic cavity. We achieved the hemostasis by repairing the lesion with a continuous suture using absorbable Vicryl® 1 (Ethicon, Somerville, NJ, USA). The permeability of the natural cervical os was indicated by the observation of spontaneous lochia drainage.

Figure 1. Bedside partogram (induction day 2).

Figure 2. Schematic presentation of observed posterior cervical tear (PCT), below unopened cervical os (CO), that permitted spontaneous delivery of the fetus.
The patient received oxytocin perfusion (20U) and 2 g of cefazolin. The hemorrhage resulted in a reduction of the hemoglobin level from 12.9 to 10.7 g/dL, thus the patient was also medicated with daily iron supplement. Having an uneventful postoperative course, the patient was discharged on day 3 postpartum and referenced to the Hospital’s Unit for Puerperal Revision and Family Planning.

**Discussion and Conclusions**

Clinically significant lesions of the cervix occur in 0.2–1.7% of vaginal deliveries [1]. Cervical tears have been frequently reported with instrumental delivery, particularly when forceps was engaged. However, large tears that mimic a full dilatation and lead to fetus delivery are ultimate rarity.

Cervicovaginal fistula represents a complication of induced midtrimester termination of pregnancy, observed in the past to follow intraamniotic hypertonic saline injections and the use of prostaglandin F2-α [5–7]. It is also a known complication of cervical cerclage [6, 8, 9]. There are reports of vaginal deliveries through these lesions leading to extension of the defect into the vaginal fornix, bladder or lower uterine segment [6, 9]. Besides, a spontaneous delivery through a cervical tear with an intact cervical os was reported in a patient with a history of cervical intraepithelial neoplasia which was treated with a loop electrosurgical excision procedure (LEEP) [4]. The cervical tearing occurred upon the labor induction with PGE2. Differently, in two other reported cases with unmentioned medical history, large cervical tears coexisted with partially [10] or fully dilated cervices [11]. Thus, cervical tearing in these cases probably occurred during, and not before the fetal passage.

In our patient, prostaglandins were used for labor induction as a standard method. It may be suspected that misoprostol itself resulted in observed cervical damage. Indeed, misoprostol increases the risk of corporal rupture in women with uterine scar [12]. Vaginal misoprostol can also decrease the macrophage function resulting in the overgrowth of clostridial organisms and development of gastrointestinal and reproductive tracts [13]. However, cervical lesions after prostaglandin application were more often reported in midtrimester abortions as highlighted before [5–7]. Generally, the drug allows appropriate cervical modification and fetal passage through the low-resistant cervical canal [8]. Although no previous cervical trauma was documented, CD&C used for the termination of preceding pregnancy opens up possibility of subclinical cervical injury. Despite the fact that a high percentage of patients with a history of CD&C have a favorable outcome of future pregnancies/deliveries, the procedure is associated with an increased rate of postpartum hemorrhages [14]. We believe that previous cervical injury, although clinically silent, should be considered as the principal reason for cervical tearing. The induction drugs were applied in a habitual and safe manner while no other obvious predisposing factors existed. Similarly to the case of patient submitted to LEEP, presumptive cervical tissue alteration did not allow dilatation of the external os due to fibrosis. Persisting rigidity of the os weakened the nearby cervical segment. Under the effect of prostaglandin-induced contractions, this resulted in tissue thinning and tearing. Alternatively, the intrinsic cervical os stiffness in our patient can be taken into account, making the above tissue prone to rupture.

Regardless of etiology, this complication could be prevented by performing a cesarean section upon the recognition of the pathologic significance of the external os rigidity. In nulliparous women at term, external cervical os remains stationary during the cervical shortening, but quickly evolves once the cervical effacement is completed [15]. Thus, identification of women at risk for significant cervical tearing is fundamentally important. It can be achieved by strict and careful monitoring of the labor progression. During the serial digital examinations of the cervical response, a particular attention should be paid to the characteristics and modification of the external os. Upon the completion of cervical effacement and in the presence of significant uterine contractility, repetitive finding of a rigid external os should be considered as a sign of alarm.

In conclusion, rigidity of the external cervical os and the lack of its dilation after the cervical effacement may indicate the imminence of cervical tearing beside a closed natural orifice. In particular, this cervical response should be valorized in women with a history of a cervical intervention, even when it is not associated with apparent complications. The case suggests that the group at risk of described complication also involves women submitted to minimally invasive procedures such as cervical dilatation. Labor induction in women with the history of CD&C is relatively common. Therefore, the prevalence of intrapartum cervical tearing may increase. Although the described event is rare, the possibility of its occurrence should not be neglected because of the serious maternal and fetal morbidities that may arise.

**Conflict of Interest**

None declared.

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