CASE REPORT

Delivery through perineal body and severed external anal sphincter with an intact vaginal orifice during a precipitous labor: a case report

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Introduction
The perineal body is pyramidal fibromuscular structure in the midline of the perineum at the junction between the urogenital triangle and 1.5 cm from the anus. It is an important structure in pelvic organ support and can be damaged during labor and delivery. The perineal body is a convergence point for the important muscles of pelvic support; namely, the external anal sphincter, bulbospongiosus muscle, superficial transverse perineal muscle, levator ani (anterior fibers), fibers from the external urinary sphincter, and the deep transverse perineal muscle.

Injury during labor is usually associated with tearing involving the vaginal orifice or anal sphincter. The rectovaginal septum is a thin membranous partition separating the rectum and the vagina. It is at the cephalad apex of the perineal body, lateral to the levator ani muscle. Injury to the rectovaginal septum is frequently associated with anal sphincter damage, while injury with an intact anal sphincter is rare. Obstetric perineal and anal sphincter injuries are divided into four grades; however, there is no classification or inclusion of rectovaginal septum or perineal body injuries.

Key Clinical Message
We present the case of a patient who delivered her baby through the perineal body with an intact vaginal introitus. Damage to the perineal body through its connection to the external anal sphincter can involve the rectovaginal septum with increased morbidity and thought to be linked to rectocele development.

Keywords
External anal sphincter, perineal body, precipitous labor, rectocele.

Case Report
A 30-year-old primigravida booked at 11-week gestation. She has no relevant medical or surgical history. Her pregnancy was uncomplicated until she received a diagnosis of gestational diabetes mellitus (GDM) at 29 weeks through an abnormal oral glucose tolerance test. She was
commenced on insulin at 33-week gestation after failing to control her blood sugars through diet and exercise alone. Her pregnancy was further complicated with diagnosis of obstetric cholestasis at 36-week gestation and she was admitted to hospital due to persistent pruritus and reduced fetal movements. A decision was made to induce labor at 37 weeks due to worsening liver enzyme profile and further progression of her pruritus. Induction of labor was achieved with 2 mg of dinoprost per vaginum. Her initial examination revealed a cephalic presentation, with a soft posterior cervix 1 cm dilated. Spontaneous rupture of membranes occurred 5 h after vaginal prostaglandin administration. Palpable contractions commenced within 20 min of rupture of membranes and reached a peak frequency of 7 in 15 min, lasting 45–60 sec each. Vaginal examination was performed 45 min after rupture of membranes due to maternal distress. The patient was found to be 4 cm dilated, vertex -2 and was transferred to a dedicated delivery room with one to one midwifery care. Within 2 h, the patient began involuntary bearing down and was found to be fully dilated, vertex +1 and rapidly advancing. Pushing was commenced.

In the second stage of labor, after pushing for 16 min, a 4 cm defect of the perineum between the vaginal orifice and the rectum was noted. The vaginal orifice itself remained intact. The fetal head was visible through this perineal defect as the head crowned. A right mediolateral episiotomy was performed to prevent an anal sphincter injury; however, the patient sustained a third-degree tear. Total time in labor from contractions starting to delivery of the baby was 2 h 45 min.

After examination under anesthesia, a 3b tear was diagnosed – namely, a tear that has severed the external anal sphincter. This was successfully repaired under general anesthesia with a combination of 3.0 polydioxanone monofilament synthetic absorbable suture in an overlapping technique to the anal sphincter, and a 2.0 serapid® OLM Limited, O’Leary Medical, Dublin, Ireland polyglycolic acid braided multifilament to the perineal muscle and skin. Particular care was made in the re-anastomosis of the perineal body to the underlying muscle. The patient was given a course of broad-spectrum antibiotics according to the local protocols and discharged home well after 4 days. She had no symptoms of flatus or fecal incontinence on discharge. A review at 3 months post delivery in the hospital’s specialized perineal clinic revealed no abnormality in anal sphincter tone, an anatomically normal perineum and an asymptomatic patient with no fecal incontinence. The patient was counseled that her next child should be born by elective cesarean section to prevent a recurrence of the injury. She was also counseled on the possibility of developing a rectocele prolapse later in life.

Discussion

Pelvic floor strength requires input from both the muscular and fascial compartments with an intact nerve supply to both. Factors contributing to the etiology of prolapse include disruption to the above three components, for example, trauma to pelvic floor musculature, insult to the endopelvic fascia, or damage to the nerve supply.

Delivery through the perineal body with an intact vaginal orifice is an unusual complication of childbirth. A previous case of perineal body injury occurring with occipitoposterior presentation of the fetal head at delivery has been attributed to the direction of the expulsive force toward the perineal body [2]. A further case with an occipitoanterior presentation is postulated to have been caused by either an anatomical variation of the pelvis, longer perineal body or a large fetal head [3].

Isolated rectovaginal defect with intact anal sphincter is rare, with only a single case reported where with a fetal foot presented transanally through a rectal defect [4].

We propose that precipitous labor may be an independent risk factor for damage to the perineal body. Further research is warranted to examine the effect of precipitous labor on the later development of rectocele.

Muscle and fascial injuries after vaginal delivery are often not immediately clinically apparent [5]. However, with aging and further vaginal deliveries, these connective tissue defects may become larger and more apparent, forming cystocele and rectocele.

Rectocele is thought to arise as a long-term result of either excessive stretching or tearing of the rectovaginal septum, typically caused by traumatic obstetric events. This can occur when the presenting part descends too quickly in the second stage of labor, as happened in this case report. The forces of labor may separate, tear, or distend the pelvic floor, altering the functional and anatomic position of the muscles, nerves, and connective tissues. The separation of the rectal fascia from the perineal body may then predispose the development of rectocele.

In addition to the above, denervation injuries due to overstretching or compression of the pudendal nerve can cause atrophy and denervation of the pelvic floor muscles resulting in vaginal support defects [6]. This neuropathy can lead to weakening of pelvic floor muscles and development of a rectocele.

Conclusion

While the perineal body and rectovaginal septum can be injured at childbirth, an isolated rectovaginal defect with intact anal sphincter during parturition is rare. Instrumental delivery, high birthweight, persistent occipitoposterior presentation and nulliparity are factors in the
etiology of the injury. Stretching or tearing of the rectovaginal septum by childbirth can lead to formation of rectocele. Identifying these factors may prevent the injury and its long-term consequences and shortening the second stage of labor may decrease the risk of denervation and subsequent pelvic floor damage. Precipitous labor, however, cannot necessarily be predicted or prevented. Further research is warranted to examine the effect of precipitous labor on the later development of rectocele.

**Patient Consent**

Consent was obtained from the patient for publication.

**Conflict of Interest**

Authors declare no conflict of interest.

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