OBSTETRICALLY-CAUSED ANAL SPHINCTER INJURY – PREDICTION, MANAGEMENT, PREVENTION

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LEARNING OBJECTIVES

On completion of this presentation, the recipient should be in a position to:

1. Understand the mechanisms by which injury to the fecal continence mechanism can occur during childbirth.

2. Have an awareness of the patients most at risk of anal sphincter injury.

3. Be able to select and interpret the appropriate investigations of the fecal continence mechanisms.

4. Understand how predisposing obstetric factors may be manipulated to reduce risk of intrapartum injury.

5. Initiate treatment measures, both at the time of injury and during the puerperium and afterwards, which will reduce or avoid fecal continence morbidity.

6. Be in a position to counsel women on appropriate obstetric management and recurrence risk in subsequent pregnancies.
7. Be aware of areas of potential development of our knowledge of this significant clinical problem.

Fecal incontinence is 8 times more prevalent in women than in men and, although clinical presentation occurs most frequently in older age groups, an obstetric injury is the presumed cause in the majority of cases.

Obstetric injury to the fecal continence mechanism can be either through direct anal sphincter disruption, which occurs especially at first deliveries, or through pudendal neuropathy which is more prevalent in multiparas; a combination of pathologies is not infrequently present.

Assessment of fecal continence is frequently omitted at postnatal examination. Many women with continence difficulties are reluctant to volunteer symptoms unless prompted. The recognized methods for evaluation of postpartum fecal continence include:

1. Standardized continence questionnaires
2. Anal manometry
3. Endo-anal ultrasonography
4. Pudendal neurological assessment, either through nerve conduction or electromyography

Using a combination of these methods, clear delineation of the type of injury is usually possible.

Risk factors for anal sphincter injury include especially primiparity and difficult or instrumental vaginal delivery. Duration of the second stage appears to be influential and midline episiotomy is an avoidable predisposing factor.

In terms of avoiding sphincter damage, primary prevention methods include oxytocin augmentation in the primiparous second stage, so as to avoid instrumental delivery, and mediolateral rather than midline episiotomy where this is indicated. In women with epidural anesthesia, delaying pushing efforts may increase pudendal nerve vulnerability. Vacuum extraction is potentially less traumatic than forceps delivery, although a combination of the two instruments is a particular risk factor. Optimal technical repair when an anal sphincter disruption is diagnosed at delivery can greatly reduce and/or prevent subsequent continence morbidity and primary sphincter muscle approximation is as effective as an overlapping technique.

In the immediate aftermath of a third/fourth degree tear antibiotic and laxative therapy should initiated for the first five days. Following a recognized anal sphincter injury, postnatal management should ideally include evaluation at 8-12 weeks using the standardized assessment methods listed above. In most cases minor incontinence symptoms can be abolished by early resort to biofeedback physiotherapy.
In general, the incidence of third degree tear is about 2 – 2.5% in primiparas and 0.5 – 0.8% in multiparas. Recurrence risk in following pregnancies is about 3-4%, provided midline episiotomy is avoided, but recurrence can not be predicted antepartum. The continence outcome following repair of recurrent injury is comparable with that after first injury.

Evidence for selection of women for prophylactic caesarean delivery in pregnancies following anal sphincter injury is relatively scant but two groups of women almost certainly require prelabor cesarean next time: those who have persistent significant incontinence symptoms lasting into the next pregnancy and that small number of women who have required a secondary surgical repair of the anal sphincter muscle to restore continence.

Postpartum pudendal neuropathy fits into four main patterns; demyelinating injury is consistent with prolonged second stage and postpartum fecal urgency while axonal damage usually follows nerve trauma at instrumental delivery and is frequently associated with persistent sphincter muscle defects on ultrasound. Non obstetric neuropathy can also present at this time and requires careful differentiation so as to optimize management.

Intra-anal biofeedback physiotherapy has been shown to be an effective primary therapy in women with postpartum incontinence symptoms. Self-administered home treatment has proved to be of at least comparable efficacy to hospital-based treatment and it may well prove most effective when commenced within one week of delivery.

Most women with fecal incontinence present after childbearing age and frequently post menopause. Estrogen hormone replacement therapy has been shown to significantly improve continence in the post menopausal cohort. While it is difficult to clearly differentiate the contributions of obstetric injury and advancing age to the prevalence of female fecal incontinence, available evidence indicates that age-related deterioration in the continence mechanism is the more potent factor.

Advances in the understanding of female fecal incontinence have been somewhat hampered by the absence of experimental models. It has recently proved possible to construct an animal model of obstetric injury, which permits evaluation of neurotrophic and sacral nerve stimulation therapies and also, through the use of functional brain MRI, to assess the contribution of cerebral cortical awareness in the aftermath of sphincter injury.

**References:**


