Clinician's guide to perineal and obstetric anal sphincter injury

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Introduction
Perineal trauma is a common consequence of vaginal childbirth. This may be spontaneous or an iatrogenic event following an episiotomy, or a combination of the two. One UK study states that up to 85% of women experience some form of perineal damage during labour.1 Many of these are minor lacerations, but up to 70% may require suturing.[2,3]

Obstetric anal sphincter injuries (OASIS) are severe perineal lacerations involving part or all of the anal sphincter complex. Improved knowledge and education in the identification and management of OASIS are essential in view of the substantial acute and chronic morbidity associated with such trauma.

Clinical significance of OASIS:
Acute complications:
Acute physical complications from perineal trauma include bleeding and possible haematoma formation. Additionally, infection and in some cases abscess formation may cause breakdown of the repair and can necessitate iatrogenic disruption of the repair to enable drainage. Severe cases may rarely be complicated by necrotising fasciitis and severe morbidity and maternal death.[4]

Chronic complications:
OASIS is often associated with chronic perineal pain. Almost half of women at 10 days postpartum who deliver vaginally still have pain or discomfort, and of those, 10% have pain which endures at 18 months post delivery[5], and may contribute to sexual dysfunction. In their study describing the relationship between postpartum sexual function and perineal trauma, Signorello et al found that 25% of primiparous women who had a vaginal delivery reported decreased sexual satisfaction, decreased sensation, and a diminished ability to attain orgasm. Additionally, when compared to women with an intact perineum after delivery, women with a second degree and third/fourth degree tear were found have an 80% and 270% increase in the likelihood of dyspareunia at 3 months. Episiotomy was associated with the same array of sexual dysfunction as perineal lacerations.[6]

Bowel dysfunction is a common complication following perineal trauma, this is expressed as a range of bowel related symptoms including anal urgency and incontinence which can encompass incontinence of flatus, liquid or solid stool[7,8] Although it is under-reported, the incidence of bowel dysfunction in OASIS has been reported to be as high as 61%.[9]

Beyond the physical implications, OASIS have can have a deleterious effect on patients' emotional and social wellbeing, and may trigger complex relationship issues.[10] This includes OASIS syndrome which includes a loss of self-esteem and severe psychological, emotional and sexual morbidity resulting from a sense of being unclean, and feelings of grief, guilt and shame amongst others.[11]

Anatomy of the perineum relevant to OASIS:
Anatomically, perineal trauma can be divided into anterior and posterior wall trauma.

Anterior wall trauma comprises injury to anterior vaginal wall, urethra, clitoris, and labia.

Posterior wall trauma includes injury to posterior vaginal wall, perineal muscles and disruption of anal sphincters.[12]

The bony pelvic outlet is paralleled by the perineum, the anterior border of which is the pubic arch. The posterior and lateral borders are the coccyx, and the ischiopubic rami/ischial tuberosities/sacrotuberous ligaments respectively.[13]

If one were to sketch an imaginary line transversely between the ischial tuberosities, the perineum can be divided into two triangular portions:

1. The Urogenital triangle (anterior division) includes the superficial muscles, namely the Ischiocavernosis, Bulbopongiosis, and Superficial transverse perineal muscles.
2. The Anal triangle (posterior division): encompasses the anal sphincter complex.

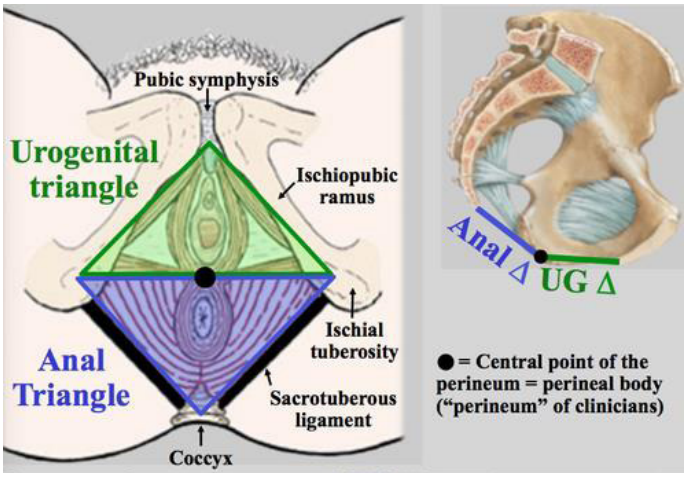

Figure 1. Diagrammatic representation of the Urogenital and Anal triangle (Source: https://quizlet.com/195582951/perineum-flash-cards/)

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The anal sphincter:
Anal continence involves interplay between a number of physiological mechanisms including a normal diet and bowel habits , normal cortical function, and a functional and structurally integral bowel and pelvic floor - specifically an effective anal sphincter complex, as well as a number of psychological factors.[7]

The arrangement of the smooth muscle of the rectum is characteristic of the GIT (layers of inner circular and outer longitudinal muscle), until the perineal flexure at which the circular layer thickens to form the internal anal sphincter (IAS).[14] This sphincter maintains all the majority of resting anal tone, the remainder produced by the EAS and anal cushions.[8] The Longitudinal layer concentrated on the rectal walls anteriorly and posteriorly, connects to the coccyx and perineal body, and passes under the external anal sphincter (EAS). The EAS and the puborectalis (forms a sling around the anorectal junction) together form a functional unit which is for the most part under sustained tonic contraction, can also be voluntarily contracted. The IAS and EAS encircle the anal canal distal to the anorectal angle (ARA) formed by the puborectalis muscle. The longitudinal muscle of the rectum and the puboanal fibres of the puboccyceal muscle travel between the two sphincters.

Table 1. A description of the portions of the anal sphincter complex[8,14,15]

Muscle	Innervation	Function
EAS	Inferior rectal branch of pudendal nerve Perineal branch of the 4th sacral nerve	Tonic contraction Exerts maximal contraction
IAS	Autonomic (excitation and inhibition) Enteric (controls tone)	Confers up to 85% of resting anal tone
Puborectalis	Inferior rectal and perineal branches of the pudendal nerve	Actual role controversial, may brace the rectum above ARA, acting as a watershed for intrabdominal pressure and enteric matter

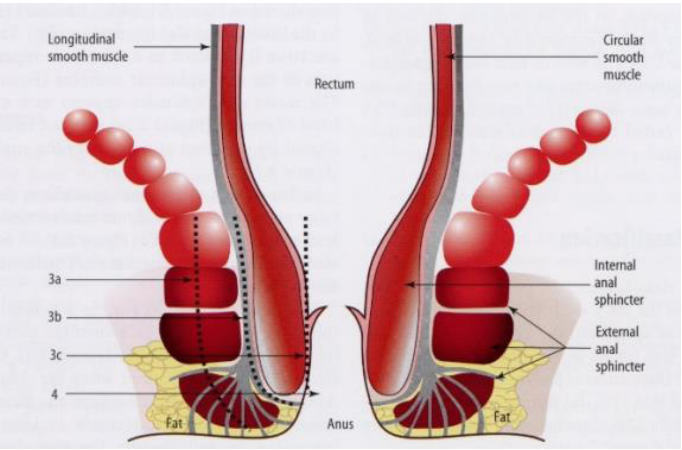

Figure 2. A coronal view of the anal sphincter.[60]

Classification:
To allow for clarity of description and better discussion around OASIS, the classification of OASIS first illustrated by Sultan has been accepted and assimilated by the Royal College of Obstetricians and Gynaecology in their green top guidelines, as well as by the WHO and the International Consultation on Incontinence.[16–18]

Table 2. Classification of perineal injury

Definition	Description	Structures involved
First degree tear	Injury to perineal skin	Skin, subcutaneous tissues of anterior/ posterior vaginal wall, perineum, in any combination
Second degree tear (corresponds to a mediolateral episiotomy)	Injury to perineum involving perineal muscle but not the anal sphincter	Bulbospongiosis, ischiocavernosis, transverse perinei, possibly perineal body. Rarely a more complex tear can occur: the injury can extend spherically behind hymenal remnants and cranially to the clitoris causing a separation of the lower third of the vagina from the underlying structures
Third degree tear	Injury to the perineum involving anal sphincter complex	Varying degrees of the anal sphincter
3A	Less than 50% of EAS	
3B	More than 50% of EAS	
3C	Involvement of both EAS and IAS	
Fourth degree	Injury involving the EAS, IAS and anal epithelium	
Buttonhole tear*	EAS intact but involvement of anal or rectal mucosa with or without involvement IAS	

*Buttonhole tears are uncommon, isolated tears of the rectal mucosa. Although it should be regarded as a separate entity, it is important to recognize and repair such defects to avoid the formation of consequent ano or rectovaginal fistulae.[10]

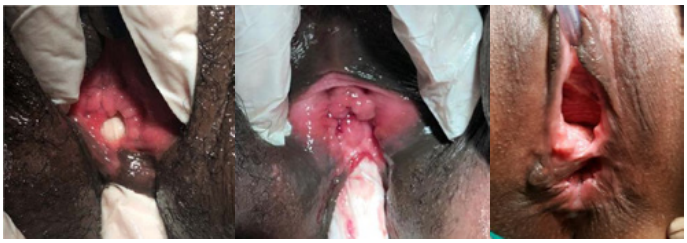

Figure 3. Various forms of injury to the anal sphincter as a result of vaginal childbirth and failed primary repair

For clinical purposes, OASIS may be further classified into subtypes according to the time frame in which it has been recognised. Although there is no standardized terminology for the OASIS subtypes, the distinction is of clinical relevance because the presentation, diagnostic evaluation, and management differs according to the delivery to repair interval.

1. Overt OASIS: Diagnosed at the time of delivery, or within 8 weeks of delivery. These patients may present with one or more of the following on history and examination:

- These patients may have sustained an obvious and timeously recognised perineal laceration at the time of the delivery.
- The laceration may have been recognised at delivery and were either inadequately repaired.[19]
- Alternatively it may have been adequately repaired but subsequently developed complications such a sepsis and wound breakdown which has been found to complicate 25% of women who sustain obstetric perineal injury
- The injury may have been sustained during unsupervised deliveries or in areas where patients unable to access prompt obstetric care.
- Patients describe perineal pain, bleeding and/or vaginal discharge. Additional symptoms may include anal incontinence and fecal urgency.[20]

2. Latent OASIS: This may be a partially repaired with remnant sphincter defect, or may have been unrecognized until months or years later. These injuries were previously named occult sphincter injuries but it is now thought that they are lacerations that have not been recognised or have been misidentified.[21,22] One study found that up to 40% of all third degree tears had not been diagnosed adequately by the midwife or doctor present at the initial perineal evaluation.[22] Patients may present months or years after the delivery. Typical symptoms include anal incontinence (the severity of the incontinence may differ according to the defect) and fecal urgency. This may arise from two entities:

- Chronic/unresolved anterior anal sphincter defect
- Rectovaginal fistula

Epidemiology of OASIS

A majority of women will sustain some form of vaginal laceration during delivery. Including superficial tears that need no suturing this can occur in up to 85% of women.[23] Obstetric anal sphincter injury (OASI) forms a subset of these injuries. Although post-partum symptoms of anal incontinence occur in some patients without OASI, they are more common after OASI[24] and symptoms are proportional to severity of injury.[25]

There is considerable variation in the reported incidence of OASIS, even between units within countries during a standard time-frame.[26] Some authors report incidences as high as 34%,[27] with others as low as 0.1%.9 Some of this variation is attributed to the over or under-diagnosis of OASI.[28] Studies have shown that midwives and junior doctors miss OASI in up to 87% of cases.[13] Standardisation of examination and classification is therefore key to determining the true incidence of OASI.[28] Although large studies of the local incidence has not been published, large review and meta-analysis of studies from low and middle-income countries found an overall reported incidence of 1.4%.[29]

Risk factors

A variety of risk factors have been evaluated, in retrospective and prospective studies, with mixed outcomes. This is reflected similarly in guidelines and systematic reviews that find conflicting roles for different factors.[16,23] The list of factors that have been considered can be divided into patient, fetal and healthcare worker factors.

Patient factors

An increased BMI over 30kg/m^2 appears to be mildly protective against OASI.30 Patient age holds direct relation to parity, but a large registry based study from Sweden of more than one million births showed that increasing maternal age is an independent risk factor.[31] There is consensus that the risk is increased in the nulliparous women, and decreases with each subsequent birth.[16,23,32]

Prolonged second stage of labour is associated with an increase in risk of OASI, with the risk increasing when comparing <1h with 1-2h, 2-3h and more than 3h.[9,16,33] A study from Hyderabad in India including more than 14 000 births over a period of five years found the same association, but when performing multivariate analysis found this association to be non-significant, suggesting that the same underlying factor that causes the prolonged second stage is responsible for the OASI.[34] In registries that enable tracking this data, a family history of OASI in a first degree female relative is associated with increased risk. Patients delivering vaginally after previous CS are also at increased risk of OASI.[30]

Fetal factors

Factors related to the fetus associated with increased rates of OASI are fetal head circumference >35cm, increased birth weight and persistent occipito-posterior position. Birth weight in most analyses divides births into binary groups of less or more than 3.5kg[34] or 4kg[30], but in either case the difference is consistent.

Intrapartum care factors

Intrapartum and delivery factors are of especial interest, as these are the factors potentially amenable to intervention. This has been demonstrated in a study conducted in two centres in Sydney and Hong Kong, including only women of Asian descent, where the anal sphincters were assessed immediately after birth, by the same investigator in all cases, in a standardised way. This showed a wide difference in outcome for these women, which the author explained by differences in labour ward practice.[27]

Assisted delivery consistently increases risk of OASI. This risk is greatest with forceps delivery increasing the risk by as much as 7-fold.[16] A similar but smaller increase in risk is found with vacuum delivery. The role of episiotomy has been studied extensively. Episiotomy is associated with increased risk of sphincter injury,[16] but may be protective in the context of assisted delivery. Midline episiotomy has greater risk than mediolateral episiotomy.[23] In a retrospective cohort study of women undergoing a second vaginal delivery after OASI, the use of episiotomy appeared to be protective against repeat OASI.[35]

Other factors associated with OASI include induction and augmentation of labour.[30] The use of an epidural has been postulated to increase the risk but has not been consistently found to have an effect on rates of OASI. Different birth positions, including sitting, squatting, all fours, lithotomy, supine, on birth seat, standing and kneeling, have been evaluated retrospectively in a large cohort, and conflicting results were found, depending on parity.[36] The most consistent finding was that sitting position was associated with lower risk than lithotomy, in all groups.

Trends, prediction and prevention

Different researchers have attempted to construct models to predict the occurrence of OASI.[30,37,38] These perform variably, with all of them having either unacceptably low sensitivity[38] or low positive predictive values,[37] making them of little clinical utility. In addition, the strongest predictive factors relate to intrapartum events that can't be foreseen at the time when the patient is counselled on mode of delivery.

In some regions a trend towards increased rates of OASI have been seen. In the light of the possibly preventable healthcare factors, different intervention programmes have been instituted in an attempt to decrease the incidence of OASI. Finland experienced a more than three-fold rise in the incidence of OASI in nulliparous women from 1997 to 2007.[39] The so-called Finnish intervention was then designed and widely instituted across the Nordic countries. This included good communication between accoucheur and patient, the birth positioning allowing visualization of the perineum during delivery, mediolateral episiotomy on indication and the Finnish manoeuvre. A review in 2015 found seven studies that showed decrease in rates of OASI, but noted that all of these were of low quality.[40]

In the UK the STOMP (Stop Traumatic OASI Morbidity Project) programme consists of encouraging upright positioning,

maintaining good communication and tactile control of the speed of delivery of the fetal head, and has likewise been found in small studies to decrease the incidence of OASI.

Evaluation and management:
A thorough general history and examination is a mandatory part of OASIS evaluation and repair.

General principles of OASIS repair:
- Who: Repair of OASIS should be performed by qualified and competent surgeon, or by a supervised trainee.
- Where: Operating theatre, good lighting. Only under very specific circumstances should it be performed in the delivery room.
- How: Regional/General anaesthesia, suitable instruments. Where possible figure of eight sutures should not be used.
- When: As soon as possible. In the interim and for transfer to theatre vaginal packing is recommended in the event of severe bleeding.[16]

Primary OASIS repair:
Overt OASIS:
According to the RCOG, all patients attempting a vaginal delivery are at risk of OASIS or a buttonhole tear. It is the responsibility of the attending physician to thoroughly assess the perineum and rectum prior to repair.[16]

The NICE perineal care guidelines state that the examination by the attending clinician should be preceded by:
- A detailed explanation of what they plan to do and why.
- Suitable analgesia with confirmation by the patient that it is adequate.
- Adequate lighting and positioning of the patient so ensure comfort as well as adequate exposure.[41]
- Lithotomy is advised, rectal examination mandatory.[16]

Imaging:
It has been found that at least 10% of OASIS are missed at delivery.[21] Immediate postpartum endoanal ultrasound (EAUS) is a straightforward and non-invasive modality used to image the anal sphincter. It has been shown to be as sensitive as MRI in the detection of sphincter anomalies. It has been postulated that the use of EAUs or transperineal ultrasound immediately postpartum could increase OASIS recognition. A Cochrane review on the subject of immediate endoanal ultrasound found one study of 752 participants, comparing outcomes of repair following either ultrasound or clinical exam. The study found that women who had ultrasound prior to repair were about 50% as likely to report severe anal symptoms, but were more likely to report severe perineal pain at three months. The review concluded that although the trial was well designed, more studies in this area are necessary before the practice can be properly evaluated.[42] Currently the application of US at delivery to identify OASIS is limited to research.[16]

Repair technique:

Table 3. Techniques of primary surgical repair of OASIS and perineal lacerations according to location of tear.[4,16,43,44]

Tissue	Suture material	Technique
Anorectal mucosa	3-0 polyglactin (Vicryl®)	Continuous or interrupted (Previously when catgut was used the recommendation was for interrupted sutures with the knot in the anal canal – not necessary with Vicryl®)
IAS	Monofilament: 3-0 PDS or Modern braided 2-0 polyglactin (Vicryl®)	End to end repair-advised to bury knots under superficial perineal muscles
EAS Full thickness (3c)	Monofilament: 3-0 PDS or Modern braided 2-0 polyglactin (Vicryl®)	End to end/Overlapping can be used with comparable outcomes
EAS Partial thickness (3a and some 3b)	Monofilament: 3-0 PDS or Modern braided 2-0 polyglactin (Vicryl®)	End to end
Second degree tears/Episiotomy repair	3-0 polyglactin (Vicryl®) Vicryl Rapide®: This can be used with caution, tensile strength is reduced by 50% in 5 days. Associated with less pain at 10 dys, and smaller risk of requiring suture removal	Continuous suture may be associated at 10 days with less pain, less analgesia use, and smaller probability or requiring removal if suture material
First degree tears	3-0 polyglactin (Vicryl®) Vicryl Rapide®: This can be used with caution, tensile strength is reduced by 50% in 5 days	Clinical acumen should be used to distinguish those tears that should be sutured and those that will heal spontaneously
Periclitoral/Periurethral/labial lacerations	3-0 polyglactin (Vicryl®) Vicryl Rapide®	If not bleeding profusely/distorting anatomy may be sutured or left to heal spontaneously

General principles of post-operative management:
Local practice, for example known pathogens and resistance models, should guide the prescription of the following

- Broad spectrum antibiotics: to decrease the probability of wound infection and breakdown. A Cochrane review found only 1 small trial on antibiotic use post repair, data showed that there was value in the use of antibiotics, but not enough evidence.
- Post-operative laxatives, not bulking agents.
- Pelvic floor physiotherapy is advisable for optimal outcomes.
- Post-operative review at expedient time (6-12 weeks if feasible), preferably by special interest clinic/clinician. If pain or incontinence is reports, referral to a Urogynaecologist/ colorectal surgeon is advised.[16]

Latent OASIS (delayed repair):
Management should begin with a thorough history and examination, with due cognizance given to the fact that clinical examination can easily miss sphincter defects.

Investigation:
Ultrasound is therefore a valuable tool in the evaluation of sphincter defects. There has been much work of late on the uses of transvaginal, transperineal, and endoanal ultrasound in the detection of pelvic floor disorders. The advantages of such ultrasonography over clinical practice in the long term treatment of OASIS is that it allows better detection of injury, allows for surveillance of therapeutic effect, as well as to predict future pregnancy outcomes.[45] MRI can also be used for the imaging of the anal sphincter, however the advantage of enhanced anatomical detail has to be balanced against the higher cost and the extra time which it consumes. It can also prove problematic when trying to image patients with highly fibrosed sphincters, as the modality is less able to distinguish between fibrosis and muscle tissue.[45]

Anorectal physiology testing such as manometry gives functional information which can be complementary to the structural information obtained from ultrasonography. EAS defects are accompanied by lower squeeze increments, while IAS defects are associated with lower resting pressures. The squeeze increment is therefore strongly associated with the bulk of the EAS.

Surgical management of delayed repair:
The general guidelines for immediate repair also hold for delayed repair.

The timing of the repair is quite contentious, but Dudding et al advise that if the defect has been missed or the primary repair has failed, any attempt to repair the defect should be delayed for at least three months.[45] The secondary repair should be performed by an experienced specialist/colorectal surgeon. The EAS (and IAS if 3c) should be mobilised en bloc, and the two ends of the muscle repaired, preferably the two ends of the muscle should be overlapped with interrupted mattress sutures if there is enough length.

The IAS is a fragile smooth muscle, too thin to hold sutures, thus with solitary IAS defects, repair tends not to improve symptoms.

In general, bowel diversion does not improve outcomes, and can cause further morbidity.

Diverting stomas should therefore be reserved for cases complicated by sepsis, previous failure, inflammatory bowel disease or cloacal deformity. Additions like perineal drains, bowel preparation and antibiotics have scant supportive literature.[45]

Other surgical treatment:
Diverting colostomy remains a salvage procedure for patients who fail the above, and who have debilitating incontinence. Complex procedures such as neosphincter procedures involving dynamic graciloplasty/artificial bowel sphincter are associated with high morbidity. As such, they should be performed by extremely specialised clinicians, and for patients with devastating symptoms as a last resort.[45,46]

Non surgical modalities of treating OASIS:
Clinicians treating OASIS must also be aware of the non-surgical modalities of OASIS treatment. These may be instituted as first line treatment of the symptoms associated with OASIS, or in patients who are not fit for surgery. They may also be a last resort in patients for whom surgery has proven unsuccessful. These include:

- Lifestyle and diet modification
- Anti-diarrhoeal drugs: increasing gut transit time and producing firmer stool consistency.
  - Loperamide hydrochloride: 1st line – if 2mg dose produces constipation, may try titrating Loperamide syrup to effect
  - Codeine phosphate (Has side effects and potential for abuse)
  - Amitriptyline: 20mg has been shown in a small study to decrease number or bowel movements and incontinence episodes (For idiopathic FI)
- Bulking agents
- Physiotherapy and biofeedback is also a valuable tool.[47]
- Anal irrigation.[47]

Sacral neuromodulation (SNM):
One of the indications for sacral neuromodulation is faecal incontinence. In patients with intact sphincters, 40% will achieve full continence, and up to 90% will report a decrease in incontinence episodes by at least half.

Patients with EAS disruption also appear to benefit from SNM, with the benefit of good durablility if effect. Given the low morbidity associated with procedure, and the somewhat poor results of sphincter repair reported in the literature, this should be discussed with patients as an alternative to sphincter repair. The added advantage being that the sphincter is not interfered with during the procedure, and repair can always be performed at another time if necessary.[45,47] In a recent study on FI following OASIS, SNM was shown to decrease the number of incontinence episodes per well by more than 90%, regardless of severity of sphincter defect. A significant decrease in faecal urgency was also reported.[48]

Outcomes
The main outcome measures of OASI and repair is anal function. This encompasses continence to solid or liquid stool and flatus, and subsequent lifestyle alterations and quality of life. Anal continence is by its nature not a single event, and a variety of assessment tools to measure anal continence have been described and validated. Most of these assess the frequency of incontinence to solid and liquid stools, incontinence to flatus, the use of pads or plugs and symptoms of faecal urgency or inability to defer defecation.

The St Marks Incontinence score (SMIS) (table) has been validated against a visual acuity score of incontinence bother.[49] Objective outcome measures include anal manometry measurements and endoanal or transperineal ultrasound findings of anal sphincter disruption, described by the span of arc of anal sphincter disruption.

The background prevalence of anal incontinence in the UK is 0.8% in men and 1.6% in women under the age of sixty, with this rising to 5.1 and 6.2% respectively over the age of 60.[50] In the post-partum period, symptoms of anal incontinence are common, occurring in up to 23% of women without OASI, and rising to up to 59% of women with grade 3c and 4 injuries.[24]

Interestingly, even in multiparous women without any OASI, symptoms of AI are fairly common, with 6.3% of women reporting a change in quality of life as a result.[51] However, women with OASI

have a two-fold risk of anal incontinence.[52] At long-term follow-up 15-23 years after delivery, the incidence of anal incontinence for formed stool is 9% in women with OASI and 1% in women without.[53]

The importance of recognition of injury is highlighted by the finding that women who have missed tears have higher rates of anal and urinary incontinence than those whose injuries were recognised and primarily repaire.[54] A prospective study from the UK assessing outcomes in women with OASI repaired by adequately trained clinicians found no difference between women with and without OASI after 4 years; indeed, they did not find any faecal incontinence at all.[55] By contrast, were primary repair has failed, the outcomes of secondary repair are much worse. A large long-term follow-up series from Denmark found that all women undergoing secondary repair had some form of incontinence at 18 years after repair.[47]

Subsequent deliveries

The evidence base for best advice for women with a history of OASI on method of delivery in subsequent pregnancies is incomplete. The relative risk of repeat OASI is increased in these women, but absolute risk remains small.[56] Compared to CS, subsequent vaginal delivery does not have increased symptoms.[57] Conversely, in women with prior OASI, those with repeat OASI have increased symptoms compared to those without.[58] Therefore, it would be desirable to attempt to identify those who are at risk of worsening of symptoms as a result of vaginal birth.

Abdul's group have devised a decision algorithm that incorporates symptoms using the SMIS, endo-anal ultrasound (EUS) and anal manometry. In women with no bowel symptoms, maximum squeeze pressures of >20mmHg and an external anal sphincter defect of <30 deg on EUS subsequent to primary repair, increased symptoms after subsequent vaginal deliveries have not been shown.[59] However, in women with pre-existing symptoms, repeat vaginal delivery appear to increase risk of development of new or worsening of existing symptoms, and it would be reasonable to advise caesarean delivery for these women.[16]

In conclusion

Women who sustain OASI and perineal injury can suffer from both short and long-term morbidity. These injuries require thorough assessment by trained clinicians and should preferably be referred to a dedicated perineal clinic. It is incumbent that all healthcare professionals involved in childbirth be familiar with basic anatomy of the perineum and anal sphincter, risk factors, and appropriate surgical treatment.

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