MALE AND FEMALE SURGICAL INTERVENTIONS (C CARSON AND P SELPH, SECTION EDITORS)

Medical and Surgical Management of Genital Lichen Sclerosus

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Abstract



Purpose of Review Lichen sclerosus (LS) is a chronic inflammatory skin condition that can cause debilitating sexual and urinary sequelae in men and women. In this review, we discuss the etiology, clinical presentation, and management options for genital LS. **Recent Findings** While medical and surgical management of LS has remained similar for quite some time, techniques such as the Kulkarni urethroplasty have made one stage reconstruction for panurethral LS strictures more feasible. Perineal urethrostomy has become an increasingly preferred treatment modality for complex LS patients. Current LS research has focused on the pathophysiology of the LS at the protein level and possible targets for treatment.

Summary While exact etiology of LS remains unknown, many theories have been hypothesized. The mainstay of medical treatment includes topical steroids. Various reconstructive techniques may be used depending on patient symptoms, extent of involvement, and the location of disease. Non-genital skin grafts, such as buccal mucosa, are recommended in LS patients requiring grafting for urethral reconstruction. Genital skin flaps are not recommended, as they have a high failure rate in this setting. Long-term follow-up is recommended, as LS can be associated with the development of squamous cell carcinoma.

Keywords Lichen sclerosus · Balanitis xerotica obliterans · Urethral stricture · Medical treatment · Surgical treatment

Introduction

Lichen sclerosus (LS) is a chronic, inflammatory skin disease that can affect any cutaneous area, however, mainly affects the anogenital region in both men and women. In males, penile LS have also been referred to as balanitis xerotica obliterans (BXO) and was first described by Stuhmer in 1928 [1, 2]. BXO typically presents with white penile lesions, plaques, and pruritus and can be associated with significant urinary and sexual morbidity. The etiology of LS is unclear, although autoimmune, chronic irritation and infectious mechanisms have all been proposed. Herein, we review the etiology, clinical presentation, and management options for genital LS.

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Epidemiology

LS can affect both women and men, with a stronger predilection toward women, estimated at 3:1 to 10:1 [3]. LS is most common in Caucasian patients, although can occur among any ethnicity. LS can occur at any age; however, the age of presentation is typically bimodal. In females, diagnosis classically is made in the pre-pubertal and post-menopausal ages (most common in postmenopausal) [1-3, 4•]. In males, presentation peaks early in childhood and then again later in adulthood [5, 6, 7..]. LS can be diagnosed by a number of different clinicians including primary care physicians, pediatricians, dermatologists, gynecologists, and urologists. The exact prevalence of LS is unknown and is likely underreported given asymptomatic presentations and a general lack of familiarity among non-specialist clinicians [8]. However, dermatology literature estimates the prevalence to be between 1:300 and 1:1000 [9].

Etiology

While the underlying etiology of LS is unknown, many theories have been hypothesized. There may be a genetic

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predisposition, as 10% of patients with LS have affected relatives [10]. The most common explanations include autoimmunity, chronic irritation, and infection.

Studies have suggested an immune etiology for LS. This has been proposed to be secondary to a localized loss of immune self-tolerance allowing a cell-mediated or humoral response to LS-specific antigens [11]. Organ specific antibodies have been found in patients with LS, and LS patients also have an increased incidence of other autoimmune disorders (vitiligo, alopecia, and diabetes).

Chronic irritation, typically from ongoing exposure of cutaneous surfaces to urine, may play a role in the development of LS. It has been hypothesized that urine and feces in occluded spaces may help lead to the development of LS in both men and women. This hypothesis is supported by the fact that LS is rarely seen in circumcised patients, and the localization of LS to occluded epithelium can be seen even in patients with perineal urethrostomies. It is thought that LS related inflammation is a result of the combination of occlusion, urinary exposure, and the phenomenon of koebnerization [12]. Also in contrast to women, men rarely have perianal disease, as the male perineum is not commonly exposed to urine [11, 13, 14].

Many infective agents have been linked to the development of LS. Acrodermatitis chronica atrophicans is caused by Borrelia burgdorferi, which shares clinical and histological features with LS. Extensive testing has not linked these conditions, however [15]. Multiple viral agents have also been investigated including Epstein Barr Virus (EBV), Hepatitis C Virus (HCV), and Human Papilloma Virus (HPV) [2, 16–18]. These infections have been found to be associated with LS in about 0–75% of cases, but currently there is insufficient evidence to conclude that any of these infections are contributing factors to the development of LS [11].

Histology and Diagnosis

LS is commonly diagnosed by clinical characteristics; histologic examination is not always essential. When biopsied, pathognomonic histological features include hyperkeratosis of the epithelium, hydropic degeneration of the basal cells, epidermal atrophy, follicular plugging, homogenized collagen in upper dermis with dermal edema, and lichenoid lymphocytic infiltrate [1, 2, 19]. Differential diagnosis includes mucosal or erosive lichen planus, eczema, vitiligo, morphea, plasma cell vulvitis/balanitis, vulval/penile intraepithelial neoplasia, and squamous cell carcinoma (SCC) [3]. Biopsy is indicated if 1. there is suspicion of neoplastic change 2. there is an area resistant to adequate treatment; 3. in cases of extragenital LS, with features of an overlap with morphea; 4. to exclude an abnormal melanocytic proliferation in pigmented areas; or 5. if second-line therapy is being considered [19, 20].

Risk of Malignant Transformation

The overall risk of progression of LS to SCC is estimated to be approximately 3–7% in females and 2–8% in males [1, 21–25]. LS can be identified in adjacent regions in over 60% of cases of vulvar SCC [26, 27]. LS is more common among uncircumcised males [22], although circumcised men can develop concealed or buried genital tissues prone to LS (and at risk for SCC) when lower abdominal obesity displaces the penopubic junction anteriorly. A high incidence of HPV 16 has been reported in patients developing penile SCC from penile LS [28]. In contrast, extragenital LS does not seem to carry a risk of malignant change [8].

Clinical Features

LS can have a benign or insidious course. While some may patients be asymptomatic for long periods of time, others will develop significant sexual and voiding dysfunction when untreated or even despite attempts at treatment. The most common clinical features at onset include white lesions, plaques, and erythema in men and atrophic skin, pruritus, and anogenital sclerosus in women [7]. Perianal disease is uncommon in men [29].

Male

LS typically affects the glans and/or foreskin in men. Progression of disease can lead to phimosis, thinning of the skin, and penile plaques, which can fissure during sexual activity [5]. Some men can develop acquired concealed or buried penis, which can be very debilitating. Meatal stenosis and urethral strictures are also commonly seen in men with LS. While urethral involvement typically starts at the meatus (Fig. 1), extensive disease can lead to mucosal involvement and spongiofibrosis proximally all the way to the posterior urethra (Fig. 2) [30]. Isolated bulbar urethral strictures secondary to LS have also been reported [31].

Female

LS in females typically affects the anogenital area. Patients commonly complain of pain in the vulvar and perianal areas, pruritus, dyspareunia, dysuria, and pain on defecation [32]. Over time, fissures and tears can develop, causing scarring and fusion of the labia, narrowing of the introitus, and burying of the clitoris [32]. In younger patients, symptoms may improve spontaneously at menarche [1].

Medical Management

Ultrapotent topical steroids are the first line of therapy in the non-surgical management of LS. The British Association of



Fig. 1 Meatal stenosis in a patient with LS

Dermatology recommends Clobetasol propionate 0.05% applied once a day for up to 3 months [7] with a success rate of approximately 50% [29]. Clobetasol was shown to be more effective treating vulvar LS than Tacrolimus in a double blind, prospective randomized study [33]. Unfortunately, no such study is available for patients with LS of the male genitalia. If no improvement is noted at 3 months, a biopsy should be considered [7].



Fig. 2 Panurethral stricture secondary to LS

Second line treatments include hormone therapy, calcineurin inhibitors, retinoids, and tumor necrosis factor (TNF) inhibitors. Good response to topical testosterone has been documented in vulvar LS [34, 35]. Others studies found testosterone to be no better than placebo [36]. Tacrolimus 0.1% and pimecrolimus, both calcineurin inhibitors, have shown response rates of 63% and 53%, respectively [37, 38]. The retinoid, acitretin, has shown to be effective with a response rate of 64% [39]. Adalimumab is a monoclonal antibody that inhibits TNF. Serial intralesional injections of Adalimumab led to the stabilization of severe and recurrent LS in one patient [40]. It is worth noting that many of these treatments have unknown side effects, including potential carcinogenesis, that have not been well studied.

Surgical Therapy

In patients that progress despite medical management or present with significant disease surgical treatment may be indicated. In women, surgical management typically entails procedures such as dissection of a buried clitoris, division of fused labia, or enlargement of a narrowed introitus [41]. Surgical treatment in male patients focuses on relieving urinary obstruction secondary to urethral strictures, eliminating cutaneous spaces prone to urinary sequestration, and alleviating pain with erections and/or intercourse.

Circumcision

Circumcision has an important role in the surgical management of early LS, especially in patients with disease limited to the glans and/or foreskin provided an adequate amount of uninvolved shaft skin is present. Depasquale et al. found that 92% of patients were successfully treated with circumcision alone and that recurrence can occur when residual moist skin folds are left or are unavoidable, such as in obese patients [21]. In those that have developed a buried penis, circumcision may not be possible, and more complex reconstruction techniques may be required.

In previously circumcised men with balanopreputial adhesions, patients may require excision and skin grafting [8]. If acquired buried penis has also developed, escutcheonectomy can be combined with excision and grafting [42]. Patients treated for LS should be continuously monitored throughout their life for recurrence of symptoms or for the development of SCC.

Meatotomy/Extended Meatoplasty

When LS causes isolated meatal stenosis, ventral meatotomy and extended meatoplasty are viable options. Because ventral meatotomy alone can result in restenosis, many advocate for an extended meatoplasty with creation of a hypospadias meatus (Fig. 3), especially if also associated with a fossa navicularis stricture [43]. Morey et al. found extended meatoplasty to be effective for refractory cases of fossa navicularis strictures in 14 of 16 patients (87%) with complex or reoperative strictures [44]. Malone also described a ventral and dorsal meatotomy with an inverted V-shaped relaxing incision with good results in LS patients [45].

Urethral Reconstruction

In severe or refractory cases of LS involving the anterior urethra, urethroplasty may be required. For short strictures limited to the bulbar urethra, excision and primary anastomosis can be performed. LS strictures, however, tend to be longer and involved the penile urethra, and thus grafting procedures are usually required. Genital skin-based repairs (graft and/or flap) should be avoided in LS strictures due to the risk of recurrence which approaches 100% [30, 46].

Buccal mucosa remains the preferred graft for urethral reconstruction for long and/or complex LS strictures, although bladder and rectal mucosa and tunica vaginalis have also proven to be effective in this setting [8, 21, 47]. For patients with non-obliterative strictures, one-stage repairs are possible by buccal graft onlay and have had good success in this patient population [48, 49]. The Kulkarni urethroplasty technique has



Fig. 3 Extended meatoplasty performed for treatment of a patient with meatal stenosis and fossa navicularis stricture secondary to LS $\,$

also been utilized with good results in patient with LS strictures that involve both the penile and bulbar urethra [49]. This technique includes penile invagination through a perineal incision, allowing access to the entire urethra without the need for a penile incision. A one-sided anterior dorsal buccal mucosal graft urethroplasty is then performed, preserving the contralateral vascular supply to the urethra [50].

When the urethral plate is not salvageable, a two-stage urethroplasty is necessary [21, 30, 43]. The first stage involves excising all diseased urethra and securing buccal mucosa graft to the tunica albuginea. After 6 to 12 months, a second stage procedure is performed, where the graft is tubularized. Success has been reported in up to 82% of cases [51]. Interestingly, some men will elect to not proceed with second stage reconstruction after 6 to 12 months, as they are content with the functional proximal urethrostomy created during the first stage [51].

Some patients are unwilling or are not fit enough to undergo complex urethral reconstruction. Perineal urethrostomy is a viable option for these patients, as many are already accustomed to sitting to void. Fuchs et al. evaluated trends in urethral reconstruction and reported perineal urethrostomy has increasingly become preferred for longer strictures, especially in those with adverse etiology, such as LS [52]. The Lahey group compared single-stage, two-stage, and perineal urethrostomy procedures for patients with LS-related strictures and reported the highest degree of success in patients who underwent perineal urethrostomy [53]. Perineal urethrostomy is usually performed by utilizing either an inverted "U" incision or midline perineal incision using a "7flap" [54, 55].

Current and Future Research

Contemporary LS studies have focused on the pathophysiology of the disease at the protein level and possible targets for treatment. Levy et al. studied differences in protein expression between strictures associated with LS and those that were not. LS associated strictures were longer in length and were associated with increased inflammatory markers. EBV RNA was more commonly identified among LS strictures [56•]. This study was recently followed-up with a comparison between LS strictures that recurred versus those that did not recur. A decrease in inflammatory markers in recurrent LS strictures may represent a transition to a static phase of impaired healing ability after surgical repair. An increase in VEGF was also noted in recurrent LS strictures [57]. Future research will likely focus on evaluating other protein targets, as well investigations into LS prevention and its impact on quality of life [58].

Conclusion

LS is a chronic inflammatory skin condition that can cause debilitating sexual and urinary sequelae in both men and

women. While the etiology remains unknown, genetics, autoimmunity, chronic irritation, and infection may all play a role in its development. As LS can be associated with the development of SCC, long-term follow-up is recommended. The mainstay of medical treatment includes topical steroids. Various reconstructive techniques may be used depending on patient symptoms, extent of involvement, and the location of disease. Non-genital skin grafts, such as buccal mucosa, are recommended in patients requiring grafting for urethral reconstruction.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflicts of interest.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal subjects performed by any of the authors.

References

Papers of particular interest, published recently, have been highlighted as:

- · Of importance
- •• Of major importance
- 1. Powell JJ, Wojnarowska F. Lichen sclerosus. Lancet. 1999;353(9166):1777–83.
- Val I, Almeida G. An overview of lichen sclerosus. Clin Obstet Gynecol. 2005;48(4):808–17.
- Kirtschig G, Becker K, Gunthert A, Jasaitiene D, Cooper S, Chi CC, et al. Evidence-based (S3) guideline on (anogenital) lichen sclerosus. J Eur Acad Dermatol Venereol. 2015;29(10):e1–43 This European guideline focuses on anogentical lichen sclerosis.
- Lipscombe TK, Wayte J, Wojnarowska F, Marren P, Luzzi G. A study of clinical and aetiological factors and possible associations of lichen sclerosus in males. Australas J Dermatol. 1997;38(3):132– 6.
- Das S, Tunuguntla HS. Balanitis xerotica obliterans-a review. World J Urol. 2000;18(6):382–7.
- Kiss A, Kiraly L, Kutasy B, Merksz M. High incidence of balanitis xerotica obliterans in boys with phimosis: prospective 10-year study. Pediatr Dermatol. 2005;22(4):305–8.
- 7.•• Lewis FM, Tatnall FM, Velangi SS, Bunker CB, Kumar A, Brackenbury F, et al. British Association of Dermatologists guidelines for the management of lichen sclerosus, 2018. Br J Dermatol. 2018;178(4):839–53 This guideline from the British Association of Dermatologists provides evidence-based recommendaitons for the management of LS.
- Pugliese JM, Morey AF, Peterson AC. Lichen sclerosus: review of the literature and current recommendations for management. J Urol. 2007;178(6):2268–76.
- 9. Wallace HJ. Lichen sclerosus et atrophicus. Trans St Johns Hosp Dermatol Soc. 1971;57(1):9–30.
- Kirtschig G. Lichen Sclerosus-presentation. Diagnosis and Management Dtsch Arztebl Int. 2016;113(19):337–43.

- Fergus KB, Lee AW, Baradaran N, Cohen AJ, Stohr BA, Erickson BA, et al. Pathophysiology, clinical manifestations, and treatment of lichen Sclerosus: A systematic review. Urology. 2019.
- Weigand DA. Microscopic features of lichen sclerosus et atrophicus in acrochordons: a clue to the cause of lichen sclerosus et atrophicus? J Am Acad Dermatol. 1993;28(5 Pt 1):751–4.
- CB B. Diseases and disorders of the male genitalia. In: Fitzpatrick's dermatology in general medicine, 7th ed. LA Goldsmith SK, BA Gilchrest, AS Paller, DJ Leffel, K Wolff, editor. New York: McGraw-Hill; 2007.
- Bunker CB. Male genital lichen sclerosus and tacrolimus. Br J Dermatol. 2007;157(5):1079–80.
- 15. Weide B, Walz T, Garbe C. Is morphoea caused by Borrelia burgdorferi? A review Br J Dermatol. 2000;142(4):636–44.
- Drut RM, Gomez MA, Drut R, Lojo MM. Human papillomavirus is present in some cases of childhood penile lichen sclerosus: an in situ hybridization and SP-PCR study. Pediatr Dermatol. 1998;15(2):85–90.
- Beattie PE, Dawe RS, Ferguson J, Ibbotson SH. UVA1 phototherapy for genital lichen sclerosus. Clin Exp Dermatol. 2006;31(3): 343–7.
- Neill SM, Lessana-Leibowitch M, Pelisse M, Moyal-Barracco M. Lichen sclerosus, invasive squamous cell carcinoma, and human papillomavirus. Am J Obstet Gynecol. 1990;162(6):1633–4.
- Fistarol SK, Itin PH. Diagnosis and treatment of lichen sclerosus: an update. Am J Clin Dermatol. 2013;14(1):27–47.
- Neill SM, Lewis FM, Tatnall FM, Cox NH. British Association of Dermatologists' guidelines for the management of lichen sclerosus 2010. Br J Dermatol. 2010;163(4):672–82.
- Depasquale I, Park AJ, Bracka A. The treatment of balanitis xerotica obliterans. BJU Int. 2000;86(4):459–65.
- Powell J, Robson A, Cranston D, Wojnarowska F, Turner R. High incidence of lichen sclerosus in patients with squamous cell carcinoma of the penis. Br J Dermatol. 2001;145(1):85–9.
- Bleeker MC, Visser PJ, Overbeek LI, van Beurden M, Berkhof J. Lichen Sclerosus: incidence and risk of vulvar squamous cell carcinoma. Cancer Epidemiol Biomark Prev. 2016;25(8):1224–30.
- Heymann WR. Lichen sclerosus. J Am Acad Dermatol. 2007;56(4):683–4.
- Thomas RH, Ridley CM, McGibbon DH, Black MM. Anogenital lichen sclerosus in women. J R Soc Med. 1996;89(12):694–8.
- Kagie MJ, Kenter GG, Hermans J, Trimbos JB, Fleuren GJ. The relevance of various vulvar epithelial changes in the early detection of squamous cell carcinoma of the vulva. Int J Gynecol Cancer. 1997;7(1):50–7.
- Leibowitch M, Neill S, Pelisse M, Moyal-Baracco M. The epithelial changes associated with squamous cell carcinoma of the vulva: a review of the clinical, histological and viral findings in 78 women. Br J Obstet Gynaecol. 1990;97(12):1135–9.
- Nasca MR, Innocenzi D, Micali G. Penile cancer among patients with genital lichen sclerosus. J Am Acad Dermatol. 1999;41(6): 911–4.
- Edmonds EV, Hunt S, Hawkins D, Dinneen M, Francis N, Bunker CB. Clinical parameters in male genital lichen sclerosus: a case series of 329 patients. J Eur Acad Dermatol Venereol. 2012;26(6): 730–7.
- Venn SN, Mundy AR. Urethroplasty for balanitis xerotica obliterans. Br J Urol. 1998;81(5):735–7.
- Liu JS, Walker K, Stein D, Prabhu S, Hofer MD, Han J, et al. Lichen sclerosus and isolated bulbar urethral stricture disease. J Urol. 2014;192(3):775–9.
- Yesudian PD, Sugunendran H, Bates CM, O'Mahony C. Lichen sclerosus. Int J STD AIDS. 2005;16(7):465–73, test 74.
- Funaro D, Lovett A, Leroux N, Powell J. A double-blind, randomized prospective study evaluating topical clobetasol propionate

0.05% versus topical tacrolimus 0.1% in patients with vulvar lichen sclerosus. J Am Acad Dermatol. 2014;71(1):84–91.

- Ayhan A, Guven S, Guvendag Guven ES, Sakinci M, Gultekin M, Kucukali T. Topical testosterone versus clobetasol for vulvar lichen sclerosus. Int J Gynaecol Obstet. 2007;96(2):117–21.
- Ayhan A, Guven ES, Guven S, Sakinci M, Dogan NU, Kucukali T. Testosterone versus clobetasol for maintenance of vulvar lichen sclerosus associated with variable degrees of squamous cell hyperplasia. Acta Obstet Gynecol Scand. 2007;86(6):715–9.
- Sideri M, Origoni M, Spinaci L, Ferrari A. Topical testosterone in the treatment of vulvar lichen sclerosus. Int J Gynaecol Obstet. 1994;46(1):53–6.
- Kim GW, Park HJ, Kim HS, Kim SH, Ko HC, Kim BS, et al. Topical tacrolimus ointment for the treatment of lichen sclerosus, comparing genital and extragenital involvement. J Dermatol. 2012;39(2):145–50.
- Goldstein AT, Creasey A, Pfau R, Phillips D, Burrows LJ. A double-blind, randomized controlled trial of clobetasol versus pimecrolimus in patients with vulvar lichen sclerosus. J Am Acad Dermatol. 2011;64(6):e99–104.
- Bousema MT, Romppanen U, Geiger JM, Baudin M, Vaha-Eskeli K, Vartiainen J, et al. Acitretin in the treatment of severe lichen sclerosus et atrophicus of the vulva: a double-blind, placebocontrolled study. J Am Acad Dermatol. 1994;30(2 Pt 1):225–31.
- Lowenstein EB, Zeichner JA. Intralesional adalimumab for the treatment of refractory balanitis xerotica obliterans. JAMA Dermatol. 2013;149(1):23–4.
- Rouzier R, Haddad B, Deyrolle C, Pelisse M, Moyal-Barracco M, Paniel BJ. Perineoplasty for the treatment of introital stenosis related to vulvar lichen sclerosus. Am J Obstet Gynecol. 2002;186(1): 49–52.
- Strother MC, Skokan AJ, Sterling ME, Butler PD, Kovell RC. Adult buried penis repair with Escutcheonectomy and Splitthickness skin grafting. J Sex Med. 2018;15(8):1198–204.
- Belsante MJ, Selph JP, Peterson AC. The contemporary management of urethral strictures in men resulting from lichen sclerosus. Transl Androl Urol. 2015;4(1):22–8.
- Morey AF, Lin HC, DeRosa CA, Griffith BC. Fossa navicularis reconstruction: impact of stricture length on outcomes and assessment of extended meatotomy (first stage Johanson) maneuver. J Urol. 2007;177(1):184–7 discussion 7.
- Malone P. A new technique for meatal stenosis in patients with lichen sclerosus. J Urol. 2004;172(3):949–52.
- Virasoro R, Eltahawy EA, Jordan GH. Long-term follow-up for reconstruction of strictures of the fossa navicularis with a single technique. BJU Int. 2007;100(5):1143–5.
- 47. Granieri MA, Zhao LC, Breyer BN, Voelzke BB, Baradaran N, Grucela AL, et al. Multi-institutional outcomes of minimally

invasive harvest of rectal mucosa graft for anterior urethral reconstruction. J Urol. 2019;201(6):1164–70.

- Dubey D, Sehgal A, Srivastava A, Mandhani A, Kapoor R, Kumar A. Buccal mucosal urethroplasty for balanitis xerotica obliterans related urethral strictures: the outcome of 1 and 2-stage techniques. J Urol. 2005;173(2):463–6.
- Kulkarni S, Barbagli G, Kirpekar D, Mirri F, Lazzeri M. Lichen sclerosus of the male genitalia and urethra: surgical options and results in a multicenter international experience with 215 patients. Eur Urol. 2009;55(4):945–54.
- Kulkarni S, Barbagli G, Sansalone S, Lazzeri M. One-sided anterior urethroplasty: a new dorsal onlay graft technique. BJU Int. 2009;104(8):1150–5.
- Peterson AC, Palminteri E, Lazzeri M, Guanzoni G, Barbagli G, Webster GD. Heroic measures may not always be justified in extensive urethral stricture due to lichen sclerosus (balanitis xerotica obliterans). Urology. 2004;64(3):565–8.
- Fuchs JS, Shakir N, McKibben MJ, Scott JM, Viers B, Pagliara T, et al. Changing trends in reconstruction of complex anterior urethral strictures: from skin flap to Perineal Urethrostomy. Urology. 2018;122:169–73.
- Patel CK, Buckley JC, Zinman LN, Vanni AJ. Outcomes for Management of Lichen Sclerosus Urethral Strictures by 3 different techniques. Urology. 2016;91:215–21.
- 54. French D, Hudak SJ, Morey AF. The "7-flap" perineal urethrostomy. Urology. 2011;77(6):1487–9.
- 55. Myers JB, McAninch JW. Perineal urethrostomy. BJU Int. 2011;107(5):856–65.
- 56.• Levy A, Browne B, Fredrick A, Stensland K, Bennett J, Sullivan T, et al. Insights into the pathophysiology of urethral stricture disease due to lichen Sclerosus: comparison of pathological markers in lichen Sclerosus induced strictures vs nonlichen Sclerosus induced strictures. J Urol. 2019;201(6):1158–63 This contemporary study is the first to investigate the pathophysiology of LS in urethral stricture disease at the protein level.
- Levy AC, Moynihan M, Bennett JA, Sullivan T, Stensland K, Browne BM, et al. Protein Expression Profiles Among Lichen Sclerosus Urethral Strictures: Can Urethroplasty Success be Predicted? J Urol. 2019:101097ju000000000000610.
- Simpson RC, Cooper SM, Kirtschig G, Larsen S, Lawton S, McPhee M, et al. Future research priorities for lichen sclerosus results of a James Lind Alliance priority setting partnership. Br J Dermatol. 2019;180(5):1236–7.

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