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Review article

Penile venous surgery for treating erectile dysfunction: Past, present, and future perspectives with regard to new insights in venous anatomy☆

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ABSTRACT

In the physiologic model of normal erectile function, a healthy veno-occlusive mechanism is essential to initiate and maintain a rigid erection. The surgical treatment of patients with venous leakage, which is synonymous with corporoveno-occlusive dysfunction (CVOD), was based on the decreased venous outflow during the erection process. The initial reports of short-term results were promising, but the long-term benefits of penile venous ligation surgery were limited. Most clinical guideline panels concluded that surgeries performed in an attempt to limit the venous outflow of the penis were not recommended. Consequently, this surgery was nearly abandoned in most medical societies worldwide. These unfavorable postoperative outcomes seemed attributable to the indispensable usage of electrocautery and insufficient venous management, based on conventional penile venous anatomy. Advances in better understanding of human penile venous anatomy has enabled the development of refined penile venous stripping surgery. The thorough stripping surgery is an even more radical procedure, which is an even more radical procedure, and seems to be a viable option for the treatment of CVOD, however, there is still a need for further study with well-defined diagnostic criteria, and standardized patient and partner outcome assessment.

1. Introduction

Erectile dysfunction (ED) is the persistent inability to attain and maintain an erection sufficient to permit satisfactory sexual performance. It is a common disorder worldwide and affects a high percentage of the male population. It remains a challenging medical problem, even after the introduction of phosphodiesterase 5 (PDE5) inhibitors, which became the first-line drugs for ED management in 1998.

Erectile dysfunction affects physical and psychosocial health and significantly impacts the quality of life of men and their partners. It is uncommon in young men, but more common in middle age, and is highly prevalent in men aged > 60 years. Thus, to some extent, ED is a natural expression of aging. Epidemiologic studies of ED suggest that approximately 5–20% of men have moderate to severe ED.[1]

Most patients with ED were previously believed to be psychogenic. This belief has given way to the realization that ED etiologically has an organic basis in most patients. Erectile dysfunction can be caused by vasculogenic, neurogenic, hormonal and/or psychogenic factors, and alterations in the nitric oxide/cyclic guanosine monophosphate pathway or other regulatory mechanisms, which result in an imbalance in corporal smooth muscle contraction and relaxation.[2]

Vascular abnormalities are the most common factors in patients with an organic etiology. There is no doubt that arterial inflow and sinusoidal relaxation are important in the erectile phenomenon; however, failure to trap cavernosal blood within the corpora cavernosa is a common cause of ED.[3] Penile corporoveno-occlusive dysfunction (CVOD), which is synonymous with venous leakage and venogenic ED, represents the most common vascular dysfunction,[4,5] and can be identified in up to 85% of men evaluated for ED, regardless of the age of the patient.[6,7] Most patients with vascular ED are in this group, however, a minority of especially

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younger patients may develop ED because of circumscribed acquired or congenital vascular abnormalities.

2. Physiology of penile erection, pathophysiology of CVOD, and diagnostic investigations

Human penile erection is likely a mechanical phenomenon in a peculiar vascular system, which is involved in three phases of the erectile phenomenon: (1) an increase in arterial inflow into the penis via paired cavernosal arteries; (2) sinusoidal relaxation and filling of the corporeal bodies from this increased arterial inflow: and (3) a decrease in the outflow from or trapping of blood within the corpora, thereby causing tumescence and ultimate rigidity. If all three of these vascular events work normally, an erection ensues. The venous closure mechanism has been described in anatomical dissections and physiological studies.[3] By contrast, if there is a defect in one or all three of these events, ED may occur.

Venogenic ED may result from an improperly functioning occlusion mechanism. Investigations stress the role of the tunica albuginea in the venous occlusion mechanism of the penis during an erection.[2,7,8] The location and degree of venous leak is variable and can occur anywhere along the tunica albuginea of the corpus cavernosum, which has been demonstrated in a canine model[8] and in ED patients.[9]

Recent studies suggest that, in most patients, CVOD results from endothelial dysfunction and damage to the trabecular smooth muscle content because of multifactorial degenerative processes.[10] Structurally based CVOD occurs in patients with ED who have a history of vascular risk factors and/or exposure to disorders that induce trabecular structural alterations such as veno-occlusive priapism, penile irradiation, and connective tissue disorders. Other investigators have reported patients with vasculogenic impotence who exhibit degeneration and atrophy of trabecular smooth muscle cells with increased fibrous connective tissue.[11,12] The possible causes of CVOD include: congenital vascular anomalies; trauma; arterial disease (e.g., hypercholesterolemia, arteriosclerosis); alterations in the cavernosal smooth muscle, trabeculae, or tunica albuginea; psychogenic factors; postpriapism; and unknown origin. It is a common belief that CVOD is an effect rather than a cause of ED.[11,12]

Objective vascular testing that provides a physiologic diagnosis may help direct appropriate therapy because not all patients respond adequately to oral ED therapy. Erectile hemodynamics is evaluated by duplex Doppler ultrasound or dynamic infusion cavernosometry. Diagnostic dynamic penile color-duplex ultrasound can assess arterial function and significant cavernosal venous leakage by measuring peak arterial flow and the calculated resistive index.[13,14] Localization and severity of veno-occlusive dysfunction may be determined by invasive dynamic intracavernosal cavernosometry and cavernosography.[15]

3. A historical overview: penile venous ligation surgery

In 1873, the Italian Francesco Parona injected the varicosity dorsal penile vein of an impotent young patient with hypertonic saline to cause sclerosis and thereby reduce excessive venous outflow. The concept that erectile disorders may be treated surgically by occluding venous channels from the penis was promoted as early as the turn of the 20th century.[16] Surgical ligation or resection of the dorsal vein was practiced by several American doctors [e.g., James Duncan (1895); Joe Wooten (1902); and particularly, Frank Lydston (1908), who reported 100 resections].[16] Beginning in the 1930s, Oswald Swinney Lowsley combined simple dorsal vein plication with a surgically more advanced perineal crural technique in which he plicated the bulbocavernosus and ischiocavernosus muscles with several mattress sutures.[16] After his initial report in 1935, he followed 273 patients from more than 1000 patients that he operated on in his later publication in 1953. After these techniques disappeared from the medical literature, the rebirth of COVD surgery began in the 1980s as a result of the new area of investigation of erectile physiology.[17]

3.1. Surgical technique

Using a parapenile inguinal incision, the shaft of the penis is delivered into the operative field in a gooseneck fashion. Buck's fascia is incised over the dorsum of the penis, the deep dorsal vein is identified and isolated. Care must be taken to avoid injuring the paired dorsal arteries and nerves. The deep dorsal vein is stripped from the symphysis pubis to 1 cm proximal to the corona, while its tributaries are transected and ligated. The suspensory ligaments are not divided and no attempt is made to ligate the cavernosal or crural veins.[18]

There has been some debate as to whether a standard surgical approach should be used, which involves ligating and excising the deep dorsal vein and its tributaries,[17,19,20] or whether the procedure should be directed specifically to the site of the leakage demonstrated on cavernosography.[21] Other investigators have routinely performed an even more extensive procedure by ligating the cavernosal and crural veins and the deep dorsal vein and its tributaries.

The technical goal of therapy addresses the identified malfunctioning or ectopic deep dorsal, crural, or cavernosal veins. The surgical procedure has, over time, been expanded from simple deep dorsal vein ligation to extensive surgical exposure and vein ligation, excision, crural placation, and spongiolysis performed alone or in combination.[22]

3.2. Outcomes of penile vein ligation surgery

Most studies have been retrospective analyses, and rely on the patient's report of his sexual functioning. No study has reported the use of standardized questionnaires for subjective improvement or has reported improvement in the quality of life as a result of the surgical intervention. There are generally no postoperative quantitative vascular assessments, except in patients with a poor response to the intervention.

The initial reports with short-term results were promising,[17,19,20,23] however, the long-term benefits of venous ligation surgery have been limited. Success rates within the 1st year range from 23% to 80%, but consistently decline on longer follow up.[18,24,25] There is a time-related decline in successful function, presumably through incomplete ligation, recanalization, cavernosa-spongiosum leak, inadequate selection of patients, or the unrecognized presence or progression of arterial pathology, and/or smooth muscle dysfunction.[17,19,20,26,27] Perioperative complications include wound infection, penile curvature, skin necrosis, painful erections, postoperative shortening of the penis, penile deformity, transient or irreversible numbness, lymphedema, and ligation of the penile artery by error.[26,28] Berardinucci et al[18] report complication rates of 15%, which include temporary paresthesia of the penis (12%), and adhesions between the skin and the penis, which result in variable degrees of penile deviation during erectile episodes. Freedman et al[21] described complications such as excessive penile edema (33%), hematoma (15%), excessive pain (17%), scarring requiring revision (2%), penile shortening (43%), and hypoesthesias (20%).

4. Important guideline publications

The diagnosis and treatment of venous insufficiency is increasingly questioned because it appears that “leakage” is a misnomer and represents an erroneous interpretation of a more fundamental problem related to alterations in the cavernosal tissue and the