Erectile Dysfunction Following Pelvic Fracture Urethral Injury

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ABSTRACT

Introduction: Although pelvic fracture urethral injuries (PFUIs) are rare, approximately half these patients will report erectile dysfunction (ED) after their injuries. The anatomic relations of the cavernosal nerves and arteries to the bones of the pelvis and the urethra put these structures at significant risk at the time of PFUI. This review examines the epidemiology, evaluation, and management of ED in this patient population.

Aim: To evaluate the epidemiology, pathophysiology, diagnosis, and management of patients with ED after PFUI.

Methods: A literature review was performed to identify articles on PubMed published before May 2017 addressing PFUI and ED.

Main Outcome Measures: Incidence, mechanisms, risk factors, evaluation, and management strategies of ED after PFUI were analyzed.

Results: Patients with pelvic fractures are at risk of post-injury ED, whereas those with PFUI appear to be at even higher risk. Different potential mechanisms contributing to the pathophysiology of ED in this setting have been described in the literature, including damage to the nervous supply to the penis, arterial insufficiency, and veno-occlusive dysfunction. However, there is a lack of consensus on the predominant etiology. Appropriate diagnostic evaluation can help to elucidate the underlying pathophysiology on an individual basis and can help guide management. Oral therapies, intracavernosal injections, and inflatable penile prostheses have shown great success. Furthermore, unlike patients with ED from other causes, select patients with isolated arteriogenic ED are potentially eligible for penile revascularization procedures.

Conclusion: Because most patients with pelvic trauma are younger than 40 years with a significant life expectancy, appropriate diagnosis and management of ED after PFUI can greatly improve quality of life and allow resumption of post-injury sexual function. Identification of the causative pathology can help tailor treatment on an individual basis. **Johnsen NV, Kaufman MR, Dmochowski RR, Milam DF. Erectile Dysfunction Following Pelvic Fracture Urethral Injury. Sex Med Rev 2018;6;114–123.**

Key Words: Pelvic Fracture; Urethral Injury; Erectile Dysfunction; Urotrauma; Urethral Trauma

INTRODUCTION

On an annual basis, blunt pelvic trauma constitutes nearly 10% of all traumatic injuries in the United States, with most of these patients younger than 40 years.[1,2] Although prior series have suggested higher frequencies, contemporary data estimate that of men with traumatic pelvic fractures, approximately 2% will have concomitant urethral disruption injuries.[1,3] Patients with severe pelvic trauma historically have high rates of inpatient mortality; however, improvements in motor vehicle safety and interdisciplinary trauma management have resulted in significantly improved survival and thus increased numbers of patients surviving with lifelong complications. As such, urologic involvement in the care of these patients has become vital not only in the acute care of pelvic fracture urethral injuries (PFUIs) but just as importantly in the long-term management of the sexual and urinary dysfunction that often follows. We review the epidemiology, evaluation, and management of erectile dysfunction (ED) in patients with PFUI.

ANATOMY

The presence of pelvic fracture after blunt pelvic trauma often serves as a marker of significant high-energy force and is frequently associated with high Injury Severity Scores representative of severe overall trauma.[3] The intimate relation of the bony pelvis with the urethra and the neurovascular supply of the penis makes these structures especially susceptible to injury in cases of severe pelvic trauma, presumably leading to an increased risk of post-injury ED.[4] For normal erections to occur, sexual

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stimulation triggers neurotransmitter release from the cavernous nerves, resulting in dilation of the cavernosal arteries and increased penile blood flow.[5,6] This influx of arterial blood causes expansion of the corporal sinusoids, which ultimately leads to compression of the subtunical venous plexuses and penile emissary veins, thus decreasing venous outflow. As a result, the corporal bodies engorge and erection ensues. Therefore, normal erections depend primarily on three factors (nervous system input, increased arterial inflow, and decreased venous outflow) that are at risk from severe pelvic trauma and any of which, if damaged, can significantly affect long-term sexual function.

Early work by Walsh and Donker[7] in the 1980s carefully traced the path of the cavernosal nerves as they branch from the pelvic plexus and pass posterolateral to the prostate. They demonstrated that the cavernosal nerve fibers run adjacent to the membranous urethra as it penetrates the urogenital diaphragm before entering each body of the corpora cavernosa. The close relation of the nerves not only to the membranous urethra but also to the prostatic apex puts these fibers at significant risk for injury during PFUI, when shearing forces to the pelvis are transmitted to the bulbomembranous and/or prostatomembranous urethral junctions, resulting in avulsion and transection of the urethra.[8,9] These fibers also are at risk of damage with injuries to the posterior pelvis and sacroiliac joint, because parasympathetic fibers originating from the S2 to S4 spinal nerve roots exit the pelvic plexus to supply the cavernous nerves.

The arterial blood supply to the penis originates from bilateral pudendal arteries branching along their respective internal iliac arteries. These pudendal arteries subsequently branch to provide the common penile arteries and then the cavernosal arteries bilaterally. In severe pelvic trauma, compression injuries or lacerations to the pudendal or even internal iliac arteries are common. These injuries can not only lead to massive hemorrhage but also result in irrecoverable arterial lesions or disruptions that affect the penile blood supply. Furthermore, in severely injured patients, extraperitoneal pelvic hemorrhage ultimately might require arterial embolization in the acute setting for damage control.[10–12] Although selective embolization of pelvic vessels during angiography is often performed after trauma, failure of initial embolization is common because of temporary vessel spasm cloaking an active bleed or significant venous extravasation that cannot be visualized by arteriography.[13] As such, nonselective bilateral internal iliac artery embolization has been advocated in select cases. Given the impact such embolization could have on long-term sexual function, previous studies have sought to evaluate the effect of internal iliac embolization on erectile function (EF). Ramirez et al[13] showed that there was no increased risk of postembolization sexual dysfunction between patients with pelvic fractures who underwent bilateral internal iliac artery embolization and patients with pelvic fractures alone. However, it should be noted that they studied patients embolized with a non-permanent gelatin slurry that eventually degraded over time, which likely allowed reconstitution of penile blood flow. In addition, using a matched cohort analysis, they found that the main determinant of post-injury sexual dysfunction was the presence of pelvic fractures, rather than the process of embolization. Nonetheless, as discussed below, a significant number of men with ED after PFUI will be found to have arterial insufficiency as the primary driver of their ED.

The mechanism behind failure of the veno-occlusive function of the corporal trabeculae in post-traumatic ED has been less extensively evaluated than that of the arterial and nervous components of ED after PFUI.[14–20] However, failure to identify veno-occlusive dysfunction in this patient population can lead to inappropriate treatment selection and unsatisfactory functional outcomes, especially for patients considering penile revascularization. As stated earlier, corporal veno-occlusion during erection is the result of penile smooth muscle relaxation and increased arterial blood flow.[18,19] Thus, the veno-occlusive function is dependent on trabecular fibroelastic compliance and trabecular smooth muscle tone.[15,20] The mechanism by which trauma results in veno-occlusive dysfunction is less clear, although previous researchers have hypothesized that traumatic injuries result in structural changes to the trabecular smooth muscle from direct injury and alterations in local wound repair. Such architectural changes lead to a functional inability to completely relax, expand, and compress the subtunical venules to prevent venous leak and maintain an erection.[14–18,20]

INCIDENCE

The true incidence of post-PFUI ED is difficult to estimate. This is due in part to the fact that preinjury EF is often poorly characterized and not well documented. Almost all studies that provide pre-PFUI data on EF obtained these data after the injury occurred and thus are significantly compromised by recall bias. Similarly, there is immense variation in the literature with regard to reported ED rates after PFUI. This variation is due in part to variable definitions of ED among studies, differences in timing from injury to data collection, and variability in the severity of trauma and concomitant injuries experienced among patients. Similarly, although some studies have suggested that patient self-assessment of EF correlates well with objective data obtained by validated instruments such as the International Index of Erectile Function (IIEF), there remains a degree of variability related to subjective reporting that makes determination of the true incidence more difficult.[21] Several prior studies have estimated that the incidence of ED after pelvic fracture alone is approximately 5% to 20%, whereas the incidence in patients with PFUI increases to as high as 42% to 62%.[22–26] A recent meta-analysis from Blaschko et al[27] estimated that the overall rate of ED after PFUI was 34%. However, of the 14 studies included in this analysis, the rate of ED ranged from 0% to 100%, making true estimation of the incidence less clear.

There also remains debate as to whether the primary management strategy for acute PFUI affects the long-term incidence of

ED. Currently, patients presenting with PFUI are generally managed by one of two methods: primary endoscopic realignment of the urethra through a retrograde or combined retrograde and antegrade approach with placement of an indwelling urethral catheter or suprapubic catheter placement with delayed urethroplasty for the inevitable fibrotic distraction defect that occurs. Proponents of endoscopic realignment argue that it provides decreased rates of stenosis, shorter strictures when they do occur, and decreased need for major urethral reconstruction.[28–33] Proponents of suprapubic catheter placement and delayed urethroplasty argue that given the high rates of failure of endoscopic realignment in preventing significant urethral stenosis (45–79%), realignment only serves to delay the time to definitive urethral reconstruction in most patients and potentially increases the risk of failure of subsequent repairs.[34,35]

Previous research has attempted to determine whether it is the mechanism of injury or the means of management that has the greater effect on incidence of ED in this patient population. Kotkin and Koch[33] suggested, based on review of 32 patients with PFUI managed at their institution, that the injury itself, rather than the means of acute management, led to post-injury ED. However, the methods used for realignment at the time of that study are outdated and not regularly used in the developed world. Nonetheless, because currently used endoscopic methods appear to be less traumatic to the urethra and surrounding tissues than methods used in the study by Kotkin and Koch, this research does support the notion that realignment is unlikely to negatively affect EF rates postoperatively.

Multiple single-institution reports exist in the literature quoting rates of 21% to 55% of ED after endoscopic realignment.[29–31,36,37] Research from our institution showed relatively higher rates of post-PFUI ED in the realigned and suprapubic catheter groups (78% and 90%, respectively), although this difference was not statistically significant.[28] A meta-analysis performed by Barrett et al[38] supported this finding and showed no difference in rates of ED between patients managed with early urethral realignment and those managed with suprapubic catheter placement alone (odds ratio [OR] = 1.19, 95% CI = 0.73–1.92). Interesting to note, however, is that like most of the currently available literature evaluating ED after PFUI, no study included in the previously cited meta-analysis used validated questionnaires to objectively evaluate and quantify the degree of ED from all categorizations of patient-reported ED. As such, rates of ED are not just variable but subjective and often based on a patient's desire to pursue treatment. This omission leaves room for future research to obtain robust data on this patient population to better elucidate the true incidence of ED associated with PFUI.

RISK FACTORS

Different studies have attempted to predict which patients are at higher risk for development of ED after PFUI. In a series of 292 patients with pelvic fracture, Wright et al[39] found that the presence of sacroiliac fractures resulted in a significantly increased risk of sexual dysfunction in men 1 year after their injuries (relative risk [RR] = 4.0, 95% CI = 2.3–6.8). However, this study excluded patients with coexistent genitourinary injuries and did not specifically define the type of sexual dysfunction experienced, but rather only asked patients whether, as a result of their injury, they were physically limited in their ability to have sexual relations. However, as stated earlier, the rates of ED in patients with PFUI are higher than in those with pelvic fracture alone, so the true risk that sacroiliac fractures portends for those with PFUI is not clear from these data. Similarly, because previous research has shown that patients can continue to recover spontaneous sexual function for up to 2 years after injury, the short 1-year follow-up could overestimate the true long-term impact.[26,40]

Other studies have specifically evaluated patients with PFUI to determine predictors of ED.[41,42] In one study by Koraitim,[41] 90 adult patients with traumatic PFUI were evaluated using the IIEF questionnaire. Koraitim reported that 40 patients (44%) exhibited various degrees of ED, with more than half these patients having severe ED (defined as IIEF score = 6–10). On multivariate analysis, the presence of pubic diastasis, lateral prostatic displacement, and long urethral gap were found to be significant predictors of ED after PFUI. Pubic diastasis alone was found to confer the highest risk of ED (OR = 15.89, 95% CI 1.9–131.6). Data by Feng et al[43] supported this finding, showing that in their series of 40 patients pubic diastasis was significantly associated with ED. Lateral prostatic displacement was found to be the second most highly associated predictor in the multivariate model. This relation was hypothesized to be due not only to direct trauma to the neurovascular bundle with significant displacement but also to dense adhesions that form during healing, entrapping the nerves.[44] Indeed, other prior studies have used magnetic resonance imaging to show a link between lateral prostatic displacement and increased rates of ED.[44,45] Further, a "long urethral gap" was found to confer two times the risk of ED after PFUI in this study (OR = 2.0, 95% CI = 1.1–3.6); the mean urethral gap in patients without ED after PFUI was 2.2 ± 1.3 cm compared with 3.3 ± 1.4 cm in the ED group ($P = .001$).

These data are quite similar to those reported by Koraitim,[42] demonstrating that the average urethral gap in potent patients was 2.5 vs 4.0 cm in those with ED. This relation is likely related to the fact that the degree of force and trauma required for significant urethral disruption injuries similarly results in significant damage to the neurovascular bundles that lie in close proximity. These data have been further supported by subsequent work in children who had experienced PFUI before puberty and were evaluated after puberty to determine EF.[42] Koraitim similarly found that increased urethral gap (≥2.5 cm) and lateral prostatic displacement were significantly associated with increased risk of ED at multivariate analysis.

At our institution, we have sought to evaluate risk factors for ED after PFUI in patients who were treated for their initial injuries and their subsequent care within our facility. We identified 30 patients with a mean follow-up of 37 ± 12.9 months who had sufficient follow-up data for inclusion. No particular fracture pattern, admission demographic, age, or acute management strategy was found to be significantly associated with development of ED. However, patients with higher-grade urethral injuries (grade 4 or 5, as determined by the American Association of Surgery for Trauma Urethral Trauma Grading Scale) had a significantly higher likelihood of ED compared with those with lower-grade injuries (unpublished data). This finding appears to corroborate that of the studies discussed earlier that higher-grade injuries (such as those that result in increased urethral gaps and lateral prostatic displacement) are more likely to result in significant neurovascular injuries and long-term ED after PFUI.

PATHOPHYSIOLOGY

Proposed causes of ED after PFUI include vasculogenic and neurogenic etiologies. As such, different studies have attempted to delineate the pathophysiologic factors involved and evaluate specific etiologies of ED to more appropriately guide treatment options. It should be noted that psychogenic ED is not uncommon in these patients, especially after significant trauma and injury, but has been very poorly evaluated in the literature. Providers should be aware of the possibility that patients have a significant psychogenic component of their ED and appropriately screen for such before further diagnostic and therapeutic procedures. However, for those with organic ED, there appears to be a lack of consensus as to the most common etiology. Feng et al[43] evaluated 40 men from their institution with ED after PFUI. Using nocturnal penile tumescence (NPT) testing, they found that only 11 men (28%) had evidence of organic ED. Interestingly, 8 of the 11 (72%) had normal vascular response at color-duplex ultrasonography after injection of vasodilatory agents, with only 3 patients having demonstrable evidence of significant vascular pathology. This finding suggested that most men in this study had a neurogenic etiology of ED. Interestingly, Feng et al noted that patients who had pubic diastasis with their initial injuries were more likely to show a lower peak systolic velocity at color-duplex ultrasound than those without and were more likely to have a vascular etiology for ED.

Different studies have supported this hypothesis that, in a large proportion of men, ED after PFUI is secondary to neurogenic causes. Mark et al[25] reported a relatively high rate of response to intracorporeal injection (ICI) therapy, suggesting neurogenic impotence as the most common ED etiology. Machtens et al[46] evaluated 31 patients with ED treated at 10 trauma centers in Europe for pelvic fractures and found that only 4 patients had vascular lesions resulting in arteriogenic ED, similar in frequency to that seen in other studies.[46–48] Interestingly, using electromyographic studies, they found further evidence that although a number of patients might display extensive vascular lesions, long-term ED frequently appears secondary to autonomic cavernosal nerve damage rather than arterial insufficiency. They further suggested that the ability to maintain normal arterial inflow to the penis despite damage to the internal iliac or pudendal arteries might be secondary to the presence of accessory penile arteries found in up to 70% of men.[46] However, Mundy[22] stated that most men (80–85%) with ED after PFUI have a vasculogenic etiology as the primary driver of their ED. This was supported by Morales et al[49] who showed that only 18% of patients had a complete response to ICI and 27% had a partial response, suggesting that the arterial inflow or venous outflow mechanisms were the primary obstacles to achieving adequate erections after injury.

However, Guan et al[50] performed one of the most comprehensive evaluations of ED after PFUI by evaluating a large cohort of 120 patients over a 5-year period. All patients filled out IIEF questionnaires and underwent NPT testing to objectively evaluate their ED. Patients with abnormal NPT results went on to have duplex ultrasonography testing after injection of vasodilatory agents. A peak systolic velocity less than 25 cm/s was deemed to indicate significant arterial insufficiency, whereas an end-diastolic velocity greater than 5 cm/s and a resistance index less than 0.85 suggested veno-occlusive dysfunction. Furthermore, for those with a high suspicion for venogenic ED, cavernosography was performed to evaluate for venous leak. All patients with abnormal NPT results also underwent neurophysiologic testing with posterior tibial somatosensory nerve evoked potentials, pudendal nerve evoked potentials, and bulbocavernosus reflex testing to evaluate for neurogenic dysfunction. At the end of the study, Guan et al found that 80% of the 120 patients had organic ED as indicated by an abnormal NPT result. Of these, 30% were found to have isolated vasculogenic ED, 43% were found to have neurogenic ED, and 27% were found to have mixed vasculogenic and neurogenic ED. Interestingly, of those with vasculogenic ED, most (56%) showed evidence of isolated veno-occlusive dysfunction, whereas only 13% had isolated arterial insufficiency (the remainder had mixed arteriogenic and venogenic dysfunction). Taken together, these data suggest that neurogenic ED is quite common after PFUI, whereas those who experience vasculogenic ED are more likely to have veno-occlusive dysfunction than arterial insufficiency.

When these data are examined in total, it is clear that there is significant variability in determining the exact etiology of ED after PFUI, suggesting that ED after PFUI is not due to a single etiology but likely presents a collection of injury patterns affecting at least one pathway involved in normal EF. This lack of consensus has led to further variability in patient evaluation before embarking on a management strategy. However, proceeding down a stepwise algorithm in determining the cause of an individual patient's ED after PFUI might allow providers the ability to tailor management to fit a particular patient and his underlying pathophysiology.

CLINICAL EVALUATION

The evaluation of patients with ED after PFUI should include a thorough history including mechanism of injury, associated injuries (especially neurologic injuries), comorbidities, and current medications. Baseline psychological evaluation also should be considered because of the risk of psychogenic ED after severe traumatic injuries of any type. A patient's ability to obtain erections at night or in the early morning is strongly suggestive of psychogenic ED and should prompt a more thorough psychological evaluation rather than continuing down a path of identifying organic causes. Attempts also should be made to determine preinjury function, although these data are likely subject to significant recall bias, as discussed earlier, because these data could provide information as to the etiologies involved.

In general, patients who are eligible should be trialed on oral phosphodiesterase type 5 inhibitors (PDE-5i) before more involved testing because these medications are generally well tolerated with minimal adverse effects. For those with failed oral therapies, further evaluation should first attempt to separate organic from psychogenic causes of ED. NPT testing has long been used for this purpose.[51,52] A normal NPT result historically was defined as one in which the patient had four to five erections per night, with a mean duration longer than 30 minutes and a maximal rigidity greater than 70% at the base and the tip.[53] However, more recent studies have used the criterion of just a single erection lasting at least 10 minutes with a minimum of 60% rigidity at the tip of the penis as normal.[54,55] The ability of a patient to obtain sustained nocturnal erections in the absence of awake, spontaneous erections is relatively specific for psychogenic ED and should prompt further psychological evaluation.

Artificial erection testing using ICI of vasodilatory agents is often helpful as a next step in evaluation for several reasons. First, ICI allows for providers to examine the erect penis to identify any deformities that might be present after the initial injury and to evaluate for curvature that might influence future treatment directions. Second, evaluating for appropriate response to injection therapy provides some degree of evidence as to the etiology of ED, because patients without a response likely have a significant vascular component of their underlying pathology.[46,50,51,56] ICI also is useful because it can be coupled with duplex ultrasonography to evaluate vascular parameters at the time of erection. A decrease in peak systolic velocity to less than 25 cm/s suggests decreased arterial inflow, whereas an increased end-diastolic velocity could suggest veno-occlusive dysfunction.[57–60]

Cavernosometry and cavernosography are less frequently used in contemporary practice but previously were performed regularly in select patients with suspected significant venous leak. This technique involves injection of saline and contrast directly into the corporal bodies with measurement of intracavernous pressures and direct radiographic evaluation of contrast distribution. Veno-occlusive dysfunction is indicated by a rapid decrease in intracavernous pressures after cessation of saline infusion or the inability to increase intracavernous pressures to mean systolic pressure.[61,62] This rather invasive diagnostic study is generally reserved for those with significant arterial insufficiency being considered for penile revascularization.

Similarly, penile angiography is reserved for those who have isolated arterial insufficiency at duplex ultrasonography after pelvic trauma with no evidence of venous leak and is intended to evaluate for anatomic lesions amenable to revascularization procedures. In this procedure, the internal pudendal artery is selectively cannulated and contrast is injected, often with concomitant injection of a vasodilatory agent, in the corpora cavernosa to obtain maximal vasodilation of the penile arterial supply. This allows for identification of distinct vascular lesions that might be bypassed with revascularization procedures. Of note, the inferior epigastric arteries also are regularly interrogated, because these are most often used for revascularization.

It should be noted that different investigative techniques have been developed to evaluate for neurogenic etiologies of ED.[50] These tests have been designed to evaluate the neurologic innervation of the corpora cavernosa on the somatic and autonomic pathways. However, to date, these techniques have had limited impact in the evaluation and management strategies used for ED after PFUI and are generally reserved for research, because they rarely, if ever, affect treatment decisions.[63,64]

MANAGEMENT

Once the particular pathophysiology of ED has been characterized, therapeutic options for management of ED after PFUI are similar to those used for non-trauma–related ED. As stated earlier, an initial trial of an oral PDE-5i is a reasonable first treatment choice. Shenfeld et al[65] evaluated 15 patients with ED after PFUI treated with sildenafil 100 mg and noted that 47% reported a favorable response with achievement of erections satisfactory for intercourse. On subgroup analysis, 60% of patients with neurogenic ED (as defined by normal vascular parameters on penile duplex ultrasound) and 20% of patients with arteriogenic ED demonstrated satisfactory responses. These data correlate with unpublished data from our institution, which found that 52% of the 30 men with ED after PFUI evaluated reported satisfactory responses to oral therapies alone. However, Fu et al[66] evaluated 41 patients treated with posterior urethroplasty for PFUI who had concomitant ED. They treated patients with sildenafil 100 mg and reported an overall success rate of 81%, with best efficacy noted in patients younger than 40 years. However, the success rates for patients with arteriogenic ED, venogenic ED, and neurogenic ED were 75%, 33.3%, and 80%, respectively. Taken together, these data suggest that patients with a neurogenic etiology are more likely to respond to oral PDE-5i therapy than those with a vascular etiology.

For those not responding to PDE-5i, ICI is often the next option, particularly for those with a neurogenic etiology. Of the

eight patients in the study by Shenfeld et al[65] who did not respond satisfactorily to PDE-5i, six (75%) responded to ICI (four with neurogenic ED, two with arteriogenic ED). Mark et al[25] found that 24 of 27 patients (89%) with ED after PFUI had adequate response to ICI after perineal repair of urethral distraction defects. However, like PDE-5i, ICI relies on an intact vascular supply and adequate venous outflow obstruction for a satisfactory response. For those who do not adequately respond, a vacuum erection device and a constriction band might be used alone or in addition to medical therapy to improve results. Although data in this population are lacking, intraurethral suppositories with vasodilatory agents could have a role in patients with ED after PFUI and should be considered before more invasive treatment options, if desired.

For patients presenting with significant veno-occlusive dysfunction, surgical attempts to limit venous leak are infrequently used as our understanding of the pathophysiology has developed. Prior attempts at surgical ligation of the extracorporal veins have been shown to be ineffective, because veno-occlusive dysfunction is the result of damage to the penile smooth muscle and endothelium, rather than to individual penile veins.[67–69] As such, the American Urological Association guidelines on ED recommend against venous ligation procedures for ED.[70] These patients are likely best managed with placement of an inflatable penile prosthesis (IPP) should they not respond to oral therapies or ICI.

Penile arterial revascularization has been shown to have a role in treating ED after PFUI unresponsive to lesser invasive therapies and in whom appropriate diagnostic evaluation has demonstrated an isolated arterial lesion and no evidence of venous leak.[67] This technique is an appropriate option only for young men with a history of trauma and isolated arteriogenic ED with focal endothelial dysfunction. Various techniques for penile revascularization have been described in the literature, with most involving anastomosis of the inferior epigastric artery to vessels of the penis, bypassing the obstructed portion of the internal pudendal or common penile arteries.[69] Most often, the inferior epigastric artery is anastomosed to the dorsal penile artery, with the intent that increased dorsal arterial perfusion pressures and flow will result in increased cavernosal artery perfusion through perforating branches between the dorsal penile artery and cavernosal artery.[67,69,71,72]

Zuckerman et al[73] reviewed their series of patients undergoing penile revascularization specifically for ED after PFUI using an end-to-side technique of the inferior epigastric artery to the dorsal penile artery. At a mean follow-up of 3 years, 11 of 13 patients (85%) reported success, defined as the ability to participate in intercourse with or without pharmacologic assistance. This distinction is important because, as discussed earlier, ED in this setting is often multifactorial and, despite correction of arterial insufficiency, many patients will have a coexistent neurogenic component. Kawanishi et al[74] reported on a large contemporary series of penile revascularization with 5-year follow-up. Using a study end point of sustained erections sufficient for intercourse without additional treatment, 85.9% and 67.5% of patients reported satisfactory responses at 3 and 5 years, respectively. However, this study was relatively selective in its inclusion criteria and included only patients younger than 50 years with no history of diabetes, hypertension, or hyperlipidemia and who had localized arterial lesions identified at arteriography. As such, Kawanishi et al stressed that revascularization surgery should be offered only to young, healthy men and that long-term functional and adverse outcomes remain unknown. To this end, a meta-analysis by Babaei et al[75] reviewed 25 studies involving penile revascularization for ED and found that men younger than 30 years responded better overall than older patients (OR = 1.8, 95% CI = 2.2–6.4).

Despite the reported successes of penile revascularization in these very select patients, complications are not uncommon. Glans hyperemia, decreased penile sensation, pain, and decreased penile length have been reported throughout various revascularization studies.[69,71,73,75–77] One recent report even described a new phenomenon of penile artery shunt syndrome resulting in persistent ED after revascularization.[78] However, penile revascularization is not a very widely used management option for ED after PFUI despite the relatively good success in well-selected patients. This is in part because patients with pure isolated arteriogenic ED with focal lesions are rarely identified, in part because appropriate arteriography is often difficult to perform and operator dependent, and, in part because the success rate and simplicity of PP placement has generally shunted many eligible candidates to implant procedures instead.[79,80]

Penile implant surgery has become the standard third-line treatment for management of ED of any etiology, including ED after PFUI, in patients whose therapies have failed or who refused more conservative therapies. In the industrialized world, IPPs are used in more than 80% of implant cases and are considered the gold standard of penile implants.[81] The IPP boasts one of the highest overall patient satisfaction rates for all ED treatments, with some studies finding greater than 90% satisfaction.[82,83] However, it should be noted that satisfaction rates in patients with significant corporal fibrosis and penile shortening, as can be seen occasionally after traumatic injuries and urethral interventions, can be lower than those in the general population.[84,85] Although these devices, like any medical implants, are subject to complications such as infection and malfunction, improvements in antibiotic coating and engineering have significantly improved device quality and decreased complications.[86] Infection rates have decreased considerably from 0.5% to 3.5% because of newer antibiotic and hydrophilic coatings.[87–90] Similarly, long-term device survival at 10 years ranges from 59% to 78%, and mechanical survival at 5 years is 88% to 98%.[89,91–94] Taken together, these data show not just high satisfaction rates but also reliable outcomes in patients undergoing IPP placement and, as such, this treatment should be strongly considered for patients with ED with any etiology after PFUI for whom more conservative management options have failed.[79]

CONCLUSIONS

ED after PFUI is a relatively common occurrence. Because most patients experiencing pelvic trauma are younger than 40 years with a significant life expectancy, appropriate diagnosis and management of ED after PFUI can greatly improve quality of life and allow resumption of post-injury sexual function. ED is much more common in patients with PFUI than in those with pelvic fracture alone and these patients should be followed closely to provide appropriate counseling and treatment as desired. ED after PFUI can be arteriogenic, neurogenic, or venogenic or a combination of etiologies. Management strategies should be tailored to the underlying pathophysiology identified at diagnostic workup and interventions should be recommended on an individual basis.

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