DOI: 10.4081/aiua.2025.13383

ORIGINAL PAPER

Factors associated with erectile dysfunction in traumatic urethral strictures following EPA urethroplasty: A single center experience

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Summary ***Introduction: Urethral repair with Excision and Primary Anastomosis (EPA) urethroplasty offers excellent outcome in managing traumatic urethral strictures. However, its impact on erectile function (EF) is largely unknown. Study to evaluate EF outcome post-operatively is still limited worldwide. We report factors associated to EF following EPA Urethroplasty performed by single surgeon in tertiary hospital. In this study, we aim to evaluate the risk of erectile dysfunction (ED) following EPA Urethroplasty.***

Materials and methods: This is a retrospective study on patients with traumatic urethral strictures who underwent EPA Urethroplasty from 2013 to 2023. Variables including age, body mass index, systemic disease, etiology, stricture length, prior procedures and erection hardness score (EHS) score prior and 12 months after surgery were recorded. Pre-Operative ED was determined using Penile Doppler Ultrasound, which was defined as a peak systolic velocity of less than 25 cm/s. Univariate and Multivariate logistic regression analysis were performed using IBM SPSS Statistic.

Results: A total of 89 patients were included. Among them, 33 patients (33.7%) suffered from initial ED prior to surgery. Pelvic fracture urethral injury (PFUI) was the predominant etiology (74%); 29% of the patients were active smokers, and 68.5% had prior endoscopic treatment. Among the 48 patients without ED prior to surgery, 7 of them (14.6%) developed ED following surgery in 12 months of follow up. After EPA, there was a reduction of mean EHS score from 2.70 to 2.53 (p = 0.176). Multivariate analysis showed that smoking status (p = 0.035; OR 4.41), PFUI as the mechanism of injury (p = 0.007; OR 2.89), prior urethrotomy (p = 0.020; OR 4.69), and prior dilatations (p = 0.046; OR 0.18) were related as risk factors of ED following EPA urethroplasty.

Conclusions: Risk of ED following EPA is inevitable, although the number is not high as expected. Smoking, PFUI and prior treatment rather than EPA, emerge as predominant risk factors associated with the development of ED subsequent to surgical repair.

KEY WORDS: EPA urethroplasty; Erectile dysfunction; Traumatic urethral strictures.

Submitted 14 November 2024; Accepted 2 December 2024

INTRODUCTION

The rate of traffic accidents in Indonesia is alarmingly high, which significantly impacts the incidence of pelvic trauma. A study on pelvic fractures in Indonesia from 2016 to 2018 involved 229 patients with pelvic injuries that lead to developing of traumatic urethral strictures especially *Pelvic Fracture Urethral Injury* (PFUI). Traffic accidents were responsible for 76% of these cases (1-6).

Excision and Primary Anastomosis (EPA) Urethroplasty is a reconstructive technique for traumatic urethral strictures, involving the complete removal of the affected urethral segment followed by tension-free anastomosis that offering excellent clinical outcomes (7). However, the impact of EPA Urethroplasty on *erectile function* (EF) has raised concerns. Existing studies on *erectile dysfunction* (ED) both prior and after EPA Urethroplasty for traumatic urethral strictures are limited. Research evaluating EF outcomes post-operatively remains scarce worldwide. The exact cause of ED in men following urethroplasty remains poorly studied (8, 9).

There is lack of available data in Indonesia concerning about EF following such procedure. This gap in information limits the understanding of the risks and effectiveness of this interventions. Our study aims to address this gap by examining the factors associated with EF following EPA Urethroplasty performed by a single surgeon at a tertiary hospital.

METHODS

Study sample and design

This study employs a retrospective design focusing on patients with traumatic urethral stricture who underwent EPA Urethroplasty at *Dr. Saiful Anwar General Hospital in Malang*, a tertiary referral hospital. Study sample consists of patients with traumatic urethral stricture who received EPA-Urethroplasty treatment at *Dr. Saiful Anwar General Hospital* between 2013 and 2023. The inclusion criteria for this study are as follows (1). Male patients diagnosed with traumatic urethral stricture, confirmed through *Bipolar Voiding Cysto-Urethrography* (BVCUG); (2) Patients with no prior EPA Urethroplasty Repair. The exclusion criteria for this study are (1) Male patients with urethral stricture caused by factors other than trauma; (2) Patients loss of follow-up before 12 months; (3) Patient who underwent Redo-Urethroplasty.

The research utilizes medical records to evaluate sexual function outcomes before and after the procedure. The variables recorded in this study include age, BMI, systemic diseases, etiology, stricture length, prior procedures, and EHS scores both prior to and 12 months after surgery. We excluded all the patients who suffered pre-operative ED from the study. Pre-operative ED was determined using Penile Doppler Ultrasound, and a Peak systolic velocity of less than 25 cm/s indicate initial ED. Postoperative ED was evaluated using the *Erection Hardness Score* (EHS) and the *International Index of Erectile Function* (IIEF-5) until 12 months following EPA Urethroplasty procedure.

Postoperative follow-up and outcome definition

We followed all the patients with no prior ED following surgery up to 12 months of follow-up using EHS score and IIEF-5 assessment at 1, 3, 6 and 12 months following the surgery. We included and analyzed all the patient who develop ED during this period of follow-up.

Statistical analysis

Univariate and multivariate logistic regression analyses were performed using IBM SPSS Statistics to analyze the data. This statistical approach allowed for the examination of potential predictors and the impact of various factors on the outcomes of ED in patients undergoing EPA-Urethroplasty.

Results

Characteristics of the research sample

The study included 89 patients with a mean age of 41.24 years (SD ± 15.59). The median follow-up period was 16.3 months (SD ± 3.15). The mean BMI was 23.05 kg/m² (SD ± 2.57). Among the patients, 29.2% were current smokers, 10.1% had type 2 diabetes mellitus, and 16.9% had hypertension. The average length of stricture was 25.4 mm (SD ± 16.3), with the mean time to surgery being 6.67 months (SD ± 4.07). Types of injuries included straddle injury (12.35%), iatrogenic (11.23%), direct (2.24%), and PFUI (74.15%). The stricture site was bulbar in 62% of cases and membranous in 27% of cases. There were 34 patients (38.20%) who had performed internal urethrotomies and 33 patients (37.07%) had performed urethral dilatation prior to surgery. Fourteen patients (15.73%) did not undergo any types of endoscopic treatment following the surgery. Types of surgery performed were bulbar mobilization (39.33%), crural separation (32.58%), inferior pubectomy (26.96%), and supracrural rerouting (1.12%). The success rate of EPA was 91%, with an 8% recurrence rate. ED was noted in 40.74% of patients pre-operatively, and 49.39% post-operatively (Table 1). Among the 48 patients without ED prior to surgery, 7 of them (14.6%) developed ED post-operatively.

Erectile function outcome following EPA urethroplasty

The statistical analysis was performed using the Mann-Whitney test. The mean EHS Score decreased slightly from 2.70 (SD ± 0.798) pre-operatively to 2.53 (SD ± 0.823) at 12 months post-operatively, but this change was not statistically significant (p = 0.176). Similarly, the mean IIEF-5 score showed a minor increase from 19.35 (SD ± 4.547) to 19.73 (SD ± 4.174) over the same period, which was also not statistically significant (p = 0.444) (Table 2 and Figure 1).

Table 1.
Patient characteristics.

Number of patient	89	
Age (year) (SD)	41.24	(± 15.59)
Median follow-up (months) (SD)	16.3	(± 3.15)
BMI (kg/m^2), mean (SD)	23.05	(± 2.57)
Current smoker, n (%)	26	(29.2)
DM Type 2, n (%)	9	(10.1)
Hypertension, n (%)	15	(16.9)
Stricture length (mm), mean (SD)	25.4	(± 16.3)
Time to surgery (month), mean (SD)	6.67	(± 4.07)
Stricture aetiology, n (%)		
Straddle Injury	13	(14.60)
Iatrogenic	10	(11.23)
PFUI	66	(74.15)
Stricture site, n (%)		
Membranacea	27	(30.34)
Bulbar	62	(69.66)
Previous endoscopic treatment, n (%)		
Internal urethrotomies	34	(38.20)
Dilatations	33	(37.07)
No prior endoscopic surgery	14	(15.73)
Type of surgery, n (%)		
Bulbar mobilization	35	(39.33)
Crural separation	29	(32.58)
Inferior pubectomy	24	(26.96)
Supracrural rerouting	1	(1.12)
EPA outcome, n (%)		
Success	81	(91)
Recurrance	8	(9.00)
Erectile dysfunction (ED), n (%)		
Pre-operative (n: 81)		
No ED	48	(59.26)
De novo ED	33	(40.74)
Post-operative (n: 81)		
No ED	41	(50.61)
De novo ED	33	(40.74)
ED following EPA urethroplasty	7	(8.64)

BMI: Body Mass Index; DM: Diabetes Mellitus; SD: Standart Deviation; EPA Excision Primary Anastomosis; PFUI: Pelvic Fracture Urethral Injury.

Table 2.
Erectile function outcome.

	Pre-operative (n: 48)	12 months of follow-up (n: 41)	
	Mean ± SD	Mean ± SD	p value (vs Pre-operative)
EHS	2.70 ± 0.798	2.53 ± 0.823	0.176
IIEF-5	19.35 ± 4.547	19.73 ± 4.174	0.444

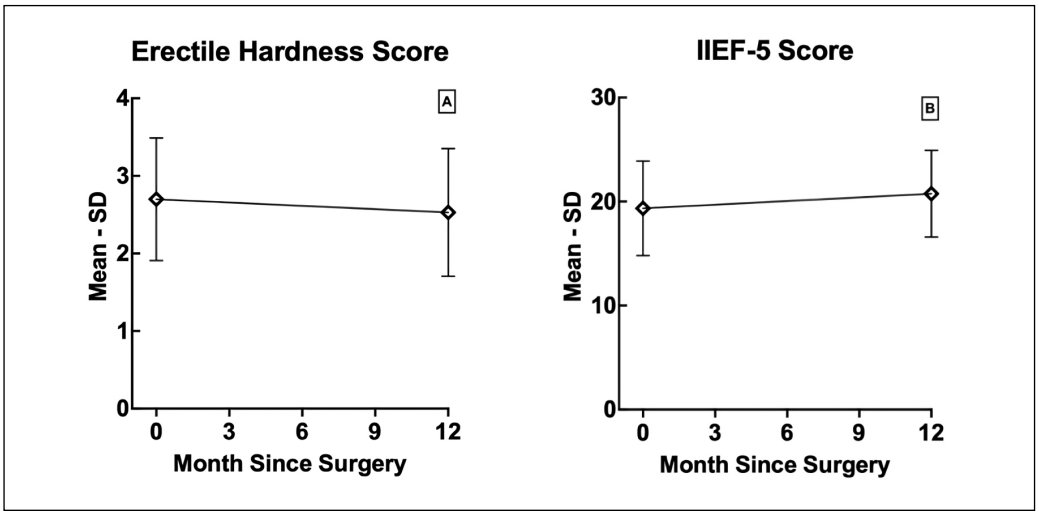

Figure 1.
EHS (A) and IIEF-5 Score (B) in 12 months of follow up.

Factors associated with the risk of ED post-operatively

In the univariate analysis, smoking significantly increased the odds of occurrence of erectile dysfunction (OR 6.78, 95% CI 1.32-34.81, p = 0.022). The type of PFUI injury also emerged as a significant predictor of occurrence of erectile dysfunction (OR 2.76, 95% CI 1.13-6.72, p = 0.026). Additionally, the number of procedures prior to EPA Urethroplasty significantly increased the risk of erectile dysfunction after EPA (OR 0.19, 95% CI 0.04-0.90, p = 0.036).

In the multivariate analysis, smoking remained a significant risk factor for occurrence of erectile dysfunction (OR 4.41, 95% CI 1.11-17.57, p = 0.035).

The PFUI injury type continued to significantly predict recurrence erectile dysfunction occurrence (OR 2.89, 95% CI 1.34-6.21, p = 0.007). The number of urethrotomies also significantly increased the odds of ED following EPA Urethroplasty (OR 4.69, 95% CI 1.28-17.24, p = 0.020), and prior procedures remained a significant factor in post-operative ED (OR 0.18, 95% CI 0.05-0.68, p = 0.011) (Table 3).

Table 3.
Uni- and multivariate Cox regression analysis for ED following EPA urethroplasty.

	Univariate analysis			Multivariate analysisore (> 3)		
	OR	95% CI	p value	OR	95% CI	p value
Current smoker	6.78	1.32-34.81	0.022	4.41	1.11-17.57	0.035 *
Diabetes mellitus (DM)	0.21	0.01-3.35	0.268			
Hypertension	1.56	0.23-10.78	0.652			
Time of symptom	0.55	0.21-1.43	0.216			
Type of Injury	2.76	1.13-6.72	0.026	2.89	1.34-6.21	0.007 *
Type of surgery	1.80	0.79-4.10	0.159			
Age	1.26	0.51-3.11	0.616			
BMI	0.90	0.43-1.86	0.773			
Stricture length	2.40	0.70-8.25	0.165			
Number of urethrotomies	2.93	0.69-12.45	0.145	4.69	1.28-17.24	0.020 *
Number of dilatations	0.19	0.04-0.90	0.036	0.18	0.05-0.68	0.011 *

* *Significant result.*
BMI: Body Mass Index; CI: Confidence Interval; DM: Diabetes Mellitus; OR: Odd Ratio; PFUI: Pelvic Fracture Urethral Injury.

Discussion

The prevalence of ED after pelvic fractures ranges from 5-28%, but it is significantly higher after PFUI, varying between 26-76% (10-12). ED after PFUI typically results from direct injury to the posterior urethra, penile nerves, and arteries. There is ongoing debate about whether neurogenic or vascular injury is the primary cause. Evidence leans towards neurogenic causes, with high response rates to intracavernosal vasoactive medications and abnormal electromyograms in affected patients, despite normal vascular flow. Some studies, however, report high occlusion rates in penile arteries, suggesting a vascular component (13).

The mechanism of ED following PFUI typically involves direct trauma that both lacerates and often avulses the posterior urethra, concurrently damaging the penile nerves and arteries as they traverse the pelvic floor behind the prostate before entering the penis. Literature debates whether neurogenic or direct vascular injury is the primary cause of ED, with more evidence pointing towards neurogenic ED (14, 15). Both vasculogenic and neurogenic causes have been proposed for ED after PFUI, and various studies have attempted to identify the specific pathophysiologic factors involved to better guide treatment.

There is no clear consensus on the primary cause of organic ED after PFUI. Feng et al. found that only 28% of men with ED post PFUI had organic ED, most of which was neurogenic (16). Studies by *Mark et al*. and *Machtens et al*. support the predominance of neurogenic factors, noting high response rates to neurogenic treatments and evidence of nerve damage (17, 18). *Guan et al.'s* study of 120 patients found that 80% had organic ED, with 30% vasculogenic, 43% neurogenic, and 27% mixed. Most vasculogenic cases involved veno-occlusive dysfunction rather than arterial insufficienc (19). These findings highlight the variability in ED etiology post PFUI and emphasize the need for a tailored, stepwise diagnostic approach to treatment.

Differentiating between ED due to PFUI and de novo ED from urethral realignment or delayed urethroplasty requires multiple assessments before and after injury and repair (20). Studies show that delayed urethroplasty increases the risk of ED by 3% over the 34% associated with PFUI alone, resulting in a 37% incidence of de novo ED. The higher rate of ED after delayed urethroplasty compared to primary endoscopic alignment likely reflects differences in injury severity and ED reporting (21). Men undergoing primary realignment generally have less severe PFUIs, as indicated by a lower ED rate compared to those needing delayed urethroplasty. However, the association is unclear due to variability in institutional practices regarding primary realignment and delayed urethroplasty (20).

Our study utilized four techniques include bulbar urethral mobilization, crural separation, inferior pubectomy, and supracrural rerouting. Despite employing these methods, the results showed no significant impact on postoperative erectile dysfunction. Using only bulbar urethral mobilization and crural separation is considered a simple perineal approach. Including either an inferior pubectomy or supracrural rerouting is classified as an elaborated perineal approach (22). In a previous study, 74 patients (30.2%) underwent transecting bulbar urethroplasty, while 171 patients (69.8%) underwent non-transecting techniques. Both groups had similar success rates (87.8% *vs.* 86.5%, p =0.93) and postoperative complications (8.1% *vs.* 7%, p= 0.73). Transient ED was more common in the transecting cohort (8.1% *vs.* 2.9%, p =0.07), but de novo permanent ED rates were comparable (4.1% *vs.* 2.9%, p = 0.65). Transecting techniques can lead to transient ED that typically improves within a year, while permanent ED is uncommon and not significantly influenced by the type of urethroplasty technique used (23).

A previous meta-analysis involving 21 studies with 6.791 patients showed consistent results across two stages. In the univariate analysis (18 studies, 5.811 patients), smoking was associated with a higher risk of stricture recurrence (RR = 1.32, p = 0.001). The multivariate analysis (11 studies, 3.176 patients) also found smoking linked to increased stricture recurrence (RR = 1.35, p = 0.049). Smoking may increase the risk of stricture recurrence after urethroplasty. Quitting smoking is recommended for patients undergoing urethroplasty (24). Smoking adversely impacts ED through several mechanisms. It disrupts the *nitric oxide* (NO) pathway by impairing both endothelial and neuronal *NO synthase* (NOS) activities. Specifically, components of burned tobacco inhibit neuronal NOS, while cigarette smoke damages the endothelium, reducing endothelial NOS-mediated vasodilation. Additionally, smoking produces superoxide anions that decrease free NO levels in the corpora cavernosa. These superoxides activate NADH oxidase enzymes, diverting NO into a peroxynitrite pathway and reducing its vasoactive availability. Smoking also affects *Rho-associated kinase* (ROK) signaling by decreasing NO levels, which leads to increased ROK activity and worsens ED.

Finally, smoking causes intrinsic vascular damage, altering elastin in the extracellular matrix and inducing calcification of medial elastic fibers, resulting in arterial stiffness and impaired vessel dilation (24). Unlike our study, previous study found no significant correlation between postoperative ED and smoking history (25), Waddel et al. reported a higher incidence of ED associated with smoking history (26).

In our study, we found that previous treatment had significant results in post-operative ED. Potential mechanisms for erectile dysfunction following DVIU include direct injury to the cavernous nerve caused by the urethrotomy knife, fibrosis resulting from fluid and urine leakage into the periurethral space, infection, or the creation of a shunt between the corpora cavernosa and the corpus spongiosum (27). Despite its lower success rates compared to urethroplasty, *Direct Vision Internal Urethrotomy* (DVIU) remains the most used treatment for anterior urethral stricture (28).

Erickson et al. used the BMSFI to assess ED following urethral reconstruction and discovered that patients over 50 years old experienced a notable decline in the mean EF domain score, unlike their younger counterparts (29). Similarly, Anger et al. indicated that both age and preoperative EF can negatively impact postoperative sexual outcomes. Their prospective review found that men with postoperative EF scores ≤ 20 on the IIEF questionnaire were older (mean age 47 *vs.* 36.8 years, p = 0.17) and had poorer preoperative EF values (mean 20 *vs.* 29, p = 0.11) compared to those with EF scores > 20, though these differences were not statistically significant (30). In contrast, other studies found no clear link between age and the incidence of ED, though older men generally had lower preoperative IIEF scores and experienced a greater decline in these scores. Chapman et al. also found no significant association between age and sexual dysfunction in their multivariate analysis (31-33). Age showed insignificant results in our study on postoperative ED due to a potentially small sample size, narrow age range, variability in preoperative function, and confounding factors. Statistical methods may not have detected subtle age effects, and the follow-up duration might have been too short. Additionally, the complex relationship between age and ED could have diluted the impact of age alone.

Coursey et al. suggested that stricture length could be a predictive factor for postoperative erectile function, finding that men with poorer erectile outcomes had significantly longer strictures compared to those with improved or stable erections (mean 6.8 cm *vs.* 4 cm) (34). However, subsequent research has contradicted this association (29, 32, 35). ED is approximately 3.5 times more prevalent in men with *diabetes mellitus* (DM) compared to those without the condition (36). In a previous study involving 878 men, logistic regression was used to examine the relationship between ED and *body mass index* (BMI). The incidence of ED was 53.1%. Men in the ED group had significantly higher BMIs compared to those in the non-ED group (p = 0.01). Obese men had a higher risk of ED compared to those with normal weight (OR = 1.97, 95% CI = 1.25-3.14, p = 0.004), even after adjusting for potential confounding factors (OR = 1.78, 95% CI = 1.10-2.90, p = 0.02). Overall, these findings indicate a positive correlation between obesity and the risk of moderate to severe ED (37). In our study, the variable BMI did not yield significant results. This lack of significance may be attributed to the fact that the average BMI of the participants was 23.05, which falls within the normal weight

range. Additionally, the study sample did not include a wide range of BMI values, leading to a limited distribution of data that could have impacted the ability to detect a significant effect of BMI on erectile function outcomes. The limitation of the study is its retrospective design, which relies on historical medical records. This approach can lead to incomplete or inconsistent data collection, potentially affecting the accuracy and reliability of the findings. Additionally, the study's reliance on patient-reported outcomes, such as the EHS Score and the IIEF-5 score, may introduce bias due to subjective interpretations by the patients. To strengthen the findings, a prospective cohort study design could be implemented in future research. This approach would allow for more controlled and consistent data collection, including real-time assessments of erectile function and other relevant variables. Additionally, incorporating objective measures, such as penile Doppler ultrasound or other physiological assessments of erectile function, could provide more accurate and reliable data on the impact of EPA Urethroplasty on erectile dysfunction.

Conclusions

The risk of ED following EPA Urethroplasty, while present, is generally lower than anticipated. Smoking, PFUI, and prior treatments rather than the EPA procedure itself emerge as significant risk factors contributing to the development of ED after surgical repair. Despite the inherent risk of ED associated with any urethral surgery, these factors play a more prominent role in influencing post-operative erectile function.

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Declarations

Ethical approval: *This study was approved by the Health Research Ethics Commission of Saiful Anwar General Hospital Number: 400/214/K.3/102.7/2024.*

Availability of data and material: *The datasets used and/or analyzed during the current study are available upon reasonable request.*

Competing interests: *The authors declare that they have no competing interests.*

Funding: *The authors report no funding.*

Authors' contributions: *PS, BD: conceptualization, methodology, writing-original draft, visualization, project administration, validation, GWDPD, AGW, FKD: conceptualization, methodology, writing-original draft, investigation, validation, data analysis.*

Acknowledgments: *Not applicable.*

Consent for publication: *Not applicable.*

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