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A Simple and Reproducible Rat Model of Urethral Spongiofibrosis: Evaluation of Colchicine as a Potential Antifibrotic Agent

RAMAZAN SAMET CETINKAYA

r.s.cetinkaya@gmail.com

Kastamonu University

MUMTAZ DADALI

Ahi Evran University

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Abstract

Objective

Urethral stricture is an abnormal narrowing of the anterior urethra caused by fibrosis of the surrounding corpus spongiosum. Since fibrosis is the main factor responsible for disease progression, this study investigated the effect of colchicine, a drug with known antifibrotic properties, on fibrosis development in a rat model of urethral injury.

Materials and Methods

Twenty male Wistar-Albino rats (250–300 g) were included. One rat was used to standardize the trauma model. The remaining 19 rats were divided into control (n = 2), sham (n = 5), and treatment (n = 12) groups. No intervention was performed in the control group. Urethral trauma was induced at the 12 o'clock position in the sham and treatment groups. The treatment group additionally received oral colchicine (1 mg/kg/day). All animals were sacrificed on day 28. Histopathological evaluation of proximal (location A) and distal (location B) urethral segments included fibrosis, mononuclear cell infiltration, and vascular proliferation.

Results

Significant differences among groups were observed at location A for fibrosis ($p = 0.013$) and vascular proliferation ($p = 0.020$). No significant differences were detected at location B or in the combined analysis. Comparison of the sham and treatment groups showed significant differences at location A for fibrosis ($p = 0.037$) and vascular proliferation ($p = 0.048$), mainly reflecting lower scores in the sham group. No significant differences were found at location B.

Conclusion

The proposed rat model successfully induced urethral fibrosis, particularly in the proximal urethra. Colchicine did not demonstrate a protective antifibrotic effect under the study conditions. Larger experimental studies are required to clarify its potential role in urethral stricture treatmentKeywords: urethral stricture, fibrosis, colchicine, urethra, antifibrotic therapy

INTRODUCTION

Urethral stricture is an abnormal narrowing of the anterior urethra caused by fibrosis of the surrounding corpus spongiosum [1]. In men, its prevalence ranges from 229 to 627 per 100,000, with incidence increasing with age due to the accumulation of risk factors [2]. Although the exact prevalence in women is unknown, the condition is considerably less common than in men.

The etiology of urethral stricture can be classified as iatrogenic, traumatic, inflammatory, or idiopathic [1]. Iatrogenic causes account for approximately 45% of cases and include traumatic catheterization, transurethral procedures, hypospadias repair, prostatectomy, and brachytherapy [3, 4]. Trauma, particularly following pelvic fractures, is another important cause and frequently results in posterior urethral strictures. Infectious and inflammatory conditions, including gonococcal urethritis and lichen sclerosus, also contribute to disease development [5]. Despite their diverse origins, these factors share a common pathological mechanism: fibrosis.

The substantial healthcare costs associated with urethral stricture management have stimulated interest in medical therapies. In the United States, the reported cost per patient is $17,747 for repeated internal urethrotomies and $16,444 for urethroplasty [6]. Several antifibrotic agents, including halofuginone, mitomycin C, botulinum toxin A, somatostatin analogs, and corticosteroids, have been investigated to prevent fibrosis and recurrence [7–11]. These medical treatments share a common goal: to slow down or inhibit the fibrotic process.

Colchicine inhibits microtubule formation and exhibits well-established antifibrotic properties. It is widely used in urology for the treatment of Peyronie's disease, another fibrosis-related condition. Based on this mechanism, we investigated its potential role in urethral stricture disease. To our knowledge, no previous study has histopathologically evaluated urethral fibrosis following colchicine treatment in an experimental urethral stricture model. We hypothesized that colchicine would reduce fibrosis in traumatized urethral tissue.

MATERIALS AND METHODS

This study was approved by the Local Ethics Committee for Animal Experiments of the Ministry of Health Ankara Training and Research Hospital (decision no. 587, meeting no. 0054, dated 01.07.2019) and conducted between 11.09.2019 and 09.10.2019 at the Hüsnü Sakal Experimental and Clinical Research Center (DEKAM) of the same institution. The animals were obtained from the Experimental Research Center of Ankara Training and Research Hospital. A total of 20 male Wistar-Albino rats weighing 250–300 grams were used.This study was conducted in accordance with ARRIVE guidelines.

All rats were housed in the same laboratory environment for one week prior to the experiment. They were maintained in standard cages, a 12-hour light/dark cycle at room temperature. During the preoperative and postoperative periods, rats were given standard laboratory chow and tap water.

A total of 20 rats were included in the study. Initially, one healthy rat was euthanized after urethral trauma and en bloc excision of the bladder and urethra to assess urethral anatomy and verify procedural success. The remaining 19 rats were randomly allocated into three groups:

Control group: 2 healthy rats with no intervention

Sham group: 5 rats with urethral trauma only

Study group: 12 rats with urethral trauma followed by oral colchicine treatment

Standard surgical instruments, sterile lubricating gel (Dispogel®, 12.5 g, Dispofarma Pharmaceutical Co.), and intravenous cannulas (Polycan Cannula®, 24G, PolyMedicure Ltd.) were used for all procedures. Colchicine (Kolsin®, İ.E. Ulagay Pharmaceutical Co.) used in the study was supplied by the research team. For histopathological evaluation, all rats were euthanized on day 28, and their urethras and bladders were excised. The specimens were placed in formalin and transported on the same day to the Pathology Laboratory of Kırşehir Ahi Evran University Training and Research Hospital.

All procedures were performed under sterile conditions using 50 mg/kg intramuscular ketamine hydrochloride anesthesia (Ketalar®, Pfizer Inc.). After shaving the abdomen, the skin and penis were disinfected with povidone-iodine. A 24G cannula needle was bent into a hook shape with a needle holder (Fig. 1). A rat was taken, and the urethra was traumatized longitudinally from proximal to distal at the 12 o'clock position using the modified needle. After laparotomy, the urethra and bladder were excised as a whole. Dissection of the specimen revealed a ventral fibrotic lesion approximately 2 cm in length involving the corpus spongiosum. The remaining 19 rats were allocated into the previously described three groups.

Figure 1: Tip of a 24G cannula bent into a hook shape

Control Group

No intervention was performed in this group. After 28 days of follow-up, the rats were euthanized, and normal urethral tissue was evaluated.

Sham Group

In this group, under ketamine anesthesia, a lubricated, bent cannula needle was inserted 2 cm into the penis after traction and introduced at the 12 o'clock position to scratch the urethra before being withdrawn (Fig. 2).

Figure 2: Determining the length of trauma after stretching the rat urethra

Study Group

Urethral trauma was induced in the same way as the sham group. Then, colchicine was administered daily via a 6 Fr feeding tube at a dose of 1 mg/kg diluted in 1 cc of 0.9% NaCl solution for 28 days until sacrifice.

Euthanasia and Evaluation

There were no losses due to surgery, complications, anesthesia, or drug toxicity in any of the 19 rats from the three experimental groups. On day 28, all rats were euthanized via intracardiac injection of 100

mg/kg thiopental. To avoid damaging the urethra, a vertical incision was made in the abdominal skin. The bladder and urethra were then excised en bloc (Figs. 3 and 4).

Figure 3: Dissection of the urethra after vertical abdominal skin incision

Figure 4: En bloc dissection of the bladder and urethra

Pathological specimens were fixed in 10% buffered formalin and sent to the Pathology Laboratory of Kırşehir Ahi Evran University Training and Research Hospital. The pathologist was blinded to group allocation. The specimens were processed using standard histological techniques and embedded in paraffin blocks. Four 4-micrometer circular sections were prepared from each urethra and mounted on slides. Two groups of samples were formed, each including two sections from proximal to distal: group A (proximal) and group B (distal). All slides were stained with Hematoxylin-Eosin and Masson's Trichrome and evaluated by an expert pathologist under light microscopy at 10x, 50x, and 100x magnification.

Each group was evaluated using three histopathological parameters: fibrosis, mononuclear cell infiltration, and vascular proliferation. A semi-quantitative scoring system was used:

0: None

1: Mild

2: Moderate

3: Severe

Each parameter was scored between 0 and 3 (Figs. 5, 6, and 7).

Figure 5: Fibrosis scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: area of spongiofibrosis)

Figure 6: Vascular proliferation scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: vascular proliferation)

Figure 7: Mononuclear cell infiltration scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: mononuclear cell infiltration)

Statistical Analysis

Data were analyzed using SPSS software (IBM SPSS Statistics for Windows, Version 22.0, Armonk, NY: IBM Corp.). Because the data were ordinal and did not follow a normal distribution, results are presented as median and interquartile range (IQR). Differences among the control, sham, and colchicine-treated groups were evaluated using the Kruskal–Wallis test, followed by Dunn's post hoc test for pairwise

comparisons. For direct comparisons between two groups, the Mann–Whitney U test was used. A p-value < 0.05 was considered statistically significant.

To better understand the magnitude of differences between groups, effect size (r) was calculated for pairwise comparisons using the formula r = Z/√N. Effect sizes were interpreted as small (0.1), moderate (0.3), or large (0.5).

Post hoc power analysis was performed to provide an estimate of the study's ability to detect differences, particularly for fibrosis and vascular proliferation at the proximal urethral segment. These estimates were based on observed effect sizes and sample distributions and are presented descriptively, acknowledging the inherent limitations of post hoc power calculations.

Confidence intervals (95%) were also estimated to evaluate the precision of the observed effects. Given the non-parametric design and relatively small sample size, these intervals were interpreted cautiously and used primarily to support the overall interpretation rather than to draw definitive conclusions.

The primary outcome of the study was fibrosis score at the proximal urethral segment (location A).

RESULTS

A total of 19 rats were included in our study and divided into three groups: control (n = 2), sham (n = 5), and treatment (n = 12). During pathological examination, each urethral specimen was divided into two sections—A and B—based on the area of intervention (A: proximal section, B: distal section) (Fig. 8). The samples were evaluated for fibrosis, mononuclear cell infiltration, and vascular proliferation. These parameters were scored on a scale from 0 to 3. Since the approximately 2 cm segment of the urethra could not be displayed on a single slide, the A and B regions were examined on separate slides.

Figure 8: Macroscopic view of locations A and B

The histopathological examination results for the groups included in the study are presented in Table 1.

Table 1
Scoring of Histopathological Findings by Groups A: proximal urethra, B: distal urethra, 0: none, 1: mild, 2: moderate, 3: severe

Group	Fibrosis (A)	Fibrosis (B)	Mononuclear Cell Infiltration (A)	Mononuclear Cell Infiltration (B)	Vascular Proliferation (A)	Vascular Proliferation (B)
Study 1	2	1	0	0	2	1
Study 2	2	1	0	0	1	1
Study 3	2	2	1	0	2	0
Study 4	3	2	1	1	2	0
Study 5	0	2	0	1	0	2
Study 6	2	0	0	3	2	2
Study 7	3	2	0	1	1	0
Study 8	1	1	0	0	1	1
Study 9	2	1	0	0	2	1
Study 10	2	1	0	2	1	0
Study 11	3	0	0	0	1	0
Study 12	1	0	0	0	2	0
Sham 1	1	2	1	1	1	1
Sham 2	1	2	0	2	1	2
Sham 3	1	1	0	0	0	1
Sham 4	1	2	0	0	0	1
Sham 5	1	2	0	1	1	1

Group	Fibrosis (A)	Fibrosis (B)	Mononuclear Cell Infiltration (A)	Mononuclear Cell Infiltration (B)	Vascular Proliferation (A)	Vascular Proliferation (B)
Control 1	0	1	0	0	0	1
Control 2	0	1	0	0	0	0

Table 1: Scoring of the obtained data by groups (A: proximal urethra, B: distal urethra, 0: none, 1: mild, 2: moderate, 3: severe)

All descriptive data are presented as median (interquartile range, IQR) (Table 1). Fibrosis, mononuclear cell infiltration, and vascular proliferation scores following urethral trauma were compared across the control, sham, and colchicine-treated groups for both location A (proximal) and location B (distal) using the Kruskal–Wallis test.

At location A, statistically significant differences were observed among groups for fibrosis ($p = 0.013$) and vascular proliferation ($p = 0.020$), whereas no significant difference was found for mononuclear cell infiltration ($p = 0.809$). In contrast, no statistically significant differences were detected at location B for any of the evaluated parameters. Similarly, when locations A and B were analyzed in combination, no significant differences were observed. These findings indicate that the trauma model produced measurable histopathological changes primarily in the proximal urethral segment.

Subsequent pairwise comparisons between the sham and colchicine-treated groups were performed using the Mann–Whitney U test. At location A, significant differences were identified for fibrosis ($p = 0.037$) and vascular proliferation ($p = 0.048$), while mononuclear cell infiltration remained non-significant ($p = 1.000$). Notably, these differences were driven by lower fibrosis and vascular proliferation scores in the sham group. At location B and in the combined analysis, no statistically significant differences were detected between the two groups.

Effect size analysis provided further insight into these findings. At location A, large effect sizes were observed for both fibrosis ($r \approx 0.53$) and vascular proliferation ($r \approx 0.50$), indicating that the differences between the sham and colchicine-treated groups were not only statistically significant but also substantial in magnitude. At location B, moderate effect sizes ($r \approx 0.35–0.42$) were noted despite the absence of statistical significance, suggesting the possibility of underlying biological differences that the study may not have been sufficiently powered to detect.

Post hoc power analysis supported this interpretation, demonstrating moderate statistical power for detecting differences at location A (approximately 50–65%), whereas power for distal and combined analyses remained low (< 30%). Confidence interval assessment further highlighted these limitations, with relatively wide intervals observed across several comparisons, reflecting variability and the small

sample size. Overlapping confidence intervals in distal and combined analyses indicate reduced precision and increased uncertainty.

Taken together, these results suggest that while statistically significant differences were primarily confined to the proximal urethral segment, the presence of moderate-to-large effect sizes alongside wide confidence intervals indicates that some non-significant findings may be attributable to limited statistical power rather than a true absence of biological effect.

DISCUSSION

Urethral stricture disease represents a fibrotic process involving both the urethral mucosa and the surrounding corpus spongiosum. Because fibrosis is the key mechanism underlying disease progression and recurrence, reliable experimental models are essential for investigating pathophysiology and potential antifibrotic therapies.

The first aim of our study was to establish a simple, rapid, and reproducible rat model of urethral spongiofibrosis. Previous experimental models have used endoscopic coagulation, urethrotome blades, or pediatric resectoscopes in rats and rabbits, with reported success rates ranging from 50% to 100% [12–15]. In our model, trauma was induced using a cannula needle modified into a hook shape. The procedure required approximately one minute per animal and used inexpensive, readily available materials, making it a practical alternative to previously described techniques.

In a rat model, urethral trauma created using a urethrotome blade at the 4 and 8 o'clock positions has been shown to induce chronic inflammation, fibrosis, and scar formation on histopathological evaluation[16].

In our study, trauma was induced using a cannula needle modified into a hook shape. This procedure, which takes approximately one minute per rat, is considerably faster than other experimental urethral stricture models reported in the literature. Additionally, the low cost and easy accessibility of the materials used represent further advantages of our method.

Numerous medical agents have been investigated in the literature for the treatment of urethral stricture and the prevention of recurrences. These agents have been administered orally, intraurethrally, intralesionally, or in various combinations. Differences in the pharmaceutical agents and dosages used have led to varying outcomes.

Several medical agents have been investigated to prevent fibrosis and recurrence in urethral stricture disease. Halofuginone, rapamycin, matrix metalloproteinase-1, mitomycin C (MMC), corticosteroids, and hyaluronic acid have all demonstrated varying degrees of antifibrotic activity in experimental or clinical studies [7, 10, 17–25]. Reported mechanisms include inhibition of collagen synthesis, suppression of fibroblast proliferation, modulation of inflammation, and promotion of collagen degradation. Clinical

studies have shown reduced recurrence rates following the use of MMC, triamcinolone, hyaluronic acid, or combinations of these agents after internal urethrotomy [10, 18, 24, 25].

Unlike these agents, colchicine exerts multiple antifibrotic effects simultaneously through inhibition of microtubule polymerization, modulation of inflammatory responses, and reduction of neutrophil activity. Because of its established use in other fibrotic disorders, including Peyronie's disease, colchicine represents an attractive candidate for the prevention of urethral fibrosis.

To our knowledge, no previous experimental study has evaluated colchicine in a rat model of urethral stricture. The only clinical study available was published by Sinanoğlu et al. in 2018 and reported a lower recurrence rate in patients receiving colchicine after internal urethrotomy compared with controls [26]. However, that study lacked histopathological evaluation, limiting conclusions regarding the underlying tissue response.

In the present study, urethral trauma successfully induced fibrosis, particularly at location A, confirming the validity of the proposed model. However, colchicine did not demonstrate a protective antifibrotic effect. Unexpectedly, fibrosis scores at location A were higher in colchicine-treated animals than in the sham group. Although colchicine is generally regarded as an antifibrotic agent, its effects may depend on factors such as dosage, treatment duration, timing of administration, and local tissue dynamics. The observed increase in fibrosis may reflect a paradoxical response related to interference with early inflammatory and healing processes.

The results should be interpreted with caution because of the study's limitations. Most importantly, the control group size was very small (n = 2), reducing the strength of statistical comparisons. Post hoc power analysis indicated only moderate power for significant findings at location A and low power for other comparisons. Consequently, some non-significant findings may be attributable to insufficient sample size rather than the absence of a biological effect.

In conclusion, the proposed rat model effectively induced urethral fibrosis and may serve as a useful experimental platform for future studies. Under the conditions tested, colchicine did not prevent fibrosis formation. Larger studies with balanced group sizes and optimized dosing protocols are required to better define the potential role of colchicine in urethral stricture disease.

CONCLUSION

We describe a simple, rapid, and reproducible rat model for inducing urethral spongiofibrosis, which reliably generates localized fibrotic changes, particularly in the proximal urethra. This model offers practical advantages, including low cost, technical simplicity, and short procedural time, making it a valuable platform for experimental studies.

Within this model, colchicine did not demonstrate a protective effect against fibrosis and was associated with increased fibrotic scores in the proximal segment, suggesting a potential context-dependent or

paradoxical response.

These findings highlight the complexity of antifibrotic modulation in urethral tissue and underscore the importance of dose, timing, and tissue-specific dynamics. Future studies with larger and more balanced sample sizes, as well as optimized dosing strategies, are needed to further clarify the role of colchicine in urethral stricture disease.

Declarations

Conflict of interest:

The authors have no conflicts of interest to declare that are relevant to the content of this article.

Funding:

No funding was received for conducting this study.

Author Contribution

R.S.C. wrote main manuscript textM.D. contributed to the data preparation and data analysis process.All authors reviewed the manuscript.

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Figures

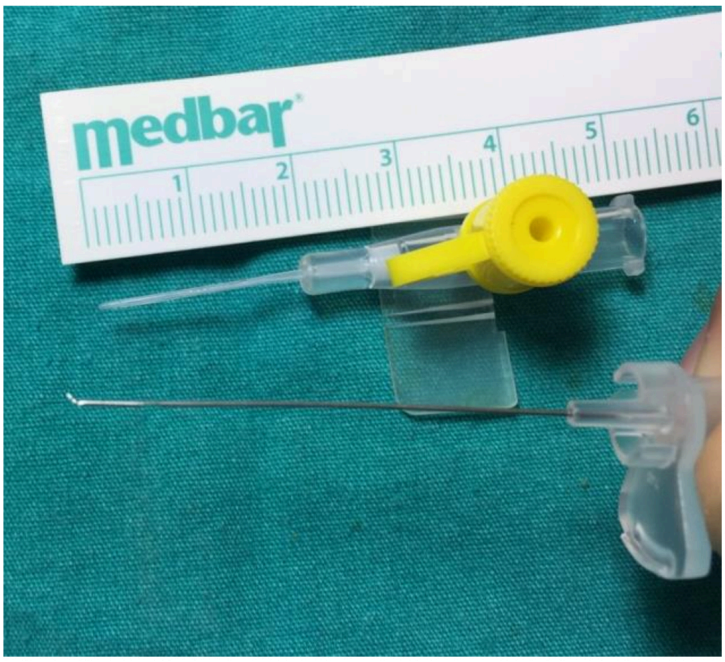


Figure 1

Tip of a 24G cannula bent into a hook shape

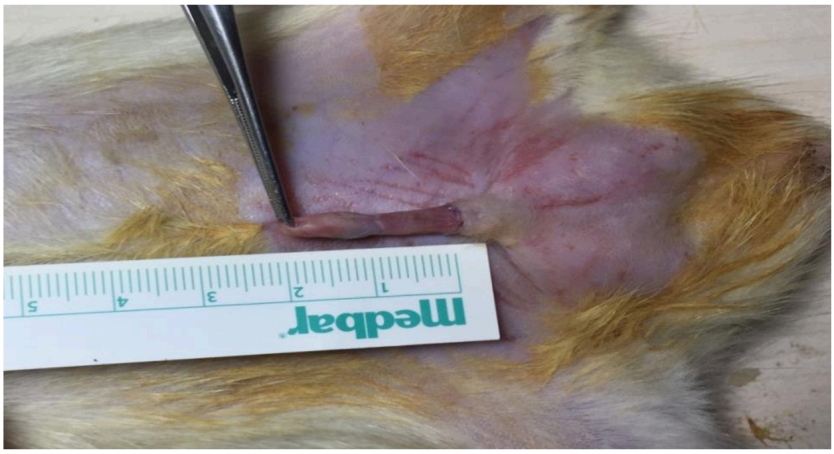


Figure 2

Determining the length of trauma after stretching the rat urethra



Figure 3

Dissection of the urethra after vertical abdominal skin incision

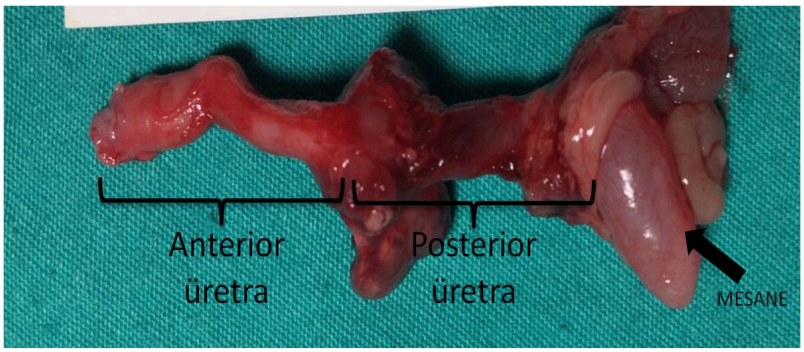


Figure 4

En bloc dissection of the bladder and urethra

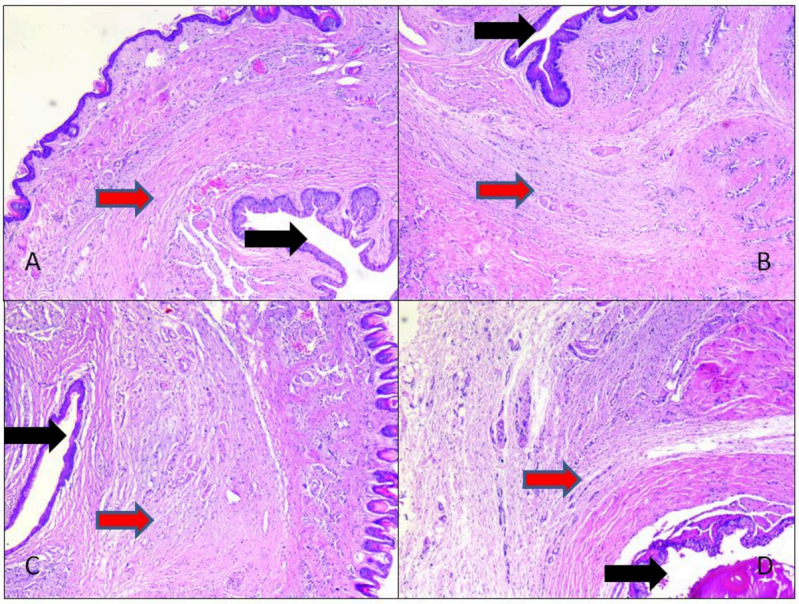


Figure 5

Fibrosis scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: area of spongiofibrosis)

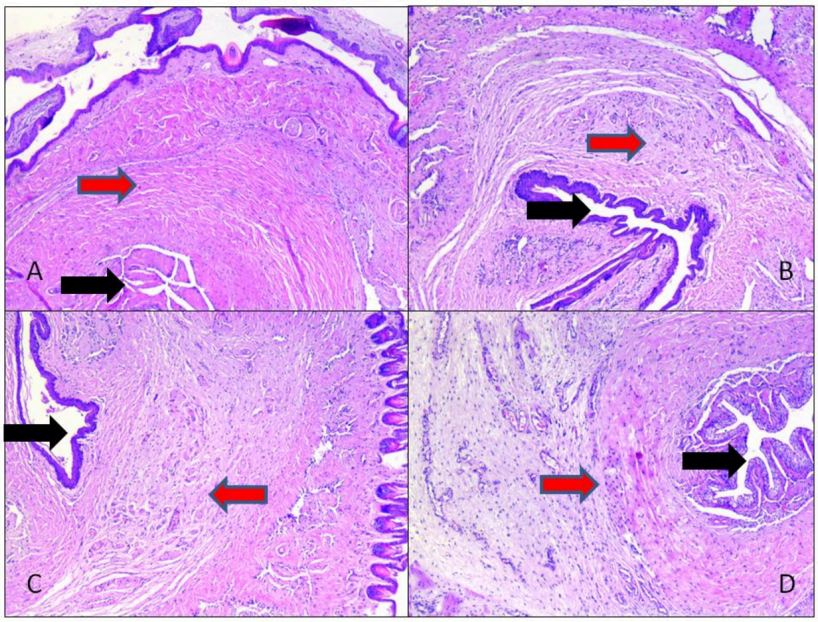


Figure 6

Vascular proliferation scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: vascular proliferation)

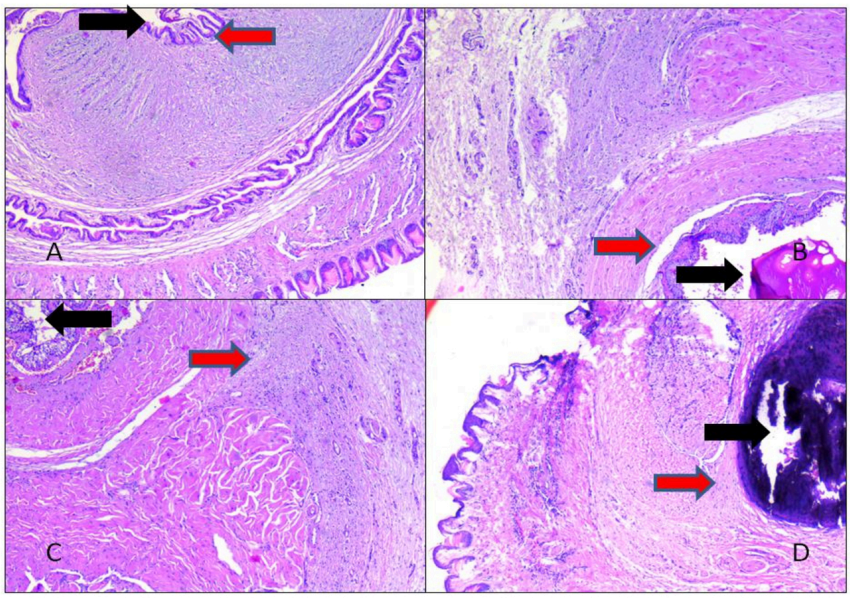


Figure 7

Mononuclear cell infiltration scores under 50x magnification (A: score 0, B: score 1, C: score 2, D: score 3; black arrow: urethral lumen, red arrow: mononuclear cell infiltration)

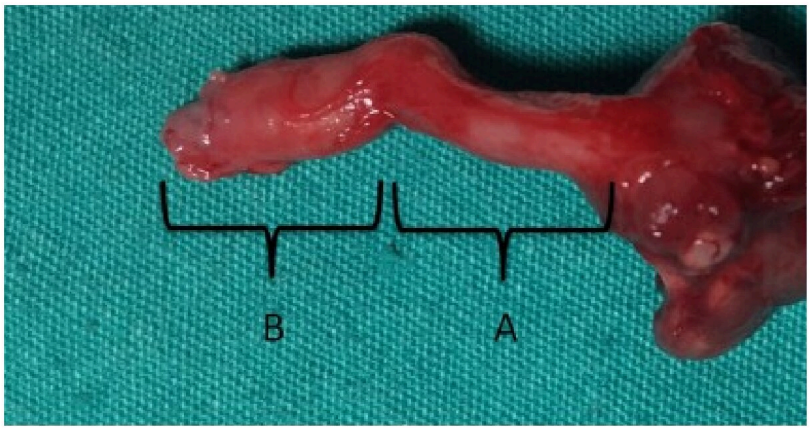


Figure 8

Macroscopic view of locations A and B

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