CASE REPORT

Nagoya J. Med. Sci. **88**. 138–148, 2026
doi:10.18999/nagjms.88.1.138

Inguinal bladder hernia with bilateral hydronephrosis: a case report of urodynamic and functional recovery assessments

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ABSTRACT

We report a case of a 50-year-old man referred to our department with a marked inflammatory response and severe renal dysfunction. Computed tomography scans revealed an inguinal bladder hernia with bilateral hydronephrosis. A urethral catheter was placed to relieve urinary retention, and antimicrobial therapy was initiated for pyelonephritis. Despite adequate urine output, renal function improved only partially, and hydronephrosis persisted. Further imaging and intraoperative findings confirmed that the hernia sac was adjacent to the bladder and the bilateral lower ureters, which were significantly stretched and displaced. Firm adhesions between the bladder and the hernia sac necessitated meticulous dissection to restore normal anatomy. Surgical repair of the inguinal hernia successfully repositioned the bladder. Although morphological upper urinary tract dilation persisted postoperatively, a mercaptoacetyltriglycine 3 renal scan demonstrated no evidence of functional obstruction, indicating improved ureteral patency. A urodynamic study (UDS) revealed significantly decreased bladder compliance, likely due to chronic displacement and compression within the scrotum, which impaired the detrusor function. The patient gradually regained spontaneous voiding after urethral catheter removal and initiation of clean intermittent catheterization. Follow-up UDS confirmed a substantial improvement in bladder compliance, indicating functional recovery. Follow-up imaging indicated the resolution of bilateral hydronephrosis and improvement of renal function. This case emphasizes the importance of recognizing lower ureteral entrapment in cases of bladder hernia and demonstrates that bladder dysfunction due to chronic displacement can be reversed by timely surgical intervention.

Keywords: bilateral hydronephrosis, inguinal bladder hernia, lower urinary tract dysfunction, post-renal acute kidney injury, urodynamic study

Abbreviations:
UDS: urodynamic study
CIC: clean intermittent catheterization

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INTRODUCTION

Bladder hernia is an uncommon condition, accounting for approximately 1–4% of all inguinal hernias.[1] While inguinal hernias are frequently encountered in clinical practice, bladder herniation within the scrotum is rare and often asymptomatic, leading to an incidental diagnosis during imaging or surgery.[2] However, in some cases, bladder herniation can have significant clinical consequences, including urinary retention, recurrent urinary tract infections, and obstructive uropathy, potentially progressing to renal dysfunction.[2]

The involvement of both ureters in a bladder hernia is particularly rare, with only a few reported cases of bilateral ureteral obstruction leading to postrenal acute kidney injury.[3] The mechanical entrapment and displacement of the lower urinary tract structures along with the hernial sac can present unique diagnostic and therapeutic challenges, especially when hydronephrosis persists despite bladder decompression.[2] Moreover, the functional impact of bladder herniation on lower urinary tract dynamics is still largely unexplored.

Here, we report a rare case of inguinal bladder hernia causing bilateral lower ureteral obstruction, leading to post-renal acute kidney injury and persistent lower urinary tract dysfunction. This case is unique in that the lower urinary tract function was assessed using a urodynamic study (UDS), providing novel insights into the physiological effects of bladder herniation and its potential reversibility following surgical intervention. In this report, we emphasize the significance of early recognition and appropriate management of bladder hernias with urinary tract involvement, as well as the role of functional assessment in guiding postoperative care.

CASE PRESENTATION

History, examination, and radiological findings

A 50-year-old man presented to our hospital with fever (39.7 °C) and fatigue.

He experienced frequent urination (more than once every 2 hours) and scrotal swelling over the past 6 months.

This patient was undergoing treatment for hypertension and sleep apnea. He had a history of diabetes mellitus; however, his blood glucose level was well controlled, and oral medication was discontinued.

Additionally, the patient had obesity (height, 170 cm; weight, 131.4 kg; and body mass index, 45.5). Excess adipose tissue obscured the physical examination findings; however, his right scrotum was markedly enlarged and apparent despite the difficulty in examination.

Table 1 presents the findings of the blood and urine tests. Laboratory tests revealed an elevated white blood cell count and elevated C-reactive protein and creatinine levels (Table 1). Computed tomography revealed bladder distention extending into the scrotum via the inguinal region. The estimated prostate volume was 25 mL (Fig. 1A, B). Additionally, bilateral hydronephrosis was observed (Fig. 1C).

Table 1 Laboratory tests at initial visit

Blood test	
WBC	27400/μL
Hb	9.3 g/dL
HCT	29 %
MCV	96 fL
PLT	28.1×10^4/μL

TP	7.1 g/dL
ALB	3.5 g/dL
AST	24.8 U/L
ALT	18.8 U/L
BUN	58.4 mg/dL
CRE	5.68 mg/dL
eGFR	9.4 mL/min/1.73m^2
Na	137.1 mEq/L
K	4.4 mEq/L
Cl	104.1 mEq/L
CRP	15.98 mg/dL
Glu	96.1 mg/dL
HbA1c	5.9 %
Urine test	
pH	8.0
Protein	(–)
Nitrite	(–)
RBC	7.1/HPF
WBC	5.8/HPF

WBC, white blood cell count; Hb, hemoglobin; HCT, hematocrit; MCV, mean corpuscular volume; PLT, platelet; TP, total protein; ALB, albumin; AST, aspartate aminotransferase; ALT, alanine aminotransferase; BUN, blood urea nitrogen; CRE, creatinine; eGFR, estimated glomerular filtration rate; Na, sodium; K, potassium; Cl, chlorine; CRP, C-reactive protein; Glu, glucose; HbA1c, hemoglobin A1c; RBC, red blood cell count; HPF, high-power field.

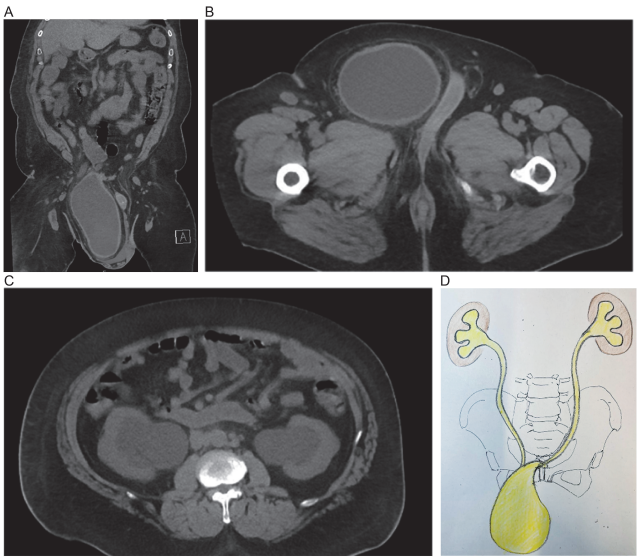

Fig. 1 Computed tomography and schematic diagram of the initial visit
Fig. 1A: The distended bladder has prolapsed into the right scrotum (coronal view).
Fig. 1B: In the axial view, the prolapsed bladder showed tension at the inguinal level.
Fig. 1C: Axial view showing bilateral hydronephrosis.
Fig. 1D: A schematic diagram of computed tomography is illustrated in Figures 1A, 1B, and 1C.

Based on these findings, the patient was diagnosed with pyelonephritis and post-renal failure secondary to urinary retention caused by a herniated bladder. A urethral catheter was inserted, leading to immediate drainage of 1,500 mL of urine. Even after the drainage, attempts at manual bladder repositioning within the pelvis were unsuccessful. Antibiotic therapy with cephalosporins was initiated.

The following day, the creatinine levels remained elevated, decreasing only slightly to 3.31 mg/dL by day 3, with persistent bilateral hydronephrosis (Fig. 2A). Further imaging confirmed that entrapment and traction of the bladder and ureters, along with the hernial sac, led to functional obstruction of the lower ureters, resulting in upper urinary tract dilation (Fig. 2B, white arrow, right ureter; gray arrow, left ureter). On the fourth day of hospitalization, we explained to the patient that surgical repair of the inguinal hernia was necessary to relieve urinary tract obstruction. The procedure was performed on the same day by general surgeons and urologists.

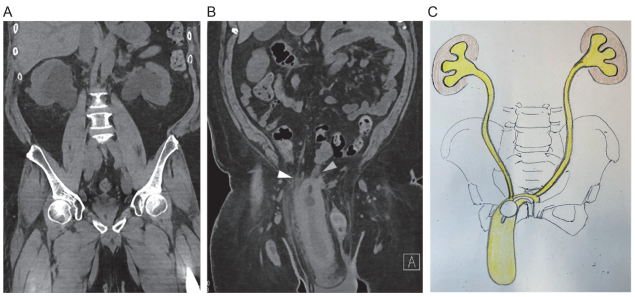

Fig. 2 Computed tomography and schematic diagram on day three

Fig. 2A: Coronal view showing persistent bilateral hydronephrosis despite bladder drainage.

Fig. 2B: An indwelling urethral catheter is used to drain the bladder successfully. However, the bladder remained prolapsed into the scrotum. Both ureters are simultaneously subjected to traction along the hernial sac. The right ureter is indicated by a gray arrow, whereas the left ureter is marked by a white arrow.

Fig. 2C: A schematic diagram of computed tomography is illustrated, summarizing the findings in Figures 2A and 2B.

Operation

Under general anesthesia, an inguinal incision was made, along with a transverse incision extending to the lower mid-abdomen. After identifying and incising the external oblique apo-

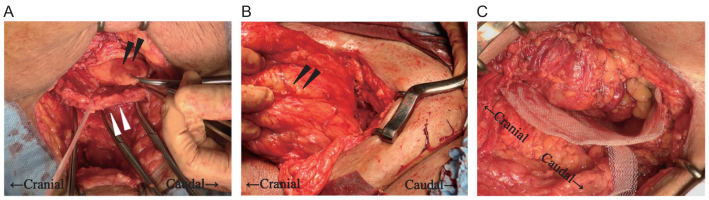

Fig. 3 Operative findings of the hernia repair

Fig. 3A: Identification of the hernial sac was challenging due to significant obesity. Based on the location of the hernial orifice (black arrows) and inferior epigastric vessels (white arrows), the patient is diagnosed with an internal inguinal hernia. The left side is cranial, and the right side is caudal.

Fig. 3B: Due to inflammation and adhesion, prolonged dissection is required to successfully detach the bladder (black arrows) from the scrotum.

Fig. 3C: The herniation is repaired using a Kugel patch.

neurosis and opening the spermatic cord, the hernial sac was found to be difficult to identify due to significant obesity. Based on its location and anatomical characteristics, the hernia was classified as an internal inguinal hernia (Fig. 3A, black arrows, hernial orifice; white arrows, inferior epigastric vessels). The bladder and scrotum exhibited inflammation and firm adhesions, necessitating an extensive dissection to separate the bladder from the scrotum. (Fig. 3B, black arrows, bladders). The bladder, prostate, and bilateral lower ureters herniated along the hernial sac. The bladder was carefully repositioned to its normal anatomical location, and Kugel patch repair was performed without complications (Fig. 3C).

Postoperative course

On postoperative day 1, computed tomography confirmed that the bladder was successfully repositioned, although bilateral hydronephrosis persisted (Fig. 4). By postoperative day 10, the creatinine levels had decreased to 2.07 mg/dL. Cystography revealed that the bladder had a pine-tree-like shape (Ogawa classification grade III), suggesting long-term bladder outlet obstruction. Vesicoureteral reflux was not observed following the infusion of 90 mL of contrast medium. In contrast to the cystographic findings, trabeculation was not prominent, and both ureteral orifices were easily visualized and positioned in their normal anatomical locations on cystoscopy. The patient's renal function and pyelonephritis improved and he was discharged on postoperative day 14 with a urethral catheter in place.

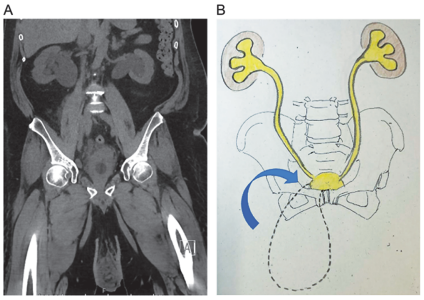

Fig. 4 Computed tomography image and schematic diagram after hernia repair

Fig. 4A: The prolapsed bladder is repositioned to an anatomically normal location. Bilateral hydronephrosis persists on the coronal view.

Fig. 4B: Schematic demonstrating successful repositioning of the prolapsed bladder following hernia repair. Despite anatomical correction, persistent bilateral upper urinary tract dilation was observed.

Progress after discharge

UDS performed on postoperative day 22 demonstrated reduced bladder compliance, indicating a decreased ability to store urine at low pressure during the storage phase. However, the patient was able to void upon initiation of the voiding command, with no residual urine observed (Fig. 5).

Despite the persistent morphological dilation of the upper urinary tract, a renal scan using mercaptoacetyltriglycine 3 revealed no evidence of functional ureteral obstruction, suggesting improved ureteral patency. Given the persistent upper urinary tract dilation and significantly reduced bladder compliance, clean intermittent catheterization (CIC) was introduced after the removal of the urethral catheter to facilitate adequate bladder emptying and prevent further deterioration of urinary function.

After removal of the urethral catheter, the patient was able to void spontaneously.

To monitor the post-void residual volume, CIC was performed four times daily after each

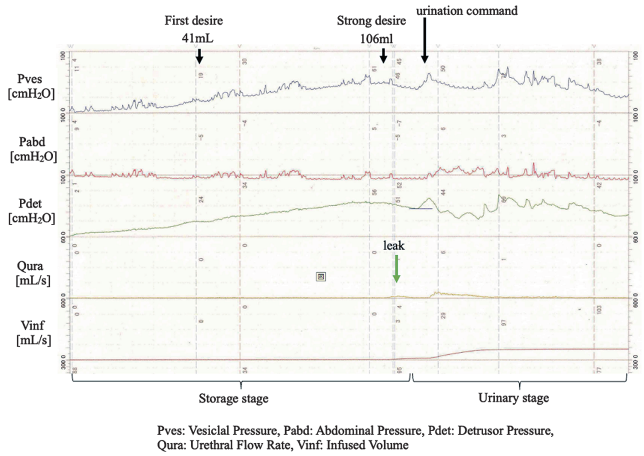

Fig. 5 Postoperative changes in urodynamic study parameters
Urodynamic study (UDS) results on postoperative day 22 reveals a markedly low bladder compliance (bladder compliance = 1.9 mL/cmH_2O). The other urodynamic findings are as follows: maximal cystometric capacity = 106 mL, no detrusor overactivity during the storage phase, detrusor pressure at maximum flow = 44 cmH_2O, maximum flow rate = 5.9 mL/s, normal flow pattern, post-void residual volume = 3 mL, bladder contractility index = 73.5.

voiding. As the post-void residual volume consistently remained below 200 mL, the frequency of CIC was reduced to twice daily after 2 weeks. On postoperative day 93, uroflowmetry showed a voided volume of 280 mL, a maximum flow rate of 20 mL/s, and an average flow rate of 10 mL/s. The urinary stream was continuous without intermittency or straining to void, and the post-void residual volume was less than 100 mL. CIC was therefore discontinued. The patient reported no symptoms of urinary urgency and did not receive any β3-agonists or anticholinergic agents during the follow-up period. Moreover, persistent bilateral hydronephrosis disappeared on computed tomography on postoperative day 109 (Fig. 6).

Fig. 6 Computed tomography image on postoperative day 109
Bilateral hydronephrosis disappears in the coronal view.

On postoperative day 337, repeat UDS demonstrated a significant improvement in UDS parameters (Table 2).

Table 2 Postoperative changes in urodynamic study parameters

Time of exam	Postoperative day 22	Postoperative day 337
Storage phase		
First desire volume (mL)	41	116
Maximum cystometric capacity (mL)	106	118
Bladder compliance (mL/cmH_2O)	1.9	118
Voiding phase		
Maximum flow rate (m/s)	5.9	16
Detrusor pressure at peak flow (cmH_2O)	44.2	4.3
Bladder contractility index	73.5	104.3

These results indicate that hernia repair and repositioning of normal anatomical structures gradually improved both storage and voiding functions, thereby enhancing the overall lower urinary tract function.

Furthermore, renal function was optimized as evidenced by a decrease in creatinine level to 1.5 mg/dL.

One year after the hernia repair, the patient had no recurrence and maintained the ability of spontaneous urination.

DISCUSSION

Surgical intervention and renal function recovery

Bladder hernia is a rare condition, occurring in approximately 1–4% of all inguinal hernias.[1] In general, bladder hernias are classified into three groups according to the anatomical relationship between the parietal peritoneum and the protruding portion of the bladder: paraperitoneal, intraperitoneal, and extraperitoneal.[2] The present case was a paraperitoneal hernia, which is the most common anatomical type of bladder hernia.[4] The herniated portion of the bladder was located along the medial wall of the hernial sac outside the peritoneal cavity.[4] As in our case, the involvement of both ureters and subsequent development of bilateral hydronephrosis leading to post-renal acute kidney injury are particularly rare.[3]

In this case, the bladder hernia was accompanied by urinary frequency. Typically, bladder prolapse remains asymptomatic and is often discovered incidentally during imaging or surgery.[2]

Our patient presented with significant obesity, which poses additional challenges for diagnosis and management.[5] Excessive adipose tissue can obscure the physical examination findings and make surgical intervention technically challenging.[6]

Furthermore, severe obesity may exacerbate intra-abdominal pressure, potentially increasing the risk of inguinal hernia.[7]

Given that hydronephrosis persisted despite initial bladder drainage, surgical intervention was deemed necessary. Although nephrostomy was initially considered, it was not performed due to the technical challenges associated with obesity.[2] Typically, percutaneous nephrostomy is preferred in patients presenting with obstructive uropathy accompanied by urinary sepsis or significant renal dysfunction, especially when their general condition is unstable and surgical risks are high. This staged approach allows infection control and renal function recovery prior to undertaking definitive hernia repair. Safavy et al reported a similar case in which nephrostomy was selected as the initial intervention, followed by surgical repair after clinical stabilization.[8] In our case, although bilateral

hydronephrosis was present and the serum creatinine level showed only a modest decrease after urethral catheterization, the patient did not meet the diagnostic criteria for urinary sepsis, and infection control was deemed acceptable. Additionally, percutaneous nephrostomy was considered technically unfeasible due to severe obesity. Based on these factors, we proceeded with early definitive hernia repair without nephrostomy.

Definitive hernia repair is generally recommended once infection has been adequately controlled and renal function has improved. Instead, early hernia repair was prioritized, leading to significant recovery of renal function and resolution of hydronephrosis over time. This suggests that in certain patients, direct hernia repair without nephrostomy may be an effective approach if renal function is closely monitored during the perioperative period.[3,8-15] The early recognition of herniation involving not only the bladder but also the ureters and its impact on renal function highlights the importance of early diagnosis and definitive surgical management in similar cases.[3,8-15]

Urodynamic evaluation and bladder function recovery

This case provides valuable insights into the functional impact of bladder herniation on lower urinary tract function. To our knowledge, this is the first report to assess lower urinary tract function using UDS. The initial UDS on postoperative day 22 revealed severely deteriorated storage and voiding functions. The gradual improvement in storage and voiding functions following surgical correction suggests that the lower urinary tract dysfunction associated with hernia impaction is at least partially reversible. On postoperative day 337, follow-up UDS revealed a marked improvement in bladder compliance, yet maximal cystometric capacity (MCC) remained relatively low. This apparent discrepancy may reflect asynchronous recovery among different components of bladder function. While improved compliance indicates reversal of ischemia-induced tissue stiffness, the limited MCC may suggest persistent impairment in neural pathways. In particular, chronic herniation involving the bladder trigone and neck may have exerted mechanical traction or compression on distal sympathetic nerves, especially those traveling via the hypogastric nerve. Anatomical studies demonstrate that the hypogastric nerves terminate near the ureterovesical junction and bladder neck, where they modulate detrusor relaxation and urine storage. Repeated displacement of this region may lead to localized sympathetic dysfunction, contributing to impaired capacity. Moreover, prolonged ischemia can cause denervation hypersensitivity, urothelial injury, inflammatory cytokine upregulation, and smooth muscle remodeling.[16,17] These factors may recover at varying rates, resulting in temporal dissociation between compliance and MCC. Despite the urodynamic limitation, the patient achieved functional recovery, as evidenced by a voided volume of 280 mL on uroflowmetry during spontaneous micturition. This finding suggests that the bladder regained sufficient storage capacity under physiological conditions, supporting the decision to discontinue CIC.

This finding is particularly relevant for patients with similar conditions because urodynamic assessments can help predict lower urinary tract function recovery and guide postoperative lower urinary tract management.

Although fluoroscopic guidance was not used during UDS, the catheter was inserted smoothly using a standard technique and advanced to a depth generally considered to place the tip in the mid-bladder region. Postoperative imaging and clinical follow-up revealed no evidence of residual herniation or abnormal compartmentalization of the bladder. While cystography was not performed specifically to assess bladder shape, the anatomical restoration was confirmed intraoperatively and supported by the resolution of hydronephrosis and restoration of spontaneous voiding. Therefore, we consider that the intravesical pressure measured during UDS accurately reflects overall bladder function, rather than pressure limited to a localized compartment.

The anatomical predisposition and longstanding displacement of the bladder in this patient may increase the risk of developing a contralateral inguinal hernia in the future. Although contralateral inguinal bladder hernia is extremely rare, it cannot be entirely ruled out. Previous reports have documented cases of bilateral bladder hernias, including one occurring after unilateral transabdominal preperitoneal repair, suggesting that chronic voiding dysfunction, abdominal wall weakness, and prior surgery may contribute to bilateral herniation.[18] At the time of surgery, we advised the patient on the importance of long-term follow-up, which has been continued to this day. Preventive strategies include weight management, avoidance of excessive straining or heavy lifting, and periodic imaging surveillance when clinically indicated. These measures aim to detect any signs of contralateral herniation at an early stage and to minimize modifiable risk factors. At present, during routine postoperative follow-up extending over 1 year, no evidence of contralateral inguinal hernia has been observed.

The possible causes of lower urinary tract dysfunction in this case were as follows:

First, lower urinary tract dysfunction may have preexisted the onset of the hernia. However, in this case, diabetes was well-controlled, reducing the likelihood of neurogenic bladder development.[19] Additionally, the patient's unremarkable prostate size and postoperative UDS findings suggested that bladder outlet obstruction was unlikely before symptom onset.[20] Therefore, this etiology was less probable.

Second, obesity may have contributed to increased intra-abdominal pressure, which could have played a role in the development of a bladder hernia.[7] The subsequent entrapment of the bladder, prostate, and ureters within the scrotum may have caused lower urinary tract obstruction, leading to distortion and angulation of the urinary tract. This may have caused increased bladder pressure, incomplete emptying, and detrusor dysfunction.

In interpreting the cause of persistent hydronephrosis, it is important to distinguish between different mechanisms, such as vesicoureteral reflux, ureteral obstruction, and ureteral peristaltic dysfunction. In our case, reflux was excluded by cystography on postoperative day 10, and mechanical obstruction was ruled out by the mercaptoacetyltriglycine 3 scintigraphy on postoperative day 30.

It is possible that the persistent upper urinary tract dilation in our case was multifactorial, involving both anatomical and functional factors. Chronic entrapment and abnormal positioning of the bladder and ureters within the scrotum may have caused excessive stretching of the supplying vasculature, leading to local ischemia and subsequent ureteral peristaltic dysfunction. Additionally, the markedly low bladder compliance observed on postoperative day 22 may have contributed to elevated intravesical pressure, potentially exacerbating ureteral kinking or urinary stasis. These combined factors likely impaired urinary flow and contributed to the development and persistence of hydronephrosis, despite anatomical correction. Chronic structural changes, such as fibrosis or ischemic remodeling of the ureteral wall due to long-standing traction and impaired perfusion, may have further delayed functional recovery even after anatomical repositioning.[16]

Unlike acute cases where hydronephrosis resolves rapidly after relief of obstruction,[21] our case showed delayed resolution over a 100-day period, likely reflecting the chronicity and structural distortion caused by prolonged herniation.

Furthermore, Kim reported that in pediatric patients, resolution of hydronephrosis may be delayed even after obstruction is relieved, depending on the initial severity and underlying anatomical or functional abnormalities.[22] These findings support the view that hydronephrosis may persist longer in cases with chronic changes, as observed in our patient.

This case emphasizes the importance of recognizing lower ureteral entrapment in cases of bladder hernia with persistent hydronephrosis. Additionally, it highlights the potential reversibility of lower urinary tract dysfunction following a hernia repair, reinforcing the need for functional

assessment through urodynamic evaluations. Given these findings, early surgical intervention should be considered when hydronephrosis persists despite bladder decompression to ensure optimal renal and lower urinary tract outcomes.

ACKNOWLEDGEMENTS

Ethical approval and consent for publication

Ethical approval was not required by the local guidelines of Komaki City Hospital for this study. Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Author contributions

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Conflict of interest

All authors declare that there are no conflicts of interest related to this study.

Financial disclosure

No external funding was received for this study.

Previous presentations

This study was presented at the 73rd Annual Meeting of the Central Section of the Japan Urology Association on 12 October, 2023 in Nara.

Miscellaneous acknowledgments

We thank all medical professionals who treated this patient, and also acknowledge Editage (www.editage.com) for their assistance with English editing.

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