

Case Report

Bilateral Hydroureteronephrosis with a Hypertrophied, Trabeculated Urinary Bladder

Showkathali IQBAL¹, Iqbal RAIZ², Iqbal FAIZ³

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¹ Amala Institute of Medical Sciences, Amala Nagar; Thrissur - 680555, Kerala, India

² Government Medical College, Kozhikode, Kerala, India

³ Madras Medical College, Chennai, Tamil Nadu, India

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Abstract

Bilateral hydroureteronephrosis involves the dilatation of the renal pelvis, calyces and ureter; it develops secondary to urinary tract obstruction and leads to a build-up of back pressure in the urinary tract, and it may lead to impairment of renal function and ultimately culminate in renal failure. Although clinically silent in most cases, it can be diagnosed as an incidental finding during evaluation of an unrelated cause. In a minority of patients, it presents with signs and symptoms. Renal calculus is the most common cause, but there are multiple non-calculus aetiologies, and they depend on age and sex. Pelviureteric junction obstruction, benign prostatic hypertrophy, urethral stricture, neurogenic bladder, retroperitoneal mass and bladder outlet obstruction are some of the frequent causes of hydroureteronephrosis in adults. The incidence of non-calculus hydronephrosis is more common in males than in females. Ultrasonography is the most important baseline investigation in the evaluation of patients with hydronephrosis. Here, we report a rarely seen case of bilateral hydroureteronephrosis associated with a hypertrophied, trabeculated bladder in an adult male cadaver, suspected to be due to a primary bladder neck obstruction, and analyse its various other causes, clinical presentations and outcomes.

Keywords: hydronephrosis, prostatic hyperplasia, urinary bladder neck obstruction, ultrasonography, urodynamics

Introduction

Hydroureteronephrosis is defined as a dilatation of the renal pelvis, calyces and ureter caused by the obstruction to free flow of urine from the kidney, leading to progressive atrophy of the renal cortex (1). It can result from an anatomical or functional process obstructing the flow of urine, which can occur anywhere from the kidneys to the urethral meatus. The urinary obstruction may be acute or chronic, partial or complete and unilateral or bilateral. The symptoms may depend on the cause, location and duration of obstruction. Acute onset is associated with renal calculus, and this is characterised by colicky pain in the flanks (2). The non-calculus aetiology of

hydroureteronephrosis varies with the age and sex of patients. Congenital pelviureteric junction (PUJ) obstruction and posterior urethral valves are common causes in children, while benign prostatic hypertrophy (BPH), urethral strictures, bladder carcinoma and bladder outlet obstruction are some of the commonest causes in adult males. It also occurs secondary to pregnancy in childbearing females, but in the elderly, cervix and ovary carcinomas are the major causes.

The gradual onset of hydronephrosis is characterised by a dull pain and discomfort in the lower abdomen due to the gradual distension of the bladder. Constant obstruction in the flow of urine result in hypertension, sepsis, urinary tract infection, haematuria and

ultimately renal failure (3). The hydronephrotic kidney is a frequent clinical condition, but hydroureteronephrosis with an enlarged trabeculated bladder is an uncommon entity in the adult population. This develops secondary to vesicourethral junction obstruction, bladder carcinoma, prostatic hypertrophy/cancer, urethral strictures and neurogenic bladder. Hydronephrosis also presents as an intra-abdominal mass with renal swelling features. Due to the widespread use of ultrasonography in clinical practice, most cases of hydronephrosis can be diagnosed before the kidney shows any clinical features or signs (4). This case report presents an uncommon entity of bilateral hydroureteronephrosis with a large trabeculated bladder in a dissection hall cadaver and discusses its causes and clinical presentations, accurate diagnostic tools for early evaluation and clinical outcomes.

Case Report

The present case report pertains to the dissection of an 82-year-old male Indian cadaver from the anatomy dissection room during routine educational dissection carried out in January 2015. The cause of death and information about urological symptoms were not available because the cadaver was donated voluntarily to the institution for educational purposes.

Gross dissection of the urinary system was performed using customary procedures. Both kidneys showed hydronephrotic features. The left kidney was smaller in size than the right kidney, measuring 84.1 mm × 29.7 mm × 34.8 mm, while the right measured 88.3 mm × 41.9 mm × 46.1 mm. The cortical thickness of the left kidney varied from 11.5 mm to 14.1 mm (average 12.8 mm), while that of right kidney was in the range of 14.7 mm–15.4 mm (average 15.05 mm). The renal pelvis of the left kidney measured 58.7 mm × 24.9 mm × 22.1 mm, while that of the right kidney measured 57.9 mm × 28.6 mm × 30.9 mm. Multiple cortical cysts of different sizes were observed on both kidneys, and some of them were found to open into the renal pelvis. There were no cysts found in the liver.

Bilateral hydroureters were observed in this case and were seen running downward into the abdominopelvic region as rambling structures traversing through the posterior wall of the bladder. The left and right ureters had maximum widths of 14.2 and 19.5 mm near the

renal pelvis and minimum widths of 3.9 mm and 4.7 mm near the bladder; they were 278 mm and 260 mm in length, respectively. The intramural lengths of left and right ureters were 27.1 mm and 26.8 mm, respectively. The distal part of the bilateral hydroureter showed no obvious narrowing or obstruction (Figure 1A & B and Figure 2A & B).

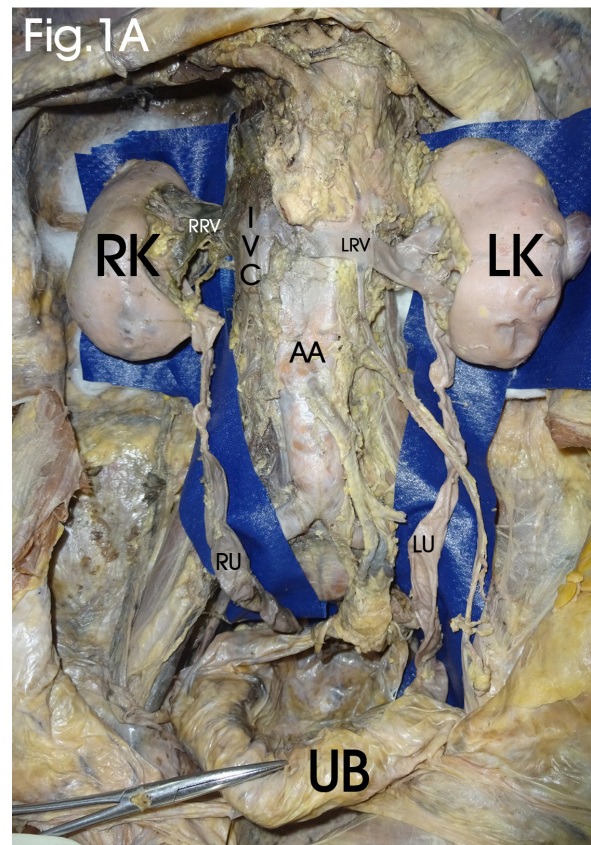


Figure 1A. Bilateral hydronephrotic kidneys, hydroureters and hypertrophied, trabeculated urinary bladder. Kidney shows bilateral, multiple communicating cysts; ureters are dilated bilaterally.
RK: Right kidney; LK: left kidney; RRV: Right renal vein; LRV: Left renal vein; AA: Abdominal aorta; RU: Right ureter; LU: Left ureter.

The urinary bladder measured 142 mm in length, 135 mm in breadth and 6.3 mm in thickness. The inner wall showed numerous trabeculae interwoven in different directions, except for that of the trigone region. The trigone measured 36.7 mm between the left and right ureteral orifices and 51.6 mm between the ureteral and urethral orifices. The ends of the

ureters formed spiral structures as they passed through bladder wall, with relatively normal left and right ureteral orifices, which were 1.5 mm and 1.2 mm, respectively, in diameter. The urethral orifice was patent, measuring 3.3 mm × 1.9 mm. The size and structure of the prostate was anatomically normal and the prostatic portion of the urethra was not obstructed (Figure 3A & B).

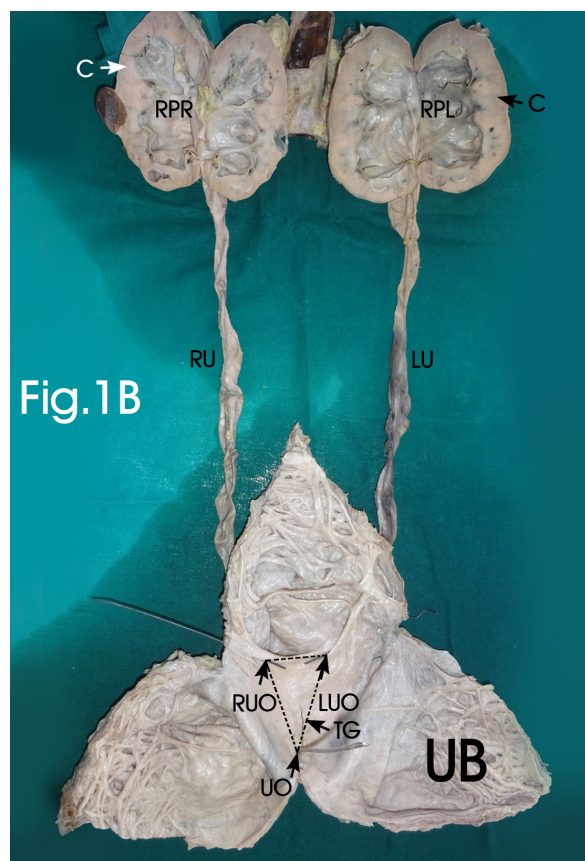


Figure 1B. Sagittal sections of hydronephrotic kidneys, hydroureters and interior of the hypertrophied bladder showing trigone. Renal pelvis and calyces are markedly dilated; pyramids and medulla are attenuated; bladder shows trabeculated anterior wall; both ureterovesical junctions are patent and probed; urethral orifice was patent and probed
RPR: Dilated right renal pelvis; RPL: Dilated left renal pelvis; RU: Right hydroureter; LU: Left hydroureter; TG: Trigone; RUO: Right ureteric orifice; LUO: Left ureteric orifice; UO: Urethral orifice.

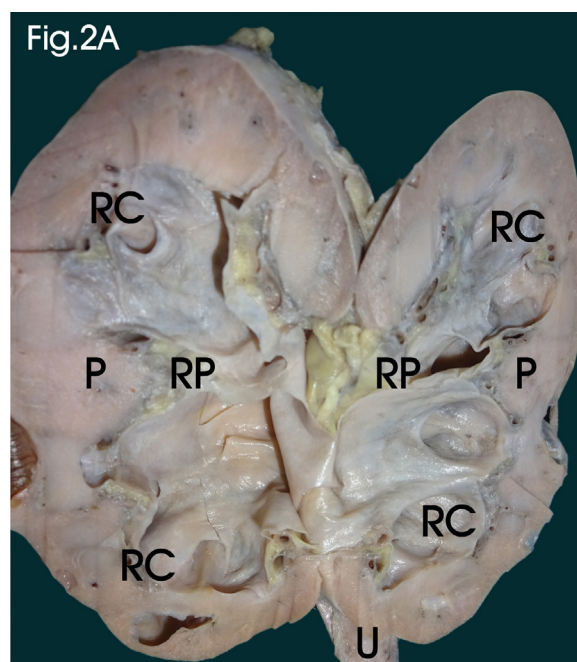


Figure 2A. Sagittal section of right kidney
Dimensions: Kidney: 88.3 mm × 41.9 mm × 46.1 mm; cortical thickness: 15.05 mm (range: 14.7 mm to 15.4 mm); renal pelvis: 57.9 mm × 28.6 mm × 30.9 mm; Ureter length: 260 mm; width max: 19.5 mm; min: 4.7 mm; intramural length: 26.8 mm.
RP: Dilated renal pelvis; RC: Dilated renal calyces; P: Attenuated renal pyramids; U: Dilated ureter

A cut section of kidney showed multiple communicating cysts, and the pelvis and calyces were markedly dilated. Pyramids and medulla were attenuated, and the ureters were dilated bilaterally. Both ureterovesical junctions were probed and the ureteric orifices were observed bilaterally. The bladder appeared enlarged, and its cut section shows a thickened and trabeculated anterior wall. Patent urethra was identified, while the prostate appeared normal. Histopathological examination of the kidney showed glomeruli and tubules with interstitial fibrosis and chronic inflammatory infiltrate in the interstitium. Many glomeruli showed periglomerular fibrosis, and a few glomeruli were completely hyalinised. The tubules showed dilation and amorphous eosinophilic material deposition in the lumen (colloid cast). The blood vessels exhibited thick, hyalinised vessel walls. Moreover, the transitional epithelium of the pelvis appeared attenuated. Sections

from the bladder showed the epithelial lining, muscularis mucosae, a hypertrophied muscular wall and adventitia. The histopathological report confirmed that the picture was consistent with chronic pyelonephritis involving both kidneys (Figure 4 A, B, C, D & E).

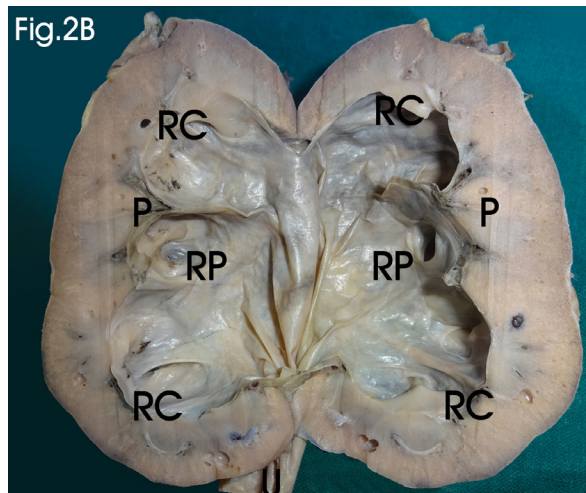


Figure 2B. Sagittal section of left kidney
Dimensions: Kidney: 84.1 mm × 29.7 mm × 34.8 mm; cortical thickness: 12.8 mm (range: 11.5 mm to 14.1 mm); renal pelvis: 58.7 mm × 24.9 mm × 22.1 mm; Ureter: length: 278 mm; width max: 14.2 mm; min: 3.9 mm; intramural length: 27.1 mm.
RP: Dilated renal pelvis; RC: Dilated renal calyces; P: Attenuated renal pyramids; U: Dilated ureter

Discussion

Hydroureteronephrosis is a common clinical condition encountered by urologists, emergency medical specialists and primary care physicians. It can be physiological or pathological, acute or chronic and unilateral or bilateral. It is always secondary to urinary tract obstruction, but it can also be present without obstruction. The aetiology and presentation of hydroureteronephrosis in adults differ from those of neonates and children. BPH or carcinoma, bladder stones, retroperitoneal or pelvic neoplasms, neurogenic bladder, bladder neck obstruction and urethral stricture are some of the primary causes in older adults; less common causes include cystocele, foreign objects, posterior urethral valves, urethral

spasms and urethral diverticula. In contrast, in young adults, calculi are the most common cause. The symptoms of bilateral hydronephrosis may vary depending on the causative factor, and they include abdominal pain, continuous feeling of a full bladder, frequent urination, acute urinary retention, dysuria, urine hesitancy, slow urine flow, urinary intermittency, nocturia, haematuria, urinary tract infections with burning micturition and ultimately the signs and symptoms of kidney failure like, nausea, fatigue and fluid retention (5, 6).

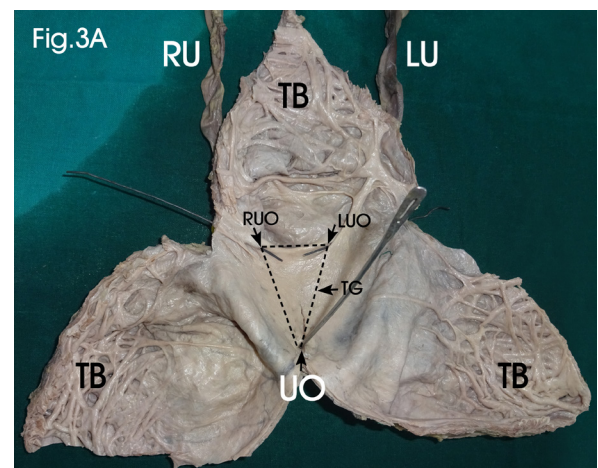


Figure 3A. Interior of hypertrophied urinary bladder
Enlarged bladder shows thickened and trabeculated anterior wall; both ureterovesical junctions are probed and ureteric orifices are made out bilaterally; urethra was patent and probed
Dimensions: 142 mm × 135 mm × 6.3 mm; Trigone interureteric distance: 36.7 mm; uretero-urethral distance: 57.6 mm; diameter of ureteric orifice left: 1.5 mm; right: 1.2 mm; urethral orifice: 3.3 mm × 1.9 mm.
TB: Trabeculated wall of the bladder; TG: Smooth trigone; RUO: Right ureteric orifice; LUO: Left ureteric orifice; UO: Urethral orifice

The incidence of non-calculus hydronephrosis is higher in the third to eighth decade of life. It is observed more commonly in males than females, at a ratio of 2:1, and the right side is most commonly affected (5, 7). Kasabe et al. (8) analysed 50 patients with non-calculus hydronephrosis with hydroureter and

concluded that PUJ obstruction is the most common cause of hydronephrosis, accounting for 38% of cases. Benign prostatic hypertrophy is the second most common cause, at 22%, followed by vesicoureteral reflex (VUR; 16%), gravid uterus (8%) and retroperitoneal mass (6%). Other less-known causes include urethral stricture, neurogenic bladder, ureteric stricture and bladder outlet obstruction, which accounted for 2% of each of the remaining cases in Kasabe et al.'s study. The most frequent complaint in patients with non-calculus hydronephrosis with hydroureter was pain followed by frequency of micturition (8).

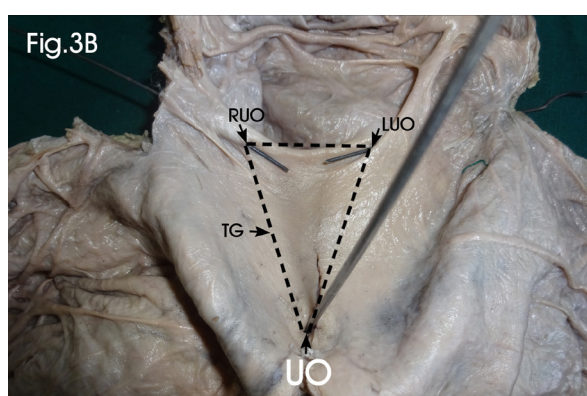


Figure 3B. Enlarged view of trigone of the bladder
TG: Smooth trigone; RUO: Right ureteric orifice; LUO: Left ureteric orifice; UO: Urethral orifice

About 30% of patients with hydronephrosis have PUJ obstruction. The usual presenting complaints are dull aching pain located in the lumbar region, increased frequency of micturition, haematuria and painful micturition. BPH is the most common cause of hydroureteronephrosis in adults over 60 years of age, accounting for 70% of the cases. The most bothersome symptoms include nocturia, followed by urgency and burning micturition. Transurethral resection of the prostate is the gold standard surgical modality for obstructing BPH, and it has gained popularity among urologists because of its success rate (9). Urethral stricture seems to be the other important factor initiating hydroureteronephrosis. Patients with urethral stricture are treated with excision with primary anastomosis, and a high success rate of 98.8% has been observed. There are few complications, and these are self-limited and of short duration. Some patients with recurrent stricture need

intermittent dilatations, urethral reconstructions and anastomosis urethroplasty (8, 10).

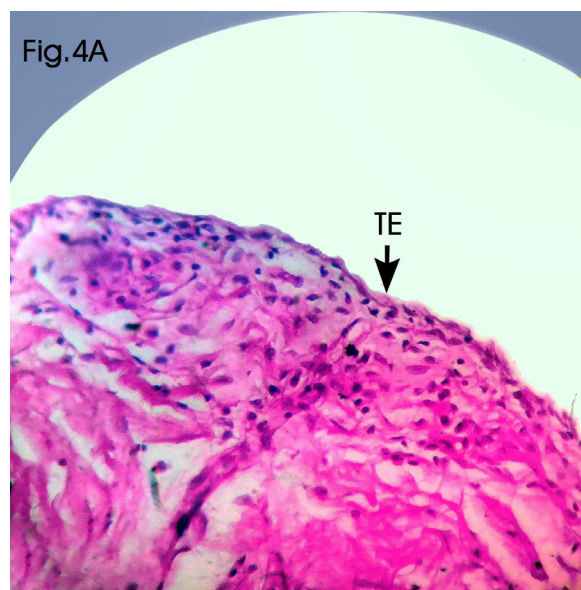


Figure 4A. Cut section of urinary bladder showing transitional epithelium (H & E stain, 10X magnification)
Sections of the bladder wall showing the attenuated transitional epithelium with hypertrophied detrusor muscular layer
TE: Transitional epithelium

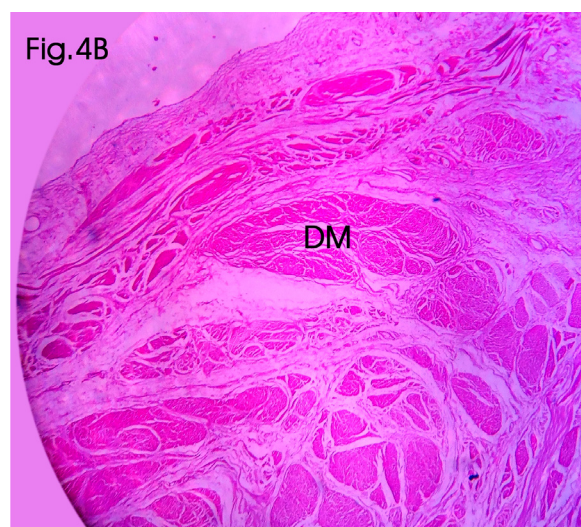


Figure 4B. Cut section of urinary bladder showing hypertrophied detrusor muscle fibres (H & E stain, 10X magnification)
DM: Hypertrophied detrusor muscle fibres

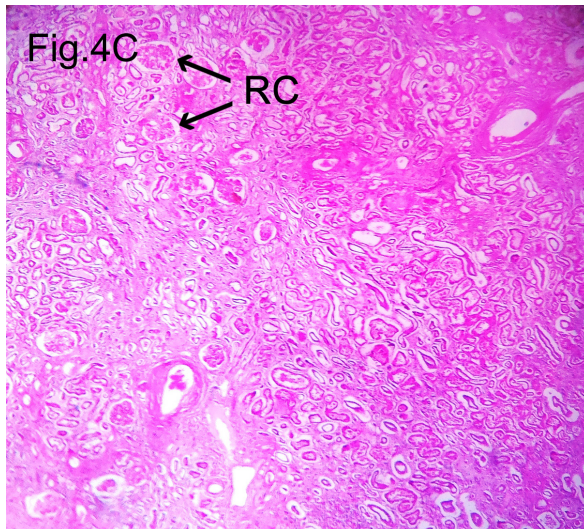


Figure 4C. Cut section of hydronephrotic kidney showing attenuated renal parenchyma (H & E stain, 4X magnification)

Sections of the kidney showing interstitial fibrosis with chronic inflammatory infiltrate in interstitium; many of the glomeruli show periglomerular fibrosis and some of them were completely hyalinised; renal tubules show dilation with amorphous eosinophilic material deposition in the lumen (thyroidisation); blood vessels show thick hyalinised vessel wall and the transitional epithelium of the pelvis was attenuated.

RC: Renal corpuscles

Mild hydronephrosis in pregnancies is considered a normal phenomenon, and it is seen in 90% of pregnancies. It is frequently observed in the right kidney in primigravida after the second trimester. This is due to the effects of progesterone, as well as the mechanical compression of the ureters at the pelvic brim. In one study, spontaneous regression was seen in all cases in the postpartum period after 6–12 weeks (11). In addition, a retroperitoneal mass may sometimes cause compression of ureters, with the development of hydronephrosis and hydroureter as the disease progresses. Pelvic inflammatory diseases in the form of tubo-ovarian abscess, uterine fibroids and other benign gynaecological neoplasms may cause ureteral obstruction, leading to hydronephrosis. Complete surgical excision has been advised in these cases, and an excellent long-term

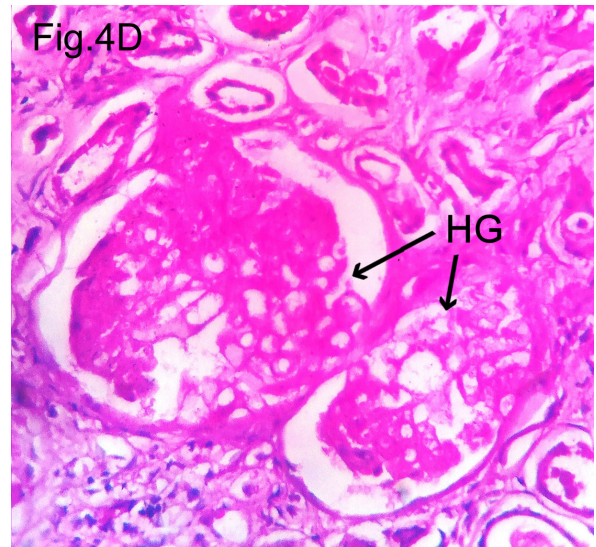


Figure 4D. Cut section of hydronephrotic kidney showing hyalinised glomerulus

(H & E stain, 10X magnification)
HG: Hyalinised glomerulus

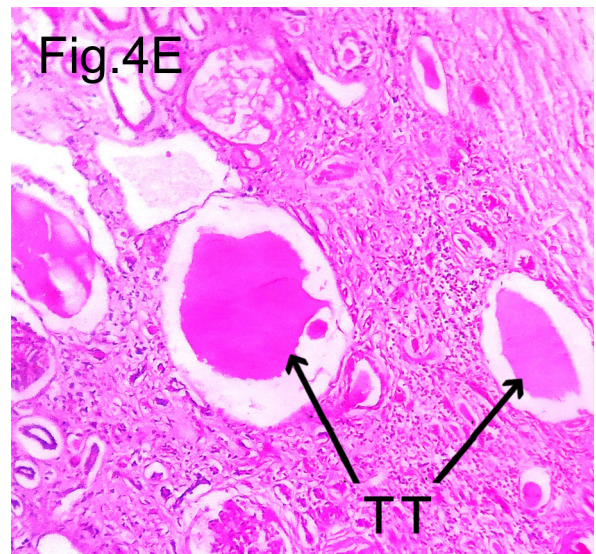


Figure 4E. Cut section of hydronephrotic kidney showing thyroidisation of renal tubules

(H & E stain, 10X magnification)
TT: Thyroidisation of renal tubules

prognosis has been reported (8). Neurogenic bladder is another aetiological factor causing hydronephrosis. Patients with neurogenic incontinence have been addressed conservatively with intermittent catheterisation and bladder relaxants (12).

Other less important factors that can induce hydronephrosis include ureteric stricture and VUR. Except in cases of primary PUJ obstruction, ureteral strictures are acquired, and they are usually iatrogenic. The treatment of choice depends on the length, location and cause of the stenosis. In one study, most patients were managed with balloon dilation and endoureterotomy, while a few required open surgical repair; the authors reported a 97% overall success rate (13). VUR usually affects children. In one study, about 30% of children with urinary tract infections were diagnosed with VUR after voiding cystourethrogram. Either these children were managed conservatively with antibiotic prophylaxis, or they underwent ureteric re-implantation (14).

A primary bladder-neck obstruction (PBNO) causes failure of the bladder neck to open adequately during voiding, resulting in obstruction of urinary flow in the absence of an anatomic obstruction. Marion first described PBNO in men in 1933. In 1973, Turner-Warwick and co-workers advocated the use of urodynamics and voiding cystourethrography to diagnose bladder-neck dysfunction in men aged 50 years or younger with a long history of lower urinary tract symptoms. The precise cause of PBNO has not been clearly elucidated. Various theories have focussed on structural changes at the bladder neck in the form of fibrous narrowing or hyperplasia; faulty dissolution of the mesenchyme at the bladder neck or inclusion of abnormal amounts of non-muscular connective tissue, resulting in hypertrophic smooth muscle, fibrous contractures and inflammatory changes; or inefficient bladder neck opening resulting from abnormal morphologic arrangement of the detrusor/trigonal musculature (15). PBNO can present with voiding symptoms, such as hesitancy, decreased force and incomplete emptying; storage symptoms like urgency, frequency and nocturia; or a combination of both. Diagnosis is usually determined through videourodynamic testing showing relatively high-pressure, low-flow voiding with radiographic evidence of obstruction at the bladder neck, relaxation of the striated sphincter and no evidence of distal obstruction. PBNO can be judiciously managed with intermittent catheterisation, alpha-blockers and bladder neck incision using a paediatric resectoscope (4, 16).

In their retrospective review of 137 male patients aged around 50 years with chronic voiding dysfunction and abnormal urodynamics, Kaplan et al. reported an incidence of 54% of

PBNO (17). Nitti et al. reported an incidence of 47% of PBNO in their prospective study of 85 patients aged 18–45 years with lower urinary tract symptoms (18). In addition, Yang et al. studied 85 Taiwanese men under 55 years with chronic voiding dysfunction and an obstructive uroflow pattern, and they reported a 33% incidence (19). Data on the incidence and prevalence of PBNO in the female population are extremely limited. Information on the incidence and prevalence rates of PBNO in Indian and other South Asian populations is also scant, and this has not been reported in the current medical literature. In addition, the studies described above excluded older men to avoid the possible contribution of BPH.

The usual investigations include blood analysis for urea and creatinine to assess renal damage; excretory, antegrade or retrograde urography to ascertain narrowing of the urethra; uroflowmetry, urodynamic testing and cystoscopy; ultrasound, contrast-enhanced computed tomography (CT) and magnetic resonance imaging scans; and urine analysis and culture to check infection. Abdominal ultrasonography is a useful, non-invasive technique facilitated by CT scans to pinpoint the accurate diagnosis of obstructive uropathy. Despite the advancement of diagnostic modalities, however, it is difficult to differentiate hydronephrosis from other abdominal cyst formations. There is a long list of differential diagnoses, which includes ovarian cysts, retroperitoneal haematoma, hepatobiliary cysts, mesenteric cysts, pseudomyxoma, cystic renal tumours, retroperitoneal tumours, ascites and splenomegaly (20, 21).

The histopathological examination in the present case showed that the pelvis of both kidneys was grossly dilated, with attenuation of pyramids and medulla of both sides; the ureters were dilated bilaterally, and ureterovesical junctions were patent. The bladder appeared grossly enlarged and its cross-section showed a thickened and trabeculated anterior wall. The urethra was patent, and the prostate appeared normal without any apparent enlargement. The haematoxylin/eosin section of the kidney showed interstitial fibrosis with chronic inflammatory infiltrate in the interstitium. Many of the glomeruli showed periglomerular fibrosis, and some of them were completely hyalinised. The renal tubules exhibited dilation with amorphous eosinophilic material deposition in the lumen. The blood vessels show thickened hyalinised vessel walls, and the transitional epithelium

of the pelvis was attenuated. In addition, the bladder wall showed an attenuated epithelium with a hypertrophied muscular wall. The histopathological report confirmed chronic pyelonephritis involving both kidneys with a hypertrophied, trabeculated bladder and attenuation of the epithelium. Speakman et al. (22) have observed that the most common pathophysiological findings in men with obstructive nephropathy is chronic interstitial pyelonephritis. In high-pressure chronic retention, there is bladder outflow obstruction and high voiding detrusor pressure with a poor flow rate, leading to persistently high pressure within bladder; this causes retrograde pressure and bilateral hydronephrosis (22). The increased intravesical pressure further causes detrusor hypertrophy with connective tissue collagen impregnation, leading to a hypertrophied and trabeculated bladder (23).

Tazi et al. (4) reported a rare case of bilateral giant hydronephrosis with a hypertrophied and trabeculated urinary bladder due to bladder neck obstruction in a 42-year-old male; they suggested that hydronephrosis is secondary to bladder-neck obstruction, which leads to a build-up of back pressure in the urinary tract, thereby causing renal function impairment (4). In addition, Ralte et al. (24) reported a case of a hypertrophied, trabeculated urinary bladder in an elderly male aged 79 years during a routine classroom dissection, and they further added that the detrusor smooth muscle underwent hypertrophy with abundant deposition of collagen in between smooth muscle fibres due to bladder outlet obstruction, which is similar to our study findings (24). Chiang et al. (25) reported four cases of giant hydronephrosis and stated that an erroneous diagnosis, such as ovarian cyst, retroperitoneal hamartoma or hepatic cyst, is often made in these cases; they suggested a two-stage procedure with slow decompression by percutaneous nephrostomy before the nephrectomy is preferred in the compromised patient (25). Sataa et al. (26) retrospectively reviewed 24 cases of giant hydronephrosis in adults and suggested that in extremely poorly functioning surgical units, nephrectomy is the procedure of choice; in contrast, in salvageable units, the anatomical configuration should dictate the type of reconstructive procedure (26).

As we could probe the ureteric and urethral orifices in the urinary bladder successfully without any resistance, and since the prostate

was normal, we suspect that PBNO due to bladder-neck dysfunction—in which the bladder neck fails to open adequately during voiding—may have been the cause in this case. This would lead to the secondary hypertrophy, causing an enlarged and trabeculated bladder. Obstruction in the lower urinary tract causes increased pressure in the renal pelvis due to a reflux of urine into the kidney. This increased pressure is transmitted back to the delicate tissues forming the filtration system of the kidneys, which eventually leads to atrophy of the renal cortex with loss of function. The back pressure also causes renal tubular atrophy, glomerular hyalinisation and fibrosis. The old age of the occurrence of PBNO in this patient can be explained by the fact that the patient was from a poor socioeconomic background; moreover, he may have chronically ignored his urological symptoms. As we are not in the possession of any antemortem medical history of the above patient, we are not in a position to confirm our present conception.

Conclusion

Non-calculus hydronephrosis is most common in adults in the third to eighth decades of life, and males are more commonly affected than females. Most patients present with abdominal discomfort, pain and frequency of micturition. PBNO due to bladder-neck dysfunction is one of the causes of the development of bilateral hydroureteronephrosis with hypertrophied bladder. Of the diagnostic modalities employed in the evaluation of patients with hydronephrosis, abdominal ultrasound is the single most important baseline investigation, followed by contrast-enhanced CT and intravenous pyelogram.

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Conflicts of Interest

None

Authors' Contributions

Conception and design: SI, IR, IF

Analysis and interpretation of the data: SI, IR, IF

Drafting of the article: SI, IR, IF

Critical revision of the article for important intellectual content: SI, IR, IF

Final approval of the article: SI, IR, IF

Provision of study materials or patients: SI, IR, IF

Administrative, technical, or logistic support: SI, IR, IF

Collection and assembly of data: SI, IR, IF

Correspondence

Dr Showkathali Iqbal
Professor of Anatomy
MBBS, MS (Anatomy)
Amala Institute of Medical Sciences,
Amala Nagar, Thrissur - 680555.
Kerala, India
Tel: +91 480 2824461 (Residence)
+91 487 2304116 (Office)
Mobile: +91 9447085253
Fax: +91 487 2307969
E-mail: dr.iqbal.s@gmail.com

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