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Case Report

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BLADDER OUTFLOW OBSTRUCTION IN YOUNG MALE - A DIAGNOSTIC DILEMMA

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ABSTRACT

Bladder outlet obstruction in young adults is uncommon and can be caused by conditions such as phimosis, calculi, urethral stricture, prostatitis, and neurogenic disorders. We present a rare case of Zinner syndrome, a Mullerian duct anomaly, causing bladder outlet obstruction. Our patient presented with obstructive urinary symptoms, underwent evaluation, and was treated with cystoscopy and excision of a right seminal vesicle cyst. Post-procedure, he was cured of his symptoms and is currently on regular follow-up. This case is reported due to its rarity.

INTRODUCTION

Zinner syndrome, a congenital malformation of the Müllerian duct (Wolffian), is characterized by a triad of ipsilateral seminal vesicle cyst, unilateral renal agenesis, and ipsilateral ejaculatory duct obstruction. Patients typically remain asymptomatic until their third or fourth decade of life, although some may present with infertility or symptoms of bladder outflow obstruction. The condition originates from an insult during the first trimester of pregnancy, leading to aberrations in the embryogenesis of the genitourinary system, affecting structures from the kidney to the vas deferens. This case report presents a young adult with an uncommon presentation of Zinner syndrome, experiencing bladder outlet obstruction. Early diagnosis and treatment significantly improved the patient's quality of life.

CASE PRESENTATION

A 23-year-old male presented with painful micturition and difficulty in passing urine freely for 10 days. He reported a history of poor urine flow for the last 2-3 years. Abdominal examination was normal; however, a digital rectal examination revealed a palpable cystic mass to the right of the midline, measuring approximately 1.5×2 cm. Laboratory parameters were normal, except for semen analysis, which showed oligo-asthenospermia.

Transabdominal ultrasound and CT urogram indicated the absence of the right kidney in the renal fossa, with compensatory hypertrophy of the left kidney. A lobulated, heterogeneously enhancing soft tissue lesion was identified posterior to the urinary bladder on the right lateral aspect, indenting into the bladder (Figure 1).

MRI of the abdomen and pelvis revealed a well- defined hyperintense tubular cystic lesion in the region of the right seminal vesicle, suggestive of a seminal vesicle cyst. Another dilated and tortuous tubular cystic lesion was closely positioned to the right seminal vesicle cyst, indicating ejaculatory duct obstruction (Figure 2) Based on the clinical presentation and radiological findings, the patient was diagnosed with Zinner syndrome. Cystoscopy was performed followed by excision of the seminal visualization of the right ureteric orifice. An exploratory laparotomy showed a cyst in the right para vesicular area with extension of a dilated tubular structure towards the right paracolic region. The cyst along with the proximal tubular structure (likely the right ureter) was excised in toto. (Figure 3)

The postoperative period was uneventful. The patient was followed up with regular monthly intervals and showed symptomatic improvement. Uroflowmetry revealed a Qmax of 30 ml/sec, and semen analysis showed improvement in sperm count and motility.

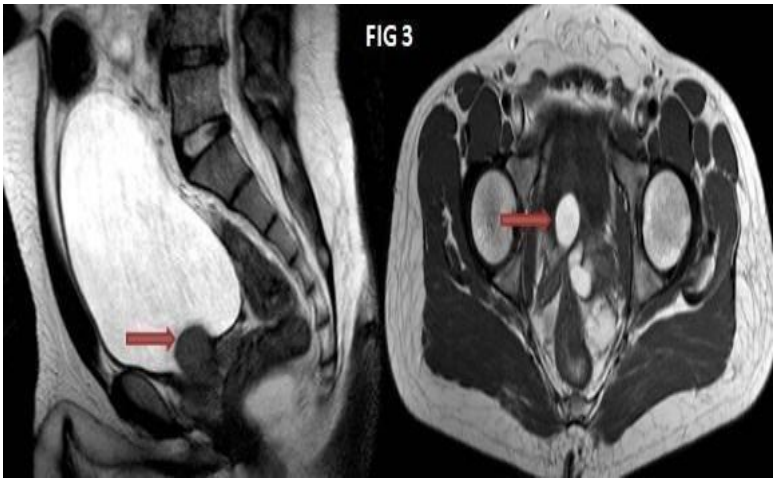

Fig. 1

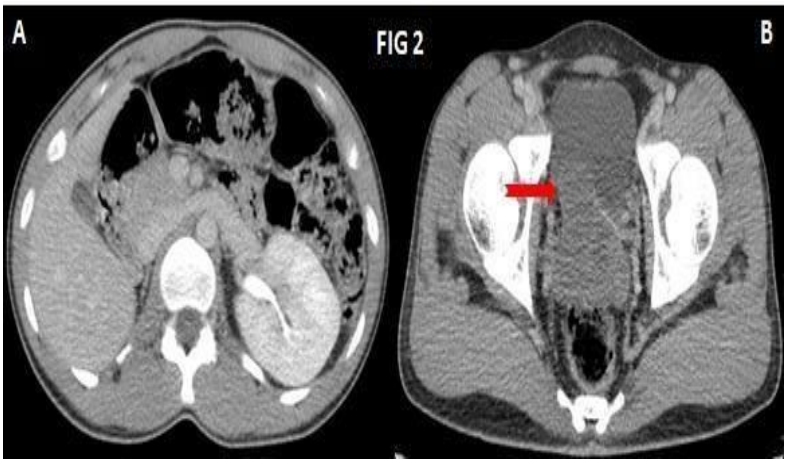

Fig. 2

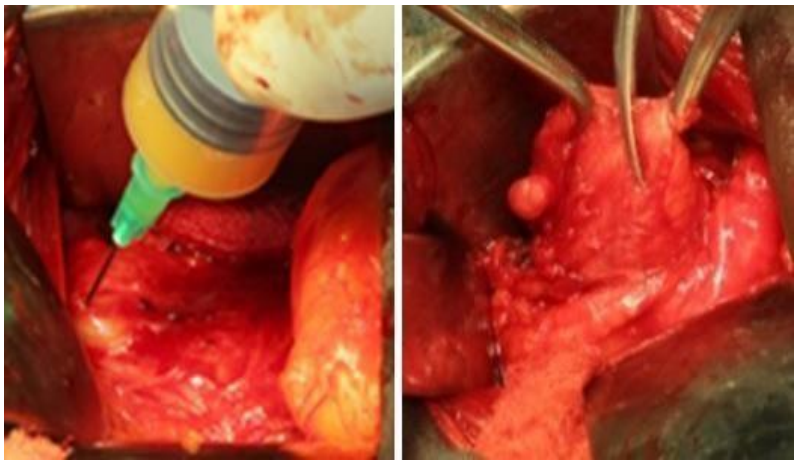

Fig. 3

DISCUSSION

Congenital malformations of the seminal vesicle are often linked to ipsilateral upper urinary tract malformations due to their common origin from the mesonephric (Wolffian) duct. This association was first identified by Zinner in 1914, and by 2000, only about a hundred cases had been reported. Zinner's syndrome is characterized by a triad of ipsilateral renal agenesis, ipsilateral seminal vesicle cyst, and ipsilateral ejaculatory duct obstruction. The Müllerian duct, a paired structure present during embryogenesis, develops into various male reproductive structures under the influence of testosterone and anti-Müllerian hormone. These structures include the hemitrigone, bladder neck, urethra (proximal to the external sphincter), seminal vesicle, vas deferens, efferent ducts, epididymis, paradidymis, and appendix of epididymis.

Disturbances during the first trimester can adversely affect the embryogenesis of the kidney, ureter, seminal vesicle, and vas deferens. Maldevelopment of the distal mesonephric duct can lead to atresia of the ejaculatory duct, causing obstruction and cystic dilation of the seminal vesicle, and abnormal ureteral budding, resulting in renal agenesis or dysplasia. Obstruction at the ejaculatory duct level leads to the gradual accumulation of secretions in the seminal vesicle, forming cysts. This developmental pathology can cause azoospermia or oligospermia, often manifesting as primary infertility. The presence or absence of associated renal agenesis or dysplasia depends on the timing of the embryogenic insult, with earlier insults (before seven weeks of gestation) more likely to result in renal agenesis.

Most patients with mesonephric duct anomalies remain asymptomatic until their third or fourth decade of life, typically becoming symptomatic during periods of high sexual or reproductive activity. Asymptomatic seminal vesicle cysts, usually smaller than 5 cm, are often incidentally discovered during digital rectal examinations or imaging studies. Symptomatic presentations can include pelvic or perineal pain, lower urinary tract symptoms (LUTS), painful ejaculation, chronic recurrent epididymitis/prostatitis, and occasionally infertility. Larger cysts (greater than 12 cm), known as giant cysts, may cause bladder and colonic obstruction. Malignant transformation of seminal vesicle cysts, though rare, has also been reported.

While ultrasound and CT imaging may not accurately distinguish the origin of a cyst, they typically reveal a retrovesical mass superior to the prostate gland. MRI is the preferred imaging modality for evaluating mesonephric duct malformations, as it can accurately differentiate seminal vesicle cysts from other pelvic cystic anomalies. Management of these cysts depends on symptomatology; asymptomatic cysts are generally managed with observation, while symptomatic cysts unresponsive to conservative treatment may require invasive procedures such as surgical resection or cyst aspiration.

CONCLUSION

Developmental anomalies of the urogenital system are often identified late due to their vague symptomatology in older patients. Our patient, who presented with a few lower urinary tract symptoms that had previously gone undiagnosed by others, was accurately diagnosed through our thorough clinical examination and radiological imaging. He subsequently received prompt and effective management. This case report highlights the importance of a proper diagnostic workup in revealing congenital anomalies, which can significantly improve the quality of life for affected individuals.

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