Wolffian duct derivative anomalies: technical considerations when encountered during robot-assisted radical prostatectomy

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ACHARYA SS, GUNDETI MS, ZAGAJA GP, SHALHAV AL, ZORN KC. Wolffian duct derivative anomalies: technical considerations when encountered during robot assisted radical prostatectomy. The Canadian Journal of Urology. 2009;16(2):4601-4606.

Background: *Although malformations of the genitourinary tract are typically identified during childhood, they can remain silent until incidental detection in evaluation and treatment of other pathologies during adulthood. The advent of the minimally invasive era in urologic surgery has given rise to unique challenges in the surgical management of anomalies of the genitourinary tract.*

Objective: *This article reviews the embryology of anomalies of Wolffian duct (WD) derivatives with specific attention to the seminal vesicles, vas deferens, ureter, and kidneys. This is followed by a discussion of the history of the laparoscopic approach to WD derivative anomalies. Finally, we present two cases to describe technical considerations when managing these anomalies when encountered during robotic-assisted radical prostatectomy.*

Design, setting, and participants: *The University of Chicago Robotic Laparoscopic Radical Prostatectomy (RLRP) database was reviewed for cases where anomalies of WD derivatives were encountered. We describe how modifications in technique allowed for completion of the procedure without difficulty.*

Measurements: *None*

Results and limitations: *Of the 1230 RLRP procedures performed at our institution by three surgeons, only two cases (0.16%) have been noted to have a WD anomaly. These cases were able to be completed without difficulty by making simple modifications in technique.*

Conclusions: *Although uncommon, it is important for the urologist to be familiar with the origin and surgical management of WD anomalies, particularly when detected incidentally during surgery. Simple modifications in technique allow for completion of RLRP without difficulty.*

Key Words: robotic prostatectomy, embryology, Wolffian duct, anomalies, technical considerations

Introduction

The incidence of congenital anomalies of the genitourinary tract is estimated to occur in 10%-14% in the general population.[1] With this frequency in mind, it is not surprising that patients undergoing evaluation and treatment for clinically localized prostate cancer occasionally present with additional urologic pathologies. Although malformations of the genitourinary tract are typically identified during childhood, they can remain silent until incidental detection during the evaluation and treatment of other pathologies during the adult years.[2] In rare instances, these anomalies are first identified intraoperatively.

Accepted for publication December 2008

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The advent of the minimally invasive era in urologic surgery has given rise to unique challenges in the surgical management of anomalies of the genitourinary tract. With continually increasing employment of robotic assistance during radical prostatectomy (RRP), the urologist must be comfortable with managing these anomalies intraoperatively. In this article, we comprehensively report on the embryologic pathways, diagnosis, and management of anomalies of Wolffian duct (WD) derivates; specifically anomalies of the seminal vesicles (SV), vas deferens (VD), ureter, and renal development. We also briefly describe two cases in which a WD anomaly was incidentally encountered during RRP, and discuss technical challenges and limitations these had during surgery.

Wolffian duct development

The WD serves as the common origin for the development of the VD, epididymis, SV, and collecting system of the kidney. Congenital aberrations of these structures can occur in isolation, or in association with other WD abnormalities. The presence or absence of renal dysplasia concomitant with reproductive ductal anomalies can be explained by the chronology of mesonephric development, Figure 1.

The ureteric bud originates from the caudal aspect of the mesonephric duct in the fourth week of gestation and separates by absorption and cranial migration in the seventh week. The ureteral bud then penetrates the metanephros to give rise to the major calyces and the primitive renal pelvis. In the 12th week of development, under androgen influence, the SVs develop as symmetric, bulbous, dorsolateral swellings of the distal mesonephric duct, just proximal to the ejaculatory ducts, where they drain into the urethra. In the 14th week of fetal life, the SV begin to separate into the ampullae and lateral diverticula. They then transform into hook like ducts during the 16th and 20th weeks of development. Together, the SV, ampullae of the VD, and the ejaculatory ducts form a functional unit that continues to develop until puberty.

Seminal vesicle anomalies

Congenital anomalies of the SV can be categorized into abnormalities of number (agenesis, fusion, duplication), maturation (hypoplastic, atretic), position (ectopia) and structure (diverticulum, cyst, communication with the ureter). In clinical practice, agenesis and cystic anomalies are the most encountered. Although rare, SV anomalies are likely to be increasingly encountered because of the continued growth in the use of pelvic CT and MRI.[3]

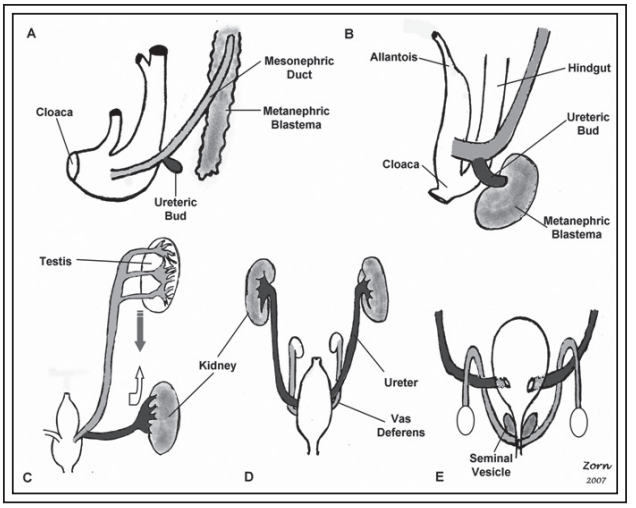

Figure 1. Chronology of mesonephric development. (A) ureteric bud originating from the caudal aspect of the mesonephric duct in the fourth week of gestation. (B) ureteric bud penetrating and inducing differentiation of the metanephros (primitive kidney) by seventh week of gestation. (C) starting after the eighth week of gestation, the testis descends and the renal unit ascends, during which time the ureteric bud bifurcates repeatedly giving rise to the renal pelvis, calyces, infundibula and collecting system. (D) kidneys and ureters in final position anterior and superior to testes and vas deferens by 10th to 11th week of gestation. (E) by the 12th week of gestation, the seminal vesicles develop as symmetric, bulbous, dorsolateral swellings of the distal mesonephric duct, just proximal to the ejaculatory ducts, where they drain into the urethra.

Anomalies of number

SV agenesis does not occur as an isolated phenomenon and is seen in two clinical scenarios; namely, renal agenesis and cystic fibrosis (CF). An insult to the mesonephric duct during embryogenesis results in associated ipsilateral or bilateral agenesis or ectopia of the VD. If the insult occurs before the seventh week of gestation, prior to ureteral budding, the patient will have associated ipsilateral renal agenesis. In patients with CF, bilateral or unilateral SV agenesis is associated with corresponding agenesis of the VD, and normal kidneys. Mutation in the transmembrane chloride conductance regulator gene, *CFTR*, result in the manifestations of CF. These patients are considered to have a primary genital form of CF with agenesis due to luminal blockade of the VD and SV by thick secretions.[4]

In addition to agenesis, anomalies of SV number include fusion (one common SV) and duplication (three or four total SV). Malatinksy et al previously reported that such SV anomalies occur at 5% in the general population.[5] In a study of 158 patients examined for sterility, deferentovesiculography was performed to evaluate for congenital anomalies of the VD, SV, ejaculatory ducts, and the utricle. The most common anomaly found was duplication of the SV (four cases). Other SV anomalies included hypoplasia (three cases), and fusion (one case).

Anomalies of structure

Although spermatogenesis is intact, the most common presentation of SV anomalies is infertility. If the ecotpic vas deferens inserts into a mullerian duct remnant, dilation occurs, and may develop into a cyst. Such cysts may cause symptoms such as hemospermia, epididymitis, perineal pain, and recurrent urinary tract infections.

Since the first case of a SV cyst with ipsilateral renal agenesis was described in 1914 by Zinner,[6] nearly 150 cases of SV cysts have been reported. Over one third of such patients have associated ipsilateral renal agenesis. SV cysts can be categorized as either congenital or acquired. Cysts of the SV usually become apparent during the second to third decade of life, related to the onset of sexual activity. Atresia of the ejaculatory ducts results in impaired drainage with build up of secretions in the gland, leading to distension and finally cyst formation. These cysts are usually unilateral without lateral predisposition. Acquired cysts, however, are more commonly bilateral, and are seen in patients with chronic prostatitis or previous transurethral resection of prostate.

SV cysts are usually smaller than 5 cm in diameter, and are often discovered incidentally as a palpable abdominal mass, or on rectal exam as a palpable fluctuant mass felt originating from the superior aspect of the prostate gland. These patients may present with a wide variety of symptoms such as dysuria, frequency, hematuria, hemospermia, epididymitis, prostatitits, infertility, and urinary tract infections.[7] Giant cysts are cysts of the SV that are larger than 12 cm in diameter, and often present with symptoms of bladder and/or colonic obstruction due to mass effect.[8] Tumors of the SV are extremely rare, with only two reported cases of papillary carcinoma in a SV cyst.[9]

Related urinary tract anomalies

Renal agenesis

Unilateral renal agenesis has been reported in 1 of 1000 (0.1%),[10] whereas clinically significant presentation may occur in only 1 in 1400 patients.[11] Cysts of the SV are associated with ipsilateral renal agenesis or dysplasia in two thirds of patients, presumably reflecting maldevelopment of the distal mesonephric duct and faulty ureteral budding (leading to renal agenesis or dysplasia), and atresia of the ejaculatory duct (leading to obstruction and cystic dilation of the seminal vesicle).[12]

With normal development, in the fourth embryonal week, the ureteric bud sprouts from the dorsal aspect of the distal mesonephric (Wolffian) duct and extends dorsocranially into the central part of the metanephric blastema. This interaction induces the differentiation of the metanephric blastema into the nephron. The ureteric bud differentiates into the ureter and the collecting system of the kidney. The WD differentiates into the hemitrigone, bladder neck, urethra proximal to the external sphincter, SV, VD, epididymis, paradidymis, and appendix epididymis. Complete failure of the WD results in absence of the ipsilateral kidney, ureter, hemitrigone, VD and SV. However, failure of the ureteral bud to develop off of the WD and meet the metanephric blastema will lead to ipsilateral renal agenesis or dysplasia with atresia of the ejaculatory duct, resulting in obstruction of the SV and formation of cysts.[13]

Ectopic ureter

Cystic dilation of the SV may also occur along with ectopic ureteral insertion into the SV. If the ureteral bud develops more cephalad on the WD, it will not achieve an independent opening into the urinary tract. Instead, the ureter would remain attached to the distal part of the WD which differentiates into the SV and ejaculatory duct.[14] Thus, the ipsilateral kidney is frequently maldeveloped, with the ureter ending in males in the ipsilateral VD or SV.[15] As the ureter inserts proximal to the urethral sphincter, males remain continent. (This is not the case, however, for females.) In males, this scenario results in an obstructed, and often atrophic kidney. Treatment of ectopic ureter include reimplantation when the ipsilateral kidney is functional, and nephrectomy or nephroureterectomy when the ipsilateral kidney is nonfunctional.

Vas deferens anomalies

Duplication of the VD is a rare condition with only a few reported cases in the literature. The embryologic etiology of this maldevelopment is not well understood, but it is theorized that VD duplication may be caused by duplication of the fetal mesonephric system. A second theory suggests that abnormalities of the VD and ureter are the result of maldevelopment of the proximal segment next to the WD that differentiates

into the VD and SV. Other rare anomalies of the VD include ectopia, agenesis, and diverticula.[16,17]

Case reports

To date, a total of 1230 RLRP procedures have been performed at our institution by three surgeons. Only two cases (0.16%) have been noted to have a WD anomaly. Herein we discuss the operative findings and difficulties encountered during dissection.

Case 1 – absence of left sided wolffian duct structures

A 69-year-old white male with known right solitary kidney was found to have an elevated serum total prostate specific antigen (PSA) level of 8.4 ng/ml during routine prostate cancer screening. His prostate was normal to palpation on digital rectal examination (DRE). Transrectal ultrasound (TRUS) guided biopsy of the prostate revealed bilateral Gleason score 3 + 3 = 6 adenocarcinoma. RRP was elected for definitive therapy.

Intraoperatively, after initial incision the posterior peritoneum, the SV and VD were identified only on the right side. No retroprostatic structures were identified on the left. Great care was taken to control the artery to the seminal vesicle during the full dissection of the right seminal vesicle, Figure 2. RRP was carried out in the standard fashion. After dorsal vein ligation, the bladder neck was transected. The right SV and VD were then brought through the open bladder neck and held on superior traction by the assistant, allowing for development of a plane between the anterior rectum and posterior prostate.

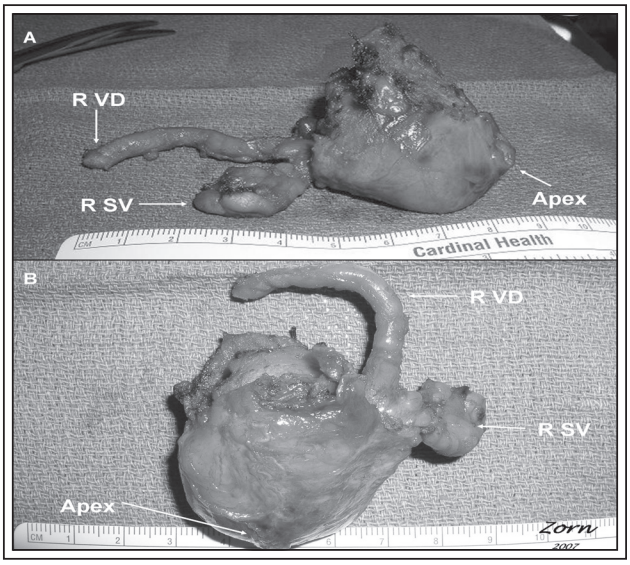

Figure 2. Anterior (A) and posterior (B) views of surgical specimen from RLRP with unilateral absence of left seminal vesicle and vas deferens (case 1). RVD = right vas deferens; RSV = right seminal vesicle; Apex = apex of prostate.

Absence of the VD and SV on the left side made the left lateral dissection more challenging. We routinely grasp the ipsilateral SV and VD to allow lateral dissection for neurovascular bundle preservation. In this particular case, traction was achieved by grasping the lateral anterior prostatic tissue as well as the back-bleed dorsal venous complex suture. This provided the necessary exposure for complete release and sparing of the neurovascular tissue. The remainder of the surgery proceeded without difficulty. Surgical pathology revealed pT2c disease, Gleason score 3 + 3 = 6 adenocarcinoma. All surgical margins and the right SV were free of disease. The patient's postoperative recovery was uneventful, and his PSA is undetectable at the time of writing 8 months following surgery.

Case 2 – ectopic left ureter with insertion into vas deferens

A 64-year-old white male with lower urinary tract symptoms, and past medical history of hypertension, diabetes mellitus, coronary artery disease, was found to have a PSA of 4.1 ng/ml. His prostate was normal to palpation on DRE. A TRUS guided biopsy of the prostate was performed revealing Gleason score 3 + 3 = 6 adenocarcinoma in the right base, mid and apical zones. RRP was elected for definitive therapy.

Intraoperatively, during initial posterior RRP dissection, the seminal vesicle and vas deferens were identified bilaterally. The left ureter, however, was found to be inserting into the left SV, Figure 3. At this point, we reviewed the preoperative abdominal imaging to assess the left upper urinary system. Preoperative MRI confirmed a normal right kidney and no functioning parenchyma on the left. With a congenitally atrophic left kidney, the decision was made to transect the left ureter. Transection of the left ureter allowed for complete dissection of the prostate and eventual release of both neurovascular bundles. The remainder of the surgery proceeded in the usual fashion. Surgical pathology revealed pT2c disease, Gleason score 3 + 3 = 6 adenocarcinoma. The surgical margins and seminal vesicles were free of disease. The patient's postoperative recovery was uneventful, and his PSA is undetectable at the time of writing 4 months after surgery.

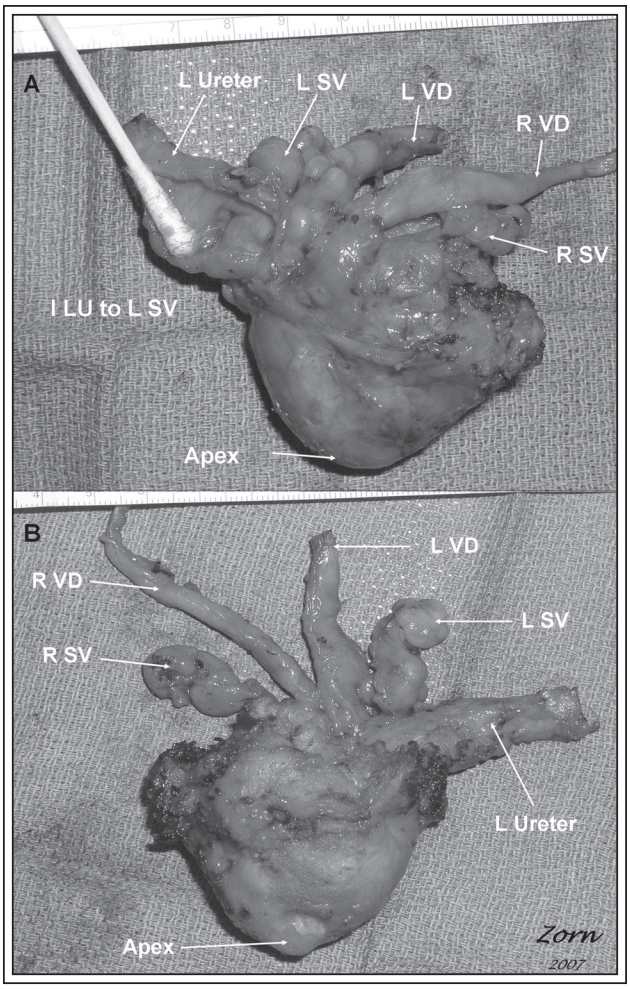

Figure 3. Posterior (A) and anterior (B) views of surgical specimen from RLRP with ectopic left ureter (case 2). RVD = right vas deferens; RSV = right seminal vesicle, LVD = left vas deferens; LSV = left seminal vesicle; L Ureter = left ureter; Apex = apex of prostate

Discussion

One third of congenital anomalies occur within the genitourinary tract. The vast majority of these, however, do not cause clinical problems, and thus remain undetected. Although uncommon, it is important for the urologist to be familiar with these anomalies, particularly as pelvic surgeries may be the first time they are diagnosed.

While the majority of SV cysts remain asymptomatic, those that become bothersome to patients usually require surgical intervention for alleviation of symptoms. Open surgery has been considered the definitive form of treatment. Although these procedures have produced excellent results, they can be associated with significant morbidity, such as rectal and bladder wall injury, ureteral injury, injury to the erectile neurovascular bundle, and pelvic urinoma.[18]

The CT guided drainage of SV cysts has also been previously described.[19] Although theoretically less invasive, the transrectal approach may be problematic. Hammad et al described a case of a SV cyst which formed an abscess after transrectal aspiration was performed.[20] This subsequently formed a fistula with the rectum which required more radical surgery for repair of this condition. In short, surgical excision of the SV cyst is the preferred definitive management because simple aspiration is often complicated by recurrence and infection.

With advances in laparoscopic equipment and experience it was natural to extend this approach to the SV cyst. To date 11 cases of laparoscopic SV cyst excisions have been reported.[21,22] The transperitoneal laparoscopic approach provides straightforward access and excellent visualization of the retrovesical SV. Its blood supply can be meticulously controlled and the seminal vesicles can be cleanly dissected free of the bladder, prostate and overlying peritoneum without entering the bladder or rectum. Among these reports, the average laparoscopic operative time has been 192 minutes, with an average hospital stay of 2.2 days. No injuries to adjacent structures and no major complications have been reported, and all patients have remained asymptomatic postoperatively.

Recently, Carmack et al have reported the novel use of the da Vinci Robotic Surgical System (Intuitive Surgical, Sunnyvale CA) to remove a seminal vesicle cyst in a previously dissected pelvis.[23] The authors felt that although the experienced laparoscopic surgeon may replicate the maneuvers that the da Vinci system can perform, the reproducibility and shortened learning curve make the robotic system attractive.

As RLRP continues to become a more common approach to managing prostate cancer, it is increasingly important that the urologist be comfortable with managing these uncommon and often surprising intraoperative anomalies. Although rare in our series (0.16%), review of the preoperative abdomino pelvic imaging is of utmost importance once a WD anomaly is incidentally discovered intraoperatively.

The portion of the RRP most affected by congenital anomalies of the WD is in the lateral dissection of the prostate, and release of the neurovascular bundles. At this point of the procedure, the presence of both left and right sided vas deferens and seminal vesicles is of greatest assistance to the surgeon. Grasping them and placing on appropriate traction by the first assistant, the prostate can then be manipulated with ease to facilitate the lateral dissection, specifically pedicle control and release of the neurovascular bundles.

WD anomalies such as unilateral or bilateral absence of either the SV or VD would make this portion of the RRP difficult. Though not impossible to perform, modifications in technique can facilitate the more technically challenging portions of the surgery. In a patient with unilateral absence of SV or VD, grasping of the contralateral SV and VD would afford some traction. In addition, the assistant surgeon can use a grasper or suction irrigator to push the lateral edge of the prostate medially, thus augmenting traction during dissection. These maneuvers would enhance traction in the area of dissection, and would allow for completion of RRP.

The existence of a SV cyst would unlikely make RRP more challenging. In the event that a very large cyst is encountered and hampers dissection, the cyst may be drained to reduce its size. This can be done either with use of electrocautery, scissors, or needle to aspirate the cystic fluid. After the cyst has been drained, RRP can be continued as usual.

The major challenge in managing an ectopic ureter encountered during RRP is assuring oneself that it is indeed the ureter. Assessment of preoperative imaging is important. In the event of a nonfunctioning kidney, transaction of the ureter is safe. If no preoperative imaging is available, administration of methylene blue or indigo carmine can be of use. If no dye is seen coming from the putative ureter, transaction is safe, whereas if dye is seen (i.e. the kidney associated with the putatitve ureter is functional), ureteral reimplantation will be required.

Conclusion

To date, no previous discussions of management of anomalies of Wolffian duct derivatives encountered during RLRP have been reported. This review comprehensively discusses the embryologic origin, diagnosis, and management of WD derived anomalies in patients undergoing RRP for localized prostate cancer. Although uncommon, it is important for the urologist to be familiar with the origin and surgical management of these anomalies, particularly when detected incidentally during surgery. □

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