



## Imaging characteristics of normal, common and uncommon diseases of seminal vesicles: A review article

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Ultrasound(US).

### ABSTRACT

Seminal vesicles (SVs) are pair of androgen-dependent accessory glandular structures of the male reproductive system. They play a critical role in male fertility. The main purpose of this review is to illustrate the imaging findings of the normal and the spectrum of seminal vesicles diseases, including congenital anomalies, inflammation, neoplastic , and nonneoplastic diseases.

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### INTRODUCTION

The seminal vesicles (SVs) are part of the male reproductive urogenital organs. The development of the SV is closely linked to ureter and kidney development. SV agenesis is the most common congenital SV pathology .<sup>1,2</sup> Infections, cysts, and neoplasms of the SVs are rare, in comparison with the adjacent prostate. <sup>3</sup> SV pathology is generally evaluated through magnetic resonance imaging(MRI). Though, multi-detector computed tomography(MDCT) and ultrasonography(US) are also useful evaluating diagnostic tools for SV. <sup>3</sup>

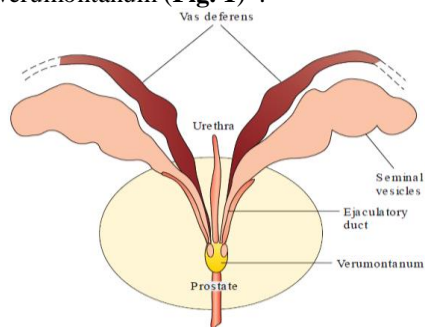
This article aims to review the imaging characteristics of common and uncommon, but significant lesions involving the SVs . Many of these

findings are incidental during imaging of the prostate or pelvis on CT. the SVs are rarely suspected to be the cause of a clinically presenting abnormality and the radiologist has a chance to be the first to propose such a diagnosis.

### ANATOMY

The SVs were described by the Italian anatomist Berengario a Carpi in 1521.<sup>4</sup> SVs are a pair of androgen-dependent accessory saccular and coiled glandular structures of the male reproductive system, which are extra-peritoneal in location, interposed between the bladder and the rectum , superior and posterior to the prostate. Each SV is about 3 to 4 mL volume and usually measures 5 to 7 cm in length and 1.5 cm in width and composed of a single coiled

tube with irregular diverticula which are connected by fibrous tissue.<sup>5</sup> The SVs are small until puberty; they reach the final dimensions about 11–13 years, because of the effect of male sexual hormones. The maximal diameters are identified about 50–60 years of age; this is also due to benign prostatic hypertrophy which compresses the ejaculatory ducts (ED), promoting the stasis of seminal liquid. The SVs usually shrink after the age of 70 years.<sup>6</sup> The main function of the SV is to secrete milky alkaline fluid which forms majority (50–80%) of the ejaculate volume; however, they are not a reservoir of the semen.<sup>1</sup> The secreted fluid contains fructose, proteins, and other enzymes that promote sperm function and provide nutrition and a variety of potent antibacterial factors to the male genital tract.<sup>7</sup> SV fluid is normally expelled in the last fractions of the ejaculate where only a few spermatozoa normally are expelled.<sup>8</sup> The ampulla of the vas deferens (VD) and excretory duct of the SV combine at the base of the prostate to form the EDs. The EDs extend further inferiorly to drain into the prostatic urethra through the verumontanum (**Fig. 1**)<sup>3</sup>.



**Figure 1. Diagram showing the relation of the seminal vesicles to the vas deferens.**



**Figure 2. Seminal vesicle normal MRI anatomy.** (A) Axial T2W image (obtained with body coil) showing normal appearance of SVs as fluid-filled tubular structures with thin walls (arrows). (B) coronal T2W image showing the paired SVs are suspended posterolateral and superior to the prostate gland. CZ, Central zone; PZ, peripheral zone; TZ, transitional zone of the prostate.

### Computed Tomography(CT)

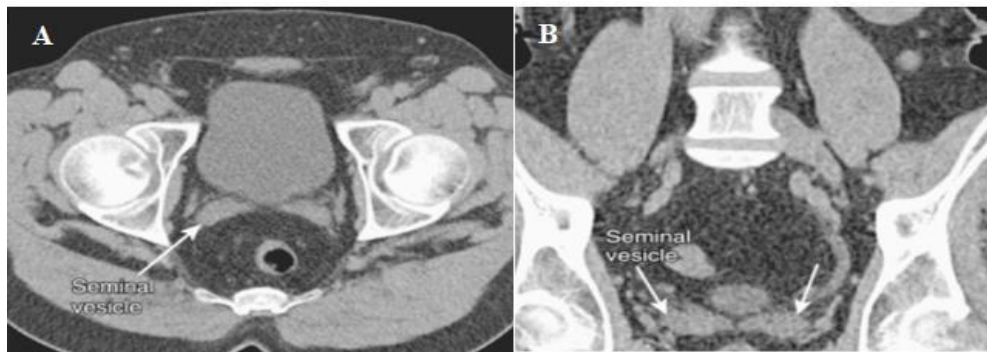
The SVs are of soft tissue attenuation (**Fig.3**)<sup>11</sup>. SVs abnormalities are probably more often encountered on CT imaging performed for nonspecific pelvic pain or for another unrelated

## IMAGING

### Magnetic Resonance Imaging(MRI)

MRI is the preferred and valuable modality in evaluating the SVs, whether for investigation of a primary SV abnormality or staging of solid neoplasms, owing to its multiplanar imaging skills, high soft tissue contrast resolution, and small field of view capabilities that allow for delicate examination of the SVs on MRI (**Fig.2**)<sup>9</sup>. Although asymmetry of size is common and a normal finding, the signal intensity on MRI should be symmetric. Post-contrast images demonstrate normal enhancement of the septa/wall.<sup>9</sup> Endorectal surface coil MRI is superior to TRUS (transrectal ultrasonography) in describing the anatomy of the prostate and distal seminal tract.<sup>10,11</sup> However, endorectal MR imaging is expensive and less available than TRUS and should be reserved for selected patients in whom results of TRUS are not conclusive. Coronal and sagittal imaging of the SVs is crucial because malignancies often arise from the glandular base or ED.<sup>12</sup> Normal SV MRI findings: the SVs appear as elongated fluid-filled structures with fine internal septations. T2-weighted (W) sequences demonstrate homogeneously hyperintense signal in the normal-appearing SVs. The T1 signal of the SVs is usually isointense to slightly hyperintense relative to skeletal muscle. A small proportion of patients demonstrate heterogeneous or increased T1 signal in the SVs, which is almost uniformly a benign finding and perhaps a function of aging and some have postulated that this increase T1 is related to amyloid deposition.<sup>13</sup>

indication. Cysts and small masses that do not deform the SV are not well seen. Large masses or inflammatory change associated with infection or abscess can be evaluated. Calcification is clearly seen.<sup>1</sup>

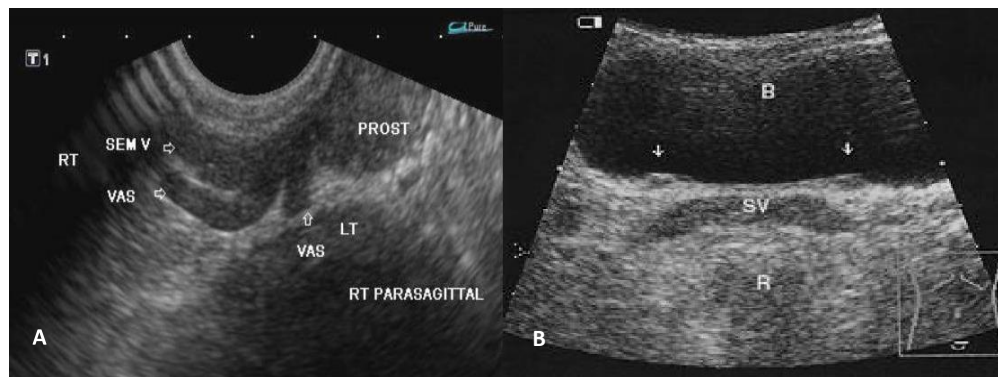


**Figure 3. Seminal vesicle normal CT anatomy.**(A and B), axial & coronal reformat CT images showing the SVs of soft tissue attenuation and form a “bow-tie” appearance posterior to the prostate.

### Ultrasound

Transrectal US (TRUS) though it is minimally invasive ,nowadays is considered superior to transabdominal suprapubic US in SV evaluation( **Fig.4** ).<sup>1,14,15</sup> . TRUS is rapid, inexpensive ,generally well tolerated ,and less time consuming than other imaging techniques such as

CT and MRI . The endorectal 5-7 MHz biplanar transducer used can detect alterations in size, echotexture , vascularization of SV and can be used to guide SV biopsy ,vesiculography or aspiration, therefore, it is used to investigate several pathological conditions.<sup>15</sup>



**Figure 4.Seminal vesicle normal ultrasonic anatomy.**(A) TRUS,RT parasagittal plane and (B) Suprapubic US axial plane showing SVs between the bladder floor (B) and rectum (R). ureteral ridges (arrows).

At TRUS , SVs have a typical ‘bow-tie’ appearance in a transverse scan and an oval configuration. Their echo-texture is usually homogeneous and slightly less echogenic than the prostate.<sup>16</sup> Very recently the European Academy of Andrology (EAA) , due to the efforts of different radiological, urological, and andrological societies ,published the results of an international multicenter study entitled “Standardization of the MGT (male genital tract ,including the SV)color-Doppler ultrasound (CDUS) parameters in healthy, fertile men” (shortened to “EAA US study”) .The EAA US study provided normal values, cut-off and classifications at CDUS (**Tab.1**).<sup>17-19</sup>

SVs are thought to be normal when > 25 mm in length, hypoplastic when > 16 mm but < 25 mm and atrophic when < 16 mm. Seminal vesicular dilatation is one of the US features of ED obstruction (EDO).<sup>12</sup>

### Minimally Invasive TRUS-guided aspiration of the SVs and seminal vesiculography (TRUS-GSV)

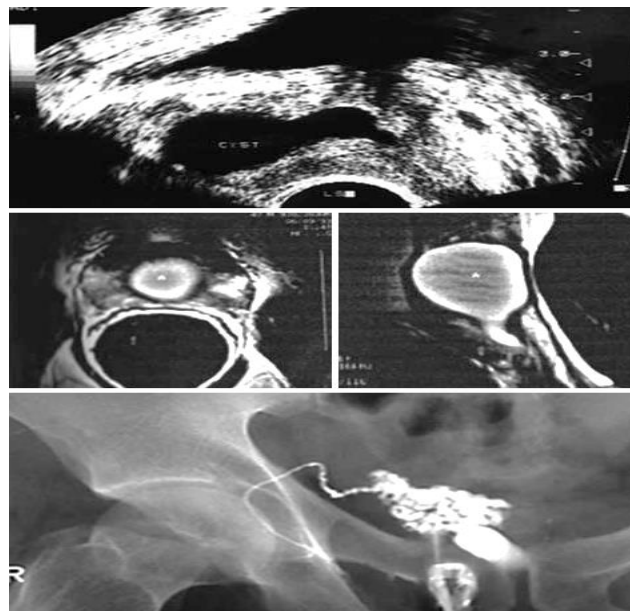
In the SV aspiration procedure, with real-time TRUS guidance, each SV was punctured transrectally using a 20-gauge, 25 cm-long echo tip Chiba needle within 2 hours after ejaculation. A scant amount up to 2 mL of seminal fluid aspirated from each SV and immediately after aspiration placed on a slide for analysis under high-power field microscopic for the

**Table 1: EAA US study derived and previously published normal values, cut-off and classifications of the main SV-CDUS parameters/characteristics**

Seminal vesicles (SV)	Previously proposed normal values, cut-off and classifications at CDUS	EAA US study normal values, cut-off and classifications at CDUS
<b>Diameters</b>	Dilation: SV apd > 15 mm (suggestive of partial or complete ejaculatory duct obstruction )  Hypoplasia: SV apd < 5 or < 7 mm and/or SV longitudinal d < 25 mm	<b>Before ejaculation</b> · apd: reference range 8–18 mm · ld: reference range 40–56 mm <b>After ejaculation</b> · apd: reference range 6–16 mm (< 6 mm suggest hypoplasia; > 16 mm suggest dilation) · ld: reference range 37–53 mm
<b>Volume</b>	No cut-off for dilation or hypoplasia	Before ejaculation: reference range 1.4–9 mL After ejaculation: reference range 0.6–6 mL
<b>SVEF</b>	SVEF < 21.6% (identifies subjects with reduced seminal volume (< 1.5 mL) and pH (< 7.2), expressing a useful indicator of EDs sub-obstruction )	Normal SVEF > 20.0% (when < 20.0% suggestive of partial and complete obstruction)
<b>Abbreviations: SVEF, seminal vesicles ejection fraction; ld, longitudinal diameter; apd, anterior–posterior diameter.</b>		

presence or absence of motile sperm. Greater than 3 sperms was considered a positive result for EDO, confirms the presence of intact spermatogenesis, and rules out more proximal obstruction.<sup>20,21</sup> If sperm are absent or fewer than 3 sperm in high power field, TRUS-GSV can be performed by filling the Rt. or left SV with 5-20 cc of dilute, nonionic water-soluble contrast under the fluoroscopic control. TRUS-GSV is a technique for imaging of the distal male reproductive tract (VD,SV, and EDs) and to evaluate

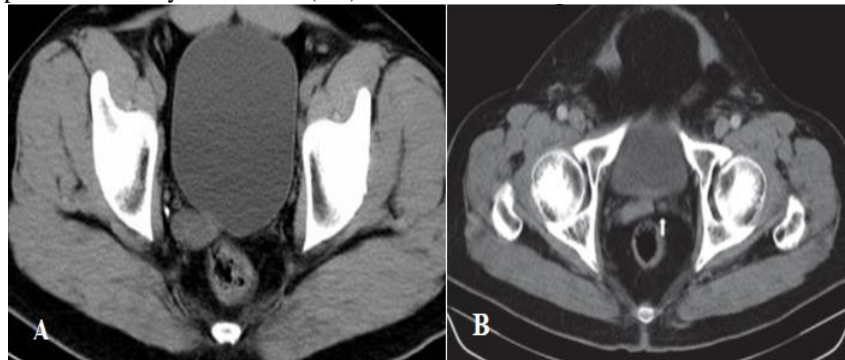
male-factor infertility (Fig.5).<sup>22</sup> The dye should not be allowed to reach the epididymal tubules otherwise chemical Epididymitis may occur . The patient there after is asked to micturate and after 10 minutes (allowing wash out of contrast medium) , then the contralateral SV is examined . Flow of the contrast in the bladder or subsequent absence of the contrast in the SV on later plain X-ray confirms the absence of the EDO.



**Figure 5. A 29-year-old man with primary obstructive infertility.** TRUS (upper image) and endorectal MRI (middle image) show a well-defined midline urogenital cyst with intra- and extraprostatic components. TRUS-contrast study (lower image) shows the SV is communicating with the urogenital cyst with non opacification of the urethra or urinary bladder denoting complete distal obstruction (N.B. the left vas and SV were absent). Trans-urethral incision of the cyst lead to improvement of sperm count

### SEMINAL VESICLES AGENESIS, HYPOPLASIA AND SV CYST

**Agnesis of the SV** is one of the most frequently seen congenital disorder (Fig.6). It may be unilateral or bilateral, and it is associated with agnesis/ectopia of the vas deference(VD) and ipsilateral kidney, as the development of these structures is strictly related.<sup>12</sup> SVs are thought to be absent when no tissue is identified. **Unilateral SV agnesis** is quite rare and its incidence is less than 1% and it is the result of an embryological insult to the mesonephric duct happening before 7<sup>th</sup> week of gestation (before the development of ureteral bud). It is often associated with ipsilateral renal agnesis (79%), and this is likely to be associated with VD (ipsilateral or/and bilateral) ectopy or agnesis.<sup>1,23</sup> If the insult occurs after 7<sup>th</sup> week of gestation, the ipsilateral kidney will be normal.<sup>24</sup> **Bilateral SV agnesis** and VD anomalies without associated urinary abnormalities are often seen in patients with cystic fibrosis(CF).<sup>1</sup>



**Figure 6. Seminal vesicle agnesis and hypoplasia.** (A) and (B) axial CT images showed LT-sided SV agnesis in A & LT-sided SV hypoplasia in B(arrow).

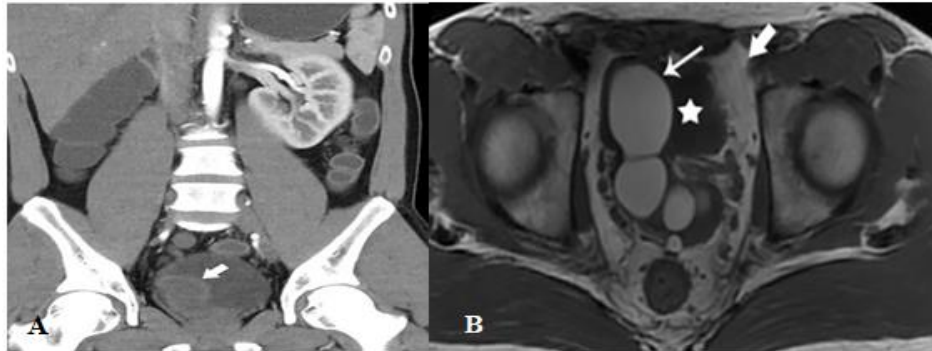
**Seminal vesicle cysts (Figs 7,8)** are rare and may be congenital or acquired and usually congenital in origin. They are located posterolateral in relation to the bladder. An occasional one is more midline in location. Most cysts are unilateral and more frequent on the right side. Usually found incidentally in 2nd or 3rd decade. SV cysts usually less than 5 cm. A giant (generally larger than 10 cm) cyst is rare and commonly associated with midline prostatic cyst(Fig.8).<sup>27,28</sup>

**Congenital SV cyst** is a rare condition that may be isolated or more frequently associated with other genitourinary (GU) anomalies, the most common GU anomalies being ipsilateral renal agnesis or hypogenesis in two-thirds of cases.<sup>12, 29</sup> **Zinner syndrome** is a rare congenital developmental anomaly of the genitourinary tract characterized by the triad of unilateral renal agnesis, ipsilateral SV cyst and ipsilateral EDO(Fig.7).<sup>27,30,31</sup> It is suggested

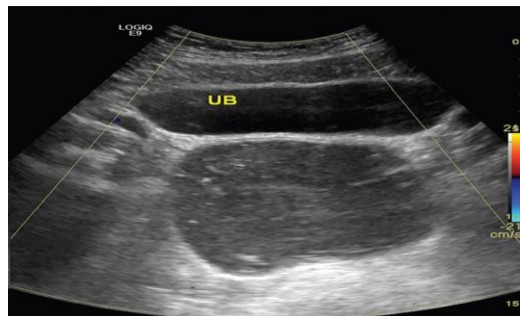
Bilateral SV agnesis is associated with mutations in the CF transmembrane conductance regulator gene in about 64–73% of cases and SV anomalies are observed in 50% of children and 90% of adults with CF, with the latter showing bilateral agnesis in half of cases.<sup>25</sup> CBAVD(congenital bilateral agnesis of the VD) in half of the cases and patients usually have normal kidneys.<sup>1</sup> The mechanism of agnesis in patients with primary genital form of CF is assumed to be luminal blockage of the SV and VD precursors from abnormal secretions after 7<sup>th</sup> week of gestation.<sup>26</sup>

**Congenital SV hypoplasia** refers to congenital underdevelopment of the SVs and may be isolated or associated with other congenital genitourinary anomalies such as absence of the VD.<sup>1</sup> Hypoplasia was defined as a maximum diameter smaller than 50% of normal or < 5 mm (Tab. 1) (Fig. 6).<sup>27</sup>

that EDO caused by maldevelopment of the distal portion of the mesonephric duct is responsible.<sup>32</sup> Ectopic ureteral insertion into the SV, ED, VD, or prostatic urethra or VD agnesis have been reported.<sup>1</sup> **Bilateral SV cysts** have been reported to occur in up to 60% of patients with autosomal dominant polycystic kidney disease (ADPCK).<sup>33</sup> **Acquired cysts** are usually unilateral and can be secondary to any inflammation-related EDO(seminal vesiculitis, prostatitis, or surgery).<sup>32</sup> Endorectal US obviously identifies the SV cysts as retrovesically located cysts. MRI is also very useful in detecting SV cysts. Generally, the simple cyst appears hypo on T1- and hyperintense on T2-weighted images, but the presence of proteinaceous (depending on its concentration), hemorrhage (depends on the age of the bleeding) and purulent material( due to infection) may increase intensity on T1-weighted images(Fig.7).<sup>34</sup>



**Figure 7. Congenital seminal vesicle cyst.** (A), Contrast-enhanced coronal reformat CT scan shows right renal aplasia is present. (B), cystic lesion attached to the right SV and present as hyperintense in T1WI sequence (thin arrow) compared with bladder (star) and fat (thick arrow) and a normal left SV, consistent with the urogenital anomalies associated with SV cyst (Zinner syndrome).



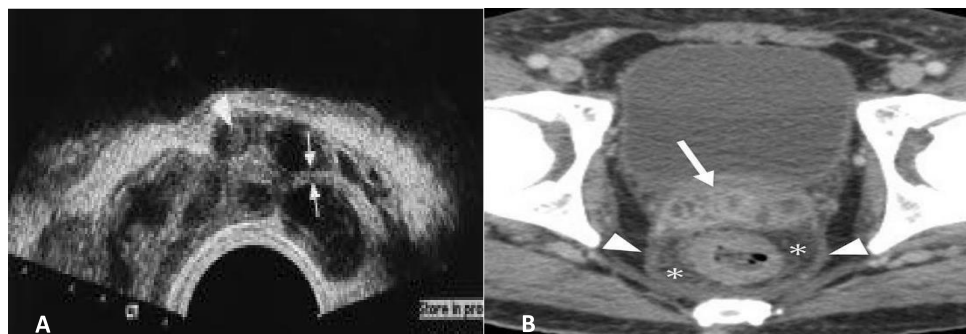
**Figure 8. Giant seminal vesicle cyst.** Ultrasound abdomen demonstrating a large midline heterogeneous pelvic cyst measuring 11.5 cm × 9 cm × 9.5 cm, with a smooth wall, anterior to the urinary bladder causing mass effect

## INFECTIOUS AND INFLAMMATORY LESIONS

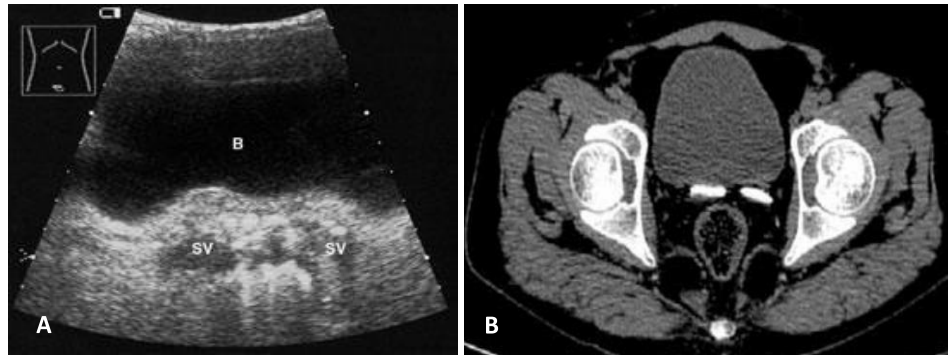
**Seminal vesiculitis.** Usually caused by bacterial infection and its a rare complication of prostatitis and/or epididymitis with a possibility of SV abscess formation. Additionally, diabetes, instrumentation, and surgery can be associated with SV abscess formation. Seminal vesiculitis presents as diffuse seminal vesicle wall thickening with diffuse enhancement of the wall and septa on CT or MRI (Figs.9,10).<sup>35</sup> Some ultrasound features have

been proposed as suggestive of vesiculitis, in specific, ‘enlargement and asymmetry’ and ‘wall thickening and calcification’ have been proposed as signs of vesiculitis.<sup>1</sup> In addition, ‘roundish anechoic areas’ within the SV described as ‘areas of endocapsulation’ also have been suggested as a sign of vesiculitis.<sup>36</sup>

Like abscesses in other locations, a **SV abscess** will classically present as a thick walled cyst on CT or MRI. Calcification may occur with end-stage disease, especially in cases of tuberculosis.



**Figure 9. Seminal vesiculitis.** (A) TRUS (coronal plane) power Doppler in a 32-year-old man, shows acute non specific deferentitis (arrow) of terminal part of VD and acute right seminal vesiculitis (ASV) with hypervascularized right SV on power Doppler (top and to the right). (B) Contrast-enhanced CT in another patient, shows diffuse wall and septal thickening of the seminal vesicle (arrow), thickening of the mesorectum (arrowhead), and increased haziness of the perirectal fat plane (asterisks). These indicate seminal vesiculitis).



**Figure 10 . Calcifications of the seminal vesicles.** (A) Calcifications of the SVs (SV) seen as focal echogenic areas. B = bladder. (B) bilateral SVs Calcifications in a Diabetic patient.

**SV stones** are rare entity. They're supposed to form due to SVs inflammation or anatomical abnormalities of the SVs predisposing to stasis and urinary reflux into the EDs and should not be confused with **SV wall calcification** that can be found in diabetes (highly associated with SV symmetric intramural pattern of calcifications) ,

schistosomiasis, and Less often due to tuberculosis, chronic renal failure and advanced age. SV calculi have the same typical imaging characteristics of urinary stones.<sup>37</sup> CT accurately demonstrates the location and TRUS may demonstrate the location and degree of SV calcification or calculi (Fig. 10)<sup>37</sup>.

**Table (2): Tumors of the Seminal Vesicle**

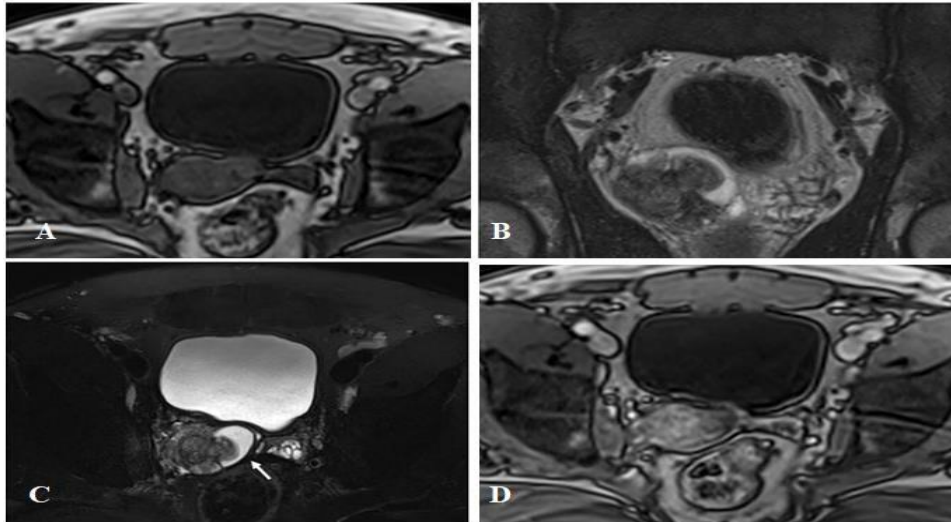
<b>Benign</b>	Cystadenoma, papillary adenoma, leiomyoma, teratoma, schwannoma, epithelial stromal tumor
<b>Malignant</b>	
<b>-Common</b>	Secondary neoplasm including bladder, prostate, or rectal cancer and lymphoma
<b>-Uncommon</b>	Adenocarcinoma, leiomyosarcoma, rhabdomyosarcoma, angiosarcoma, müllerian adenosarcoma-like tumor, carcinoid, cystosarcoma phyllodes, seminoma
<b>Nonneoplastic</b>	Amyloidosis, hydatid cyst

### TUMORS OF THE SEMINAL VESICLE

Primary neoplasms of the SV, which arise from the epithelial or mesenchymal elements, are very rare. In most cases, SV tumors are benign. When a SV tumor is detected on imaging, it is usually an incidental finding and even large SV tumors are most often asymptomatic. A variety of tumors have been reported (Tab. 2).<sup>38</sup> No imaging modality can distinguish between benign and malignant solid SV masses. On MRI, a SV tumor appears as a mass of

heterogeneous mass of T1-intermediate and as T2-weighted high signal intensity. On Ultrasound, a SV solid tumor may appear isoechoic to the prostate but relatively hyperechoic to the normal SV texture.

cystadenomas (epithelial stromal tumor) of the SV is the most common **benign primary SV tumor**. Tumors are usually present as a large usually multilocular well-demarcated cystic appearance in an asymptomatic elderly male patient or a patient complaining of hematuria (Fig. 11).<sup>39</sup>



**Figure 11. Seminal vesicle cystadenoma** .MRI shows a RT-SV mass with an inhomogeneous low signal intensity on (A) axial T1w, (B) coronal T2w and (C) axial T2-STIR images with associated peripheral cystic components (arrow in C), and (D) Axial post-contrast image shows inhomogeneous enhancement of the lesion.

leiomyoma(smooth muscle tumor) is another benign primary SV tumor. MRI demonstrates low T2 and iso to low T1 signal of the lesion with post-contrast enhancement, and CT may demonstrate coarse, dense calcifications.<sup>4</sup> A mass in the SV with an infiltrative growth pattern is suggestive of a malignancy. adenocarcinoma (glandular epithelium tumor) is extremely rare tumor and it is the most common **primary malignant SV tumors** and can be observed at a wide age range (**Tab.2**) (**Fig.12**)<sup>41</sup>. Due to rarity of the disease, unfortunately, there are no reliable imaging features that distinguish a primary from a secondary form of malignancy.<sup>42</sup> Several types of sarcomas and seminomas originating in the SV have been reported in the literature.

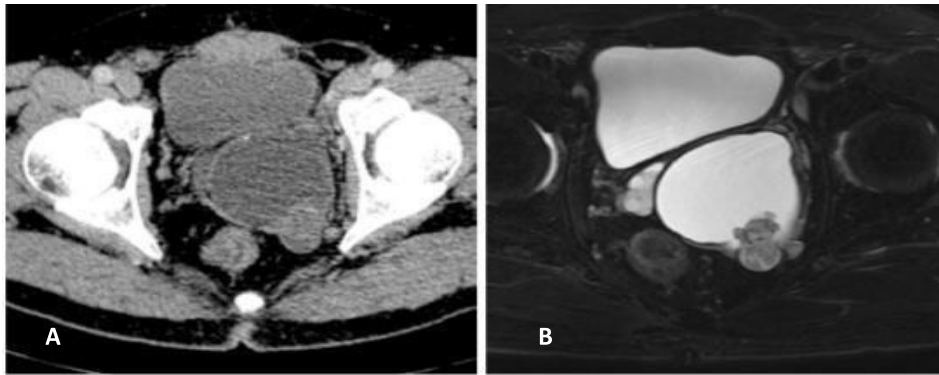
The diagnosis of primary SV carcinoma should be made only when all the established strict criteria be fulfilled.<sup>42</sup> These include:

The tumor is completely or essentially localized primarily in the SVs; no evidence of concurrent primary prostate, bladder, or colonic malignancy(Specifically the prostate should be carefully evaluated for possible carcinoma); presence of mucus production in anaplastic variant (to distinguish from anaplastic prostate carcinoma) ; prostate specific antigen (PSA) and prostatic acid

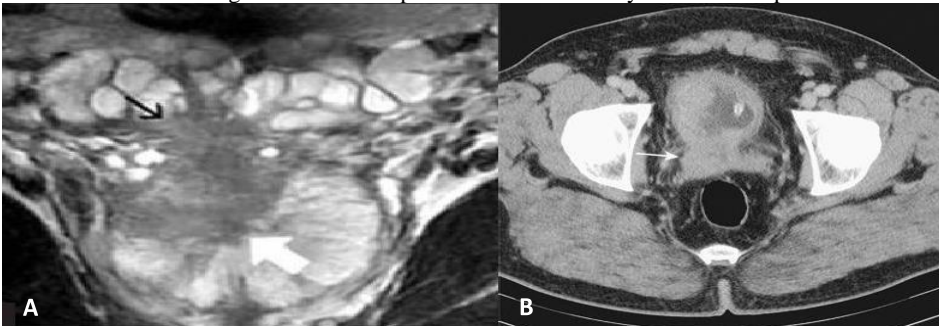
phosphatase (PAP), and carcinoembryonic antigen (CEA, which can be interpreted as absence of invasion of a colon carcinoma) should be negative. Adenocarcinoma of the SV is also usually positive for CA-125.<sup>41</sup>

Malignancy within the SV is most commonly secondary to carcinoma of the prostate, rectum, or bladder. Route of SV invasion in prostate cancer is indefinite and the probabilities include retrograde tumor extension through the ED, direct spread across the prostatic base, spread from periprostatic nerve involvement, or tumor deposits. Tumor extension from the bladder or rectum can be identified as a large contiguous soft tissue mass(**Fig.13**).<sup>1,43</sup> MRI endorectal coil imaging have significantly improved the sensitivity of determining SV invasion in prostate carcinoma . The most sensitive and specific features on MRI to determine SV invasion are low signal intensity within the SV and lack of preservation of the normal SV architecture, respectively (**Fig.13**).<sup>3,44</sup>





**Figure 12. Primary seminal vesicle adenocarcinoma imaging findings.** A: Axial dynamic contrast-enhanced CT revealed a cystic-solid mass. B: Axial T2-weighted MRI of the pelvis demonstrated a cystic-solid component mass.



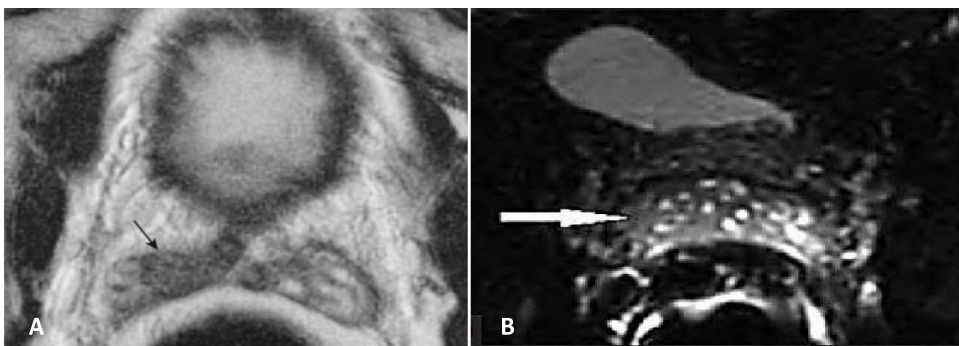
**Figure 13. Secondary SV malignancy.** Coronal T2-W MRI (A) and axial contrast-enhanced CT (B) images, show in (A) a right base prostate adenocarcinoma (white arrow) invading through the capsule the right SV (black arrow), the SV is of low T2-W SI due to infiltration by tumor, and in (B) a bladder cancer invading the right SV (arrow). The SV is enlarged by tumor. a prostatic mass at the right base (white arrow).

### Nonneoplastic

Amyloidosis, rarely infiltrates the SVs. It may be localized, part of systemic amyloid, or a mixture of both.<sup>45</sup> Local senile amyloid deposits are a common finding at autopsy and occur subepithelially, but the amyloid is located in the blood vessels walls or within muscle in the systemic amyloidosis. MRI

findings of the SV show wall thickening with luminal narrowing and low T2 signal, and usually lack of normal SV wall T1 post-contrast enhancement, and lack of restricted diffusion within the SVs (Fig.14).<sup>3,46</sup>

Primary hydatid cyst of the seminal vesicle is extremely rare.<sup>47</sup>



**Figure 14. Seminal vesicle amyloid deposition.** (A) Axial T2-W MRI image, shows areas of low T2-W SI within the right SV (arrow) was confirmed to be amyloid on pathologic evaluation. (B) axial diffusion-weighted image of the SV, shows lack of diffusion restriction, diffusely thickened SV wall and luminal narrowing.

### CONCLUSION

The association between SV pathology and other GU system diseases requires complete GU system evaluation that includes the SVs. SV anomalies can

be accurately diagnosed with all imaging modalities and the practicing radiologist should be familiar with the evaluation of these pathologic processes and the common radiological imaging findings,

## References:

1. Kim B, Kawashima A, Ryu JA, Takahashi N, Hartman RP, King BF., Jr Imaging of the seminal vesicle and vas deferens. *Radiographics*. 2009;29:1105–1121. [PubMed] [Google Scholar]
2. Batur A, Karakose S, Karalezli G. Congenital seminal vesicle cyst accompanying ipsilateral renal and ureteral agenesis. *Urol J*. 2014;11:1203–1203. [PubMed] [Google Scholar]
3. Reddy MN, Verma S. Lesions of the Seminal Vesicles and their MRI Characteristics. *J Clin Imaging Sci*. 2014;31(4):61–61. [PMC free article] [PubMed] [Google Scholar]
4. Standring S. *Gray's Anatomy: The Anatomical Basis of Clinical Practice*. London, UK Churchill Livingstone; 2008: 1261-77.
5. Parviz K, Kavoussi. *Surgical, Radiographic, and Endoscopic Anatomy of the Male Reproductive System* In A. W. Partin, C. A. Peters, L. R. Kavoussi, R. R. Dmochowski, & A.J. Wein (Eds.), *Campbell-Walsh-Wein Urology*; 2020 (12th ed.): 6333-6405.
6. Yuruk E, Pastuszek AW, Suggs JM, 3rd, Colakerol A, Serefoglu EC. The association between seminal vesicle size and duration of abstinence from ejaculation. *Andrologia*. 2017;49: e12707.
7. Ernst LM, Ruchelli ED, Huff DS. *Color Atlas of Fetal and Neonatal Histology*. New York, Springer; 2011:157-61.
8. Rowland D, McMahon CG, Abdo C, Chen J, Jannini E, Waldinger MD, Ahn TY. Disorders of orgasm and ejaculation in men, *J Sex Med*, 2010; 7:1668-1686.
9. Donkol RH. Imaging in male-factor obstructive infertility. *World J Radiol* 2010;2: 172–9.
10. Mohsen, Y., Al-Kutoubi, A. Imaging of seminal vesicles: a pictorial review. *Abdom Radiol* 2023;48: 3469–3487 . <https://doi.org/10.1007/s00261-023-04019-4>.
11. Carmel C., Joseph R. G.. " urogenital imaging: Seminal Vesicles lesions ." In *Abdominal Imaging: Expert Radiology Series*, edited by Dushyant V., Anthony E. 2nd edition Philadelphia, Elsevier, 2017.
12. Heshmat S, Lo KC. Evaluation and treatment of ejaculatory duct obstruction in infertile men. *Can J Urol* 2006; 13 Suppl 1:18-21
13. Cassidy FH, et al: MR imaging of scrotal tumors and pseudotumors. *Radiographics* 2010; 30(3):665–683
14. Lotti F, Corona G, Colpi GM, et al. Seminal vesicles ultrasound features in a cohort of infertility patients. *Hum Reprod*. 2012 Apr;27(4):974-82.
15. Lotti F, Maggi M. Ultrasound of the male genital tract in relation to male reproductive health. *Hum Reprod Update*. 2015 Jan-Feb;21(1):56-83.
16. Seaman EK, Kim ED, Lipshultz LI. Evaluation of the infertile couple. *JAMA*. 1996 Jan 17;275(3):185-6. PMID: 8604166.
17. Lotti F, Frizza F, Balercia G, et al. The European Academy of Andrology (EAA) ultrasound study on healthy, fertile men: An overview on male genital tract ultrasound reference ranges. *Andrology*. 2022 Oct;10 Suppl 2(Suppl 2):118-132. Doi 10. 1111 / andr.13260. PMID: 35930758; PMCID: PMC9828651.
18. Lotti F, Frizza F, Balercia G, et al. The European Academy of Andrology (EAA) ultrasound study on healthy, fertile men: clinical, seminal and biochemical characteristics. *Andrology*. 2020;8(5):1005-1020.
19. Lotti F, Frizza F, Balercia G, et al. The European Academy of Andrology (EAA) ultrasound study on healthy, fertile men: scrotal ultrasound reference ranges and associations with clinical, seminal, and biochemical characteristics. *Andrology*. 2021;9(2):559-576.
20. Engin G., Celtik M., Sanli O., et al. Comparison of transrectal ultrasonography and transrectal ultrasonography-guided seminal vesicle aspiration in the diagnosis of the ejaculatory duct obstruction. *Fertility and Sterility*, Volume 92, Issue 3, 2009, Pages 964-970)
21. Orhan I, Onur R, Cayan S, et al. Seminal vesicle sperm aspiration in the diagnosis of ejaculatory duct obstruction. *BJU Int* 1999; 84:1050±1053
22. Donkol RH. Imaging in male-factor obstructive infertility. *World J Radiol* 2010; 2(5): 172-179
23. Resorlu M, Adam G, Uysal F, Bas S, Karatag O, Sancak EB. Seminal vesicle hypoplasia with contralateral renal agenesis. *Urology*. 2014;84:e7.
24. Campbell MF, Walsh PC, Retik AB. *Campbell's Urology*. Philadelphia: Saunders; 2002. p. 3869-85.
25. Rathaus V, Werner M, Freud E, Mei-Zahav M, Mussaffi H, Blau H. Sonographic findings of the genital tract in boys with cystic fibrosis. *Pediatr Radiol* 2006;36:162–166).
26. Arora SS, Breiman RS, Webb EM, Westphalen AC, Yeh BM, Coakley FV. CT and MRI of congenital anomalies of the seminal vesicles. *AJR Am J Roentgenol* 2007;189:130-5.
27. Farooqui A, AlDhahir L, Mahfooz AB. Massive seminal vesicle cyst with ipsilateral renal agenesis – Zinner syndrome in a Saudi patient. *Urol Ann* 2018;10:333-5.

- 28-Zhang W et al: Relationship of seminal megavesicles, prostate median cysts, and genotype in autosomal dominant polycystic kidney disease. *J Magn Reson Imaging*. 2019; 49(3):894-903
- 29- King BF, Hattery RR, Lieber MM, Williamson B Jr, Hartman GW, Berquist TH. Seminal vesicle imaging. *Radiographics* 1989;9:653–676.
30. Jiang X-S, Wang H-J, Lin J-H, et al. Zinner's syndrome: clinical features and imaging diagnosis. *Asian J Androl* 2018;20:316–7.
31. Florim, S., Oliveira, V., Rocha, D.. Zinner syndrome presenting with intermittent scrotal pain in a young man. *Radiology case reports*, 2018; 13(6), 1224–1227
- 32- Patel B, Gujral S, Jefferson K, Evans S, Persad R. Seminal vesicle cysts and associated anomalies. *BJU Int* 2002;90:265–271.
- 33-Danaci M, Akpolat T, Bas, temir M, Sarikaya S, Akan H, Selc, uk MB, Cengiz K. The prevalence of seminal vesicle cysts in autosomal dominant polycystic kidney disease. *Nephrol Dial Transplant* 1998;13:2825–2828.
34. Livingston L, Larsen CR. Seminal vesicle cyst with ipsilateral renal agenesis. *AJR Am J Roentgenol* 2000;175:177–80.
- 35- Shibuki, S., Saida, T., Hoshiai, S. et al. Imaging findings in inflammatory disease of the genital organs. *Jpn J Radiol* 2024. <https://doi.org/10.1007/s11604-023-01518-8>.
- 36-Vicari E. Seminal leukocyte concentration and related specific reactive oxygen species production in patients with male accessory gland infections. *Hum Reprod* 1999;14:2025–2030.
37. Yadav R. et al. Incidentally detected bilaterally symmetrical seminal and vas calcification in young infertile male: a case report, literature review and algorithm for diagnosis. *Can Urol Assoc J* 2012;6(5):E206-E208.
38. John N. Eble, International Agency for Research on Cancer. *Pathology and Genetics of Tumours of the Urinary System and Male Genital Organs*. (2004)
39. Franca RA, Crocetto F, Pandolfo SD, et al. Cystadenoma of the seminal vesicle – Potential diagnostic pitfall: A case report and literature review. *Journal of Clinical Urology*. 2023;16(6):631-642..
40. Shiotani T, Kawai N, Sato M, Minamiguchi H, Takeuchi T, Tanihata H, et al. Leiomyoma of the seminal vesicle. *Jpn J Radiol* 2009;27:218-20.
41. Yao Y, Liu S, He YL, Luo L, Zhang GM. Primary seminal vesicle adenocarcinoma with a history of seminal vesicle cyst: A case report and review of literature. *World J Clin Cases* 2023; 11(14): 3261-3266.
42. Katafigiotis I, Sfoungaristos S, Duvdevani M et al. Primary Adenocarcinoma of the Seminal Vesicles. A Review of the Literature. *Arch Ital Urol Androl*. 2016;88(1):47. doi:10.4081/aiua.2016.1.47 - Pubmed
43. Ohori M, Scardino PT, Lapin SL, et al. The mechanisms and prognostic significance of seminal vesicle involvement by prostate cancer. *Am J Surg Pathol*. 1993;17:1252–1261. [PubMed] [Google Scholar]
45. Maroun L, Jakobsen H, Kromann-Andersen B, Horn T. Amyloidosis of the seminal vesicle: A case report and review of the literature. *Scand J Urol Nephrol*. 2003;37:519–21. [PubMed] [Google Scholar]
46. Kaji Y, Sugimura K, Nagaoka S, Ishida T. Amyloid deposition in seminal vesicles mimicking tumor invasion from bladder cancer: MR findings. *J Comput Assist Tomogr*. 1992;16:989–91. [PubMed] [Google Scholar]
47. Mushtaque M, Mir MF, Nazir P, et al. Primary isolated echinococcal cyst of the seminal vesicle: a rare case. *Urol Ann*. 2012;4(2) :122–125