Magnetic Resonance of the seminal pathway (MRSP): our experience applying a specific protocol

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Learning objectives

To analyze the reasons for medical appointments and findings in MRSP, describing the advantages of employing a specific protocol.
Background

All of the structures that compose the seminal pathway can be studied safely in a single step without the use of contrast in a non invasive procedure. Using a dedicated protocol provides great anatomical detail, characterization of the content of the seminal vesicles and detection of congenital anomalies.
Findings and procedure details

Anatomy on the Seminal Pathway (SP):

It consists of the seminal vesicles (SV), deferens ampulla, vas deferens and ejaculatory ducts.

The SV have an anteroposterior diameter of 1.5 cm ± 0.4 and a length of 3 cm ± 0.8. They produce and store seminal fluid, with an average volume of 10 to 16 ml; This represents 70% of the seminal fluid and is rich in semenogelin, among other proteins. [1, 2, 3]

The vas deferens has a diameter of 0.4 cm ± 0.1, rises from the posterior edge of the testicle, passes through the inguinal canal and runs parallel to the bladder wall, ending in a dilatation (ampulla); this last fusion with the seminal vesicles forms the ejaculatory duct. This duct has a length of 4.5 cm and anteroposterior diameter of 6 mm ± 2, enters the prostate, continues towards the central area of the same and ends in the posterior urethra, outside the verumontanum. [1, 3, 4]

The peristalsis and the sphincter function of the ejaculatory system is performed due to the Denonvilliers' fascia. [4]

Transrectal ultrasound (TRUS) has been used as a less invasive study to investigate seminal vesicles and ejaculatory ducts.
This method only allows the evaluation of SV and proximal ducts and does not provide information of the contents of the seminal vesicles.
Currently, the development of ultrafast MR sequences allows the study of vas deferens, ejaculatory ducts and seminal vesicles in a single examination without the use of an endorectal bobbin. [5] (Fig. 1 on page 9 and Fig. 2 on page 9)

Protocol:

All patients were studied with 1.5T equipment, applying a Magnetic Resonance (MR) specific protocol, with a duration of 20 minutes (Table 1 on page 10). The specific protocol include an oblique coronal plane along the major axis of the seminal vesicles and volumetric 3D of the minor pelvis that allowed a multiplanar reconstruction of the deferent ducts (from verumontanum to epididymis) (Fig. 3 on page 11). Sexual abstinence of 7 to 10 days was recommended.

239 patients who had MRSP performed between 08/2008 and 08/2018 were studied.

Reason for medical appointment:
A medical appointment was requested in 117 cases due to infertility (49%), in 76 due to hemospermia, (31.8%), in 28 due to perineal pains (11.7%) and in a further 18 cases due to other various causes (7.5%).

**Image Findings:**

All the patients were studied with the same protocol for the seminal route. The most frequent findings were congenital anomalies in 59 cases (24.7%) and seminal bleeding in 53 (22%), followed by cysts in the midline in 34 patients (10%), parasagittal cysts in 6 (2.5%), 2 (0.8%) had tumor invasion and 1 had a primary tumor (0.4%). (Fig. 4 on page 12 and Fig. 5 on page 13)

**Congenital anomalies:**

They can be classified as anomalies due to number (agenesis, fusion, duplication), maturation (hypoplasia), position (ectopia) or structure (diverticulum, cyst, communication with the ureter). [6]

Among these anomalies, the agenesis of the SV is the most frequent . [6]

Usually it is associated with other alterations. If the lesion occurs before 7 weeks of gestation (before ureteral sprouting), patients will also have ipsilateral renal agenesis [7] (Fig. 6 on page 13). The bilateral or unilateral agenesis of the seminal vesicle can be associated with the corresponding agenesis of the vas deferens (Fig. 7 on page 14), with unaltered kidneys. These findings may correspond to an underlying mutation in the gene regulating the transmembrane conductance of cystic fibrosis CFTR (Fig. 8 on page 15). Kim B. et. al. reports that bilateral agenesis of the seminal vesicles is associated with mutations in the gene regulating the transmembrane conductance of cystic fibrosis in 64% -73%, as well as the vas deferens is observed in 99% of patients with cystic fibrosis, but only two thirds of patients with bilateral agenesis have a CFTR gene mutation.[7]

*Hypoplasia* of SV is a congenital deformation that results in a smaller than normal SV. The size of the seminal vesicle usually decreases in patients older than 70 years. Hypoplasias of the seminal vesicles are usually bilateral and they are defined by the anteroposterior diameter, which in normal patients must be above 1.5 cm when we measured in the sagittal T2WI sequence.(Fig. 9 on page 16) [5,9]

The ectopic ureteral insertion is less frequently among the anomalies of structural development. It can occur in the posterior urethra (50% of cases), seminal vesicle (30%), vas deferens or ejaculatory duct (20%).[8](Fig. 10 on page 16)
Another group of congenital anomalies are the cystic lesions, that can be classified in midline cyst and parasagittal cyst. Because of the frequency of these findings we are going to describe them separately.

**Seminal bleeding:**

Haemospermia are a common symptom of seminal hemorrhages, with an incidence that varies between 25-52% [5]

They can be commonly caused by cystic dilations, obstructions of the seminal pathways, prostate carcinoma, lithiasis of the seminal vesicle and other less common pathologies.

Magnetic resonance has proven to be a reliable method to demonstrate the presence of blood in the seminal vesicles, and is widely considered a gold standard [1]. The hemorrhage of the seminal vesicle shows changes in the intensity of the signal in T1 and T2 according to the evolution of the bleeding.

A seminal fluid with hyperintense signal in sequences weighted in T1 and hypointense in T2WI is suggestive of acute-subacute bleeding (less than 7-12 days of evolution) (Table 2 on page 17) (Fig. 11 on page 18). The change in signal intensity in the sequences weighted in T2 to hyperintense, attributable to cell lysis, correspond to a subacute-chronic state (between 1 week to 1 month) [10](Fig. 12 on page 18).

Hyperintensity in T1WI sequences is highly suggestive of hemorrhage, but it can exclude other causes such as infections and mucinous material. [2]

Hematospermia and ejaculatory pain are the most frequent symptoms [11], with infertility being the main reason for consultation [12].

One of the most remarkable causes of seminal bleeding is the presence of lithiasis.

*Lithiasis* of the SV is considered a rare entity, of unknown prevalence [2,11], as well as its pathophysiology and etiology [11,12], but in which obstructions of the ejaculatory ducts could appear, aggravated by comorbidities such as diabetes, or chronic prostatitis [12].

Transrectal ultrasound has been considered traditionally the gold standard for the study of lithiasis in SV[12], because it is available and economical, but it does not provide information on the content of the seminal vesicles, generates discomfort for the patient and it is an invasive method.

In magnetic resonance they are visualized as foci of low intensity of signal in sequences weighted in T1 and T2, finding that must be confirmed with the contribution of the CT for the identification of the calcium content [2, 3]. (Fig. 13 on page 19)
Cystic lesions:

Can be classified as midline and parasagittal cysts. [9]

**Midline Cyst:**

These are the prostatic utricle and the Müllerian duct cysts. The former are of endodermal origin, communicated with the posterior urethra or the ejaculatory duct, and sometimes contain sperm. They are more frequent in men under the age of 20 and occur in 1 to 5% of the general population[3]. (Fig. 14 on page 19) they can be associated with genitourinary abnormalities, such as hypospadias, intersex disorders, cryptorchidism, and ipsilateral renal agenesis, usually having a length of 8 to 10 mm.

Prostatic utricle cysts do not extend above the base of the prostate and remain confined in the prostatic limit. They contain fluid, having a high signal intensity in images weighted in T2, in some cases also showing a high signal intensity in both T1 and T2 due to hemorrhage. [3] Dinamyc micturition MR confirmed the communication with the prostatic urethra and make diagnostic of the prostatic utricular cyst. (see video in Fig. 15 on page 20)

Müllerian duct cysts have a mesodermal origin and are not associated with congenital genitourinary disorders. Their maximum incidence is in men from 20 to 40 years of age, with an informed prevalence of less than 1%. Appearing as typical tear-shaped or oblong-shaped midline cysts that extend over the posterior superior margin of the prostate and best seen in the sagittal plane, they do not communicate with the posterior urethra and do not contain sperm. [3] (Table 3 on page 20)

**Parasagittal Cyst:**

Cysts of the seminal vesicles are most prevalent in patients aged between 20 and 30 years, perhaps in relation to the onset of sexual activity [7]. They can be visualized as a thin-walled unilocular cyst on the posterolateral aspect of the bladder [3] and usually have a diameter of less than 5 cm [6,13]. Images weighted in T1 show them with a variable signal intensity, having an increased signal in cases of hemorrhagic cysts or with proteinaceous content [7].(Fig. 16 on page 21)

Although its morphological characteristics are difficult to differentiate between congenital and acquired, the presence of an obstructive process (scar, tumor, inflammation, etc) leads us to the latter as well as findings of associated genitourinary anomalies [14], suggests a poor development of the distal mesonephric duct and defective ureteral sprouting [7].
Rarely, cystic dilatation of the vas deferens can be observed, frequently in relation to obstructive causes. Among these are cysts of the midline, seminal vesicle cysts, stones and fibrosis.

A dilated vas deferens is observed as a fusiform cystic mass, hyperintense in sequences weighted in T2 and hypointense in T1, being able to increase its signal intensity in T1 due to the presence of hematic content [13]. (Fig. 17 on page 21)

There are some cystic anomalies in associations with congenital anomalies and genetic conditions.

The Zinner syndrome, characterized by unilateral cystic dilation of the seminal vesicle with ectopic ureter drainage and atrophy or agenesis of the ipsilateral kidney, is a congenital alteration of Wolff's ducts, which occurs when an injury affects this embryological structure before the 7th week of gestation.[3, 6] (Fig. 18 on page 21)

In some patients with autosomal dominant polycystic kidney disease, can be associate bilateral seminal vesicle cyst. [3]

**Tumor lesions:**

*SV tumors may be primary tumors or secondary dissemination of adjacent organs such as the bladder, prostate, rectum, or lymphoma.*

**Tumoral Invasion:**

The involvement of the seminal vesicles by prostatic adenocarcinoma is the most common occurrence. Approximately 12% of patients with a clinical stage under prostate cancer will have the participation of the seminal vesicles due to adenocarcinoma of the prostate. [15] (Fig. 19 on page 22)

**Primary Tumor:**

Primary tumors of the seminal vesicle are extremely rare. There is a spectrum of tumors derived from both the epithelium and the stroma. Among them are benign tumors such as papillary adenoma, cystadenoma, hydatid cyst and amyloid deposition or adenocarcinoma malignant tumors, sarcoma, primary and carcinoid seminoma. [15]

Cystadenoma is often seen as a unilateral multiseptate cystic mass in the retrovesical space. [16] The contour is clear with a well-defined capsule. Contiguous anatomical structures are compressed without signs of infiltration.(Fig. 20 on page 23)
Fig. 1: Schematic diagram illustrating comparison between transrectal ultrasound (TRUS) and MRSP: Graphic representation of the area studied by TRUS (A), only studies the seminal vesicle and ampulla of ducts defers. The same graph in RMVS (B), studies the complete seminal path and its content.

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Fig. 2: Anatomy of seminal pathway. Graphic representation in sagittal plane (A) and its correlation with magnetic resonance in T2 WI (B), demonstrating its relationship with the prostate (p) and the bladder (b). Graphic representation in oblique coronal (C) and its correlation in T2 WI (D); seminal vesicles (Yellow arrow), ampullary portion of the vas deferens (Blue arrows) and the ejaculatory ducts ending (Green arrow)

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### Table 1: Sequences used in magnetic resonance of the seminal pathway (MRSP)

<table>
<thead>
<tr>
<th>Group of sequences</th>
<th>Sequence</th>
<th>Plane</th>
<th>RT</th>
<th>ET</th>
<th>FOV</th>
<th>Thickness</th>
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<td>Locators</td>
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<td>8.0</td>
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<tr>
<td></td>
<td>HASTE</td>
<td>Sagittal</td>
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<td>97</td>
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<tr>
<td></td>
<td>HASTE</td>
<td>Coronal</td>
<td>1000</td>
<td>97</td>
<td>450</td>
<td>8.0</td>
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<tr>
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<td>125</td>
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<tr>
<td></td>
<td>TSE T2</td>
<td>Sagittal</td>
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<td>123</td>
<td>300</td>
<td>4.0</td>
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<tr>
<td>Seminal vesicles</td>
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<tr>
<td></td>
<td>TSE T1</td>
<td>Major axis</td>
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<td>9</td>
<td>210</td>
<td>3.0</td>
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<tr>
<td></td>
<td>TSE T2 FS</td>
<td>Major axis</td>
<td>3000</td>
<td>125</td>
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<td>3.0</td>
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<td>145</td>
<td>370</td>
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</table>

**Table 1:** Abbreviations: HASTE: Half Fourier Acquisition Single Shot Turbo Spin Echo, ET: Echo Time, RT: Repetition time, FOV: Field of view, VIBE: Volumetric interpolated breath-hold examination. TSE: Turbo spin-echo, FS: Fat Saturation

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Fig. 3: Principal sequences of our specific protocol: Sagital T2W (A). The green line represents coronal oblique plane following axis of seminal vesicles. Coronal oblique in T2W (B). Sagital T2W(C): purple line shows the reconstruction path through the anterior middle deferent duct, and yellow line posterior middle deferent duct. Deferent Ducts MPR reformation (D): both deferent duct deployed from testicle to the veru mantarum, anterior half (purple arrow) and posterior half (yellow arrows).

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**Fig. 4:** Graphic of reason for magnetic resonance of the seminal pathway

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**Fig. 5:** Graphic of image findings in magnetic resonance of the seminal pathway

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Fig. 6: Unilateral seminal vesicle agenesis: Coronal T2WI oblique (A). Agenesis of the left vesicle seminal. Sequence for the retroperitoneum (B) with homolateral kidney agenesis. (yellow arrows in A and B)

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Fig. 7: Unilateral seminal vesicle. Agenesis. Sequence for the retroperitoneum with normal kidneys (A). Coronal oblique in T2WI (B), agenesis of the left SV (purple arrow). Multiplanar reconstruction (MPR) of deferent ducts (C) between testes (T) and prostate (P) confirms the deferent ducts integrity at the right side (yellow arrows) and absence on the left (green arrows).

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Fig. 8: Bilateral agenesis and hypoplasia: Two different patients, ( brothers). ABOVE: Patient 1, 31 years old: Axial T2WI (A) with bilateral agenesis (green arrows). Coronal T2WI oblique (B) with bilateral agenesis (green arrows). Sequence for the retroperitoneum with normal kidneys (C). UNDER Patient 2, 32 years old: Axial T2W (D) with bilateral hipoplasia (yellow arrows). Coronal T2WI oblique (E) with bilateral hypoplasia (yellow arrows). Sequence for the retroperitoneum with normal kidneys (F). Both brothers presented mutation in the gene CFTR of cystic fibrosis.

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Fig. 9: Bilateral seminal vesicle hypoplasia. sagittal T2WI right vesicle (A) with anteroposterior diameter. Axial T2WI bilateral hipoplasia (B) (yellow and purple arrows) Sagittal T2WI left vesicle (C) (purple arrow)

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**Fig. 10:** Sagittal T2WI (A), left ureteral bud and left seminal vesicle with common termination in the prostate near the veru montanum (yellow arrow). Correlation with laparoscopic surgery (B). UB: ureteral outbreak, LSV: left seminal vesicle, RSV: right seminal vesicle, VD:vas deferens

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<table>
<thead>
<tr>
<th>Time</th>
<th>Component</th>
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<th>T2 W</th>
<th>Liquid aspirated</th>
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</thead>
<tbody>
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<td>&lt; 1 Week</td>
<td>Deoxyhemoglobin</td>
<td><img src="image1" alt="A" /></td>
<td><img src="image2" alt="B" /></td>
<td>Abundant red blood cells with conserved membranes</td>
</tr>
<tr>
<td>&gt; 1 Week</td>
<td>Metahemoglobin and Red blood cells lysates</td>
<td><img src="image3" alt="C" /></td>
<td><img src="image4" alt="D" /></td>
<td>Scarce red blood cells with signs of lysis and macrophages loaded with hemosiderin</td>
</tr>
</tbody>
</table>

**Table 2:** Hematological stages: Acute hemorrhage: Coronal oblique in T1W (A), hyperintensity in the right seminal vesicle (Yellow arrow). Coronal oblique in T2WI(b), hypodensity in right seminal vesicle (Yellow arrow). Chronic subacute hemorrhage: Oblique coronal in T1WI (C), hyperintense signal of seminal fluid in the right seminal
vesicle (yellow arrow). Coronal oblique in T2 WI (D), hyperintensity of seminal fluid in left seminal vesicle (yellow arrow).

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**Fig. 11:** Left seminal vesicle with acute hemorrhage: Coronal oblique in T1 WI (A), hyperintensity in the left seminal vesicle (Yellow arrow). Coronal oblique in T2 WI (B), hypodensity in left seminal vesicle (Green arrow).

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**Fig. 12:** Left seminal vesicle with acute hemorrhage: Coronal oblique in T1 WI (A), hyperintensity in the left seminal vesicle (Yellow arrow). Coronal oblique in T2 WI (B), hypodensity in left seminal vesicle (Green arrow).

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Fig. 13: Lithiasis of seminal vesicles in patient with hepatorenal polycystosis. (A) Coronal T2WI, lithiasis in the excretory (eyaculatory) duct of the left seminal vesicle (Arrow). (B) Coronal oblique T1WI and (C) Coronal oblique in T2W, focal hypointense, correspondent to lithiasis in right seminal vesicle (Arrow). (D) Axial TCMD and (E) Axial T2WI, confirms the presence of lithiasis (arrows) and mild degree of ectasia in both seminal vesicles. (F) Coronal T2WI sequence for the retroperitoneum, demonstrates the coexistence of renal polycystosis.

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Fig. 14: Midline Cyst: T1W (A) hyperintense content (yellow arrow). Coronal oblique T2W (B) with fat suppression liquid level for hematic-seminal fluid in midline cyst (green arrow) and seminal vesicles (red arrow in A and B). Communication with the urethral lumen (purple arrow) (C).
Fig. 15: Video 1: Midline Cyst. Dinamyc micturition MR confirmed the communication with the prostatic urethra and make diagnostic of the prostatic utricular cyst

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**Table 3:** Differences between prostatic utricle and the Müllerian duct cysts.


**Fig. 16:** Cyst of the right seminal vesicle. Coronal oblique T1 WI (A), hypointense content cyst (yellow arrow). Coronal oblique T2 WI (B), same patient, hyperintenseness of the cyst is observed (yellow arrow). T2 WI coronal sequence for the retroperitoneum (C) with normal kidneys.

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**Fig. 17:** Cyst of the ampulla of the vas deferens. Oblique coronal axis of seminal vesicle in T1WI(A). Same plane T2WI(B) cyst with hematic-protein content in subacute-chronic stage (yellow arrows). Sagittal T2WI(C), the saccular form of the seminal vesicle cyst is observed (Yellow arrow).

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**Fig. 18:** Zinner syndrome. Oblique coronal in T2 WI (A), hyperintense content in cystic dilatation of the rudimentary ureter (yellow arrow), left seminal vesicle (purple arrow) and left deferens ampulla (Purple arrow head). T1 WI (B), with hyperintensity inside the aforementioned structures (yellow arrows), Due to subacute-chronic hemorrhage. Coronal T2 WI retroperitoneum (C), rudimentary left kidney (Yellow arrow). Axial CT (D), ectopic outlet of the right ureter (Yellow arrow).

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**Fig. 19:** Tumoral Invasion: prostatic solid formation. Axial T2 WI (A). Sagital T2 WI (B). Coronal T2 WI(C). Tumoral prostatic with dissemination to bladder and seminal vesicles (yellow arrows)

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**Fig. 20:** Cystadenoma: T2 WI axial (A), coronal oblique in T1 WI (B) and T2 WI (C). Tumor with hyperintense cystic component in both sequences attributable to hematico-protein content and associated with solid vegetation (yellow arrow). Fusion, by way of illustration, of axial image weighted in T2 and surgical piece (D). Macroscopic surgical piece (E). Microscopy (F) with hematoxylin-eosin, tumor proliferation constituted by cysts and glands (star), upholstered by few layers of cubic or cylindrical epithelial cells (yellow arrow head) and variable amount of stroma (green arrowhead).

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Conclusion

The pathologies of the seminal pathway are a heterogeneous group of anomalies that appear with varied manifestations. The MRSP protocol, exposes advantages in comparison with the US transrectal. It is a non-invasive method, allowing to study the seminal pathway in its entirety and characterize its content; as well as to show congenital structural anomalies, causes of obstruction, differentiate solid or cystic masses and tumor pathologies.
Personal information

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