

I'm sharing with you a description of my 30-year history with CPPS.

First, let me remind you of the guidelines of the American Urological Association (AUA), which classify many causes of chronic pelvic pain:

<https://www.auanet.org/guidelines-and-quality/guidelines/male-chronic-pelvic-pain>

Possible non-bacterial causes of prostate pain according to the AUA:

1. Inflammation/immune, mast cell hyperreactivity (consultation with an immunologist recommended)
 - a) Chronic inflammation of the prostate/vesicles (often without bacteria in cultures)
 - b) Semen retention → activation of inflammatory cytokines.
 - c) "Neurogenic inflammation" (release of substance P, CGRP from nerve endings) - consultation with a pain management neurologist is recommended.
2. Myofascial pain - requires physiotherapy.
 - a) Excessive tension in the pelvic floor muscles (hypertonia).
 - b) Trigger points in the levator ani and coccygeus muscles.
 - c) Overexertion after sexual activity, prolonged sitting, or stress.
3. Neuropathic pain - consultation with a pain management neurologist is recommended.
 - a) Irritation of the pudendal and sacral nerves (S2–S4).
 - b) Neuralgia following injuries, surgeries, or infections.
 - c) Central sensitization → hypersensitivity to mechanical stimuli (e.g., ejaculation causes disproportionate pain).
 - d) Long-term post-inflammatory changes in the nerve region
4. Visceral/Structural
 - a) Benign prostatic hyperplasia (BPH) → obstruction of the ejaculatory ducts, stagnation of secretions.
 - b) Sternal duct stenosis, stones in the prostate/vesicles.
 - c) Adhesions following infections, inflammations, or surgeries.
5. Vascular/Microcirculation
 - a) Disturbances in pelvic venous outflow, microemboli.
 - b) Local ischemia → promotes fibrosis and post-ejaculatory pain.
6. Psychosocial
 - a) Stress, anxiety, depression → intensify pain perception.
 - b) Post-ejaculatory pain may persist through psychophysiological mechanisms.
7. Other causes.

And now my case was rather from the beginning it was hyperreactivity of mast cells and "Neurogenic inflammation" - strong stimulation of nerves to act in the area of seminal vesicles and prostate (secretion of large amounts of fluid), hypersensitivity to any stimuli (including ejaculation), nerve reconstruction after multiple, frequent ejaculations (release of substance P, CGRP from nerve endings).

My parents are genetically very empathetic, highly sensitive. They worked in professions requiring empathy, such as veterinarians and nurses. They are now 85 years old and retired. They have their own age-related illnesses, including my father, who has had prostate cancer for 10 years. He takes

hormone injections, and the cancer is under control. They do not have CPPS. My parents are sensitive and empathetic.

Since birth, I have had some of the hypersensitivity and hyperactivity characteristics of ADHD (although this doesn't qualify me as ADHD 100%, but at least 75%, as being raised with clear guidelines helped me greatly). These characteristics are called neurodiversity. Statistically, ADHD is associated with many "genetically programmed" somatic complications throughout a person's life. When I was playing, I was more sensitive, anxious, and fearful than other boys. I also have other ADHD traits—high emotional intelligence, sometimes racing thoughts, and a tendency to overanalyze events. What distinguishes me from severe ADHD is that I never used substances. As a teenager, I was rather reserved, serious, less playful than my peers, and sometimes idealistic. During my teenage years, I didn't smoke, didn't get drunk, and didn't party often, but occasionally.

My first sexual experience occurred at age 6 or 7 and involved several dry ejaculations. The first time was accidental, I accidentally brushed against something. The next time was out of curiosity. But I didn't pursue the matter further; rather, I exhausted my curiosity and stopped. At that time, the hormones that stimulate sexual activity weren't yet in my bloodstream. I didn't have any psychological or religious problems, nor did I feel any guilt about it. I was raised Catholic, but without any guilt.

When the hormones started kicking in (around the age of 14-15), I usually ejaculated once a week. Sometimes twice.

Later, after the age of 15, the hormones began to have a stronger effect. The nervous system around the seminal vesicles also became more active. I became aroused quickly and felt a strong pressure to ejaculate – I could feel this in the seminal vesicles, as well as the penis being very sensitive to touch, friction against underwear, and frequent erections. My body was producing a lot of seminal fluid, and if I didn't ejaculate for a week, I could feel it in the seminal vesicles, as well as the penis being very sensitive to touch and the rapid arousal.

After age 17, I usually ejaculated 2-3 times a week. The pressure from the nervous system, after a 2-3 day break, was significant for ejaculation. Rubbing and touching quickly caused erections, arousal, and the feeling of full seminal vesicles.

At that time, I also had other fluid excesses: gastritis due to excess bile, and hyperacidity (endoscopies were performed). Dentists told me I was producing a lot of saliva.

I lived in a small town and wanted to go away for college. My first girlfriend was after I left for college at the age of 20 (in the early 1990s).

Studying, observing my peers, and living in a dormitory in a big city toughened me up mentally.

Around the age of 21, I went on vacation to the mountains with my girlfriend from college, who was also very energetic and very curious about life. It was the first time we spent several days alone without interruption, undisturbed. We hiked in the mountains during the day and had sex every evening. After a few days, the weather worsened. We stayed in bed. From the morning on, we had vaginal sex, two or three times over the course of two or three hours. I always ejaculated quickly on the first try. Subsequent intercourse extended the penetration time. And by the third time, it was very long, and I even had trouble maintaining a long erection in my vagina – the hormones had already been released by sex. We remained curious about each other's bodies, so for the next few hours, we took baths and touched each other. Thanks to the release of sexual tension, there were no quick ejaculations, we could stimulate each other longer, and we engaged in oral sex. After the

fourth ejaculation, which contained a small amount of clear fluid, I felt a slight pain above the pubic symphysis on both sides (I now know these are the ejaculatory ducts, the base of the seminal vesicles). But it stopped after an hour. And we went out for dinner. That evening, I had my fifth ejaculation of the day. After that, the pain in the ejaculatory ducts became more intense and intensified over the next few hours. It lasted for two or three days, then gradually subsided. During this time, there were no further ejaculations for about a week. The pain stopped after a week. I forgot about it. I never felt guilty about it. I considered it a pleasant experience. I didn't remember it until I was about 50, when the pain returned in the same place.

Today, I believe I have a genetic predisposition that triggered MCAS or other neurological mechanisms in the highly innervated seminal vesicles.

I never had any sexual eccentricities, interrupted intercourse, delayed ejaculation, anal sex, or casual sex. I didn't use stimulants or other substances.

I never had so many ejaculations in a single week or day again. Usually 2-3 per week. After a week without sex, ejaculation into the vagina without a condom lasted about 15 seconds, so 99% of the time I had sex with a condom because it delayed the sensitivity of my penis and allowed for longer penetration. Therefore, I believe I never had any problems due to bacteria. I had a very high sex drive until I was 40, and ejaculations were regular because they were unavoidable and there was no reason for them. My illness only forced me to limit them in my 40s. Many months later, after that vacation, when I washed my penis with cold water after ejaculating, I would experience discomfort for several hours after ejaculation (this only happened after ejaculation). At 24, I graduated from college and went to work. My work usually involved sitting and walking. However, sometimes I would sit for 3-5 hours with short breaks.

At 25, during the summer, after three ejaculations in a week, one day after ejaculation and after prolonged sitting at work, I felt increased discomfort in my seminal vesicles, and after a few hours, my prostate "began to swell" and I needed to urinate. The next day, I felt mild pain and even more frequent urination. No fever. That's how my first prostate infection began. I went to a urologist and had cultures done. After that, I was given a weak antibiotic for three weeks. The urine cultures were sterile. The pain and LUTS subsided – my body quickly recovered.

My libido was strong then. After a while, I had about three ejaculations in a week again, and after prolonged sitting at work (squeezing the prostate and seminal vesicles), and the inflammation returned. I was in my early 30s and had prostatitis 2-3 times a year, mainly in the summer, when there's more stimulation for ejaculation (e.g., girls at the beach). Subsequent cultures were also taken, including for chlamydia and semen. The tests came back negative. Next time, I was prescribed stronger antibiotics (this was the late 1990s). Abstinence due to discomfort helped the body regenerate quickly. A urologist at the university hospital was then introducing laser biostimulation treatment (low-level laser therapy). I started seeing him because it helped. After just one laser treatment (the head was aimed at the prostate behind the testicles on the perineum), there was significant improvement. I had four treatments, one every week.

Laser physical therapy (biostimulation) is a non-invasive treatment for prostate complications, providing anti-inflammatory, analgesic, and swelling-reducing effects.

At age 27, an ultrasound revealed a thickened cover of prostate and occasional calcifications. The nervous system of the seminal vesicles was stimulated to action and production of seminal fluid. It was easily aroused sexually. Laser biostimulation helped, but again, after a situation where, after about three ejaculations a week and sitting long, I felt discomfort from the seminal vesicles and after a few hours from prostate. There was never a fever or severe pain. There was mild pain

and frequent urination during the day and night. If I had about three ejaculations per week and sat for long periods, after 1-2 days after ejaculation I would experience seminal vesicle discomfort and prostate pain. I could feel the prostate "swelling, " then after 2 days I would experience prostate and seminal vesicle pain. After turning 30, my seminal vesicles slowly shrank (ultrasound showed enlargement, with a small prostate of about 30 ml). I could feel the vesicles enlarge after ejaculation (not due to abstinence), and then I would experience pain. In addition to pain, ultrasound revealed changes in the prostate – thickening of the capsule, calcifications, post-inflammatory changes, and enlargement of the vesicles up to 6 cm. Lasers quickly helped, and the seminal vesicles shrank within the next week. I never had chlamydia (or scarring from it) or bacteria in my urine or semen. I never engaged in risky sexual behavior. I had regular ejaculations. Physiotherapy helped: low-level lasers up to 400mW under the testicles, towards the prostate. Once a week, it helped, and after two, I was fine. I had these post-ejaculatory inflammations several times a year, often in the summer. After ejaculation and sitting, they would worsen and hurt similarly. This exacerbated LUTS. When I learned to live with my problem, I avoided ejaculations above three times a week, despite my sexual drive, and then there were no problems. But I didn't always manage. I was 30. I knew this mechanism and tried to control it. Since I was 30, I have been regularly taking a saw palmetto supplement to this day. When I was 30, I met my future wife – an internal medicine doctor. She examined me completely. She's also a very empathetic person, with highly developed emotional intelligence, and a very holistic view of people. From time to time, I'd go for these lasers in advance, even when I knew I have my third ejaculation in a week and had to be at work. After successful conception I became a father without any problems, and two years later I became a father again.

Up to 40 years of age, the disease had been somewhat controlled. Saw palmetto also stabilized my condition. I had laser treatments without waiting for worsening after frequent ejaculations, and I avoided sitting after frequent ejaculations. It seemed to be okay. I had about 4-6 laser sessions per year. However, the sex drive to ejaculate, the sensitivity of the penis to touch, and the production of seminal fluid (i. e., libido) were still high. Unfortunately, after the age of 40, the disease became apparent again due to ejaculations and age. On ultrasound after ejaculation, I sometimes had long seminal vesicles, measuring 5-8 cm, and I felt they were inflated. My body no longer regenerated as quickly after ejaculations, even those two a week. And my libido was still high. I always took care of my health, including light runs up to 5 km once a week and occasional swimming on the pool. After swimming in the pool I always had to pee more often at night for 1-2 nights, but nothing hurt. No stimulants or unhygienic lifestyles. I avoided pornography because after watching it I always felt a strong and frequent need to ejaculate, and this caused problems later. My body was generally healthy, so I still demanded two ejaculations a week, otherwise, I experienced severe sensitivity to touch and seminal vesicles discomfort. I had lasers if a flare-up occurs: sessions averaged 5 times a year. Around the age of 42, I had another case of prostatitis – after a trip to the seaside in September (it was cold) – it started with the prostate, not the seminal vesicles. I was given antibiotics. I couldn't ride a bike anymore because it left me with a strong urge to urinate for several hours (the saddle and the crushing). I hadn't ridden much before. Sometimes I went to a modern, clean swimming pool, and afterward, I developed severe LUTS for 48 hours (probably MCAS from the temperature, but I didn't know it was MCAS at the time). After the second ejaculations, I still couldn't sit on a hard surface for long because my seminal vesicles hurt slightly and became inflamed. I could sit on a toilet seat. However, it wasn't the pudendal nerve, but rather the crushing of the prostate and seminal vesicles. I bought a horseshoe-shaped (U) foam cushion and it helped a little with prolonged sitting, but didn't eliminate the problems.. My prostate was then above the seat. Perhaps the pudendal nerve can also somehow transmit some impulse. However, the problems always occurred only after the next ejaculation and not after every sitting I couldn't sit with a belt on my pants because my seminal vesicles hurt from the pressure on my abdomen (changes in intra-abdominal pressure), and after every ejaculation, even a rare one, they would become inflamed. That's why I used suspenders. I also couldn't wear tight pants around my thighs like I used to. When

I sat down, they became too tight, which also caused pressure in my thighs and crotch. I even had a slight tingling sensation from the tight pants, like hypersensitivity in the skin of my thighs and crotch. So I only wear pants a size larger with looser legs.

At age 42, after temporarily stopping the saw palmetto supplement, after a few days I felt a strange sensation in my prostate (probably fibrosis). I immediately bought another pack of saw palmetto. I had no erection problems. At age 42, I underwent another TRUS scan with a very good specialist, and it revealed some changes.

TRANSRECTAL ULTRASOUND (TRUS) OF THE PROSTATE The seminal vesicles are of normal size, without retention of contents. A single small calcification is present in the right seminal vesicle. The terminal portions of the vas deferens are normal. The prostate measures $41 \times 34 \times 39$ mm, with a volume of approximately 28 ml. In the peripheral zones, areas of increased echogenicity are visible, suggestive of fibrosis. In the right peripheral zone, a longitudinal fibrotic focus measuring $11 \times 15 \times 3$ mm is located approximately at mid-gland level. On the left side, a similar focus measuring $8 \times 5 \times 6$ mm is located in the anteroinferior part of the peripheral zone. No other focal lesions are identified in the peripheral zones. The transitional zones are mildly enlarged (benign prostatic hyperplasia – BPH). Echogenic calcifications are present within the prostate gland, around the prostatic urethra. The median lobe protrudes into the bladder lumen to a depth of 8 mm. The ejaculatory ducts are not dilated. The contours of the anatomical capsule are smooth. Conclusion: No inflammatory infiltrates or abscesses are identified. The fibrotic foci represent a typical ultrasonographic feature of chronic prostatitis. Mild benign prostatic hyperplasia (BPH).

I also visited another urologist:

Visit Date: 23 June 2014 Medical History and Physical Examination Current Medical History The patient has a history of seminal vesiculitis and mild enlargement of the prostate gland. He reports lower abdominal pain. Nocturia: up to 2–3 times per night. The patient was previously treated with antibiotic therapy. Currently, he experiences intermittent, stabbing pain in the lower abdomen. The patient denies other medical conditions. Current medications: dietary supplements. Physical Examination • normal gait • testes without abnormalities • digital rectal examination (DRE): prostate not enlarged, with glandular consistency Additional Information (23 June 2014) The patient previously took Celexa, without improvement, and reported cardiac rhythm disturbances. He was treated with laser therapy, which resulted in improvement; however, symptoms have since worsened again. The patient underwent TRUS on 17 April 2014, which described: • prostate volume approximately 28 cm³ • features consistent with chronic prostatitis Laboratory Tests • Total testosterone (TST): 4.58 (31 December 2013) • PSA: 0.4 ng/mL (31 December 2013) • Urine culture and urinalysis: no abnormalities detected Physical Examination (Repeat) • DRE: prostate not enlarged, with glandular consistency Test. I performed the 4-glass test twice, six months apart, in different labs – and they showed nothing. Later, I also had: Diagnosis (as recorded) • Chronic prostatitis • Chronic pelvic pain syndrome (CP/CPPS) Services Provided During the Visit • 163 – Follow-up urological visit Specialty: Urology Recommended Treatment • 2149 – Extracorporeal Shockwave Therapy (ESWT) in urology Planned sessions: 7 • 159 – Prostate massage Planned sessions: 5 Prescribed Medications Tamsulosin Naproxen

I had several shockwave sessions. However, too frequent ejaculation and prolonged sitting caused further inflammation. I underwent several sessions of pelvic floor exercises, massage of the muscles around the prostate, and biofeedback with a very good pelvic floor physiotherapist, but I didn't feel

any significant muscle tension. There was initial improvement after the biofeedback, but the currents caused irritation after the first few sessions. I also underwent INDIBA treatments – a physiotherapy method that uses a patented radiofrequency current at a constant frequency of 448 kHz to stimulate cells and accelerate tissue regeneration. Too frequent ejaculation and prolonged sitting = increased inflammation. My PSA was always low, and my testosterone levels were normal. At around age 45, I had a CT scan with contrast, which showed no changes in my prostate or pelvis. I consulted a gastroenterologist and had a colonoscopy – both also revealed nothing. I'd never had any visceral problems, aside from hyperacidity in my youth, which gradually subsided in my thirties when my wife started cooking me healthy meals. For 45 years, I avoided ejaculation more than ONCE a week. Fortunately, my libido declined. I was now infertile. A examination of my semen revealed the presence of individual sperm and there were Semen microscopy revealed the presence of lipid bodies / lipophages (lipid-laden macrophages), consistent with chronic inflammatory changes. When I turned 48, I experienced discomfort after 3-4 hours ejaculation, so I also took antispasmodic medications for smooth muscles. After I took stronger antispasmodic medications before ejaculation to prepare myself. When I took large doses of them orally, I felt currents and nerve fibers, but only in the seminal vesicles and prostate. After I turned 50, low-emission lasers stopped helping. That was in 2022. One very hot summer day, I spontaneously went with friends to a clean, modern outdoor pool with thermal water at 82°F (28°C). However, then I developed prostatitis. The urologist prescribed antibiotics because it occurred after swimming. A week later, things were better and I ejaculated. Unfortunately, the inflammation worsened again, this time even more severely. Two days later, while lying down, I felt the weight of my bladder pressing against my seminal vesicles, which were incredibly painful. The pain was very localized – at the base of the seminal vesicles, near the prostate. For several days, I couldn't change position without pain. I didn't go to work. I couldn't sit at all because the pain was getting worse. In a short time, two inflammatory mechanisms overlapped – low body temperature after swimming and ejaculation. My body had already lost its ability to regenerate quickly. I was restarted on naproxen. I took it for a long time, but with no improvement. I had an MRI 3T of my spine, but it revealed nothing. I also took pregabalin at a dose of 75 mg once daily plus 75 mg twice daily (total 225 mg/day), without significant improvement, together with baclofen 10 mg once daily. Tadalafil was prescribed, but it increased sexual drive and provoked ejaculation, there-fore I did not take it. We searched for information about the condition online and from other urologists who use modern treatments. I've tried several other medications, including diazepam suppositories. No improvement. In 2024 I repeated the TRUS but with the same radiologist as in 2014.

EXAMINATION: DIAGNOSTIC EXAMINATION – REPORT TRANSRECTAL ULTRASOUND (TRUS) OF THE PROSTATE – 2024 The seminal vesicles are of normal size, without retention of contents. Several small calcifications are present in both the right and left seminal vesicles. The terminal portions of the vas deferens are normal. The prostate measures 45 × 37 × 45 mm (transverse, AP, height), with a volume of 39 ml. Fibrotic foci are present in the peripheral zones in the upper half of the gland. No other focal lesions are identified in the peripheral zones. The transitional zones are mildly enlarged (BPH) with a glandular echostructure. Calcifications are present within the prostate gland, mainly around the prostatic urethra. The median lobe protrudes into the bladder to a depth of 11 mm. The ejaculatory ducts are not dilated. The contours of the anatomical capsule are smooth. Conclusion Post-inflammatory changes in the form of calcifications within the seminal vesicles and fibrosis within the peripheral zones. No ultrasonographic features of an active inflammatory process are identified. Compared with the examination from 2014, the prostate volume is increased by approximately 10 ml. Calcifications within the seminal vesicles have appeared. The echostructure of the prostate has not changed significantly.

I had a 3T MRI done in early 2024

DESCRIPTION, RESULTS, CONCLUSIONS Multiparametric MRI (mpMRI) of the Prostate Examination performed according to PI-RADS v2. 1 protocol. Protocol includes: T2-weighted sequences (sagittal, axial, coronal), T1-weighted imaging, DWI, ADC, DCE. • Prostate volume: 35 ml • PSA: 0.644 ng/ml (2022) • PSA density (PSAD): 0.02 • Hemorrhage: none Transitional zone: features of benign prostatic hyperplasia (BPH) – PI-RADS 2. Peripheral zone: heterogeneous, with wedge-shaped hypointense foci – appearance consistent with evolution of an inflammatory process – PI-RADS 2. Seminal vesicles: decreased signal intensity – appearance consistent with evolution of an inflammatory process. Lymph nodes: up to the level of the aortic bifurcation – not enlarged, no features of lymphadenopathy. CONCLUSIONS • BPH – PI-RADS 2 • Inflammatory / post-inflammatory changes in the peripheral zone – PI-RADS 2 DIAGNOSIS Primary diagnosis: Inflammatory diseases of the prostate

My urologist, whom I had been seeing for many years, was very kind but couldn't help me. He recommended pain medication and that I seek treatment elsewhere. However, his lasers healed me for many years, and I am very grateful to him. I also found another well-known urology professor. He suggested a new medication, Silodosin (8 mg Silodosin), which, in combination with quercetin, proved helpful. I also avoided ejaculation. After three months (without ejaculation), the pain was 80% gone. Three months after ejaculation, I experienced mild pain and had to avoid sitting for a few days. When the pain subsided significantly, my libido increased, and ejaculations returned every eight weeks. This was a mistake, as this caused inflammation to develop after each subsequent ejaculation. I tried various medications and supplements, including hydroxyzine, SNRIs, and others. Some medications caused difficulty initiating and maintaining urination. After all this, this new urologist performed a cystoscopy on me. During this visit I told him that I wanted to HOLEP that I couldn't live with such uncertainty about whether I would pee.

VISIT REPORT Visit Title Cystoscopy Medical History Patient with chronic pelvic pain syndrome (CPPS) and non-bacterial prostatitis. Symptoms: PSA < 1 ng/mL MRI: calcifications and inflammatory changes No erectile dysfunction (ED) Prostate volume: approximately 39 mL Nocturia: up to 6 times per night, worsened after prolonged sitting Previous Treatment Silodosin, baclofen, pregabalin, Specialist Examination (ICD-9) Transurethral cystoscopy [57.32] Findings: entry into the urethra under direct visual control, external sphincter contracted, spastic. Does not open in response to irrigation flow verumontanum elevated, paradoxically closing the urethral lumen. Prostate enlarged in the lateral lobes, creating a "curtain sign", bladder neck elevated with a third lobe, constricted, urinary bladder normal. Overall appearance consistent with benign prostatic hyperplasia and trapped prostate ICD-10 Diagnosis Benign prostatic hyperplasia (BPH) Treatment Silodosin, dosage: 1 capsule once daily in the morning Baclofen – tablets (10 mg), dosage: ½ tablet in the morning + 1 tablet in the evening. Pregabalin – hard capsules (75 mg) dosage: 1 capsule in the morning + 2 capsules in the evening Recommendations Continue treatment for 3 months After 3 months: repeat PSA, semen culture, and reassessment of symptoms If symptoms persist - qualification for HoLEP (Holmium Laser Enucleation of the Prostate)

I underwent the HoLEP procedure in May 2025. There were no complications. After the procedure, the urologist explained that I had a small prostate, but its interior was hard and fibrotic, and the laser penetrated it slowly. (People with large adenomas usually have soft tissue.) The procedure took over an hour. Initially, I noticed a slight pain when urinating after surgery, but after one or two days, the pain subsided. After two days, I was urinating in a strong stream, like a teenager. After three weeks, without sitting, the post-procedure pain subsided. I continued taking Silodosin (8 mg). After six weeks, I ejaculated. Immediately after the ejaculation, I felt the inside of my prostate when

urinating, experienced mild discomfort in my seminal vesicles, and after a few days, pain, even without sitting. This ejaculation was too early after the procedure, because the tissues were still sore after the HOLEP and I gave them an additional shock from MCAS. The pain returned, just like it did immediately after the procedure. After a few days, it began to subside. I also started taking PEA, Quercitin (you need to take breaks from taking it when your joints hurt and after reduce the dose) and LDN. The inflamed area decreased because my prostate is now empty. I also tried antihistamines and steroids. The antihistamine Claritin worked too well – it was very stimulating – I felt a very strong urge and high pressure to ejaculate, but ejaculation caused inflammation. I stopped taking it. I also take the anti-allergy medication Allegra – it doesn't stimulate the nerves in the seminal vesicles as much. I also tried the antihistamine Vistaril (hydroxyzine). I take both of them when I have a flare-up (after ejaculating too frequently for my age and the condition of my seminal vesicles). I tried prednisone 10 mg. It works well (I take it in the morning, the day before, and two to three days after ejaculation). However, it shouldn't be taken in the evening or in high doses for long periods. It worsens the pain in the long term. So I'm testing various medications under the 24/7 supervision of a doctor-my wife. The ones I've recommended work best for me. Others may have different results. Now I schedule my ejaculations every 4-6 weeks because, fortunately, I no longer have a strong libido. Some medications, including healthy foods with phytohormones, cause me to ejaculate during sleep unplanned more often, meaning I have after it a flare-up. I took a CPPS test. When my symptoms stabilized after the HOLEP, I went swimming in the pool with 10 mg of prednisone (the day before and two days after). My prostate was fine. After swimming at home, I felt slight discomfort on these seminal vesicles. I urinated once during the night. Two weeks later (without ejaculation), I went swimming without medication. I could feel the edges of my seminal vesicles from the cold water. After swimming at home, I felt more intense sensations in my seminal vesicles and slightly in my prostate. I urinated about four times during the night. The next day, I took 10 mg of prednisone to treat this.

Information on what helps and what hinders can be found here:
<https://www.UCPPS.men/viewtopic.php?f=53&t=9338>

While on vacation after my HOLEP, I was looking for more information about the condition. I came across the American Urological Association (AUA) guidelines, which classify the many causes of pelvic pain, and a CPPS forum <https://www.UCPPS.men/>

In October 2025, after my HOLEP, I had a 3T MRI.

Description, Results, Conclusions Multiparametric MRI (mpMRI) of the prostate:
Examination performed according to PI-RADS v2. 1 protocol. Protocol: T2-weighted (sagittal, axial, coronal), T1-weighted, DWI, ADC, DCE. • Prostate volume: 17 ml • Compared with the previous examination dated 22 Feb 2024 Transitional zone: status post HoLEP. Peripheral zone: heterogeneous with wedge-shaped hypointense foci – appearance consistent with evolution of an inflammatory process – PI-RADS 2. Seminal vesicles: small, symmetrical, with decreased signal intensity – appearance may correspond to post-inflammatory changes; compared with the previous examination, changes are slightly more pronounced. Lymph nodes included in the examination: not enlarged, no features of lymphadenopathy. Diagnosis Primary diagnosis: Inflammatory diseases of the seminal vesicles

I know I already have significant post-inflammatory changes in my seminal vesicles, including fibrosis, which reduces their elasticity. After ejaculation, they hurt when I bend down. Pressing them while sitting causes inflammation. My reaction to medication suggests MCAS and nerve hypersensitivity. The changes are already significant after 30 years of illness. Fortunately, my libido has decreased and I can limit my ejaculations. Subsequent ejaculations still cause inflammation and

further tissue fibrosis. When I try to introduce medication now, I do it one at a time for at least 10 days to see what helps and what hinders. I've actually tested the medications listed on the website. <https://www.UCPPS.men/viewtopic.php?f=53&t=9338> They worsen after ejaculation. If I don't ejaculate for 2-3 weeks, recovery occurs, and some of them don't bother me. I'm very happy with HOLEP. I can now eat different foods, travel, take various medications, and I have no problem peeing. My case is quite rare, but there are likely many similar cases worldwide.

Each of you may have a different case and treatment should be under the supervision of doctors. However, you must undergo evaluation by specialists: a) urologists, b) pelvic floor neurologists and pain management specialists, c) immunologists, d) gastroenterologists, e) allergists, f) pelvic floor physiotherapists. g) psychiatrists, psychologists, sexologists, h) radiologists, i) orthopedists and surgeons - to rule out other causes, h) others.

Long-term use of steroids can significantly reduce inflammation in the seminal vesicles, prostate, urethra, etc. Damaged, reactive, or adjacent nerves to post-inflammatory tissues can intensify pain sensations in such cases. Similarly, but to a lesser extent, long-term use of high doses of antihistamines can also significantly reduce inflammation. Sometimes, in such cases, damaged, reactive, or adjacent nerves to post-inflammatory tissues can intensify pain sensations. Long-term use of high doses of quercetin can cause joint pain. Therefore, medications should be dosed judiciously, and not multiple medications should be tested simultaneously, but individual doses should be used to determine what helped and what harmed. In extreme cases, a neurologist treating chronic pain may suggest local (or intravenous) anesthesia to restore nerve function or a nerve block. An interesting example might be the use of anesthesia in patients with chronic prostate and seminal vesicle pain undergoing prostate biopsy. Local anesthesia is used during prostate biopsy. I wonder how much it reduces post-procedure neural reactivity in the long term? Medicine is constantly evolving and becoming increasingly sophisticated. Artificial intelligence will be very helpful in the future because it can work with large datasets. This will enable the identification of common turning points for a given group of conditions, characteristic of a given condition. I changed jobs – in mine, I don't sit still for long periods. I also often work from home. I have a special office chair and desk. Desk work places more stress on the back than the pelvis. My ADHD traits and deep analytical skills have been very helpful in my professional life. They have helped me achieve success. I am happy with my life. It's just that I'm over 50, and like most people my age, I have some health issues.