

# 14 Different Treatments for Vulvar Vestibulitis Syndrome

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Please see artist renderings of the surgery by clicking here. As Awareness Grows, So does Diagnosis and Treatments: Vulvar vestibulitis syndrome (VVS) is a subset of vulvodynia that is characterized by severe pain during attempted vaginal penetration to pressure localized to the vulvar vestibule (see diagram), and redness (erythema) of the vulvar vestibule. A subset of vulvar vestibulitis is called vestibulodynia, which combines the three symptoms with constant pain at the vestibule. Increased awareness of VVS has led to exciting new research. This review will examine current concepts regarding the diagnosis, etiology, and treatment of VVS.

Until recently, neither the cause of VVS nor what causes the underlying pain was known. However, in the last few years, increased awareness of vulvar vestibulitis syndrome (VVS) has led to exciting new research. This research focuses on many different aspects of VVS including possible genetic, infectious or allergic causes, and on multiple treatment regimens. Even basic assumptions of early researchers have been questioned, leading to a greater understanding of VVS.

We now know that women with VVS feel pain at the vestibule because they have an increased number of nerve endings in the vestibule. A woman with VVS can have up to 30 times the number of nerve endings (3000%) as compared to women who do not have sexual pain. The nerve endings are called nociceptors. Nociceptors are the nerve endings that are responsible for sensing pain and stretch.

So what caused this increase in the number of nerve endings?

Women with Primary VVS are women who have pain ever since their first attempt at intercourse. In fact, most women with primary VVS have had pain when they first started to use tampons. Approximately 20-25% of women with VVS have primary VVS. Women with primary VVS almost never have had completely pain free intercourse. The current hypothesis is that the neuronal proliferation in primary VVS is a congenital problem (birth defect). Current evidence supporting this hypothesis is that the tissue of the vestibule is completely different tissue than the tissue of the vagina above the hymen (embryologically, morphologically, and histologically). Therefore, it is plausible that a woman could have a congenital problem in the vestibule without having any problem in the vagina. This hypothesis is further supported by the fact that there is a very high concordance of women with VVS and interstitial cystitis (a similar pain syndrome of the bladder and urethra). The bladder and urethra are derived from the same tissue as the vestibule.

Women with secondary VVS have pain beginning after some period of pain free intercourse. Women with secondary VVS also have a proliferation of nerve endings, but unlike women with primary VVS, they are not born with it. Instead, women with secondary VVS have acquired these nerve endings. To explain this unfortunate acquisition is slightly more complicated than in primary VVS. Over the last 5 years scientist have been pasting together several different pieces of information to get a congruent explanation of secondary VVS. The pieces of information are as follows 1) women with VVS have much more sensitive skin throughout their whole body as compared to women without VVS. 2)

In addition to a proliferation of nerve endings in the vestibule, there is a proliferation of mast cells in the vestibule. Mast cells are the white blood cells that are responsible for allergic and inflammatory reactions. 3) Up to 50% of women with VVS have a defect in one of 2 genes (IL1-RA, IL-1 beta) that are responsible for limiting inflammatory conditions in the body. If we put these (and other) pieces of information together, a new hypothesis is emerging. (Actually, we must give credit to Dr. Stanley Marinoff who first published this hypothesis in 1986- long before there was data to support it.)

VVS may be initiated by an allergic reaction to a chemical irritant in the vulvar vestibule. This irritation &ndash; possibly to topical antifungals, other medications, or chemicals- causes mast cells to migrate to the vestibule. If the irritation persists, activation of mast cells leads to an uncontrolled proliferation of nociceptors in the mucosa.

This hypothesis explains why up to 80% of women with VVS complain of an acute onset of symptoms that includes burning and itching, which then progress to severe pain on touch. The pain on touch often then persists even after the initial symptoms of itching and burning disappear. Of course, further studies are ongoing to assess the validity of this hypothesis.

Treatments of VVS

Unfortunately, studies examining the possible treatments of VVS have not been as numerous as studies to identify the cause of this disease. The two most widely studied treatments for VVS are surgery (25 studies), interferon (6 studies) and biofeedback (3 studies). In addition, there are many other treatments that are being used without supporting medical literature. While this does not invalidate any of these treatments, patients and their practitioners are left with a &ldquo;lets try it and see&rdquo; approach that can be very frustrating (and potentially dangerous.)

1) Tricyclic antidepressants (amitriptyline, desipramine, nortriptyline). There have been no published studies on TCA use in VVS. However there is currently an ongoing NIH funded study examining desipramine (with and without lidocaine) for VVS. The current theory behind using TCAs for VVS is that they &ldquo;numb nerves&rdquo; and VVS is a disease of too many nerves. It is not known if treatment of VVS with TCA is curative or just palliative.

2) Lidocaine is a topical anesthetic. Traditionally, lidocaine has been prescribed to numb the vestibule enough to have intercourse. More recently, investigators have been examining the possibility that lidocaine- if applied for long periods of time may cause the nerves to regress or become permanently numb. A study published in July 2003 by Zolnoun and colleagues showed that long-term, nightly application of 5% lidocaine ointment significantly improved the ability to have

intercourse. It is not known how long this effect will last after stopping the lidocaine.

3) Estrogen: While there have been a few reported cases that estrogen can be used to treat VVS, no studies have been published. It is possible that estrogen treats VVS by numbing nerves or by improving the health of the vestibular tissue. In addition, it is possible that a woman has been incorrectly diagnosed with VVS and has instead a condition called atrophic vaginitis that is caused by a lack of estrogen. Using topical estrogen will dramatically improve atrophic vaginitis.

4) Low oxalate diet: In 1992 an organization was founded called the Vulvar Pain Foundation (VPF)

(vulvarpainfoundation.org) that was founded on the belief that the symptoms of VVS were caused by very minute crystal of oxalic acid in the urine. Oxalic acid is found in many foods, so they proposed "low oxalate diet" that limits foods that have high levels of oxalate (an organic acid). In addition to the low oxalate diet, they suggest adding calcium citrate supplements to help neutralize the acid. The VPF claims that many women have been helped with this treatment approach. Unfortunately, the only study examining this theory showed that women with VVS have the same amount of oxalate crystals in their urine as women without VVS.

5,6,7) Biofeedback, Intra-vaginal physical therapy, BOTOX: Some women with VVS -especially those with vestibulodynia and dysesthetic vulvodynia- have developed pelvic floor dysfunction (PFD). PFD is when the muscles of the pelvis become very tight and tender. This type of pain, called myofascial pain, can be very severe. Biofeedback is a technique by which you use a probe in the vagina to learn to control and relax the muscle of the pelvic floor. Intravaginal physical therapy is akin to a massage of the pelvic floor muscles to slowly stretch and relax them. BOTOX is botulism toxin that has been approved by the FDA for the treatment of wrinkles. BOTOX works by temporarily paralyzing muscle in which it is injected. Ongoing research is examining if injections of BOTOX into the pelvic floor muscles can treat PFD and thereby help VVS. I suggest that you go to Dr. Howard Glazer's website ([www.vulvodynia.com](http://www.vulvodynia.com)) for a discussion about biofeedback and Talli Rosenbaum's website ([www.physioforwomen.com](http://www.physioforwomen.com)) for an in depth discussion of intravaginal physical therapy.

8) Steroids: A small study of 15 women in 2001 examined injections of steroids combined with lidocaine for VVS in which 32% were cured and 36% were significantly improved. No data exists to show if this was cure or just temporary palliation of the symptoms of VVS. More recent evidence suggests that VVS is not caused by chronic inflammation and therefore steroids should not be the correct treatment. To date, no larger, or long term, studies have been done. The paper did not distinguish between primary and secondary VVS.

9) Interferon: Interferon (INF) is a naturally occurring biological chemical that improves the immune system and decreases inflammation. INF was first proposed as a treatment for VVS in 1989. The rationale for using INF was the hypothesis that the cause of VVS was human papillomavirus (HPV) infection. However, more recent studies have cast doubts on this hypothesis. Several studies, including a large study by this author of 214 patients treated with a series of 12 intra-vestibular injections of INF showed an overall 42% success rate. Patients were more likely to have success if they had secondary VVS for less than three years. Very few patients with primary VS were improved. As INF is a very potent mast cell inhibitor, it is possible that this is why INF may work for VVS.

10) Nitroglycerine: a small pilot study was published in 2002 that examined the affect of application of nitroglycerine cream for the treatment of vulvodynia.

No distinction was made between women with VVS and dysesthetic vulvodynia. While the majority of women had decreased pain during intercourse, most participants had headaches associated with the nitroglycerine. In addition, it is not known if this is a cure or temporary palliation of symptoms. Lastly, no distinction is made between primary and secondary VVS.

11) Antihistamines: There have not been any studies examining anti-histamines for the treatment of VVS. However some practitioners do use them as part of a treatment regimen. A similar medication, Cromolyn sodium that inhibits mast cells did not help women with VVS in a placebo-controlled trial.

12) Capsaicin: Capsaicin is the purified extracted alkaloid from red chili peppers (capsicums). This is the substance that makes chili peppers hot. The purified form capsaicin has been found to relieve pain by reducing substance P, which is found at nerve endings and is involved in transmitting pain signals to the brain. There has been only one published study about using capsaicin for VVS, but it is also being used at several referral centers including the Mayo clinic. Anecdotally, this is a very painful treatment.

13) Atropine: Atropine is a medication that affects blood vessels and has a wide range of uses in anesthesia, ophthalmology, and is even used as a treatment for "nerve gas." While there have not been any published studies using atropine for VVS, some practitioners prescribe a 2% atropine cream to be applied daily. Success rates are not known.

14) Surgery &ndash; Vulvar vestibulectomy with vaginal advancement. The theory behind surgery is that the underlying problem with VVS is that there are too many nerve endings in a very small area of tissue (about one square inch) and if the mucosa (skin) with these abnormal nerve endings is removed and replaced the healthy tissue, that this will solve the problem. There have been 25 published studies examining this procedure for VVS and 21 of the 25 have shown greater than an 80% success with the surgery. Unfortunately, surgery has gotten a bad reputation.

If surgery is not done by an expert (who has done many of these operations) there can be significant side effects including infections and scar tissue formation. In addition, if not enough tissue is removed, there can be areas that still hurt after surgery. In fact, initial success rates for this operation were only about 50-60%, but continued advance in surgical technique has pushed the success rate to almost 90%. In one study, more than 85% of women who had the surgery would recommend the surgery to another woman similarly affected.

Many authors suggest that surgery should only be used as a "last resort." However, more recently, as the success rates are now so high, many experts in the field suggest surgery after failing 1-3 alternatives discussed above. Lastly, many experts also believe that because primary VVS is probably a congenital operation, surgery is the only

treatment that will work for primary VVS. 02/2005    buying viagra without prescription fast