

# Shingles/Post-Herpetic Neuralgia

## Early Treatment Options to Prevent

### Post Herpetic Neuralgia (PHN)

In this article we describe some of the treatments available today for shingles and post-herpetic neuralgia, a condition that can cause severe incapacitating pain. As with most medical conditions, prevention is the best course. Let's begin with some background on these conditions. Shingles or Acute Herpes Zoster (AHZ), is a reactivation of the varicella-zoster virus (commonly known as chicken pox), and is usually a self-limited disease, but in some patients AHZ can result in a long term, severely painful condition called Post Herpetic Neuralgia or PHN. It normally involves a single dermatome (nerve), and is most commonly seen in the thoracic region (chest), or in the trigeminal nerve distribution (second most common area is V1 - the facial region above the eye), but shingles may involve any nerve/dermatome in the body. The incidence of PHN increases with age and the group that is at highest risk are those over 70, where the incidence has been reported to be greater than 50%.

PHN is caused by nerve damage that follows the nerve inflammation associated with AHZ infection. Patients who develop PHN typically experience a severe burning pain with light touch sensitivity such that even wearing clothes over the affected area can be difficult. PHN can last for more than 10 years and can be extremely debilitating, resulting in a drastic reduction in quality of life. Unfortunately, once established, PHN is very difficult to treat and so prevention of this condition is extremely important. Today, vaccines that help improve immunity and decrease the incidence of AHZ are being more widely used and are recommended for at risk individuals. Routine therapy for shingles outbreak, including the use of Acyclovir, steroids, and oral analgesics, does not significantly change the chance of developing PHN after acute infection. Prevention through vaccination, by decreasing the incidence of acute herpes zoster, is effective at decreasing the incidence of PHN, but does not appear to change the risk of developing PHN if an outbreak of shingles should still occur.

You cannot have shingles if you have never had chicken pox because shingles is caused by the reactivation of the varicella-zoster virus which resides in the nervous system of anyone who has ever had chicken pox. Most patients who develop shingles have a weakened immune system that predisposes them to this occurring, i.e. - chemotherapy, AIDS, stress (yes, stress can lower your immunity), older age, and many other causes. Following primary varicella-zoster infection (chicken pox), live virus particles hibernate in the cell body of spinal nerves, just outside the spinal canal, called the dorsal root ganglion. Once the virus reactivates, live virus particles begin to travel down the nerve (axon) and virus is released where the sensory nerve terminates at the level of the skin causing a blistering eruption; the rash distribution tells your physician which nerve has been affected. The pain from shingles usually precedes the blistering skin eruption by several days. The fluid within the blisters contains live virus particles and that is why patients with shingles are highly contagious to immunocompromised patients (infants, those receiving

chemotherapy, etc.) or those without prior exposure (immunity) to varicella. Note: Chicken Pox in an adult can be very serious as they are more prone to develop viral pneumonia than kids; this form of pneumonia can be deadly.

Shingles typically occurs in a single nerve and primarily involves the sensory component of the nerve, but in severe cases motor function can also be affected (although typically this is a sensory nerve phenomena). I have found that when there is motor involvement these patients often have secondary nerve issues such as chronic nerve damage or compression of the effected nerve at the spinal level.

A consequence of AHZ is nerve inflammation, edema formation, and subsequently compromise of the vascular supply to the nerve from the edema. Loss of blood supply can result in neuronal death and this effects nerves based on their size, with larger nerves more likely to experience cell death/neuronal loss. Note: older individuals are more prone to PHN because they are more likely to have compromised blood flow than someone who is younger (strokes and heart attacks typically occur when people are older, not younger). Cross section of a nerve from a patient who has developed PHN reveals preferential sparing of small fibers and predominant loss of large diameter neurons (larger nerves are more sensitive to the compromise in blood flow). These large diameter fibers are involved in the transmission of normal sensory function. Small diameter neurons are involved in the transmission of pain. At the spinal level, large diameter neuron firing acts to inhibit small diameter (pain nerve) fiber input at the spinal level. When this inhibitory influence is lost, it leaves unopposed small fiber firing which leads to the increased perception of pain at the central level. The longer this goes on, the greater the central nervous system 'wind-up' and the more difficult the condition is to treat.

Based on the mechanism of this injury and my extensive review of the literature, I have developed a highly effective treatment to **PREVENT** PHN. Not every patient with AHZ requires treatment, but those at high risk for PHN should consider early treatment, as the best outcome requires early intervention. To prevent PHN in these patients, early treatment is recommended with a series of sympathetic blocks, typically through an epidural injection approach. To achieve this, an epidural catheter is inserted with its tip placed at the involved level, this way repetitive injections can be performed with a single needle insertion. Epidural blockade results in both immediate pain relief from the local anesthetic, but also localized dilation of the vasculature to the area (from sympathetic blockade). This results in increased blood flow to the involved nerve, which prevents cell death and thereby limits the extent of permanent nerve damage.

Patients at high risk are those that are older than 65-70 years of age and younger patients with co-existing vascular disease (i.e. – a patient who may already have compromised blood flow). While others can progress to PHN, these are the groups for which early treatment is recommended. For lower risk patients who develop shingles, if the painful symptoms have not abated within 3-4 weeks, early treatment should be strongly considered, especially for those with more significant symptoms. Those with more severe pain during the initial AHZ outbreak seem to be more prone to develop PHN. Time sensitivity is important because late treatment is less effective and/or may require more aggressive treatment.

The longer the period of time from the onset of shingles to the start of treatment, the more difficult the condition is to treat and the less optimal the long-term prognosis; late treatment is associated with more permanent nerve damage. Treatment instituted in the first 2 weeks following the initial onset of symptoms prevented PHN in up to 95% of patients, treatment instituted in the first 4 weeks was over 90% effective at preventing PHN and before 2 months it was approximately 80% effective. After 2 months of pain, treatment success with simple nerve blocks drops off drastically and falls to approximately 20%, presumably due to the extent of permanent nerve damage that has already occurred.

When patients fail this type of therapy, there are other interventions that can be tried, but the success rate is more limited and these treatments often require long term management with either medications or more invasive therapies (many of these treatment options are listed below). Early treatment and prevention of PHN results in a better long-term outcome because it prevents the chronic pain associated with PHN and the need for chronic treatment. Anesthetic blocks will treat the pain, speed healing of the lesions, decrease the need for antiviral medications, minimize narcotic usage, shorten the duration of the painful and infectious period, and most importantly, help prevent the development of PHN.

### **Options for treatment of longstanding PHN**

- 1) Oral Medications—Anti-seizure, antidepressant, and other neuropathic medications.  
Note: narcotics are not typically effective for nerve pain.
- 2) Topical medication creams which must be repetitively applied 3-4 x/day including various combinations of local anesthetics, amitriptyline, gabapentin, NSAID's, baclofen, etc.) —Available from a compounding pharmacy –
- 3) Capsaicin (Zostrix)—Application is associated with a temporary increase in pain and it must be used 3-4x/day (in my experience poorly tolerated). This may be more effective when applied as a topical patch in very high concentration (Qutenza).
- 4) Anesthetic blocks—May be worth a trial even though the long-term success rate is significantly lower, try less invasive approaches first for established PHN.
- 5) Nerve Lesioning—Radiofrequency or cryoanalgesic nerve blocks may provide longer term relief when a diagnostic local anesthetic injection helps.
- 6) Spinal Cord Stimulation, DRG Stimulation, or Peripheral Nerve Stimulation—can be trialed. When effective during a trial, permanent implantation of a stimulator device is required for long term management of PHN.
- 7) For recalcitrant cases associated with significant pain, Neurosurgical destructive lesioning has been tried in selected cases.

(Listed in order of least invasive to most invasive)