Postherpetic neuralgia

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Introduction

POSTHERPETIC neuralgia is one complication of herpes zoster (shingles) and was the presenting diagnosis in 11 of 144 new patients attending the pain relief clinic at Edgware General Hospital in 1984. Herpes zoster is a secondary infection, caused by the varicella/zoster virus, which is characterized by pain and a vesicular eruption limited to the dermatome innervated by a single spinal or cranial sensory ganglion.[1]

It is believed that during an attack of chickenpox the varicella virus passes from the skin lesions to the cutaneous sensory nerve endings and is then transported to the sensory ganglion where it remains latent, but with the capacity to revert to full infectivity.[2] If reactivation occurs it usually does so in older adults, over two-thirds of patients with herpes zoster being more than 50 years of age. Malignant disease and immunosuppressive therapy (radiation, antimetabolites, antilymphocyte serum and corticosteroids) predispose patients to herpes zoster.[3-5] Once the virus is reactivated it multiplies within the sensory ganglion and spreads antidromically down the sensory nerve to the skin.[6]

Herpes zoster occurs in 20–50% of patients with Hodgkin's disease with the highest incidence in those undergoing combined radiotherapy and chemotherapy.[4]

Definition

Postherpetic neuralgia has been defined in a variety of ways. Pain persisting after the skin crusts of the herpetic lesions have fallen off was the original definition.[7] This was later modified to pain of greater than 30 days from the formation of the vesicles.[8] Further definitions include pain at six weeks,[3] eight weeks,[9] six months[10] and one year[11] after the initial eruption. Other definitions have included pain severe enough to interfere with daily routine,[8,12] and disturbed sleep and no relief from simple analgesics following acute herpes zoster.[13]

Features

Postherpetic neuralgia appears to consist of two elements. First, there is a continual burning or aching pain. Secondly, there is an associated uncomfortable sensitivity of the skin (dysaesthesiae); the patient may be unable to withstand the lightest pressure either on or near to the affected area.[14]

Incidence

The incidence of postherpetic neuralgia varies according to the definition considered but is between 18%[12] and 35%[3] of all patients suffering from herpes zoster. The incidence is about 45%[3] for patients aged more than 50 years and about 50% for those aged more than 60 years.[11] Only 5% of patients with postherpetic neuralgia are less than 50 years of age.[3] The distribution between the sexes is equal.

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It is possible that those patients who suffer severe acute pain with herpes zoster are more liable to develop postherpetic neuralgia.[10] Distribution of the disease in the trigeminal area is often more severe and prolonged than in other nerve areas.[11] Although the neuralgia usually improves with time it can remain a disabling lifelong disease.[11]

Underlying mechanism

The cause of postherpetic neuralgia is obscure. Pain in the acute phase is presumed to be due to an abnormal discharge in the dorsal horn, secondary to inflammation of the dorsal root ganglion. Chronic pain may be associated with postinflammatory fibrosis in the root ganglion but appears to be perpetuated by central mechanisms.[15] There is a preferential destruction of the large myelinated afferent nerve fibres in this condition.[16]

Postherpetic neuralgia supports the gate theory of pain. Part of this theory is that a neural mechanism in the dorsal horn of the spinal cord acts like a gate which can increase or decrease the flow of nerve impulses from the periphery to the central nervous system. The theory implies that the stimuli via the large diameter afferent fibres close the gate and those from small diameter fibres open the gate and so influence the perception of pain. The loss of the larger fibres in postherpetic neuralgia leaves a relative excess of smaller fibres functioning and hence an 'open gate'.[16]

Why should the neuralgia occur more often in older people? It may be that the neurophysiological responses to injury are decreased in the elderly[1] but it is also worth noting that the proportion of large fibres to small fibres decreases throughout life and hence there is a tendency for an 'open gate' in the aged.[14]

Clinical features

Examination of a patient with postherpetic neuralgia shows evidence of scarring in the skin over the affected dermatome. There is occasional spread across the midline and this is seen more often posteriorly. The scars themselves are anaesthetic and the areas of skin between them are especially sensitive. Often the patient will tolerate firm pressure on these areas but not a light touch.[14]

Prevention of postherpetic neuralgia

There is no known reliable preventive treatment for postherpetic neuralgia. Clincial trials are difficult as many patients who have pain in the acute phase of herpes zoster recover spontaneously. Early treatment of groups who are at risk is now advocated and agents which may be helpful are listed in Figure 1.

Topical agents

Idoxuridine shortens the course of acute herpes zoster and also reduces the pain of the acute episode.[17] Juel-Jensen found that idoxuridine when applied as a 40% solution in dimethyl sulphoxide paste prevented postherpetic neuralgia in 32 patients studied, 23 of whom were aged 50 years or more.[18] It is stated that to be effective this paste should be applied four times a day for the first four days as soon as the vesicles appear.

Systemic agents

The use of corticosteroids remains controversial in the treatment of acute herpes zoster. It would seem logical to use these drugs to inhibit the acute inflammation which appears to be responsible for the stimulation of the pain pathways. In 1964 Elliot

treated 16 patients with acute herpes zoster with prednisone. Using a dose of 60 mg per day and decreasing the dose over three weeks he found that no postherpetic neuralgia occurred.[19] The best known controlled trial was conducted by Keczkes and Bashear who gave 20 patients over 50 years of age with acute herpes zoster but otherwise healthy 40 mg of prednisolone per day and decreased the dose over one month. Only three patients developed pain and resolution in this group occurred in six months.[15] Eaglestein also found that the incidence of postherpetic neuralgia decreased from 73% to 30% following the use of oral steroids.[9] However, many doctors consider the risk of systemic viral dissemination following the use of steroids to be too great to justify their use,[18,20,21] particularly for those patients with already depressed immunity.

In 1973 Galbraith used the antiviral agent, amantadine, in a double blind placebo controlled trial and found that a reduced proportion of treated patients experienced severe pain.[22] In another trial carried out by Galbraith 67 patients aged greater than 60 years who presented with acute herpes zoster of duration less than 72 hours from vesicle formation, were given 100 mg of amantadine twice daily. This trial showed that the duration of the pain experienced by the treated patients was significantly shorter than for the untreated patients and therefore this implies that amantadine is effective against postherpetic neuralgia.[23]

One trial studying the use of cyclic adenosine monophosphate (AMP) has been reported.[24] Patients with acute herpes zoster have an abnormally low level of cyclic AMP in their serum. In the trial 130 patients were given injections of cyclic AMP (1.5–2.0 mg per kg) on alternate days for between 12 and 15 injections. Of these patients, 107 were aged 50 years or more and no toxicity or postherpetic neuralgia was reported after a two-year follow up.

Merigan reported a significantly diminished severity of postherpetic neuralgia following treatment with interferon.[25]

Drugs which appear to be ineffective in the prevention of postherpetic neuralgia include acyclovir[26] and L-dopa (L-dihydroxyphenylalanine).[27]

1.	*Topical agents*	Idoxuridine in dimethyl sulphoxide paste
2.	*Systemic agents*	Corticosteroids
		Amantadine
		Cyclic AMP
		Interferon
3.	*Nerve blockade*	Local infiltration
		Epidural
		Sympathetic
4.	*Percutaneous nerve stimulation*	

Figure 1. *Possible methods for preventing the establishment of postherpetic neuralgia.*

Nerve blockade

Local anaesthetics used alone or in combination with corticosteroids have been used extensively for nerve blockade and are claimed to prevent postherpetic neuralgia and reduce the acute pain.[28] In 1971 Epstein reported an incidence of postherpetic neuralgia of approximately 4% following subcutaneous injections of procaine and triamcinolone into the inflamed skin.[29] It is not known why an injection of a local anaesthetic should alter the course of an infectious disease. Recently Riopelle studied the use of bupivacaine for stellate ganglion, epidural, and peripheral nerve blockade.[10] He also used bupivacaine with triamcinolone for local infiltration. He treated 72 patients, of whom 40 were more than 60 years of age, with various nerve blocks and found that in most patients pain abated within six months irrespective of the treatment. Riopelle considered nerve blocks to be ineffective as a method of prevention.

Since the acute disease involves the spinal ganglion it would seem logical to inject drugs directly into the organ involved. For this reason epidural blockade has been extensively used. Perkins gave epidural injections of bupivacaine (0.25%, 8–12 ml) with or without methylprednisolone (80 mg) to 12 patients and found that postherpetic neuralgia was prevented if the block was performed within three months of the episode of acute herpes zoster.[30] Recently, Schreuder treated 113 patients with acute herpes zoster of less than 10 weeks duration with epidural injections of bupivacaine (0.25%, 6–8 ml) and methylprednisolone (80 mg). This provided immediate relief of pain and prevented postherpetic neuralgia in 100% of cases providing the injection was adjacent to the nerve ganglion. Only 20 of the patients were less than 50 years of age[13] and follow-up was for one year. However, no indication of how pain was assessed was given in the paper.

Inflammation of the posterior root ganglion may cause an increased sympathetic vasoconstriction segmentally and indirectly enhance any pain present. Sympathetic blockade of the ganglion may interrupt the sensory afferent impulses through the sympathetic fibres and produce a vasodilation which may stop any chronic inflammatory process occurring.[14] Colding treated 483 patients, of whom only 55 were less than 40 years of age, with regional sympathetic blocks using 1% lignocaine with noradrenaline in 5–10 ml injections.[31] Only 5% of the patients developed postherpetic neuralgia in his series. Similarly Marmer using sympathetic blockade in patients with acute herpes zoster found no postherpetic neuralgia in a follow up of between one and four years.[32] However, Lipton remains unconvinced of the worth of sympathetic nervous blockade in the treatment of the acute phase of herpes zoster.[14]

Percutaneous nerve stimulation

There are few reports of percutaneous nerve stimulation for the prevention of postherpetic neuralgia. Good improvement in the acute pain and no postherpetic neuralgia were reported in a small study.[33]

The prevention of postherpetic neuralgia is probably best achieved by topical idoxuridine. Nerve blockade with a suitable local anaesthetic agent, for example 1% lignocaine, may also be helpful. The use of oral systemic agents is still not commonly accepted and while amantadine may be beneficial the risks of steroids do not justify their use.

Treatment of established postherpetic neuralgia

There is no treatment that reliably relieves the pain of postherpetic neuralgia. Treatment will in general be of long duration and, while many doctors do not expect a cure, there are a variety of measures available to assist in the alleviation of the pain in this condition. Figure 2 lists the methods available for the management of established postherpetic neuralgia.

Many patients are elderly people living alone, who perhaps have a tendency to worry about and dwell upon their pain. While the pain is very real and often debilitating it is wise to encourage activity and outside interests.

Explanation and support

Initially the disease process should be explained to the patient and support should be given. It should be stated that many people suffer from the disease and that in many cases it resolves spontaneously.

1. *Explanation and support*
2. *Simple analgesics, narcotic analgesics*
3. *Antidepressants, major tranquillizers, anticonvulsants*
4. *Ultrasound therapy*
5. *Percutaneous nerve stimulation*
6. *Nerve blockade*
   a) Using local anaesthetics and corticosteroids
      Subcutaneous
      Direct
      Sympathetic
      Epidural
   b) Intrathecal — phenol/alcohol
7. *Acupuncture*
8. *Topical applications*
   Carbon dioxide cryocautery
   Ethyl chloride spray
   Trichlorofluoromethane 85% in dichlorofluoromethane 15% spray
9. *Vitamin E, vitamin B12*
10. *Neurosurgical intervention, percutaneous cordotomy*
11. *Vincristine iontophoresis*

Figure 2. *Possible methods for the management of established postherpetic neuralgia.*

Simple analgesics, narcotic analgesics

Simple oral analgesics may have been prescribed unsuccessfully by the general practitioner prior to referral to a pain relief clinic. Simple analgesics are rarely successful in the management of postherpetic neuralgia.[3] Opiates are occasionally indicated, but the risk of addiction is real. Analgesics may relieve the pain but they have no effect on dysaesthesiae.[14]

Antidepressants, major tranquillizers, anticonvulsants

Oral psychotropic drugs have been used with some success. Amitriptyline was shown in a recent trial to provide effective pain relief for two-thirds of patients.[34] The dosage used was 25 mg at bedtime gradually increasing to 70 mg depending on the side-effects experienced. Watson and colleagues suggest that the action of the drug is different at these two levels. Monoamine-oxidase inhibitors have also been suggested and 15 mg of phenelzine three times per day has been used with appropriate dietary restrictions.[33] Major tranquillizers have been used alone or in combination with the above drugs. Chlorprothixene (50–100 mg intramuscularly followed by 50 mg orally at six hourly intervals for 10 days), fluphenazine (4 mg per day for 14 days) and pericyazine (2.5–5 mg twice daily and 10–25 mg at night, continuously) have been used with some success.[33,35] Carbamazepine in anticonvulsant doses has been suggested for the lancinating pain but there is little evidence to support the success of its use.[21] Similarly it has been suggested that phenytoin may be beneficial to some patients.[33]

Ultrasound therapy

This has received widespread publicity but there is little scientific evidence that it is successful. One study of eight patients receiving ultrasound therapy showed that seven of these patients found relief but one patient found that it aggravated the pain.[36] Interestingly one of the patients who was cured had a 14 year history of the neuralgia. The treatment was given daily and each patient received between six and 12 applications. Ultrasound therapy is normally provided in outpatient departments by physiotherapy staff.

Percutaneous nerve stimulation

The use of percutaneous nerve stimulation is based on the theory that electrical pulses stimulate the larger low threshold nerve fibres and 'close the gate'. Haas reported that of 11 patients with postherpetic neuralgia, of more than three weeks duration, nine benefited and two were made worse by percutaneous nerve stimulation.[33] Nathan and Wall reported good results for 11 out of 30 patients.[16] The electrodes of the stimulator are placed on the painful areas and a stimulus applied until a tingling and relatively pleasant sensation occurs. Initially, treatment lasts between half-an-hour and one hour, spread over three to four applications per day. The advantages of the machine are that it is small, portable and can be used in outpatient departments. The disadvantages include a failure to understand and use the machine.

Nerve blockade

Local anaesthetics with and without corticosteroids have been used for nerve blockade. Epstein found that injecting triamcinolone in lignocaine subcutaneously produced relief in many cases of postherpetic neuralgia and he confirmed his findings by injecting triamcinolone in saline subcutaneously into the lesions.[37] Repetitive direct nerve blockade using the intercostal and paravertebral routes has been advocated to relieve the pain acutely and sometimes permanently.[38] Colding in his large study treated 67 patients with postherpetic neuralgia using repetitive sympathetic blocks and found that 50% of the patients felt relief or an improvement in their condition.[31] Schreuder has reported the cure of a 93-year-old patient with a five-year history of postherpetic neuralgia using epidural injections of bupivacaine and methylprednisolone[13] although this treatment was not advocated in a report by Perkins.[30] Intrathecal blocks have been suggested to be of some use. Phenol and alcohol have been used but there are technical difficulties associated with these blocks.[14] Many specialists believe that any form of destructive lesion is contraindicated and may well make the pain worse.

Acupuncture

This has been claimed to be successful in eight out of 20 cases of postherpetic neuralgia in one series and it has been suggested that the low incidence of postherpetic neuralgia found in China may be attributed to acupuncture.[39] The use of acupuncture has been supported[40] but the results of a recent study by the original advocators were not favourable.[41] In this trial Lewith and colleagues performed a single blind randomized controlled study of auricular and body acupuncture compared to placebo treatment (mock percutaneous nerve stimulation) in 63 patients and found no difference between the results.

Topical applications

Local applications of cold sprays and dry ice have been used. A cryocautery using a stick of solid carbon dioxide applied for one minute to hyperaesthetic areas afforded excellent relief for five out of 14 patients and good relief for a further five. Repeated applications, often as many as eight, for between two and three weeks, were necessary to ensure success.[42] Ethyl chloride spray has been used with some success,[43] but a safer alternative, 85% trichlorofluoromethane in 15% dichlorofluoromethane spray is now preferred as a topical agent.[14]

Vitamin E, vitamin B12

It has been claimed that vitamin E will almost completely control the pain for nine out of 13 patients suffering from postherpetic neuralgia.[44] However this result was not confirmed in a small study comparing vitamin E taken orally and topically.[45] Vitamin B12 has been suggested, but there appears to be no evidence to support its use.[14]

Neurosurgical intervention

Should all other treatment fail and suicide become a real possibility then there is an argument for neurosurgical intervention and perhaps a technique such as percutaneous cordotomy should be performed.[14]

Vincristine iontophoresis

Recently there has been interest in the use of vincristine iontophoresis although further controlled trials will be necessary to see if it is to have an established place in the management of this condition. Vincristine is thought to block information travelling proximally along the axon — 'axoplasmic transport inhibition' — and thereby relieve pain. Iontophoresis involves passing of an electric current across the skin and since vincristine is positively charged it is carried with the current to the neural tissue. Rashes have been reported following the treatment, which is demanding with 40 minutes of treatment needed daily for up to four weeks before beneficial effects are seen.[46]

Experience of the Edgware General Hospital Pain Clinic

The definition of postherpetic neuralgia held by the clinic was of persistent pain for more than eight weeks from the onset of the vesicles. The results from the treatment of 11 new patients with this neuralgia confirm the difficulty of managing this condition.

Five men and six women were treated and their age range was 58–82 (mean 73) years. The sites of the lesions were trigeminal nerves (four patients), thoracic nerves (four) and sacral nerves (three). The duration of the condition ranged from three months to 10 years from onset of the neuralgia to referral at the clinic; however, seven patients had neuralgia of between one and three years duration. Three patients considered themselves cured following treatment. Nerve blocks of local anaesthetic injected into the supraorbital nerve effected two of these cures and the other patient was cured by 20 sessions of ultrasound therapy carried out by the physiotherapy department. Four of the remaining patients received moderate help from percutaneous nerve stimulation carried out at home and ultrasound therapy carried out in the outpatient department and four found all the treatment tried ineffective in relieving the symptoms. These four patients were given trials of simple analgesics, antidepressants, tranquillizers, acupuncture, local sprays and nerve blocks of local anaesthetic without success and ultrasound therapy and percutaneous nerve stimulation were found to aggravate the pain.

Conclusion

The results of treating postherpetic neuralgia are poor. There is no known cure and the most effective management involves preventive care of patients who are at risk. Once the neuralgia is established the best initial treatment appears to involve treatment with simple oral analgesics and then a trial of oral psychotropic drugs. This should be followed by nerve blockade with local anaesthetics and corticosteroids. If all these treatments are unsuccessful then ultrasound therapy and percutaneous nerve stimulation should be tried. If these fail then acupuncture, topical spray applications and vitamins can be tried. If the pain is still unabating and the patient's standard of life is compromised then perhaps narcotic analgesics should be used and opinion sought as to whether a percutaneous cordotomy should be performed.

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Continuity of care in general practice

Although continuity of care in the sense of consistency seems inherently desirable, the case cannot always be made for striving for personal care in all situations in general practice. Therefore, to deliver good quality consistent care in general practice, we need first to develop agreed standards for the management of common and important conditions. To help us adapt these standards to the individual patient we need adequate records. These must contain details of diagnoses, tests and prescriptions and also perhaps items such as patients' beliefs and concerns and the doctor's advice, which are increasingly recognized as important in patient care.

Secondly, it may be helpful to regard personal continuity as an aid to consistency rather than its essence. In this case far more needs to be known about which patients feel it is particularly important, and why; also about how personal continuity is actually achieved by patients faced with appointment systems and receptionists, what priority it is given compared with other everyday factors and how the disadvantaged patient who is unable to plan ahead may best be helped. After all, it is possible that those least able to organize their own lives might benefit most from seeing the same doctor.

Thirdly, a clearer understanding of the possible benefits of personal continuity is needed. At each end of the life-cycle these benefits are clear. Both maternity patients and those terminally ill often get personal care even outside normal working hours. Is it possible to demonstrate better outcomes for patients who are less obviously special? Easily measurable improvements in morbidity may be difficult to demonstrate in general practice but we may learn more by studying a wider range of outcomes, including patients' knowledge and understanding of their problems and how these interact with their lives.

Finally, in any assessment of a process such as continuity of care it is important to be aware of the costs while counting the benefits. Perhaps the doctor who knows his patient well does not notice the insidious onset of myxoedema. But perhaps an unfamiliar doctor does not recognize a patient's distress and so fails to provide comfort. Before appropriate guidelines can be drawn, there is a need for much more knowledge and a deeper understanding of the various facets of continuity of care.

Source: Freeman G. Continuity of care in general practice: a review and critique. *Family Practice* 1984; **1:** 245-252.