

Chapter 29

Medical Management of Trigeminal Neuralgia

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ABSTRACT

The first-line treatment of trigeminal neuralgia can be very effective, but side effects are often difficult for patients to tolerate. This chapter is a guide to the broad selection of medical therapies currently available for the treatment of trigeminal neuralgia which includes oral therapies and other non-surgical methods of treatment such as IV medications, nasal sprays, topical ointments, and injections. The discussion of each treatment includes discussion of its evidence in current literature, its proposed mechanism of action, its dosing and appropriate setting for clinical use, and its side effect profile.

INTRODUCTION

The aim of all trigeminal neuralgia therapies is to modulate the hyperexcitable state of the trigeminal afferents to relieve pain. The first-line treatment of trigeminal neuralgia is oral medication. These medications can be very effective for the treatment of trigeminal neuralgia, but their side effects are often difficult for patients to tolerate. If a patient fails first and second line oral therapies, and if the patient is not a surgical candidate, then the wide array of next-line oral medications and combination therapy should be considered. In addition, there has been exploration into other non-surgical and non-invasive methods of treatment such as IV medications, nasal sprays, topical ointments, and injections. This chapter is a guide to the broad selection of medical therapies currently available for the treatment of trigeminal neuralgia. For each treatment, this chapter will cover multiple aspects including 1) discussion of its evidence in current literature; 2) its proposed mechanism of action; 3) its dosing and appropriate setting for clinical use; and 4) its side effect profile.

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PART 1: FIRST-LINE THERAPY

Treatment of trigeminal neuralgia should begin with either carbamazepine or with oxcarbazepine, as agreed upon by the American Academy of Neurology and the European Federation of Neurological Societies (Cruccu & Truini, 2013). When a patient does not respond to either carbamazepine or oxcarbazepine, or if the patient cannot take these medications because of contraindications or intolerable side effects, then the patient is defined as having refractory trigeminal neuralgia (Cruccu, 2013).

Carbamazepine

Carbamazepine has level A evidence and is the first line therapy for initial and long-term treatment of trigeminal neuralgia (Gronseth et al., 2008; Chesire, 2015). Patients who respond to the medication will often experience pain relief within a few days of starting carbamazepine. However, this medication should be used cautiously, especially in the elderly, because of its many side effects.

Carbamazepine's mechanism for pain relief is unknown. It is an iminostilbene whose structure resembles tricyclic antidepressants and phenytoin. One hypothesis is that it decreases conductance in sodium channels, which results in stabilization of hyper excitable membranes and inhibits ectopic discharges (Tremont-Lukats, Megeff, & Backonja, 2000). The medication usually reaches peak concentration in 2-6 hours. After 2-4 weeks of starting carbamazepine, the half-life decreases from about 30 hours to 13-17 hours because of auto-induction. The drug is excreted by the liver. As an anticonvulsant, the therapeutic drug level is 4-12 mcg/dl. In the context of treating trigeminal neuralgia, there is poor correlation between pain relief and drug concentration (Green & Selman, 1991).

Carbamazepine is the best studied medical treatment for trigeminal neuralgia. There are four, high quality (Class I or II), placebo-controlled studies demonstrating that carbamazepine can, not only reduce the frequency and intensity of painful episodes, but is equally efficacious for spontaneous and trigger-evoked attacks (Gronseth et al., 2008). The dose range for carbamazepine in these studies was 300mg to 2,400mg daily. The treatment response in these trials were very strong, with 58-100% of patients on carbamazepine experiencing complete or near complete pain control, compared to 0-40% of patients on placebo experiencing complete or near complete pain control (Jorns & Zakrzewska, 2007). However, there has only been one study that evaluated the long-term efficacy of carbamazepine over a 16-year period. In this study of 146 patients, there was an initial reported success in 60% of patients. But by 5-16 years, only 22% of participants were still experiencing benefit from carbamazepine. About 44% of patients required additional or alternative treatment (Taylor, Brauer, & Espir, 1981). Observational studies have shown that there is a decrease in efficacy over time, but this may be related more to increased severity of the disease rather than development of tolerance (Zakrzewska & Patsalos, 2002).

Carbamazepine should not be used in patients with a history of bone marrow depression or hypersensitivity to tricyclic compounds such as amitriptyline and nortriptyline (Ferrell & McLeod, 2008). Also, before starting carbamazepine, it is imperative to evaluate the patient's risk for potentially developing carbamazepine-induced Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). Though the occurrence is rare, SJS and TEN are two forms of the same life-threatening skin disease that can cause rash, skin peeling, and sores on the mucous membranes. The HLA-B*1502 allele is highly associated with carbamazepine-induced SJS and TEN. The association has been found mostly in the Han Chinese. The association has not been found in Caucasian patients. The HLA-B*1502 allele frequency is 10.2% in Han Chinese living in Asia and 5% in Asians living in North America (Ferrell & McLeod,

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