Trigeminal Neuralgia Treatment: A Case Report on Short-Term Follow up After Ultrasound Guided Autologous Platelet Rich Plasma Injections.

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**3420 Conflict of Interest:** The patient is a family member and works as practice manager for the author. The treatment was carried out on the basis that all non-invasive options were exhausted with no symptom relief, it was a very safe option compared to other available procedures and the author is a trained neurointerventional radiologist who was the only person in the region who could offer this treatment.

**Additional Files:**
- Manuscript
- Appendix
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Introduction

Autologous Platelet Rich Plasma (PRP) contains natural growth factors and is increasingly used in various painful musculoskeletal conditions. This report is on the use of autologous PRP in a case of Trigeminal Neuralgia (TN).

Case Report

In August 2011 a 40 year mother of two young children, part time manager, complained of tingling and numbness around the right nasolabial fold and episodes of vague pain around the right lower jaw. Within a few days there was rapid escalation to lancinating pain with electric shock like symptoms to the right side of the face, radiating to the right orbit, right hemivertex and right occipital region without warning. In a matter of weeks symptoms were severe enough during the day and night to prevent normal activities of daily living, sleep and work. She lived in constant fear that pain could strike at any time. Her social life was non existent as she had to retire to bed at 6pm and could not report to work. Acupuncture was tried on two occasions and this provided relief for about half an hour. Several months prior she had undergone dental filling followed by malocclusion that required correction of the dental filling. For a couple of years she had been using a peroral device to prevent grinding of teeth during sleep. Since the onset of TN she stopped using this device.

An MRI of the brain with thin section images showed suspicion of neurovascular conflict around the right trigeminal nerve but there was no compressive lesion. There was no evidence to suggest demyelination elsewhere in the brain. Following neurology review a diagnosis of TN was made and oral Gabapentin was commenced with increasing doses. In a few weeks she reported memory problems and a general lethargy upon increasing doses of Gabapentin to achieve symptom control. Therefore oral Tegretol 100mg was commenced under the supervision of a neurologist after gradually weaning Gabapentin. She developed urticaria with small skin rashes whilst on Tegretol without any definite features to suggest Steven Johnsons Syndrome. Symptoms of lancinating pain around the right nasolabial fold persisted. She was reluctant to increase the dose of Tegretol due to possible worsening side effects and fear of Steven Johnsons Syndrome. Oral high dose methyl prednisolone and Famcyclovir was commenced for two weeks. She had right patellofemoral chondromalacia and had good relief from autologous Platelet Rich Plasma (PRP) knee joint injections in the past. She requested PRP treatment for TN and provided full informed consent after explanation of small risks, lack of published evidence and unknown outcome of the procedure. Between October and December 2011 she underwent five injections of PRP as per the Doss Trigeminal Neuralgia Treatment (DTNT) protocol (see appendix) in the clinic with each procedure taking about half an hour. She was discharged immediately after each procedure and resumed normal activities without any need for recovery. She maintained a pain diary for most days from the start of symptoms. Since the PRP injections, she has been on 50 mg of Tegretol twice a day for about 3 months. She continued with oral vitamin, omega three fatty acid supplements during the follow up period. For 6 months post PRP, she has had a normal social and work life with resolution of symptoms and no clinical features to suggest peripheral nerve damage.

Discussion

Trigeminal Neuralgia (TN) is the most painful condition known to man and severely affects quality of life in some patients. The mechanism of pain production is controversial. However there is a view that TN is typically caused by a dysfunction in the peripheral nervous system with a lesion of the trigeminal root or the nerve itself. One theory suggests peripheral nerve injury or disease of the trigeminal nerve increases afferent firing probably by ephaptic transmission between afferent unmyelinated axons and partially damaged myelinated axons. Over time there is retrograde degeneration of the ganglion cells and failure of central inhibitory mechanism (1). Irritation of
the trigeminal root by blood vessel, aneurysm or a mass may occur at the level of the pons. Rarely patients with multiple sclerosis present with TN due to demyelination. Trauma or prior surgical procedures to the face have been associated with TN type symptoms and some authors refer to this as Atypical TN, Trigeminal ‘Neuropathy’ or Post Traumatic TN. This is seen following cranio-facial trauma, dental trauma, sinus trauma and post rhizotomy (used for treating TN). This is due to permanent damage to the trigeminal nerve and secondary hyperactivity to the trigeminal nerve nucleus (2).

Animal studies have shown retrograde degeneration of trigeminal ganglion cells is related to degeneration of trigeminal afferents in the brain stem following transection of the infraorbital nerve (3). This suggests that injury to a distal branch of the trigeminal nerve may predispose to pathological changes of the proximal trigeminal nerve and ganglion. Treatment of TN by radiosurgery results in focal axonal degeneration of the trigeminal nerve in a primate model. This has implications in patients with TN treated by radiosurgery (4).

Treatment of neuropathic pain is an important unmet medical need because this pain is often refractory to many medical interventions (5). An important factor in the development of neuropathic pain is a dysfunction in the activity of peripheral nerves. The role of abnormal sensory input from the peripheral nervous system in TN is a subject of study in a NINDS funded study. NIH funded research examines functional and chemical changes in sensory neurons in the peripheral and central nervous systems and evaluates the roles of nerve growth factor and sympathetic nerves in neuropathic pain (6).

Modulating the activity of peripheral nerves has shown potential for normalizing neural activity with reduction in signs of neuropathic pain in animal models and in clinical studies (5). In a nerve crush rat model study, nerve regeneration and restoration of erectile function was shown in rats that were treated with activated PRP application to the crushed cavernous nerve (7). PRP contains insulin growth factor-1 (IGF-1) and vascular endothelial growth factor (VEGF). There is evidence that IGF-1, VEGF, neuroimmunophilin ligand and other growth factors including brain-derived growth factor (BDNF) play a significant role in neural regeneration and up-regulation of neuronal nitric oxide synthase (nNOS) (7-15).

PRP and neural induced mesenchymal stem cells promote facial nerve regeneration and the greatest myelination of axon fibres when used in combination compared to when used alone in a facial nerve axotomy injury in a guinea pig model (16). Remyelination may help to ensure relief of symptoms are sustained in TN (17).

PRP injection around peripheral nerves may modulate neuronal activity and play a role in regeneration or myelination. This may affect ephaptic transmission of impulses in the sensory pathway of these nerves. PRP of the trigeminal nerve or ganglion involves risk of needle placement at the base of the brain. Pain in the distribution of the distal branches of the maxillary nerve is the commonest presentation of TN. PRP around the distal branches of the maxillary nerve is likely to carry a very low risk profile as seen with PRP use maxillofacial surgery, cosmetic medicine and musculoskeletal medicine. Ultrasound imaging guidance for needle placement does not carry risks of radiation as with Xray guidance for injection of the maxillary or trigeminal nerves at the skull base. An internet search for PRP in TN did not reveal any articles in Pubmed, Google and Google scholar. The favourable short term response in our patient may be due to remission as part of the natural course of TN or a combination of very low dose of Tegretol (50mg twice a day), natural supplements, two weeks of oral cortisone and antiviral treatment and Platelet Rich Plasma. For patients who suffer refractory TN despite oral medications or inability to continue oral medications due to adverse events, there are invasive treatment options. These include microvascular decompression (MVD) at the level of the root entry zone of the trigeminal ganglion and neurolysis with sclerosing agents eg: alcohol or phenol at the level of the foramen rotundum, both of which carry a risk profile. Gamma knife radiosurgery is a non-invasive procedure with a high success rate with some centers using this as the first line option. Recurrence of pain and long-term facial numbness are well recognized in some of these patients. Patients who are not candidates for any of these options or have recurrent pain following invasive percutaneous or surgical procedures may have to endure the symptoms of TN until definitive new treatment options emerge. This paper is a report on a single case of refractory TN treated with PRP and does not validate the routine use of PRP in TN. However this paper provides the first step towards a new approach in the treatment of TN.

Conclusion

As far as the author is aware this is the first report of ultrasound guided PRP of the distal branches of the trigeminal nerve in Trigeminal Neuralgia. In comparison to other percutaneous or surgical
procedures, DTNT is a very low risk, outpatient clinic procedure that can be repeated and should be further evaluated in a large patient group with long term follow up.

References


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