The Long-term Treatment of Atypical Odontalgia with Tapentadol: A Case Report

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Abstract

A typical Odontalgia, typically related to changes in barometric pressure persisted for over 5 years. Despite extensive dental interventions and multiple analgesics the pain continued to have a significant impact on the quality of life of the 64 year old male. Following the introduction of Tapentadol (a dual action opioid and noradrenaline re-uptake inhibitor) a significant improvement on the pain pattern was noted and remained stable for over 12 months.

Keywords: Atypical odontalgia; Atypical; Facial pain; Neuropathic pain

Introduction

Atypical odontalgia, also known as Persistent idiopathic facial pain (PIFP), atypical facial pain, phantom tooth pain, or neuropathic orofacial pain, is characterized by chronic pain in a tooth or teeth, or in a site where teeth have been extracted or following endodontic treatment, without an identifiable cause. The pain usually presents along the territory of the trigeminal nerve that does not fit the classic presentation of other cranial neuralgias. The pain is usually of long duration, lasting most of the day (if not continuous), is unilateral, and is without autonomic signs or symptoms. It is described as a “severe” crushing sensation, or burning sensation. Investigations are aimed at excluding other causes such as infection, fractures or malignancy. Neuralgia inducing cavitational osteonecrosis (NICO) is a bone disease characterized by degeneration and death of marrow and bone from a slow or abrupt decrease in marrow blood flow [1,2]. The diagnosis of NICO is however difficult due to the lack of specific investigation or treatment options. The true incidence of this as cause of atypical odontalgia is unknown.

Unfortunately many of these individuals are misdiagnosed or their pain is attributed to a prior event, such as a dental procedure or facial trauma. Psychiatric symptoms of depression and anxiety are prevalent in this population, further compounding the diagnostic conundrum. Treatment of atypical odontalgia is typically less effective than that of other facial pain syndromes, and a multidisciplinary approach is required to address the many facets of the pain syndrome.

We present a case of atypical odontalgia that demonstrates the importance of neuropathic pain management in such situations.

Case

A 64-year-old gentleman with a 5-year history of right-sided persistent atypical oral/facial pain that was refractory to medical and dental management was referred for pain management.

Originally the patient presented with a “toothache” that radiated from the right upper gum into the medial aspect of nose. Extensive investigation at that time included facial fluoroscopy, magnetic resonance imagery (MRI) and multiple dental examinations but they all failed to identify a cause. During this time the pain persisted and the second molar was proposed to be the cause of the pain. The patient was referred to an endodontist, who concluded that the second premolar was the likely source of the pain and performed endodontic therapy in that tooth. Subsequently, the patient’s general dentist extracted the second premolar and second molar, but the pain continued in the edentulous region. Antibiotics were prescribed in an effort to exclude an underlying infection but these too failed to resolve the pain. The dental surgeon made the diagnosis of “atypical facial pain” and he was subsequently referred to pain management.

Clinical Features

On presentation this patient reported that it was “normal” to be in constant pain on a daily basis. The Numerical Rated Pain Score (NRS) was reported as 8 out of 10 (where 0 is “no pain” and 10 is the “worst” pain). The symptoms were worst at night and these were related to “changes in the weather”. He described features of neuropathic pain (burning, pins and needles, allodynia) and scored high on the DN4 pains core. Features of migraine or migraine like symptoms, trigeminal neuralgia and other causes of atypical facial pain were excluded. No source of infection was ever identified. The patient was heavily dependent on codeine products to try and contain his pain at this level. He felt he was becoming intolerant of the medication and while he wanted to reduce the codeine he was “completely dependent on it in order to have some quality of life”. Daily codeine consumption had continued to increase and averaged 240-300 mgs daily. Previous treatment with pregabalin, gabapentin, tramadol, oxycodin, DFI118 and anti inflammatories’ had all failed to offer any relief. He admitted to feeling “down and depressed with the pain”. His general practitioner would prescribe diazepam occasionally when the symptoms become unbearable.

Other side effects of long-term opioid use were noted, including fatigue, reduced appetite, constipation and dry skin. He had a very supportive wife and was self-employed which allowed him manage his career around his chronic pain. The Brief Pain Inventory (BPI), which is a general assessment of the impact the chronic pain was having on his quality of life, was reduced by 70% in all domains.

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The patient had learnt to manage acute episodes by combining codeine/paracetamol (30 mg/1 gram) every 6 hours with non-steroidal anti-inflammatory medication (Diclofenac 75 mgs, 12 hourly) and Tramadol (100 mgs orally every 6 hours). The frequency of the severe episodes had increased over the years and was typically reported on a weekly basis.

**Barometric Pressure**

The only "trigger" factor that the patient recognized was that inclement weather aggrivated the symptoms. He was resident in a coastal region in South-West Ireland where changes in barometric pressure would occur regularly and often without warning. The severity of pain was related to weather changes. During these periods, despite a combination of 30mgs Codeine and1g Paracetamol every 6 hours, non-steroidal anti-inflammatory agents nothing would resolve the pain. He would expect to experience 6 of these events weekly each lasting several hours. The numerical rating pain score (NRS) during these events would be 9/10.

**Treatment Proposed**

Tapentadol slow release (150 mgs, twice daily) and the rapid acting film-coated tablet (F.C.T, 50 mgs orally every eight hours if required) were prescribed. These doses were chosen based on:

a). The presence of neuropathic pain;

b). The severity of the pain;

c). The need for prolonged steady treatment; and

d). The need for an agent to deal with the sudden onset of symptoms.

It was agreed that in order to assess the impact of the treatment on the pain we would monitor the relationship between the pain intensity and local barometric pressure patterns provided by the metrological services in the region for 7 days prior to the treatment starting in order to act as a reference. The codeine products could be used as usual during this time. We would continue to monitor the relationship between the pain pattern and barometric pressure once the Tapentadol treatment had commenced.

**Results**

Pain intensity and barometric pressure were recorded for 8 consecutive days before and after the commencement of Tapentadol. Table 1 shows that there was a significant reduction in the pain intensity (p<0.05) after the treatment started. The patient reported 2 episodes of "acute pain" during the 8-day treatment with Tapentadol. The maximum severity score was still significantly less that previously reported (4/10).

**12 Month Follow up**

12-month follow up has shown that Tapentadol SR (150 mgs orally twice daily) continued to provide effective analgesia. The use of the Tapentadol F.C.T (50 mgs every 8 hourly) for break through management was only required intermittently and it did provide adequate analgesia when required. On direct questioning there were no side effects related to the constant use of the Tapentadol (such as gastrointestinal upset, constipation). The most common reason for requiring break through use opioid products is indeed very high. It is also acknowledged that a single case report and on its own is no more than an observation. However, the long-term control of atypical facial pain. However, given the severity of the symptoms associated with such cases we feel it warrants highlighting the treatment option. The novel mechanism of action of the product we feel matches the pain pathway there by making it a sensible option.

<table>
<thead>
<tr>
<th>Duration(days)</th>
<th>Baseline</th>
<th>Tapentadol</th>
<th>T-Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Codeine Phosphate (mg/day)</td>
<td>240mg</td>
<td>300mg</td>
<td>-</td>
</tr>
<tr>
<td>Pain Intensity (mean(SD))</td>
<td>9.00(0.95)</td>
<td>12(1.8)</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Pain Intensity 8day Range (Min-Max)</td>
<td>8-10</td>
<td>0-4</td>
<td>-</td>
</tr>
<tr>
<td>Mean Barometric pressure (hPa)(SD)</td>
<td>993(99.5)</td>
<td>985(195.9)</td>
<td>0.12</td>
</tr>
</tbody>
</table>

Table 1: Shows the relationship between the steady state treatment of the pain 8 days before and after Tapentadol was introduced.

**Discussion**

This case highlights that neuropathic pain should be considered a feature of atypical odontalgia. It also underlines that fact that treatment of atypical odontalgia may lead to dependency on analgesics can bring significant side effects due to the polypharmacy. The temptation to over use opioid products is indeed very high.

In this case the management of persistent atypical orofacial pain with the dual action analgesic agent (Tapentadol) provided a positive outcome. Tapentadol is a centrally acting analgesic with dual mechanisms of action: mu-opioid receptor agonist and norepinephrine reuptake inhibition. It has been found to be an effective and well-tolerated medication for a wide variety of chronic pain conditions where there is an element of neuropathic pain involved.

The source of pain in such cases of atypical odontalgia can be explained by changes locally in the bone such as elevated intramedullary pressures, ischemia and recurring micro infraction. These changes would in turn (a) prolong and enhance neuropetide release (especially of substance Porcalcitonin-gene-related peptide); (b) promote neural control of vasoreactivity and stimulate the local mitogenic events; and (c) promote ephaptic effects occurring at sensory nerve endings with injured and healing tissues. Together these changes would in turn lead to sensitization of the nerve fibers and generate continuous nerve pain referred to as neuropathic pain.

The response to the barometric pressure changes we believe suggest that a diagnosis of Neuralgia induction cavitational osteonecrosis (NICO) should be proposed in this case to explain the symptoms reported. NICO is a bone disease characterized by degeneration and death of marrow and bone from a slow or abrupt decrease in marrow blood flow.

We accept that the existence of NICO as a distinct disease entity is controversial. There is no precise and widely accepted definition and epidemiologic evidence of this condition. It has been proposed that the symptoms of NICO result largely from the fluid dynamics associated with reduced marrow outflow, elevated intramedullary pressures, ischemia and recurring micro infraction. As there is no other plausible cause of atypical odontalgia, (irrespective of the actual cause) illustrated by this particular case is encouraging. The possible role of NICO is not a very common cause of atypical facial pain. However, given the severity of the symptoms associated with such cases we feel it warrants highlighting the treatment option. The novel mechanism of action of the product we feel matches the pain pathway there by making it a sensible option.

Overall we suggest that Tapentadol could be considered early in the treatment of Atypical Odontalgia.

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Reference


