Journal of Arthritis
ISSN: 2167-7921

Vickers, J Arthritis 2017, 6:4
DOI: 10.4172/2167-7921.1000247

Case Report Open Access

Iatrogenic Resorption of a Mandibular Condyle Following 3rd Molar Wisdom Tooth Surgery

Russell Vickers E*

Clinical Stem Cells Pty Ltd, Australia

***Corresponding author**: Russell Vickers E, Clinical Stem Cells Pty Ltd, PO Box 6471, Alexandria, NSW 2015, Australia, Tel: +61427711888; E-mail: director@clincalstemcells.com

Received date: August 21, 2017; **Accepted date**: August 23, 2017; **Published date**: August 24, 2017

Abstract

Unilateral osteoarthritis of the mandibular condyle is typically associated with trauma to the temporomandibular joint from sports activities, falls and criminal assaults. In this unusual case the complete destruction of the condyle occurred after a long difficult extraction of a 3rd molar wisdom tooth in a young adult. Downward and lateral pressure from a dental forceps extraction that was applied anterior to the angle of the mandible, combined with the masseter muscle acting as the fulcrum resulted in rapid and complete resorption of the condyle. Clinical signs showed a marked deviation of the maxillary-mandibular incisal midline and formation of an open bite malocclusion on the unaffected side. There was a restricted oral opening, difficulty in chewing food and the patient was psychologically depressed. Acute pain management was achieved with a soft diet, heat applications and non-steroidal anti-inflammatory drugs. Long term pain management strategies included an explanation of current surgical techniques of temporalis muscle flap advancement and joint prosthesis, and the future potential of the 'stem cell in bioscaffold' approach for joint regeneration in this young patient.

Keywords Osteoarthritis; Mandibular condyle; Iatrogenic; Third molar; Stem cell; Bioscaffold

Introduction

The surgical removal of 3rd molar wisdom teeth is a common operation performed in dentistry throughout the world. Potential complications from surgery that are usually discussed with the patient in the preoperative assessment include paresthesia of the inferior dental nerve, 'dry socket', and postoperative infection. Less common risks include jaw fracture at the angle of the mandible in the atrophic mandible, or presence of a large cyst associated with the 3rd molar and the possibility of chronic neuropathic pain. Osteoarthritis (OA) of the temporomandibular joint (TMJ) is associated typically with the aging patient or from significant direct physical trauma to the joint from sports activities, falls or due to criminal assault. The case study presented is an unusual outcome and the consequence of a dental iatrogenic cause – upward pressure on the jaw joint from excessive downward pressure and lateral movements from a tooth extraction with the masseter muscle acting as the hinge.

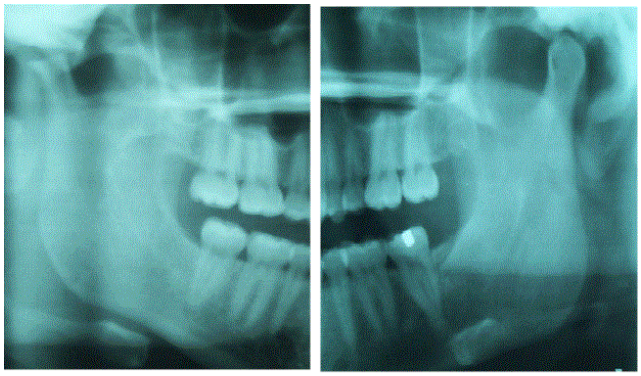
Patient Case Presentation

A 27 year old female complaining of constant right-sided jaw and facial pain was referred to the author (an oral and maxillofacial surgeon). She was on a brief overseas holiday when severe breakthrough pain resulted in her referral. She reported a dental history of the surgical extraction of four impacted third molar teeth under local anesthetic by a dentist two years previously. Her description of the procedure was one of a long duration of mouth opening with heavy pressure on the lower jaw during the extraction of the mandibular right 3rd molar wisdom tooth. The pain origin was the right TMJ, was getting progressively worse and spreading to the right side of the face. She had difficulty in eating solid food, jaw opening and lateral excursive movements of the mandible. Psychologically the patient had good coping skills but was getting depressed and produced a significant negative impact on her social life. Clinical examination revealed a lower central incisor midline shift to the right, a right sided buccal malocclusion and left sided open bite (Figure 1).

Figure 1: Patient holding lips apart demonstrating a substantial limitation of jaw opening. The image reveals a lower teeth incisal midline shift to the patient's right side and a left sided open bite.

Radiographic examination (orthopantomogram) identified complete loss of the right mandibular condyle (Figure 2a) compared with the normal left condyle (Figure 2b). Detailed computerized tomography (CT) was declined by the patient due to financial reasons. A diagnosis of iatrogenic trauma induced condylar OA was given with a secondary diagnosis of myofascial pain involving the right masseter and temporalis muscles. Temporary pain relief was achieved with non-steroidal anti-inflammatory drugs with additional local physical

therapy and heat applications. Possible short term options of arthrocentesis and long term surgical options were explained using temporalis muscle flap advancement or joint prosthesis. The patient elected to have further pain and surgical management back in her home country.



2a 2b

Figure 2: a) OPG radiograph of the right TMJ shows complete erosion of the mandibular condyle. b) OPG of the left TMJ shows normal volume, architecture and positioning of the mandibular condyle.

Discussion

Trauma induced OA of extremities from sporting activities such as knee is well described in the literature. The TMJ is also prone to OA subsequent to extracapsular, intracapsular or condylar neck fracture as a result of direct or contracoup trauma arising from sports injuries and assaults such as punch to the jaw. Loss of the condyle is also an iatrogenic complication from orthodontics and orthognathic surgery [1]. However, to the author's knowledge this is the first published case report of TMJ condylar OA from an iatrogenic cause of a wisdom tooth removal. The longer duration of the procedure performed under local anaesthetic (LA) by a general dentist can be substantially longer compared with 3rd molar surgery undertaken by a specialist oral surgeon using intravenous sedation or short general anesthetic. The LA approach would have significantly contributed to the likelihood of the pathology.

Typically, radiographic features of generalized TMJ OA and rheumatoid arthritis (RA) are similar and include osteophytes, flattened appearance of the condylar head and a reduced joint compartment space [2], although erosions of the condyle are more common in RA. Clinically, bilateral loss of condylar height from orthodontic / orthognathic treatment results in mandibular protrusion and an anterior open bite malocclusion and difficulty in biting through food. This case report showed a patient with an original Class 1 normal occlusion developed an atypical occlusion and bite pattern due to the complete loss of the condylar head. Severe jaw discrepancy has shown that Class 2 (maxillary protrusion 43%) and Class 3 (mandibular protrusion 20%) malocclusions have higher rates of OA compared with normal Class 1 occlusion (3%) [3].

At the molecular level recent bioinformatic gene meta-analysis of OA has revealed only three genes (including VEGF) that are significantly correlated with the disease [4]. VEGF displays unusual chronological effects where it is expressed during the growth phase of articular cartilage, non-functional in mature cartilage but re-expressed in OA [5]. While VEGF is critical for cartilage angiogenesis, an animal study has showed that prolonged mechanical jaw opening resulted in increased VEGF expression of the chondrocytes in cartilage and increased proliferation of osteoclasts [6]. Research into hypoxic joints (in this case from prolonged pressure to the TMJ from tooth extraction) triggers overexpression of VEGF, and this has been demonstrated in other ischemic tissues observed in myocardial and retinal pathology. Activation of VEGF transcription occurs via the erythropoietin gene in cells by hypoxia-inducible factor 1 protein (HIF-1) [7]. Prevention of OA through molecular diagnostic tools has revealed that early detection of the disease with synovial fluid sampling showed high correlation with raised interleukin-6 and may offer a screening method for traumatized TMJs [8]. A further set biomarkers are the tissue degradation enzymes with a significant increase in OA joints of the matrix metalloproteinases (MMPs) of MMP-1, MMP-3, and MMP-9 [9].

Chronic pharmaceutical drugs (typically opioids or non-steroidal anti-inflammatories with side effects) can only manage pain associated with OA. As an alternative several plant derived compounds, usually with nil or minimal side effects, are being actively investigated. For example, the polyphenol compounds gallic acid, baicalein, nobiletin, tangeretin and baicalin have been identified to significantly inhibit VEGF in ovarian cancer cells [10]. An advantage is these compounds are readily accessible and found in food that humans consume augmenting their safety. In addition, there is great interest in translational research for reconstructive approaches to damaged joints using bioscaffolds, stem cells and regenerative signaling peptides and growth factors. Advances in biomolecular approaches should advance the treatment outcomes in difficult to manage OA situations such as the TMJ. Human autologous stem cell therapy administered to the oral and facial region has been shown to be safe and have excellent therapeutic effect for treating trigeminal neurological pain [11]. Interestingly, the biodegradable scaffolds showing most promise for human tissue regeneration are the well-known dental materials of alginate and polyacrylic acids. High resolution CT imaging combined with 3D printers capable of equally high resolution (0.1 mm) layered extrusion that can manufacture bioscaffolds for 'personalised joint surgery' are available. In the clinical case presented of a young female, the most logical and successful outcome of the future would see the oral surgeon surgically placing a TMJ bioscaffold followed by the specialist prosthodontist to open the TMJ space and guide the mandible back to a normal occlusion during the regenerative phase.

References

1. Arnett GW, Milam SB, Gottesman L (1996) Progressive mandibular retrusion—idiopathic condylar resorption. Part I. Am J Orthodont Dentofac Orthoped 110: 8-15.
2. Gynther GW, Tronje G, Holmlund AB (1996) Radiographic changes in the temporomandibular joint in patients with generalized osteoarthritis and rheumatoid arthritis. Oral Surgery Oral Med Oral Pathol Oral Radiol Endodontol 81: 613-618.
3. Krisjane Z, Urtane I, Krumina G, Neimane L, Ragovska I (2012) The prevalence of TMJ osteoarthritis in asymptomatic patients with dentofacial deformities: a cone-beam CT study. Int J Oral Maxillofac Surg 41: 690-695.
4. Nagao M, Cheong CW, Olsen BR (2016) Distinct roles of vascular endothelial growth factor in cartilage development and osteoarthritis. Osteoarth Cartil 24: S365-S366.

5. Lingaraj K, Poh CK, Wang W (2010) Vascular endothelial growth factor (VEGF) is expressed during articular cartilage growth and re-expressed in osteoarthritis. Ann Acad Med Singapore. 39: 399-403.
6. Tanaka E, Aoyama J, Miyauchi M, Takata T, Hanaoka K, et al. (2005) Vascular endothelial growth factor plays an important autocrine/paracrine role in the progression of osteoarthritis. Histochem Cell Biol. 123: 275-281.
7. Forsythe JA, Jiang BH, Iyer NV, Agani F, Leung SW, et al. (1996) Activation of vascular endothelial growth factor gene transcription by hypoxia-inducible factor 1. Mol Cell Biol 16: 4604-4613.
8. Nishimura M, Segami N, Kaneyama K, Suzuki T, Miyamaru M (2002) Proinflammatory cytokines and arthroscopic findings of patients with internal derangement and osteoarthritis of the temporomandibular joint. Br J Oral Maxillofac Surg. 40: 68-71.
9. Kanyama M, Kuboki T, Kojima S, Fujisawa T, Hattori T, et al. (2000) Matrix metalloproteinases and tissue inhibitors of metalloproteinases in synovial fluids of patients with temporomandibular joint osteoarthritis. J Orofac Pain 14: 20-30.
10. He Z, Li B, Rankin GO, Rojanasakul Y, Chen YC (2015) Selecting bioactive phenolic compounds as potential agents to inhibit proliferation and VEGF expression in human ovarian cancer cells. Oncol Lett 9: 1444-1450.
11. Vickers ER, Karsten E, Flood J, Lilischkis R (2014) A preliminary report on stem cell therapy for neuropathic pain in humans. J Pain Res 8: 255-263.