Management of Long Standing Temporomandibular Dysfunction in an Edentulous Patient with Gunning Type Splint: A Clinical Case Report

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ABSTRACT

Objective: The aetiology of temporomandibular dysfunction (TMD) is multifactorial and controversial. It is associated with pain, clicking sounds and structural derangement within the joints. TMDs are believed to be a complex interaction between the temporomandibular joint (TMJ), the masticatory muscles, and possibly the occlusion of the teeth. The prevalence of TMDs in edentulous populations is similar to that in the dentate population. The objective of this study was to observe the effect of altering occlusal vertical dimension on the course of TMD and the overall rehabilitation of an edentulous patient.

Clinical Case Report: This work reports a treatment protocol on a single edentulous woman with TMD. It was carried out at a tertiary centre in Kano, Nigeria. A misdiagnosed, and wrongly treated patient was referred to the prosthodontic clinic at this centre. Clinical and radiological assessments suggested a diagnosis of TMJ arthrosis secondary to neglected TMD. Gunning type splints were used to reduce pressure on the TMJs. Thereafter, conventional complete dentures were prescribed. The symptoms of TMD began to reduce after about 4 weeks and were self-reported to have completely disappeared after 12 weeks of treatment. Treatment with splint therapy was effective and patient was successfully rehabilitated.

Conclusion: In this study, success was achieved with the use of splint therapy in the treatment of TMD. Gunning type splints are a viable option for splint therapy in the edentulous.

Keywords: Gunning type splints, Temporomandibular dysfunction (TMD), Temporomandibular Joint (TMJ),

Citation: Ikusika OF, Adenuga-Taiwo OA, Igweagu CE. *Management of long standing temporomandibular dysfunction in an edentulous patient with gunning type splint: A clinical case report. Nig J Dent Res 2020; 5(2):191-200.*

INTRODUCTION

Temporomandibular dysfunction (TMD)is a symptom complex that causes chronic orofacial pain and headache.[1] The condition has been referred to by many synonyms in the literature.[2-5] The plurality of names for this condition underlies the diagnostic dilemma this condition often poses to the clinician.

This disorder is said to have varying prevalence in various regions. There is a reported increased prevalence with increasing age, and another report of peak age incidence during the 3rd and 4th decades of life.[6] A Nigerian study in Lagos State reported a mean age incidence of 42+/-10 years,[7] while another study in Kano State reported dual peaks in the 3rd and the 6th decades of life.[8] The disorder is also reported to have a female predilection,[6] although the findings from Nigerian studies did not strongly agree with this. One study showed a female preponderance

without statistical significance,[9] while another showed a male: female ratio of 1.5: 1. The study carried in Kano had a relatively small sample size,[8] but had an almost equal gender distribution.

The commonest signs and symptoms of TMD include pre-auricular pain, limitation of mandibular movement and; clicking or other sounds from the temporomandibular joints (TMJ) on opening and closing.[10-12] Other symptoms that may be obtained during a patient interview include headaches, toothaches, diminished auditory acuity, tinnitus and vertigo.[13,14] There may also be glossodynia and a feeling that the teeth are not touching properly (sensation of altered occlusion).[10] There has also been an association between psychological disturbances and TMD. Depressive illnesses and anxiety disorders have been most strongly associated.[5,15] Trauma has also been implicated as a causative factor.[16]

The establishment of TMDs is said to be influenced by several factors which some have categorized as local and systemic factors. Anatomical, genetic and hormonal factors are some so called systemic factors that may predispose individuals to TMD.[17,18] Trauma, parafunction and occlusal changes are some of the so called local factors that precipitate the disorder, while stress and parafunction are said to perpetuate it.[17,18] Parafunction (especially bruxism) and occlusion are becoming less emphasized as local causes of TMD in contemporary thinking.[19,20] Bruxism is now thought to be centrally mediated and independent of occlusion.[19]

Attention was drawn to this symptom complex by an American otolaryngologist called James Bray Costen in 1934.[13] He published his paper describing a new syndrome of auricular and sinus symptoms which he postulated to be dependent on the TMJs. TMDs were thought to be neuralgias occurring in and around the temporomandibular joint.[13,14] It was postulated that inflammation within the mandibular fossa created a pressure effect which led to complete, or partial closure of the internal auditory canal; and varying degrees of nerve compression.[13] It is not uncommon to see individuals with glossodynia and hearing loss with this disorder.[14,21,22] This disorder is now known to be a complex interaction between the TMJ, the masticatory muscles, the muscles of the neck and scalp and possibly the occlusion of the teeth.[23,24] This interaction is thought to be mediated by areas of abnormal muscle activity.

The cause of the disorder is still not firmly established,[22,23] but many investigators are inclined to believe that it has its origins in some form of muscle hyperactivity.[24] This hyperactivity is thought to make changes to the resting length of the muscles.[24] The muscles are believed to adapt to this new position from the effects of chronic unbalanced loading. This muscle hyperactivity is thought to be responsible for the derangements seen in the TMJs.[25] It is also thought to account for the headaches that are associated with the disorder.[26]

The muscle hyperactivity is said to follow the creation of trigger points within the muscles which occasionally lead to uncoordinated spastic contractions.[24] These contractions, if they affect the small head of the medial pterygoid muscle may lead to an anterior disc displacement within the joint. An anterior disc displacement of this kind will leave the posterior innervated part of the disc vulnerable to pressure from the head of the condyle at rest.[24] This would contribute to the pain felt in the pre-auricular area.[22,23]

The joint is supplied by branches of the trigeminal nerve which has its ganglion just above and anterior to the joint within Merkel's cave in the temporal bone.[18,20,22] Compression of the branches of this nerve within the joint may be the source of the ubiquitous nature of the disorder. Disorders to this nerve may produce symptoms in a wide area similar to what is seen with trigeminal neuralgia. The effect of compression of the nerve may be responsible for the glossodynia and other remote symptoms described by Costen.[20, 22]

Shad B Smith leading several authors in reporting the OPPERA case control study provided evidence of an association between the HTR2A and COMT genes and TMD. These genes are closely associated with psychological states like depressive illness.[27] The HTR2A (5-Hydroxytryptamine Receptor 2A) gene is related to major depressive and obsessive-compulsive disorders.[28] The COMT (Cathechol-o-Methyl transferase) gene encodes an enzyme of the same name. This enzyme inhibits dopamine and also contributes to depressed mood.[29]The association with these enzymes may explain why TMD is associated with emotional disturbances.[17]

Serotonin has been observed to serve anti-inflammatory and immune-modulating functions.[30,31] These functions are proposed to be centrally modulated but active peripherally. The up regulation of the HTR2A receptors may be an attempt to combat the inflammation that is present in TMD. Similarly, the up regulation of the COMT receptors may be a response to the high numbers of estrogenic receptors on the articular discs. The enzyme produced by this gene is known to reduce

the mitogenic and mutagenic potential of estrogen,[32] which is one of the hormonal factors associated with the establishment of TMD.

TMDs are commoner in the female gender.[33] This observation is corroborated by the presence of estrogen receptors on articular discs.[33] While these receptors are expressed in both males and females, they are more expressed in symptomatic females compared to the general population.[33] The presence of estrogenic receptors in the TMJ and its association with TMD is in keeping with the relationship of estrogens and other female sex hormones with degenerative joint diseases generally.[34]

Most studies on TMDs have been on dentate or partially dentate cohorts. However, there have been a number of studies carried out on edentulous populations. Epidemiologic studies on the prevalence of TMDs in the edentulous shows a reduced incidence in the edentulous, although edentulous denture wearers were shown to have similar prevalence with the dentate population.[35-38] TMDs in the edentulous have been associated with changes in the vertical dimension of occlusion following prolonged tooth loss and the concomitant atrophic changes in the alveolar ridges.[38,39] The change in vertical dimension that ensues is proposed to affect resting muscle homeostasis and possibly create trigger points within the muscles of mastication which may contribute to the establishment of the disorder.[24]

These signs of TMD in the edentulous may be elicited even when the patients seem to be otherwise asymptomatic.[38,40] The fact that signs may be present in the absence of symptoms only adds to the opacity that surrounds the etiology and management of this group of disorders. It must also be borne in mind that the fact that TMDs also occur in the edentulous would validate the assertion that dental occlusion is not necessarily an etiologic factor for these disorders. Habitual positioning following long term edentulism may also disrupt resting muscle homeostasis and trigger TMDs.[41]

The diagnosis of TMD is mainly clinical. The diagnosis has been greatly enhanced with the development of the Research Diagnostic Criteria for TMD (RDC/TMD) which divides the symptoms into 2 axes.[42] The axis 1 being physical symptoms and axis 2 being psychological symptoms. Medical imaging may act as an adjunct to clinical assessment.[43] Imaging may be used to assess anatomical structures, observe the extent of damage if any and monitor treatment

The discs are best visualized with MRIs[44], and if this is unavailable, by the use of ultrasound-imaging. Computerized tomograms are ideal, but when unavailable may be replaced by plain views or pantographs. The plain views of choice are the trans-pharyngeal and trans-cranial TMJ views.[44,45] Pantographs may show the necks of the condyles, but there may be super-imposition in some cases. Where they are not available, Reverse Towne's projections may be employed.[46,47]

TMDs must be differentiated from other causes of chronic orofacial pain. Some disorders that may mimic TMD, or involve the TMD include osteoarthritis, Rheumatoid arthritis, Eagle syndrome (Stylohyoid syndrome) and even trigeminal nerve neuralgia.[43] A thorough clinical assessment based on the RDC/TMD criteria bearing in mind that TMDs are not usually generalized in nature, may help to differentiate these entities.[41] Other differential diagnoses may include pain from dental origin, ontological conditions like otitis media and mastoiditis; inflammatory conditions like parotitis and even psychogenic pain and atypical facial pain.[15,48]

Prosthodontic management of these disorders mainly involves the prescription of occlusal splints to open the bite.[8,24]The opening of the bite brings the heads of the condyles forwards and downwards and opens up the joint. This allows a diminution of pressure within the joint space and on the disc. This reduced pressure allows for repair and the establishment of newer patterns of muscle action.[24] Occlusal splints has been shown to produce good results in diminishing and even eliminating the symptoms of this disorder.[8]

CASE REPORT

A 70 year old lady was referred to the prosthodontic clinic of Aminu Kano Teaching Hospital with a 15 year history of recurrent headaches and bilateral pre-auricular pain. Patient had visited several health facilities prior to presentation and had been routinely prescribed analgesics and non-steroidal anti-inflammatory drugs (NSAIDs). Patient had developed some level of peptic ulceration which was likely due to abuse of NSAIDs.[48] The patient had lost her teeth to misdiagnoses and the effects of the neglect of oral hygiene that the symptoms of her disorder caused (Figure 1). Patient had undergone extractions over a 15 year period, the last of which was about 2 weeks before presentation at the

prosthodontic clinic. Patient had never worn dentures prior to presentation.

There were clicking and grating sounds from both TMJs. The patient had developed a path of opening and closure of convenience: tilting the jaw to the left on opening and closing excursions. There was significant pain associated with wide opening of the mouth.

Reverse Towne's projections showed sclerotic changes around the right condylar head and neck (Figure 2). Left and Right TMJ views showed sclerotic changes in the articular discs, the joint spaces were generally reduced. However, the right joint space appeared slightly enlarged on opening (Figure 3).

An assessment of TMJ arthrosis secondary to neglected TMD was made. The articular discs were assessed to be non-reducing and a treatment with a non-permissive splint followed by rehabilitation with complete upper and lower dentures was planned. Impressions were made and casts poured. Rest vertical dimension was measured from the philtrum/collumella angle to the tip of the chin at 73mm. Wax bite blocks were fabricated and adjusted intra-orally to obtain an occlusal vertical dimension of 78mm. These were used in the laboratory to fabricate a Gunning Type splint which the patient wore over a 3 month period (Figure 4).

A visual analogue scale was constructed by drawing a line on a piece of cardboard and dividing it into 10 equal parts with equally spaced marks from 0 to 10 and administered on the patient from presentation. The scores were made to represent ascending levels of severity. The patient self-reported a score of 9 at the beginning of treatment. The patient reported a score of 6 after 4weeks and a score of 3 after 6 weeks. At 8 weeks a score of 1 was reported. The patient was free of pain and other symptoms at 12 weeks.

The patient was rehabilitated with acrylic complete full-full dentures with non-anatomic teeth set to bilateral balanced occlusion (Figure 5). The dentures were fabricated to an occlusal vertical dimension of 71.5mm to provide the patient with a reduced inter-occlusal space at 1.5mm. This reduction of inter-occlusal space was designed to still maintain some measure of relief on the joint space for a prolonged period to prevent a possible relapse. The denture teeth were non-anatomic and the occlusal scheme selected for setting teeth was designed to reduce pressure on the ridge when the dentures are in function. The patient found the dentures comfortable after the initial appointments to correct minor over-extensions and inadequate relief.

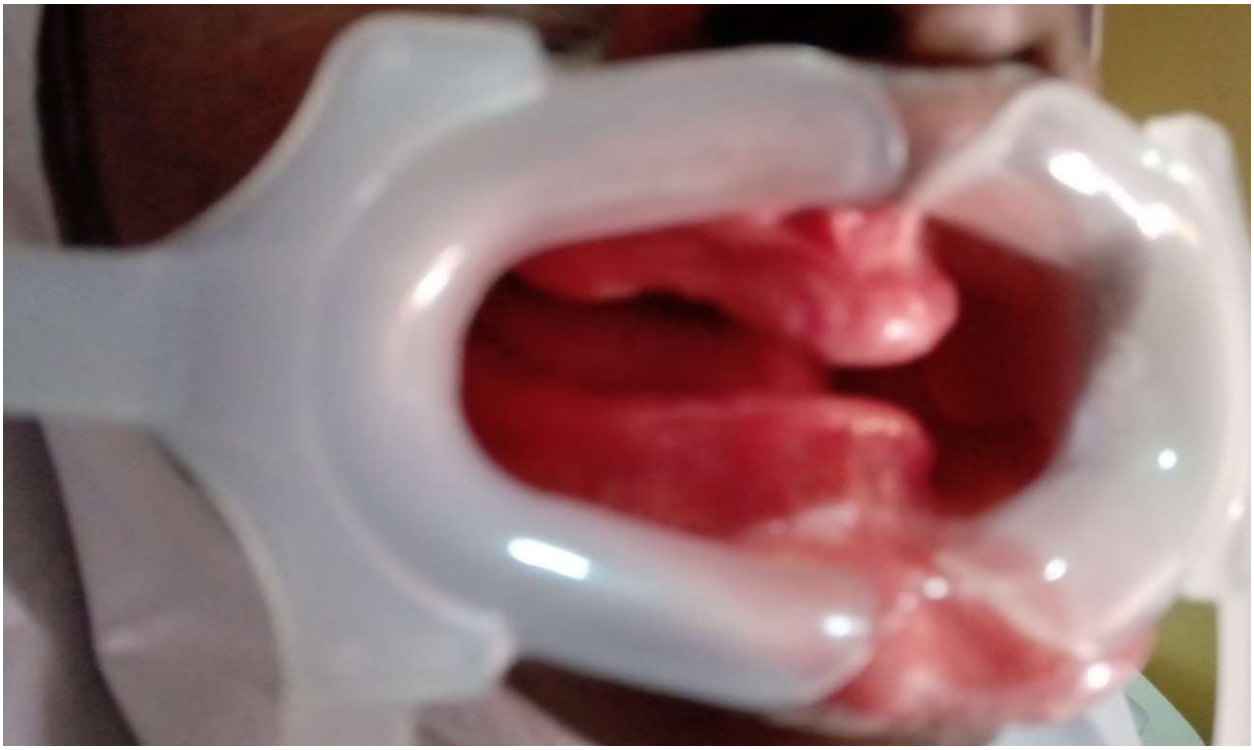

Figure 1: Patient showing edentulous ridges

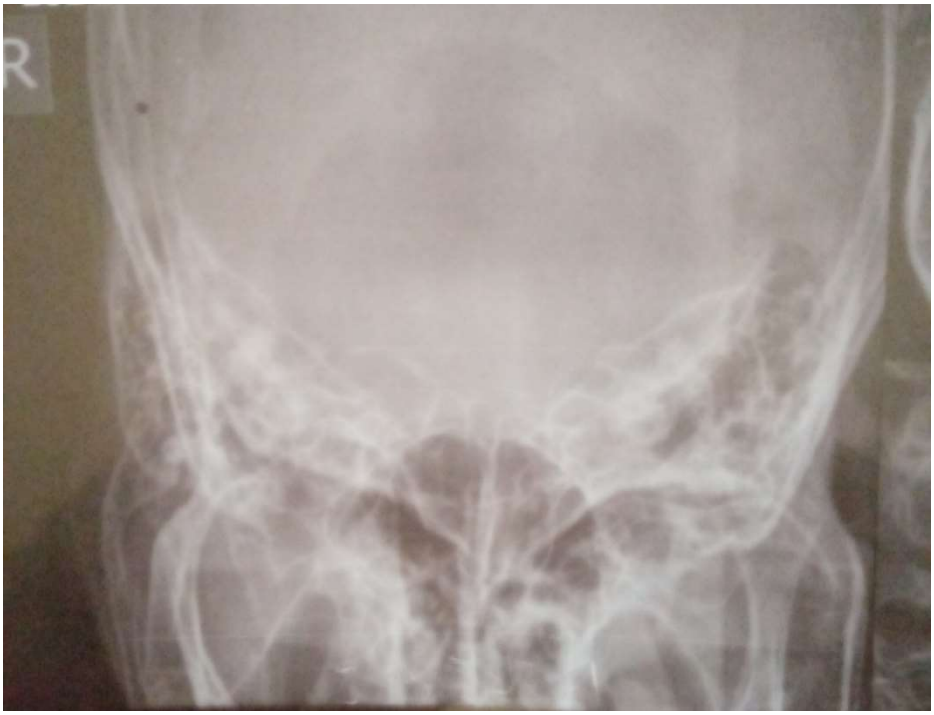

Figure 2: Reverse Townes Projection

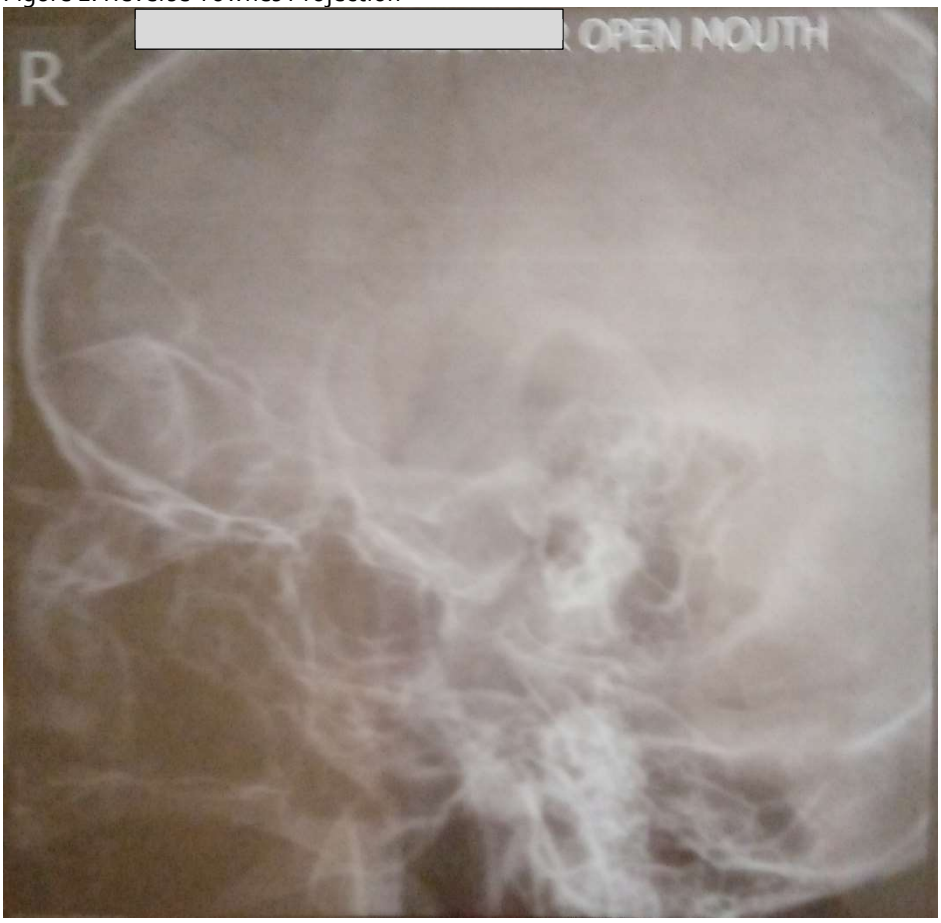

Figure 3: Right TMJ view

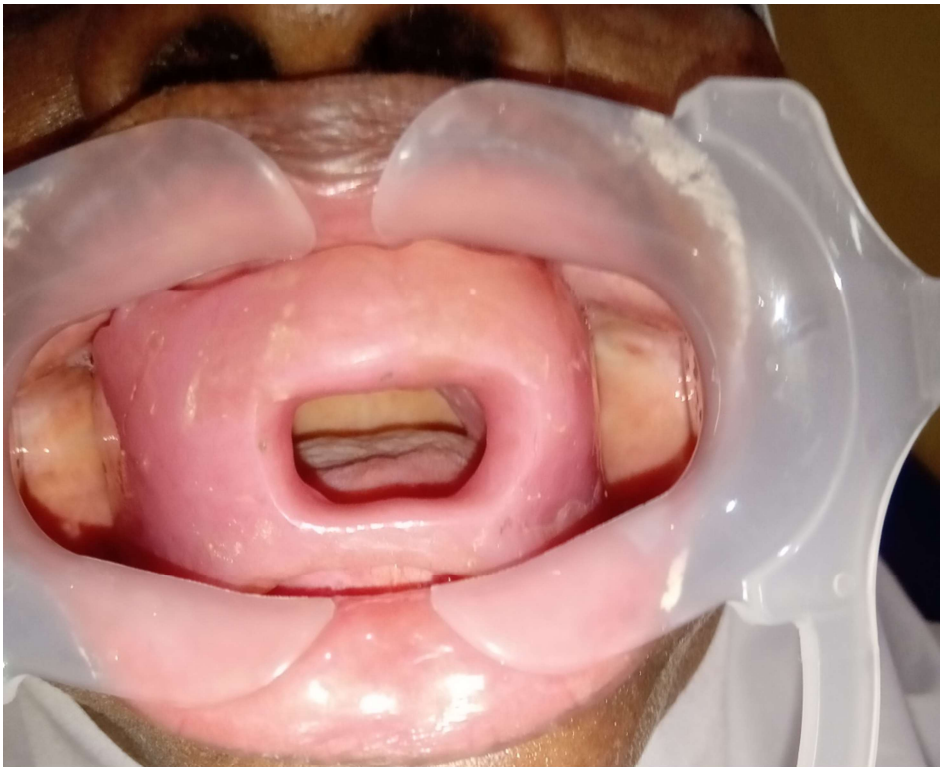

Figure 4: Gunning type Splint intra-orally

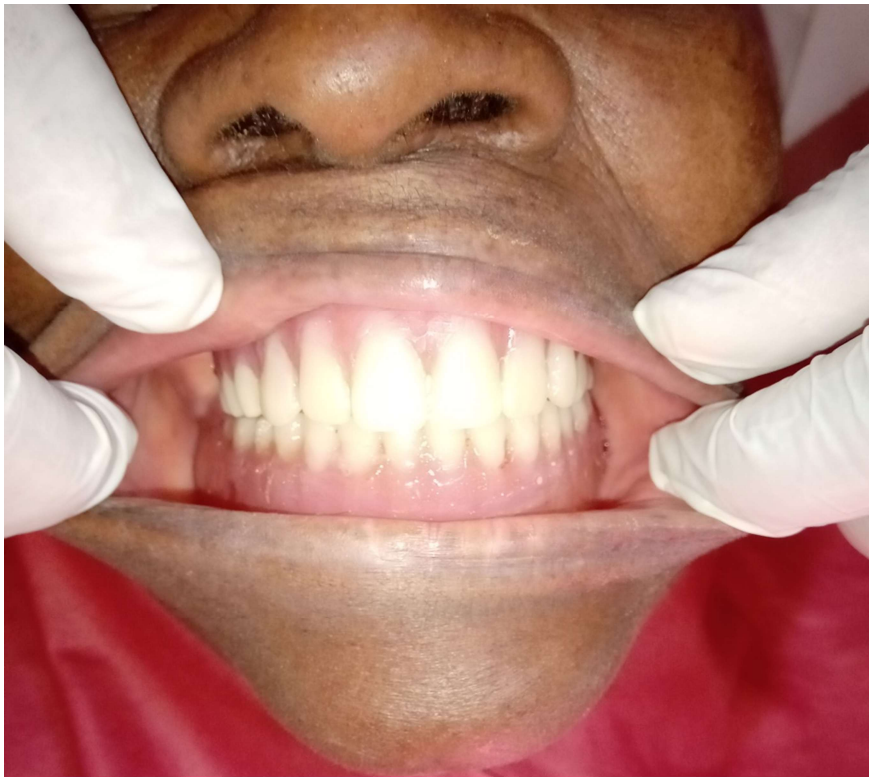

Figure 5: Patient with complete dentures intra-orally

DISCUSSION

The treatment of TMD may be pharmacologic or non-pharmacologic.[43] It may be conservative or surgical. The current trend is to limit irreversible treatments to a treatment of last resort. Conservative treatment may include non-pharmacologic measures like prescription of a soft diet, application of warm compresses and passive jaw exercises. There may be a need for behavior management and psychological counselling if there is suspected psychic affectation.50It may involve referral to a physiotherapist for specialized exercises and treatments like infra-red therapy, are being proposed.[51, 52] These treatments may be used alone, or they may be used in combination with prescription of anti-inflammatory drugs and muscle relaxants. The use of high grade analgesics like centrally acting opiods are not recommended due to the risk of addiction.[43] In view of the emotional differences associated with coping with pain, referral to a pain specialist may be indicated on rare occasions.[43]

Active treatment of TMDs may be non-surgical or surgical. Non-surgical treatment usually involves referral to a prosthodontist for splint therapy.8,24 In the past occlusal adjustment was also practiced, but this is discouraged currently. Other non-surgical management modalities include the use of cognitive behavioral therapy and habit breakers for bruxism. Surgical treatment ranges from intra-articular botox injections to actual intra-articular surgeries.[43]

We may never be able to categorically state the cause of the patient's symptoms, but the clinical evidence points at possible trauma to one TMJ. We arrived at this hypothesis due to the disparity in symptoms between both TMJs. This trauma may have led to the establishment of the altered path of opening and closure of the mandible. The new muscular patterns that would have followed this may have led to the creation of trigger points in the muscles.[8,24] These trigger points would have encouraged hypertonicity within the muscles. This hypertonicity would probably have led to an anterior displacement of the disc and the compression of retrodiscal tissue by the head of the condyle.[8,23,24]The pain and inflammation that would have ensued would further reinforce the tendency for hypertonicity within the muscles. Muscle hypertonicity has also been known to be associated with the headaches that are often associated with TMDs.[24]

The headache may be as a result of pain from the hypertonic muscles referred to the cortex through the nucleus of the spinal tract of the Trigeminal.[24]There is another possible pathway for the headaches that may follow TMDs. The up-regulation of estrogens and serotonin receptors along with the down-regulation of dopamine via the action of COMT gene may contribute to the production of migrane-like headaches in individuals with TMD.[53,54]The grating sounds from the joint were suggestive of structural damage to the disc following internal derangements. The absence of clicking convinced us that the discs were non reducible and would require a non-permissive splint.[55-57]

Our choice of Gunning type splints was motivated by a need to provide a non-permissive splint for the patient (since our diagnosis was a TMD without reduction),[24] and by the need to provide retention and stability for the splints to be used. We decided on splint therapy as patient had already been on prolonged medication without much success over a 15 year period. We decided to increase OVD 5mm beyond rest vertical dimension because of our familiarity with the promising therapeutic effects of increased OVD in relieving pain from TMD. However, the evidence for this is at best still uncertain.[58,59] Two mechanisms have been proposed through which splints may bring relief in TMD. They have been proposed to relief the pressure on the joint by increasing the OVD. This is said to occur because the condylar heads are pulled forwards and downwards when the OVD is increased.[58] Splints are also believed to reduce pain by eliminating muscle hypertonicity and providing the opportunity for the formation of a favorable balance in the stomatognacthic system.[59]

Our choice of radiographs was guided by the economic capacity of the patient. The patient could not afford an MRI, CT scan or a pantograph. We did not order post treatment radiographs as we were of the opinion that there was no need to unnecessarily expose the patient to radiation as there was a complete resolution of symptoms. We were also mindful of the economic implication of such radiographs on the patient.

The patient had lost the teeth over a 15 year period and there was obvious irregular resorption of the ridges (Figure 1). Resorption has been proposed to be a reaction to changing strains within the alveolar bones after tooth loss. It is said to continue and only slows down when the strain levels approach pre-extraction levels. We expect an irregular ridge like the one in our patient (Figure 1) to undergo relatively rapid resorption as it remodels.[60] We are also aware that changes in interocclusal space may be

compensated for by ridge resorption.[61] While we are aware that encroachment on the interocclusal space may hasten ridge resorption by increasing the forces acting on the residual ridge, we considered an 0.5mm encroachment to be modest. Our confidence was later justified as there were no untoward reactions to the dentures by the patient.

CONCLUSION

TMDs in the edentulous population are amenable to splint therapy. Gunning type splints are a viable option for splint therapy in this patient cohort. Complete denture rehabilitation in these patients must look at the long term maintenance of the results achieved with splint therapy.

There has been an increased understanding of TMD since the work of pioneers in the field. However, there is a need for randomized controlled trials to develop stronger scientific evidence in its diagnosis and management.

Consent for Publication

Informed consent was obtained from the patient for publication of this case report and accompanying images.

Source of Support

Nil.

Conflict of Interest

None declared.

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