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Review of Masseteric nerve neurectomy in masseter hypertrophy

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ABSTRACT

Masseter muscle hypertrophy is an unusual state which may be unilateral or bilateral. It is identified by pain in masseter muscles, limited mouth opening, trismus, bruxism, and prominent mandibular angle. Due to alteration in facial configuration, it remains a cosmetic concern to the patients. The treatment approaches include surgical and non-surgical means. Though non-surgical means provide relief, its success rate on clinical therapy is yet to be determined. Hence this narrative review highlights the effects of masseteric nerve neurectomy in masseter hypertrophied patients.

KEYWORDS: Masseter, hypertrophy, neurectomy, trismus, bruxism

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1. INTRODUCTION

Masseter muscle hypertrophy (MMH) is an unusual state indicated by upsurge in volume of the masseter muscle which may be unilateral or bilateral [1]. Bruxism, temporomandibular disorders, malocclusion, congenital, clenching and stress may contribute to its appearance [1-4]. There are many treatment modalities available for MMH like botulinum toxin type A injection, radiofrequency coagulation and other surgical approach. However, there is very little literature and case reports available that focuses on the masseteric nerve neurectomy for treatment of MMH. This review focuses on masseteric nerve neurectomy for masseter hypertrophy and highlights its effects on patients.

2. FEATURES OF MMH

The trilayered quadrangular masseter muscle is of importance for facial aesthetics. It gets inserted into mandibular angle and ramus and helps in mastication. Hence its hypertrophy can lead to protrusion, trismus, and bruxism [1,2]. Generally pain or tension in the hypertrophied region, facial asymmetry and limited mouth opening are its symptoms [5,6]. It also alters facial line angles and results in prominent mandibular angle which may be of cosmetic concern to the patients [7]. As a consequence of MMH, the facial configuration appears rectangular or square-shaped [1-4].

3. CLASSIFICATION OF MASSETER MUSCLE HYPERTROPHY

As explained by Teixeira et al,[8] MMH can be classified as

- I. congenital or familial
- II. acquired which is attributed to hyper-functionality

The acquired form is noted to occur most frequently. This could be due to malocclusion, habit of chewing gum, TMJ disorders, bruxism, and psychological stresses.

4. DIAGNOSIS

It is only by clinical examination and by imaging modalities we can differentiate in distinction to other findings such as oral submucous fibrosis, parotid inflammation, and tumors, benign or malignant muscle and mandibular tumors, and vascular tumors [9]. Radiographically it appears as an enlargement on mandibular angle region. This is because of hyperactive stimulation of the muscles which results in periosteal alterations and uneven bone accumulation leading to formation of bony spurs. Upon posteroanterior view, mandibular angle shows a lateral projection called bone spur [5,6]. The patient can be advised to clench his or her teeth for digital palpation. Upon clenching, prominence in the masseter muscle is the indicator for hypertrophied muscle [2,3]. Therefore, preoperative evaluation is mandatory as it helps in determining the amount of bone to be resected. In addition, CT and ultrasonography remains an established method to understand anatomical landmarks and soft tissue structures.

5. PROPOSED TREATMENTS FOR MMH

The treatment modalities include surgical and non-surgical approach. The nonsurgical approaches are counseling, application of mouth guards, muscle relaxants, anxiolytic drugs, analgesics, physical therapy, dental restorations, relaxation therapy and correction of premature contacts by occlusal adjustments [10,11]. The Conservative approach consists of botulinum toxin type A injection and radiofrequency coagulation. Surgical approach comprises partial resection of the masseteric muscle and/or mandibular angles. Though conservative approaches provide relief, its predictability for longer duration remains questionable. Hence surgical therapy may be considered for permanent outcome.

A. Botulinum toxin type A injection: It was initiated by Smyth [12], Moore, and Wood [13].

It acts by hindering the release of acetylcholine leading to inhibition of muscle contraction [14]. This in turn causes masseter muscle paralysis which eventually connects to its atrophy and decrease in muscle volume. Despite its conservative act, the effect of the toxin lasts only for a short period. Its effect reverses to its original state in six months period [15]. Hence multiple injections would be required which adds onto its disadvantage.

- B. Radiofrequency coagulation:** When compared to surgical removal, this procedure is learnt to be safer and long lasting. Powell et al [16] introduced this method by using radiofrequency energy. This causes resistance which leads to heat ranging within the tissue which coagulates muscle tissue and forms scar tissue that shrinks the overall volume of involved muscle. This decrease in muscle volume generally initiated three weeks following the procedure and extends to a period of six to eight weeks.
- C. Extraoral surgical approach:** Gurney et al. described by placing submandibular incisions followed by muscle mass removal [17]. Adams et al. in 1949 [18] introduced described mandibular angle ostetomy by inframandibular approach where in two-thirds of the lower and medial portions of the muscle and the hyperostotic areas of the mandibular angle are removed. Care must be taken as the possibility of facial nerve injury is high. Despite good access to the muscle and mandibular angle, it may be unacceptable as it shows out surgical scarring.
- D. Intraoral surgical approach:** Ginestet et al. [19] first described this approach by decreasing fusiform muscle fibers thereby causing change in the efferent system followed by rearrangement of muscle control. This technique is also known to avert relapse. Kim and Kameyama et al. in their cases performed initially mandibular angloplasty followed by myotomy [20]. In 1951, Converse resected both bone and masseter muscle by the intraoral approach [21]. Hankey et al. in 1968[22] and Beckers at al. in 1977 [23] described good cosmetic results by resending, intraorally, the medial layer of the masseter muscle from the inferior border to just beneath the zygomatic arch. Later Riefkohl et al. [24] in 1984 also treated ten patients with myotomy and resection of the bony spurs by this way. It was only in 1989 where Chee and Fe [25] shared that clamping near the zygomatic attachment is not required as muscle bulk tends to result deformity at the inferior region. Bony resections has also been suggested to reduce mandibular angle. Though it has shown better aesthetics care has to be taken as excess reduction could damage inferior alveolar nerve bundle. Whitaker et al. [26] in 1989 conveyed that lateral cortex reduction along with posterior margin of the ramus and resection of medial half of masseter caused about 5 to 6 mm reduction in each side or about 10 to 12 mm in the width of the lower face. Baek et al. [27] in 1989 also conducted bone resection in 24 out of 42 patients. With time Rispoli et al.,[2] Biruktawit Kebede et al.[1], and Shetty et al [28] prosed the removal of inner layer of the hypertrophied muscle as the main goal followed by mandibular angloplasty only if required. Anehosur V et al. [29] 2020 did genioplasty to

enhance esthetics. The muscular hyperactivity resulted the growth of mandible in horizontal direction instead of vertical manner leading to decrease in lower facial height and alteration in facial configuration. This in turn results in retruded mandible or retrognathia.

E. Masseteric nerve neurectomy: Masseter hypertrophy, characterized by the enlargement of the masseter muscle, can lead to a squared mandibular angle, which may be undesirable for individuals. The proposed solution in the study performed by Kun Hwang et al. In the year 2004 involves a selective neurectomy of the masseteric nerve to induce muscular atrophy, thereby reducing the hypertrophic mass of the masseter muscle. The study, conducted in Korea, employed cadaveric dissection and surgical procedures on patients with masseter hypertrophy to investigate the precise course of the masseteric nerve and assess the impact of neurectomy. The masseteric nerve and artery were consistently found around the mandibular notch. Measurements indicated that the masseteric nerve was approximately 11.3 ± 2.6 mm horizontally from the anterior border of the ramus and 11.3 ± 2.6 mm above the mandibular notch. Surgical procedures involved the selective neurectomy of the masseteric nerve, which was performed in combination with other techniques such as angle ostectomy in some cases. The study emphasized the constant existence of the masseteric nerve and artery, making the procedure feasible and consistent [30].

According to Vincent AG et al, In the described surgical technique, the masseteric nerve is isolated through blunt dissection at a specific anatomical location, typically located 3 cm anterior to the tragus, 1 cm inferior to the zygomatic arch, and 1.5 cm deep to the muscle fascia. [31]

According to a case report by Van Der Kelen L et al, describes a successful treatment of masticatory muscle hypertrophy, particularly affecting the right side of the face, through neurectomy of the right masseteric nerve performed via an extra-oral approach. Clinical examination revealed significant improvement in facial symmetry following the procedure, with complete paralysis and atrophy of the right masseter muscle. Additionally, the impaired function of the frontal branch of the right facial nerve was fully restored within 10 weeks postoperatively. The patient reported no further headaches or discomfort when eating or sleeping on her right side [32]

Masseteric nerve neurectomy stands out as a promising treatment option for masseter muscle hypertrophy (MMH), offering several advantages over other available modalities. Unlike nonsurgical approaches such as botulinum toxin injections and radiofrequency coagulation, which provide only temporary relief, neurectomy induces long-lasting muscular atrophy, ensuring sustained results and minimizing the need for repeated interventions.

Moreover, neurectomy offers a targeted and precise approach to addressing MMH by selectively disrupting the neural pathway responsible for muscle hyperactivity. This not only reduces the risk of recurrence but also preserves the function of surrounding structures, ensuring optimal aesthetic and functional outcomes for patients.

Additionally, the flexibility of performing neurectomy through both extraoral and intraoral approaches allows for customization based on patient anatomy and preferences. While extraoral neurectomy offers direct access with minimal risk, intraoral neurectomy provides cosmetic advantages and reduces the likelihood of visible scarring.

Furthermore, evidence from clinical studies and case reports supports the efficacy and safety of masseteric nerve neurectomy, with successful outcomes including improved facial symmetry, restored nerve function, and resolution of associated symptoms. These findings underscore the potential of neurectomy as a reliable and effective treatment option for MMH.

In conclusion, masseteric nerve neurectomy emerges as a valuable therapeutic strategy for MMH, offering durable results, precise targeting, and favorable cosmetic outcomes. As further research and clinical experience accumulate, neurectomy may become increasingly recognized as a primary treatment modality for patients seeking long-term relief and aesthetic improvement.

CONCLUSION

Despite advances, the etiology of MMH remains unknown. It is essential to treat the condition due to esthetic and functional reasons. Along with intraoral approach for the correction, genioplasty and coronoidectomy may be considered along with debulking of the hyperactive muscle. Postoperative care includes suction drainage, pressure dressings, and physiotherapy to address the functioning at the earlier stage. There is limited literature still on the success rate of therapies. Hence more investigations is required.

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