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The role of lateral pterygoid insertion type and pathology in TMJ internal derangement: An MRI study

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ABSTRACT

Background and purpose: While the two heads of the lateral pterygoid muscle (LPM) are well-defined in most anatomical descriptions, variations in their insertions have been reported. In some cases, the superior head of lateral pterygoid muscle (SHLPM) inserts solely to the condyle (Type I), whereas in others, it inserts both to the condyle and the disc (Type 2). Such anatomical variability is hypothesized to influence the biomechanics of the TMJ and contribute to the development of TMDs. The purpose of this study was to evaluate the role of type of lateral pterygoid muscle insertion and pathology in TMJ internal derangement using MRI.

Methodology: This prospective study included 54 patients (96 joints) presenting with clinical features of TMJ internal derangement. Joints were categorized into disc displacement with reduction (DDWR) and disc displacement without reduction (DDWoR) on MRI. LPM insertion type and pathological changes such as atrophy, hypertrophy, and fibrosis were assessed. Statistical analysis was performed using Pearson Chi-square test and ordered logistic regression, with significance set at $p < 0.05$.

Results: Type 2 LPM insertion was the predominant pattern with 80% joints compared to type 1 LPM insertion pattern in 20% joints. No statistically significant correlation was found between the type of LPM attachment and disc displacement (with and without reduction), the type of insertion pattern of the LPM with the pathology associated with the muscle, or between the pathological changes of the LPM with the type of disc displacement (with or without reduction). Pathological changes to the LPM were more common in type 2 insertion type than type 1, in both the superior head as well as inferior head of the LPM. However, these differences were not statistically significant.

Conclusion: LPM insertion type does not have a statistically significant association with TMJ internal derangement. Muscle pathological changes appear to be secondary adaptations rather than primary etiological factors.

Keywords: Lateral pterygoid muscle insertion type**,** lateral pterygoid muscle pathology**,** disc displacement

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INTRODUCTION: The temporomandibular joint (TMJ) is a paired synovial (diarthrodial) joint situated on either side of skull, formed by articulation of mandibular condyle with mandibular (glenoid) fossa of temporal bone [1]. Lateral pterygoid muscle (LPM) is implicated in control of articular disc movement and plays a distinct and complex role in functional biomechanics of temporomandibular joint (TMJ) [2].

The role of SHLPM (superior head of lateral pterygoid) and IHLPM (inferior head of lateral pterygoid) is however debated. Understanding the function of a muscle is challenging due to disagreements on its origin and insertion places especially of the superior head of lateral pterygoid (SHLPM) [3]. Anatomical investigations conducted by on cadaveric specimens demonstrated that the superior head of lateral pterygoid muscle inserted exclusively into the pterygoid fovea of the mandibular condyle in approximately 30% of cases. In the remaining specimens, although the primary insertion of the superior head was also into the pterygoid fovea, the uppermost 20% of its muscle fibres exhibited an additional secondary insertion into the joint capsule, located inferior to the site of attachment of the articular disc to the capsule [2].

Some clinicians believe that SHLPM participation in disc and condyle rotation is a contributing factor to ID and TMD [1,2]. These variations in insertion have led to longstanding debate regarding the role of the lateral pterygoid muscle in the etiology of internal derangement of the TMJ. It has been postulated that insertion of the superior head of the LPM (SHLPM) into the articular disc may predispose the joint to anterior disc displacement by exerting excessive or unbalanced anteriorly directed forces, particularly during parafunctional activity or muscle hyperactivity [4]. In addition to insertional variations, pathological changes within lateral pterygoid muscle have been implicated in temporomandibular joint disorders. These changes include muscle hypertonicity, hypertrophy, atrophy, fatty infiltration, fibrosis, and altered signal intensity on magnetic resonance imaging [5,6]. Conversely, muscle pathology may also develop secondary to chronic joint degeneration or altered mandibular biomechanics, indicating a complex and bidirectional relationship between muscular and articular components of the TMJ [7].

Despite extensive investigation, the role of lateral pterygoid muscle insertion and pathology in the etiology of internal derangement remains controversial. While some studies suggest a significant association, others indicate that LPM changes may represent secondary adaptations rather than primary causative factors. These conflicting findings highlight the need for further imaging-based studies to clarify the relationship between LPM anatomy, muscle pathology, and TMJ disorders.

The purpose of this study was to evaluate the role of lateral pterygoid muscle insertion and pathology in internal derangement of TMJ. Further, the objectives of this study were to assess the correlation between the insertion pattern of the lateral pterygoid muscle with the type of anterior disc displacement (with or without reduction), to assess the correlation between the insertion pattern of the lateral pterygoid muscle with the pathology associated with the muscle, and finally to assess the correlation between the pathological changes of the lateral pterygoid muscle in with the type of anterior disc displacement (with or without reduction).

MATERIALS AND METHODS: This prospective study was conducted on 54 patients who presented to the Department of Oral and Maxillofacial Surgery between June 2024 and January 2026 with clinical signs and symptoms of temporomandibular joint internal derangement, including pain and tenderness over the TMJ, clicking, intermittent locking, crepitus, mandibular deviation, or restricted mouth opening. All patients underwent TMJ tomography and magnetic resonance imaging (MRI). This study was approved by the Institutional Ethical Committee of the institute as per protocol number MADC/IEC/II/25/2024.

Patient selection

Inclusion Criteria: Patients who consented to be part of the study, over 18 years of age, and clinically diagnosed with TMJ internal derangement.

Exclusion Criteria: Patients who had not consented to be part of the study, below 18 years of age, with acquired developmental deformities of the temporomandibular joint like TMJ ankylosis, previous condylar fractures, condylar hyperplasia, condylar hypoplasia, Idiopathic condylar resorption, arthritic conditions, and patients with congenital/syndromic deformities of the temporomandibular joint.

Study Group

All patients were subjected to MRI scans of the bilateral TMJ which confirmed the diagnosis of disc displacement with reduction or disc displacement without reduction. These joints were divided into 2 groups based on the MRI interpretation of disc displacement.

Group 1: Joints diagnosed with anterior disc displacement with reduction (ADDWR).
Group 2: Joints diagnosed with anterior disc displacement without reduction (ADDWoR).
Both the groups were studied for lateral pterygoid muscle insertion and pathology associated with the muscle.

Criteria to assess lateral pterygoid muscle insertion (MRI):

Lateral pterygoid muscle attachment was categorized into 2 types.
Type 1: SHLPM was attached to the disc and IHLPM attached to the condyle **(Fig. 1a, 1b, 1c. 1d).**
Type 2: SHLPM was attached to the disc and condyle and IHLPM attached to the condyle **(Fig. 2a, 2b, 2c, 2d)**.

Assessment of lateral pterygoid for pathology on MRI (atrophy, hypertrophy, and degeneration):

1. Fibrosis was identified as low signals in the muscles on proton-density (PD) and T2-weighted images. **(Fig 3a)**
2. Atrophy of the LPM was observed by fatty infiltration, exhibiting high signal intensity across extensive regions of the muscles in both short and long (TR) relapse time /short (TE) echo time images. **(Fig. 3b)**
3. Hypertrophy was visible as the enlargement of the middle part of the belly. It represents as convexity of upper and lower edges of SHLPM or IHLPM **(Fig. 3c)**.

Correlations:

1. The type of lateral pterygoid muscle (LPM) insertion was correlated with the type of disc displacement

(with or without reduction)

2. Pathology in the superior head of lateral pterygoid muscle (LPM) was correlated with the type of disc displacement (with or without reduction).
3. Pathology in the inferior head of lateral pterygoid muscle (LPM) was correlated with type of disc displacement (with or without reduction)

Sample size calculation: The sample size calculation was performed using STATA statistical software version 17 (STATA, College Station, Texas, USA). After assigning the power of the study as 95% and alpha error as 5% the effective sample size for the study has been computed to be 54 joints per group using two samples proportion test (study parameters: alpha error = 0.05, power = 0.90, delta =0.5172, p1=0.58, p2 = 0.30).

Statistical methods: Statistical analysis was performed using SPSS version 27 (IBM Corp., Armonk, USA). Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. After excluding normal joints, only ADDWR and ADDWoR joints were analyzed and associations between categorical variables were evaluated using Pearson Chi-square test. Binary logistic regression was used to assess the independent effects of lateral pterygoid muscle insertion type and muscle pathology on the type of internal derangement, with results reported as odds ratios and 95% confidence intervals. A p value < 0.05 was considered statistically significant and < 0.01 statistically highly significant.

RESULTS: The study included 54 subjects (96 temporomandibular joints) with a mean age of 30.33 ± 9.04 years. Females constituted 66.7% (n = 36) of the study population, while males accounted for 33.3% (n = 18). Among the joints evaluated, 60 (62.5%) demonstrated anterior disc displacement with reduction (ADDWR) and 36 (37.5%) demonstrated anterior disc displacement without reduction (ADDWoR). Type 2 lateral pterygoid muscle insertion was the predominant insertion pattern in both ADDWR and ADDWoR groups. However, no statistically significant association was observed between insertion type and internal derangement ($\chi^2 = 0.214$, $p = 0.643$) **(Table 1).**

The distribution of superior and inferior head muscle pathology was comparable between the ADDWR and ADDWoR groups. None of the superior head muscle abnormalities (atrophy, hypertrophy, or fibrosis) or inferior head muscle abnormalities demonstrated a statistically significant association with the type of internal derangement (all $p > 0.05$) **(Tables 2 and 3).**

Analysis of the relationship between lateral pterygoid muscle insertion and muscle pathology demonstrated a predominance of Type 2 insertion across all superior and inferior head pathological changes. However, these associations were not statistically significant **(Tables 4 and 5).**

Binary logistic regression analysis showed that neither lateral pterygoid muscle insertion type nor superior or inferior head muscle pathology independently predicted the type of internal derangement. The overall model was not statistically significant (LR $\chi^2 = 4.53$, pseudo $R^2 = 0.036$, $p = 0.717$). **(Table 6)**

Overall, the study demonstrated a female predominance and a higher frequency of ADDWR compared with ADDWoR. Although Type 2 lateral pterygoid insertion was the most common insertion pattern, neither insertion type nor muscle pathology was significantly associated with the type of internal derangement.

DISCUSSION: There has been a general consensus for many years that the lateral pterygoid muscle (LPM) originates from two distinct heads. [8] Conventionally the LPM is described as being a single muscle with two heads, the superior head of the LPM (SHLPM) arising from the infratemporal surface of the skull made up by the greater wing of the sphenoid bone and squamous part of the temporal bone, and the inferior head of the LPM (IHLPM) from the lateral surface of the lateral pterygoid plate. The superior head fibers are inserted into the anterior, anteromedial surface of capsule and disc. The IHLPM runs posteriorly and laterally to the insertion into the pterygoid fovea and inserts onto the anteromedial surface of the condyle. The two heads converge and fuse in a short tendon, which is inserted into the mandibular fossa just below the medial pole of the condyle [9].

The function of the LPM is complex. The two heads of the lateral pterygoid muscle are reciprocally innervated so that during opening of the mouth the inferior head contracts and the superior head relaxes, whereas during closure of the mouth the situation is reversed [9, 10]. As the mouth opens, contraction of the IHLPM muscle which is attached to the neck of the condylar process, pulls the condyle downward and forward along the posterior slope of articular eminence towards its peak. Because of the attachment of the disc via the collateral ligaments to the condylar poles, the disc also moves downward and forward in unison with the condyle. However, the disc pivots posteriorly relative to the anteriorly rotating condyle because of the pull of the stretched fibers in the retrodiscal tissue and lack of opposition by the relaxed reciprocally innervated SHLPM, thus maintaining its position between the rotating condyle and eminence [9, 10]. During closure of the mouth the IHLPM relaxes, and the condyle is pulled back toward its original position by the action of the masseter, medial pterygoid, and temporalis muscles. At the same time, the gradual contraction of the SHLPM ensures repositioning of the disc to the posterior slope of the articular eminence.

In summary, as a result of the combined action of the alternate contractions of the LPM, the tension offered by the retrodiscal tissues, and the discal attachments on the condyle, during mouth opening as the condyle is translating anteriorly, the disc glides posteriorly over it, relative to the movement of the condyle. The reverse happens during closure - as the condyle is seating back into the glenoid fossa, the disc glides anteriorly over the condylar head to maintain its position relative to the posterior slope of the eminence.

The LPM makes a unique contribution to jaw movement control by virtue of the attachments to temporomandibular joint (TMJ) disc and condyle. The SHLPM primarily contributes to the apposition of the disc, condyle, and eminence in jaw closing, whilst the IHLPM primarily contributes to jaw opening. [11] However, the role of the superior head in influencing rotation and translation of disc and condyle and the relationship with internal derangement and temporomandibular dysfunction has not been fully understood.

In a study by Taskaya-Yilmaz et al [4], the authors stated that the reasons for the atrophy observed in a high proportion of type 1 muscle attachments may be related to the fact that since the SHLPM inserts only into the

disc, the SHLPM will displace the disc more anteriorly. This will reduce the function of the SHLPM and will cause muscle atrophy. According to the authors, the high ratio of atrophy observed in ADD may be explained by the reduction in activity of the SHLPM, since the disc was permanently dislocated in TMJs with ADD. Spasm of the LPM will cause disc displacement and then atrophy and degeneration of the LPM will follow disc displacement. In their study, atrophy was seen in normal TMJs, and this may predispose to anterior displacement of the discs.

However, in the present study, the authors found muscle atrophy of the superior head in only 3 joints out of 19 (15.8 %) with type 1 LPM insertion type as compared to 17 out of 77 joints (22%) with type 2 LPM insertion. **[Table 4]**. In the present study, 10 joints (10.4%) with atrophy of the SHLPM had DDWoR; however, 10 joints out of 96 (10.4%) with atrophy of the SHLPM also had DDWR **[Table 2]**

In a study on the structural and pathological alterations involving the LPM in patients with TMJ hypermobility using MRI, Yang et al [12] found that laxity of the TMJ capsules and ligaments has been implicated in TMJ hypermobility which destabilize the condyle-disk relationship. In that setting, the SBLPM might be abnormally over-loaded to maintain stability. This in turn might cause hypertrophy of the SBLPM. Atrophy or contracture of muscles was regarded as secondary to excessive muscle activity, stretching, and work overload when there is no history of disuse after trauma, immobility, debilitating disease or denervation disease. [13]

In an anatomic study of the TMJ, Grunert et al. [14] noticed that hypertrophy in the SBLPM was observed in some cases with normally positioned disks. He also suspected that hypertrophy changes of the SBLPM might be caused by chronic overload. Lafreniere et al. [15] suggested that IHLPM activity is significantly higher when the SHLPM has lost its disc-stabilizing function. They also stated that the abnormal function and alterations of the IHLPM might be considered as a secondary change of the SHLPM. In their report, excessive work of the IHLPM was found when the SHLPM seemed to have lost its function.

In the present study, 11 (11.4%) joints showed hypertrophy of the SHLPM and 2 (2%) joints showed hypertrophy of the IHLPM. **[Tables 2, 3].** Muscular atrophy was the most common pathological finding; however, the absence of a any statistically significant association between muscle pathology and disc displacement further suggests that muscle pathology may influence TMJ function but is unlikely to be the primary cause of internal derangement.

Pathological changes to the LPM were more common in type 2 insertion type than type 1, with 85% atrophy, 81.8% hypertrophy, and 70.6% fibrosis present in the SHLPM of the joints studied. Furthermore with 80.6% atrophy, 50% hypertrophy, and 83.3% fibrosis were present in the IHLPM of the joints studied. However, these differences were not statistically significant. **[Tables 4, 5]**

The lateral pterygoid muscle (LPM) has a distinct anatomical relationship with the TMJ, and variations in its insertion pattern have been extensively studied for their role in internal derangement [16]. Anatomical studies have demonstrated considerable variability in superior head insertion into the disc, capsule, condyle, or a combination of these structures [17]. Three insertion types (types 1, 2, 3) have been identified by both cadaveric

[2] and MRI studies [1,5]. The authors of the present study evaluated the LPM insertion patterns classified as type 1 and type 2, as proposed by Taskaya-Yilmaz et al [4], because for all practical purposes essentially the SHLPM inserts solely into the disc (type 1) or into the disc and condyle (type 2). **(Fig. 3, 4).**

Litko et al. [18] reported that LPM insertion patterns may contribute to disc displacement but are unlikely to be the primary cause, instead promoting the progression of internal derangement in the presence of other risk factors. Similar findings were reported by Imanimoghaddam et al. [5] and Omami and Lurie [19], who found no correlation between LPM attachment pattern and anterior disc displacement on MRI.

According to biomechanical research carried out by Tanaka et al [20] increased or hyperactivity of the lateral pterygoid muscle can cause excessive stress inside the articular disc and retrodiscal tissues, potentially leading to disc deformation and anterior displacement. This conclusion is consistent with an earlier review by Manfredini et al [21] which suggests that disc displacement cannot be explained purely by the lateral pterygoid muscle's attachment type or disease. However-Yılmaz et al. [4] suggested that Type I insertion may predispose to anterior disc displacement due to reduced stabilisation of the disc-condyle complex.

In the present study, type 1 LPM insertion was associated with more joints with ADDWR (57.9%) compared to ADDWoR (42.1%), but this the difference was not significant. At the same time, although the type 2 LPM insertion was associated with more joints with ADDWR (63.6%) compared to ADDWoR (37.5%), the difference was not significant. **[Table 1]**

Since the disc is attached to the lateral poles of the condyle via the lateral collateral ligaments and circumferentially to the capsule, the discal attachments are thus reinforced to the condyle and the capsule, and any anterior disc displacement (ADD) is not solely dependent on the insertion of the LPM alone – it is one among several factors. Hence the type of attachment of the LPM will not be the only factor influencing disc displacement and eventual LPM pathology.

According to Dergin et al [1], in contrast to the classical view that the superior head of the LPM is active only during closing, retrusion, and ipsilateral jaw movements, in fact, most of the superior head of the LPM does appear to be active during opening, protrusion, or contralateral jaw movements. This means that this single-motor unit not only pulls the disc but also pulls the condyle. This single-motor unit theory, that both the superior head of the LPM and the inferior head of the LPM are active during jaw movements was consistent with the findings of their study, showing that there was no correlation between the LPM attachment type and the presence or absence of disc displacement, disc degeneration or articular surface degeneration.

The etiology of temporomandibular joint (TMJ) internal derangement is multifaceted and complex. It is influenced by the bony anatomy/morphology of the TMJ like depth of the glenoid fossa, inclination of the articular eminence, morphology of the condyle, as well as the soft tissue components of the TMJ like overstretching of the retrodiscal tissue, laxity of the capsule, lateral pterygoid muscle hyperactivity, and the overall dynamic relationship and interactions between these hard and soft tissues. Moreover, factors like masticatory muscles, the occlusion, and

parafunctional habits have a significant role and significantly contribute to the etiology and progression of internal derangements of the TMJ. [18, 21, 22]

The drawbacks of this study include a limited sample size and the fact that each of the joints were studied only in isolation, without factoring-in the influence of the contralateral side. The temporomandibular joint on any side is highly influenced by the function and activity of the other side. Hence, internal derangements of the TMJ cannot be studied in isolation of only a single side but the role of the contralateral side has to be factored in as it invariably influences the pathogenesis and progression of disease on the affected ipsilateral side.

CONCLUSION: The present MRI-based study evaluated the association between lateral pterygoid muscle (LPM) insertion patterns, muscle pathology, and temporomandibular joint (TMJ) disc displacement in 54 patients (96 joints). Anterior disc displacement with reduction (DDWR) was more common (62.5% joints) compared to anterior disc displacement without reduction (DDWoR) found in 37.5% joints. Type 2 LPM insertion was the predominant pattern with 80% joints compared to type 1 LPM insertion pattern in 20% joints.

No statistically significant correlation was found between the type of LPM attachment and disc displacement (with and without reduction), the type of insertion pattern of the LPM with the pathology associated with the muscle, or between the pathological changes of the LPM with the type of disc displacement (with or without reduction).

Pathological changes to the LPM were more common in type 2 insertion type than type 1, in both the superior head as well as inferior head of the LPM. However, these differences were not statistically significant.

Logistic regression analysis further confirmed that neither LPM insertion nor muscle pathology significantly predicted internal derangement, indicating no meaningful relationship between these variables.

DECLARATIONS:

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Author Contributions: All authors contributed to the study conception and design. Material preparation, data collection and analysis were performed by Dr Eitisha Gupta and Dr Thomas Zachariah. The first draft of the manuscript was written by Dr Eitisha Gupta and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript

Ethical approval: This article does not contain any studies with animals performed by any of the authors.

Ethical approval: All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This study was approved by the Institutional Ethical Committee, Meenakshi Ammal Dental College and Hospital, Chennai, India, as per protocol number MADC/IEC/II/25/2024.

Consent to publish: The authors affirm that human research participants provided informed consent for publication of the MRI images in figures 1a to 1d, 2a to 2d, and 3a to 3c.

Consent to participate: Written informed consent was obtained from all individual participants included in the study.

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FIGURE CAPTIONS:

Fig. 1a and 1b Type 1 LPM insertion in DDWR - closed and open mouth. Fig. 1c and 1d Type 1 LPM insertion in DDWoR - closed and open mouth.

Fig. 2a and 2b Type 2 LPM insertion in DDWR - closed and open mouth. Fig. 2c and 2d Type 2 LPM insertion in DDWoR - closed and open mouth.

Fig. 3a Fibrosis in superior head of LPM, Fig. 3b Atrophy of inferior head of LPM, Fig. 3c Hypertrophy of superior head of LPM

Fig. 4 Graphical representation of type 1 LPM insertion

Fig. 5 Graphical representation of type 2 LPM insertion

Supplementary Files

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