Temporal Tendinitis in Craniomandibular Dysfunction (CMD) – Does it Really Exist? A Temporomandibular MRI Investigation

Tendinitis temporalis bei kraniomandibulärer Dysfunktion (CMD) – Welche Rolle spielt sie wirklich? Eine MRT-Studie der Temporomandibularregion

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Key words
temporal tendon, tendinitis, temporomandibular joint (TMJ), craniomandibular dysfunction (CMD), magnetic resonance imaging (MRI), head/neck, MR-imaging, inflammation

ABSTRACT

Objectives The aim of the study was to analyze the role of temporal muscle and particularly tendon pathology in patients suffering from craniomandibular dysfunction (CMD) using magnetic resonance imaging.

Materials and Methods Retrospective analysis of MRI examinations was carried out with regard to temporal muscle fibrosis and fatty degeneration and particularly temporal tendon rupture, thickening, and degenerative signal alterations. Descriptive statistics and the Mann-Whitney U-test were used for statistical evaluation.

Results Structural lesions of temporal muscle parenchyma were the absolute exception. PD hyperintensity, pronounced contrast enhancement, or peritendinous fluid collections along the temporal tendon were found only to a small extent, and a (partial) rupture occurred in only one case. The tendon diameter showed only slight variability. The Mann-Whitney U-test provided no results indicating a causal connection between degenerative joint or disc disease and temporal tendon pathology.

Conclusion A large sample of 128 magnetic resonance imaging examinations provided no evidence of a major role of temporal tendinitis in clinical CMD syndrome.

Key Points:
▪ Retrospective analysis of temporal tendon in CMD patients.
▪ Abnormal structural findings along the tendon seen only rarely.
▪ Obviously no crucial role of temporal tendon lesions in CMD syndrome.

ZUSAMMENFASSUNG

Ziel Ziel der vorliegenden kernspintomografischen Untersuchung ist es, die Bedeutung von Läsionen des Musculus temporalis und insbesondere der Temporalissehne für das klinische Syndrom der kraniomandibulären Dysfunktion (CMD) zu untersuchen.

Material und Methoden In einer retrospektiven Analyse von MRT-Untersuchungen der Kiefergelenke wurden Fibrose und fettige Degeneration des Musculus temporalis und insbesondere Veränderungen der Temporalissehne beurteilt. Zur statistischen Aus-
Eine retrospektive Analyse der Sehne des Musculus temporalis bei CMD-Patienten.

Nur selten strukturelle Läsionen der Temporalissehne.

Offensichtlich keine wesentliche Rolle von Läsionen der Temporalissehne beim klinischen CMD-Syndrom.

**ABBREVIATIONS**

CMD Craniomandibular dysfunction
MRI Magnetic resonance imaging
PD Proton density
TMJ Temporomandibular joint
MR Magnetic resonance
T Tesla
PACS Picture archiving and communicating system
API Average pixel intensity
PD Proton density weighted
SPAIR Spectral attenuated inversion recovery
ADC Apparent diffusion coefficient

**Introduction**

Craniomandibular dysfunction (CMD) ist ein weitverbreitetes und variabeles klinisches Syndrom, das in der weiblichen Population bei etwa 8 % bis 15 % Frauen und 3 % bis 10 % Männer auftritt [1]. In den letzten Jahrzehnten ist die dokumentierte Behandlungsbedürftigkeit gestiegen [2]. CMD wird durch Gelenk- und Gelenkverbundenschmerzen, Arthrose, Gelenkverweichung, Mazeration, Synovialveränderung, Kapselveränderung, Tendinitis, Myositis, Myalgien und Schmerzen in der Zahnhalsregion gekennzeichnet [3].

Die temporomandibulären Gelenke (TMJ) sind strukturell stark komplex, insbesondere die intraartikulären Strukturen wie die Gelenkknorpel, die Gelenkkapsel, die Gelenkknorpelprothese, die Muskeleigenschaften und die Muskelläisionen werden unter klinischen Bedingungen häufig zur Ursache von Schmerzen und Dysfunktionen genannt [3].

**Slovakia**

The Slovakian language term is not available in the provided text.

**Swiss German**

Deutschsprachige Version ist nicht verfügbar in der bereitgestellten Textversion.

**The temporalis muscle** ist die wichtigste Kiefermuskulatur, die aus einer großen kranialen Ursprungsgegend besteht. In der Literatur werden drei Teile der temporalis Muskeln beschrieben: der oberflächliche Teil, der zygomaticus Teil, und ein komplexer tiefen Teil [11–14]. Der obere Teil gliedert sich aus dem temporalen Aponeurosen und der temporalen Sehne. Der zygomaticus Teil der temporalis Muskeln entspringt vom rostral- und medialen Zygoma und Zygoma-Bogen und entspringt dem oberflächlichen Teil und Coronoidealhöcker. Der komplex tiefen Teil entspringt der vorderen Oberflächenflächen der Temporalis, einige Fasern inserieren in die Innenseite der oberflächlichen temporalis Muskeln, die meisten Fasern inserieren in den inneren Aspekt des Coronoidealhöckers. Diese anatomischen Befunde wurden in mehreren Magnetresonanztomographie-Untersuchungen [11, 12, 14] (▶ Fig. 1).

**The origin of temporal tendon pain syndromes** wurde der Insertion der Sehnen von Sharpeys' Fasern in den Coronoidealhöcker zugeschrieben [15]. Midfacial pain, temporal headache, and painful sensations over the ear to the occiput were classified as typical clinical signs of temporal tendinitis. In a large sample of 449 CMD patients, these symptoms were localized midfacially in...
68 %, temporally in 53 %, and aurally in 9 % of cases [3]. Palpation of the temporal tendon and tendon blocks using local anesthesia should have improved diagnostic reliability. However, the authors concede that it is difficult to differentiate complex pain syndromes induced by temporal tendon lesion from primarily joint-related symptoms [3].

Despite this difficult clinical definition and differential diagnosis, a major role of temporal tendinitis in CMD-like pain syndromes is repeatedly postulated in the literature [3, 15, 16]. Our impression of divergent clinical interpretation and radiological findings in daily routine work paved the way for an MRI examination of the temporal tendon in a large and clinically heterogeneous group of CMD patients. Magnetic resonance imaging is generally considered the best method for diagnostic assessment of temporomandibular joint status [17]. MRI studies evaluating the masticatory muscles and their tendons are limited [18–20]. A brief review of the current literature did not identify any recent study of this kind.

Materials and methods

A total of 64 patients underwent an MRI examination of the temporomandibular joint (TMJ) and surrounding structures (128 joints) in the years 2013 to 2019 and were evaluated in a retrospective study. The patients were all referred from the Department of Oral and Maxillofacial Surgery with the diagnosis of craniomandibular dysfunction (CMD). The patients’ age was between 12 and 74 years with an average age of 39.1 years. That means that the study population is inhomogeneous in age due to the retrospective study design. In accordance with clinical experience, most of the patients were female (68 %). All patients were examined with the same 3.0 T MRI scanner (Philips Ingenia) using a dedicated four-channel temporomandibular joint coil (Philips D-Stream-Flex Type S). The 3.0 T high-field MRI scanner is considered superior to the 1.5 T scanner for imaging of the TMJ because of significantly better resolution of the anatomical structures, particularly of the articular disc and also the surrounding periaricular muscles and tendons. We used the same examination protocol for all patients (▶ Table 1). Gadoteric acid (Dotagraf, Jenapharm, 0.2 ml/kg) was used as the MRI contrast agent. The examinations were all saved in the internal PACS system and were evaluated by two experienced radiologists and one neuroradiologist in consensus (▶ Table 2).

The MRI scans were evaluated with respect to signs of osteoarthritis and articular disc dislocation. Special attention was focused on the temporal muscle tendon, the converging temporal muscle fibers, and the insertion of the tendon at the coronoid process. The temporal tendon was depicted in all three planes with a focus on the sagittal plane. The tendon was completely shown in each case and also by far the largest part of the temporal muscle. The temporalis muscle parenchyma was analyzed with respect to fatty degeneration and fibrosis while the tendon was inspected for irregular contour and rupture. Furthermore, images were examined for local fluid collections around the temporal tendon as a sign of irritation and overstraining as well as for contrast enhancement in the insertion area. For this purpose, the average

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Technical parameter MRI.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sequence</strong></td>
<td><strong>TR</strong></td>
</tr>
<tr>
<td>PD SPAIR parasag</td>
<td>1978</td>
</tr>
<tr>
<td>T1 parasag</td>
<td>741</td>
</tr>
<tr>
<td>T1 parasag + KM</td>
<td>741</td>
</tr>
<tr>
<td>T1 parasag + KM max.</td>
<td>741</td>
</tr>
<tr>
<td>T1 cor + KM</td>
<td>677</td>
</tr>
<tr>
<td>T1 cor + KM max.</td>
<td>677</td>
</tr>
<tr>
<td>T2 ax</td>
<td>2124</td>
</tr>
</tbody>
</table>

FOV: field of view; KM: contrast medium; KM max: contrast medium and maximal opening; MRI: magnetic resonance imaging; PD SPAIR: proton density spectral attenuated inversion recovery sequence; TR: repetition time; TE: echo time.

<table>
<thead>
<tr>
<th>Table 2</th>
<th>Descriptive statistics: Summary of gender, arthrosis, disc position (n = normal, yes = anterior disc dislocation). All patients were symptomatic in particular with pain and movement restriction.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>gender</strong></td>
<td><strong>Count</strong></td>
</tr>
<tr>
<td>male</td>
<td>21</td>
</tr>
<tr>
<td>female</td>
<td>43</td>
</tr>
<tr>
<td><strong>arthrosis (R)</strong></td>
<td><strong>Count</strong></td>
</tr>
<tr>
<td>no</td>
<td>50</td>
</tr>
<tr>
<td>yes</td>
<td>14</td>
</tr>
<tr>
<td><strong>arthrosis (L)</strong></td>
<td><strong>Count</strong></td>
</tr>
<tr>
<td>no</td>
<td>42</td>
</tr>
<tr>
<td>yes</td>
<td>22</td>
</tr>
<tr>
<td><strong>disc position (R)</strong></td>
<td><strong>Count</strong></td>
</tr>
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</tr>
<tr>
<td>yes</td>
<td>17</td>
</tr>
<tr>
<td><strong>disc position (L)</strong></td>
<td><strong>Count</strong></td>
</tr>
<tr>
<td>no</td>
<td>44</td>
</tr>
<tr>
<td>yes</td>
<td>20</td>
</tr>
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</table>

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pixel intensity (API, dimensionless) of the proton density – spectral attenuated inversion recovery (PD – SPAIR) signal in the temporal tendon immediately at the point of insertion was measured. We chose an ROI diameter of 5 mm to reduce the effects of very small signal inhomogeneities. With the same procedure, a signal increase after the application of contrast medium was detected as a possible expression of overstraining and concomitant inflammatory changes of the temporal tendon. In addition, the diameter of the temporal tendon was measured immediately at the point of insertion in the coronoid process of the mandible.

In a further step the association of signs of supposed temporal tendinitis and typical CMD findings like osteoarthritis and disc dislocation were analyzed.

Statistical evaluation was performed with the support of the Institute for Medical Statistics and Epidemiology in our clinic using the following methods:

Descriptive statistics was applied in the absence of a larger healthy control group and non-availability of normal values. Box-Whisker plots were used for graphic visualization of frequency distributions.

The Mann-Whitney U-test was applied to evaluate whether two independent samples selected from populations have the same distribution. P-value as the level of statistical significance was 0.05.

**Results**

We did not find a single muscle with typical signs of fibrosis (and consecutive contracture) appearing as areas of low signal intensity on T1 and PD-weighted MR images. This is remarkable because all patients showed clinical CMD signs including movement restriction of the jaw. Fatty replacement of muscle parenchyma characterized by hyperintensity on T1-weighted images and corresponding signal loss in PD SPAIR was found in only two cases (≈1.6%) to a minor extent (less than one third of muscle fibers). In only one case, a partial rupture of the temporalis tendon near the insertion was documented showing discontinuity of tendon fibers, irregular margins, enlargement of the tendon diameter, and strong PD hyperintense signal alterations. We accordingly analyzed our MRI scans for pronounced fluid signal along the tendon and peritendinous junction of the temporalis muscle. Clearly increased fluid signal was only found in one patient on both sides (two joints) (≈1.6%) (Table 3).

In addition to these qualitative analyses, the average pixel intensity on PD and contrast-enhanced images was measured in the tendon itself as described above, as a possible sign of tendon damage and accompanying inflammatory irritation. Furthermore, the diameter of the temporal muscle tendon near the insertion was measured. For statistical evaluation we used descriptive statistics and corresponding boxplot visualization.

In our sample the bandwidth of measured tendinous PD signal intensity and contrast enhancement was narrow. Very few statistical outliers suggesting tendon alterations were found. This assessment is supported by the finding of a largely stable and symmetric tendon diameter in nearly all patients with a mean tendon thickness of 7.0 millimeters, standard deviation of 1 millimeter and a small range of 5 to 9 millimeters. Only one exception was found in the case of a partial tendon rupture.

The Mann-Whitney U-test shows that the distribution of the PD signal of the tendon, contrast enhancement of the tendon, and tendon diameter do not correlate with the extent of arthrosis and disc displacement. There is no evidence of a causal connection in this test.

**Conclusion**

In conclusion, lesions of the temporal muscle parenchyma like fibrosis and fatty degeneration could only be detected in a very low percentage of cases. Pathologic changes of the temporal tendon (peritendinous fluid collection, rupture) were also the exception.

**Table 3** Descriptive statistics: Summary of temporal muscle and tendon lesions. Remarkably low number of morphologically perceptible lesions on temporalis muscle and tendon.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Count</th>
<th>Column N %</th>
</tr>
</thead>
<tbody>
<tr>
<td>arthrosis (R)</td>
<td>no</td>
<td>50 78.1%</td>
</tr>
<tr>
<td>arthrosis (L)</td>
<td>yes</td>
<td>14 21.9%</td>
</tr>
<tr>
<td>lesion of myotendinous junction (R)</td>
<td>no</td>
<td>60 93.8%</td>
</tr>
<tr>
<td>lesion of myotendinous junction (L)</td>
<td>yes</td>
<td>4 6.3%</td>
</tr>
<tr>
<td>muscle atrophy (R)</td>
<td>no</td>
<td>63 98.4%</td>
</tr>
<tr>
<td>muscle atrophy (L)</td>
<td>yes</td>
<td>1 1.6%</td>
</tr>
<tr>
<td>muscle fibrosis (R)</td>
<td>no</td>
<td>64 100.0%</td>
</tr>
<tr>
<td>muscle fibrosis (L)</td>
<td>yes</td>
<td>0 0.0%</td>
</tr>
<tr>
<td>tendon rupture (R)</td>
<td>no</td>
<td>63 98.4%</td>
</tr>
<tr>
<td>tendon rupture (L)</td>
<td>yes</td>
<td>1 1.6%</td>
</tr>
</tbody>
</table>

In conclusion, lesions of the temporal muscle parenchyma like fibrosis and fatty degeneration could only be detected in a very low percentage of cases. Pathologic changes of the temporal tendon (peritendinous fluid collection, rupture) were also the exception.
Moreover, temporal tendinitis cannot be regarded as a typical component of degenerative temporomandibular joint disease or the clinical CMD complex.

**Discussion**

Patients with myogenous CMD [4, 5] are reported to have even more severe pain syndromes and comorbidity than patients with arthrogenous CMD [4]. Among these presumed myogenous pain syndromes, temporal-tendon-related symptoms, also classified as temporal tendinitis, and its clinical importance was discussed in one study [10]. In a retrospective study, up to 78% of CMD patient are reported to show typical signs of temporal tendinitis [3, 10]. However, this classification is based purely on clinical findings like facial pain, temporal pain, pain radiating over the ear to the head and neck, and referred pain to the temporomandibular joint, each aggravated when the temporal tendon was palpated, according to the authors [3, 10]. However, these results are not substantiated by a systematic MRI study including a larger number of patients.

On detailed investigation of the examined temporal muscle parenchyma, our study showed only isolated cases of fatty replacement and no fibrosis. Obviously, the common symptom of movement restriction of the jaw is not caused by muscle fibrosis, rather, joint structures (disc or capsule) are likely to be the reason.

In musculoskeletal radiology, peritendinous T2-hyperintense fluid collections are considered a significant indicator of tendon damage caused by overstrain and degeneration [21]. The finding of only one partial tendon rupture and of peritendinous fluid collections in just two cases stands in marked contrast to published clinical evaluations and interpretations. These results suggest that the

**Fig. 2** Temporal muscle and tendon – fatty muscle fiber replacement. PD fat sat sagittal plane. Fatty replacement of muscle parenchyma (arrow), normal signal of temporal tendon.

**Fig. 3** Temporal muscle and tendon – peritendinous fluid collection. PD fat sat sagittal plane. PD hyperintense fluid collection along the temporal tendon (arrow). The tendon itself is intact.

**Fig. 4** Temporal muscle and tendon – partial rupture of the tendon. PD fat sat sagittal plane. Partial disruption of tendon fibers (arrow), irregular contour of the tendon. PD hyperintense signal alteration in and around the tendon.
role of temporal tendinitis in CMD syndrome might be overestimated. The analysis of PDw signal alteration and contrast enhancement in the tendon of CMD-patients showed only 3 (left) and 4 (right) markedly elevated values that are considered pathological in descriptive statistics. These findings in descriptive statistics strongly argue against substantial lesions of the temporal tendon in our CMD patient group. Our morphological findings on MR imaging cannot confirm the existence of a chronic inflammatory disease of the temporal tendon (▶Fig. 5, 6).

The criterion of tendon thickening [10, 21] was also considered. Given a lack of generally recognized norms, we analyzed the range of tendon thicknesses, which proved to be small between 5 and 9 millimeters, with a mean of 7 millimeters. All measurement results are within twice the standard deviation. The tendons were consistently smoothly contoured.

The Mann-Whitney U-Test showed no clear correlation (with one less significant exception) between disc displacement or osteoarthritis and temporal tendon signal alterations or pronounced contrast enhancement. This speaks against temporal tendinitis being a typical component of degenerative or inflammatory disease of the temporomandibular joint region (▶Table 4).

A clear tendency to a substantial elongation of the coronoid process was also not recognizable in our group of patients [22].

These results may also have an impact on the choice of therapy. Various local infiltration therapies are propagated for CMD patients, including the use of local anesthetics, corticosteroids [23], and especially botulinum toxin [10, 24–27]. Such therapeutic applications in the temporal tendon region have to be discussed in view of the morphological results on MRI, especially considering the side effects of this therapy. The described symptom relief from such treatment could also be the result of diffuse denervation or anti-inflammation in the craniomandibular region and not a specific effect on an irritated or inflamed temporalis tendon. It should be taken into account that medium- or long-term botulinum toxin application causes significant side effects like masticatory function decline [28].

In summary, the present retrospective analysis of MRI examinations of the temporomandibular joint in symptomatic patients with craniomandibular dysfunction did not reveal substantial chronic degenerative or acute inflammatory changes of the tendon of the temporalis muscle.

The fact that inflammatory processes in the synovial membrane can indeed play a role in clinical CMD syndrome was described in a newer MRI study of the temporomandibular joint from 2019 [29].

In a further step, the assessment of the masticatory muscle and tendon pain may be improved using diffusion-weighted MRI. The apparent diffusion coefficient (ADC) values of the masticatory muscles on the pain side proved to be significantly greater than those on the contralateral side without pain. Even quantitative validation of masticatory muscle myalgia should be possible [30].
Clinical relevance

- Up to now, temporal tendinitis has been assigned an important role in CMD.
- In our MRI study structural lesions were only rarely found in the temporal tendon (and muscle).
- According to our investigation, temporal tendinitis does not play a major role in clinical CMD syndrome.
Ethical approval

Ethical approval was given by the Ethics Committee of the Technical University Munich (relevant judgement reference number 491/19 S-SR). Governing law (Bay KHG Art. 27/4) explicitly allows the use of stored clinical data (in anonymized form) for research purposes. Furthermore, every patient signs a corresponding treatment contract.

Patient consent

The study records of human patients (stored MRI images) were used in anonymized form for this retrospective analysis. All patients were examined for a clinical indication. There was no MRI examination performed primarily for study purposes.

Statistical assistance

Statistical evaluation was performed with the support of the Institute for Medical statistics and Epidemiology in our clinic.

All authors have viewed and agreed to the submission.

Conflict of Interest

The authors declare that they have no conflict of interest.

References