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

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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.

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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 Verdickung, degenerative Signalveränderungen und Ruptur der Temporalissehne beurteilt. Zur statistischen Aus-

wertung kamen deskriptive Statistik und der Mann-Whitney-U-Test zur Anwendung.

Ergebnisse Strukturelle Läsionen des Musculus temporalis waren die absolute Ausnahme. Nur vereinzelt wurden ein Signalanstieg in PD-gewichteten Sequenzen, eine vermehrte KM-Aufnahme oder peritendinöse Flüssigkeitsansammlungen entlang der Temporalissehne gefunden, eine (Teil-)Ruptur der Sehne nur in einem Fall. Der Sehnendurchmesser zeigte nur eine geringe Variabilität. Der Mann-Whitney-U-Test erbrachte keine Hinweise auf eine kausale Verbindung zwischen degenerativer Gelenkerkrankung und Läsionen der Temporalissehne.

Schlussfolgerung In einer größeren Stichprobe von 128 MRT-Untersuchungen der Kiefergelenke fanden sich keine Hinweise auf eine wesentliche Rolle der temporalen Tendinitis beim klinischen Syndrom der kraniomandibulären Dysfunktion (CMD).

Kernaussagen:

- 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

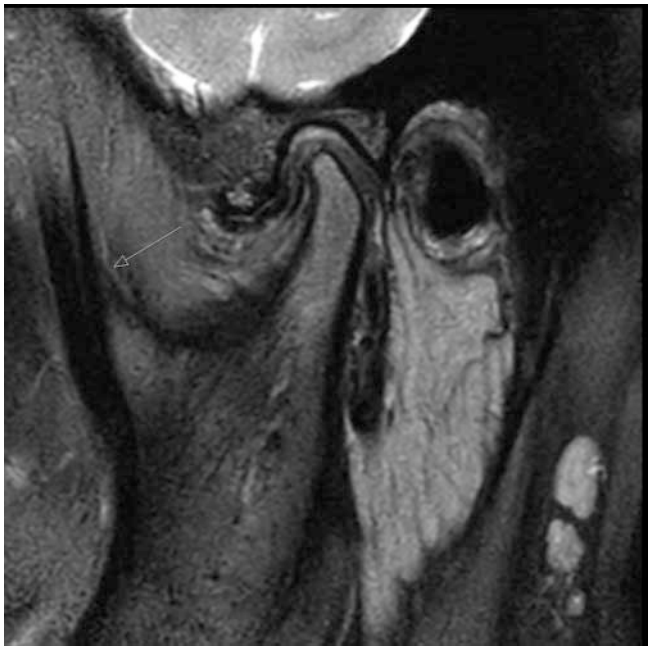
Introduction

Craniomandibular dysfunction (CMD) is a widespread and variable clinical syndrome, the prevalence of which is 8 %to 15 % in women and 3 % to 10 % in men [1]. In recent decades, the documented treatment need has increased [2]. CMD is characterized by joint pain including referred pain in other head and neck areas, joint clicking, limited mouth opening, and jaw deviations. It is assumed that the pain syndrome arises **due to a disorder or malfunction** in several joint or joint-related structures like bone and cartilage, disc, synovial membrane, capsule, tendon and ligaments, and especially also the masticatory muscles [3].

The temporomandibular joint (TMJ) structures, especially the intraarticular disc, are analyzed in great detail in a lot of imaging studies. Beyond that, however, there are numerous clinical reports that state that extracapsular structures like the muscles of mastication and their tendons also play an important role in temporomandibular joint pain triggering. Therefore, a differentiation between arthrogenous and myogenous CMD has been established [4–7]. Particularly pathologic changes of the temporal muscle and its tendon in CMD-like pain syndromes are emphasized in the literature [3, 8–10].

The temporalis muscle is the most important jaw closing muscle with a large cranial muscle origin area. In the literature three parts of the temporalis muscle are described: the superficial part, the zygomatic part, and a complex deep part [11–14]. The superficial part originates from the temporal aponeurosis and the temporal lines of the parietal bone and inserts into the coronoid process of the mandible. The zygomatic part of the temporalis muscle originates from the rostral and medial zygoma and zygomatic arch and inserts into the tendon of the superficial part and coronoid process. The complex deep part originates from the anterior surface of the temporal fossa, some of the fibers insert into the inner surface of the superficial temporalis muscle, most of them into the internal aspect of the coronoid process. These anatomical findings are confirmed by several MRI studies [11, 12, 14] (▶ **Fig. 1**).

The origin of temporal tendon pain syndromes was attributed to the insertion of the tendon's Sharpey's fibers into the bone of the coronoid process [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



▶ **Fig. 1** Temporal tendon – normal finding. PD fat sat sagittal plane. Homogenous temporal tendon (arrow) showing normal signal and regular contour, no peritendinous signal alterations. Normal signal of muscle parenchyma near the myotendinous junction.

▶ **Table 1** Technical parameter MRI.

Sequence	TR	TE	Slice [mm]	Distance [mm]	FOV [mm]
PD SPAIR parasag	1978	40	2	2.2	80
T1 parasag	741	12	2	2.2	80
T1 parasag + KM	741	12	2	2.2	80
T1 parasag + KM max.	741	12	2	2.2	80
T1 cor + KM	677	14	2	2.2	80
T1 cor + KM max.	677	14	2	2.2	80
T2 ax	2124	100	4	8	230

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.

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 periarticular 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**).

▶ **Table 2** 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.

descriptive statistics (n = 64; R = right; L = left)			
		Count	Column N %
gender	male	21	32.8 %
	female	43	67.2 %
arthrosis (R)	no	50	78.1 %
	yes	14	21.9 %
arthrosis (L)	no	42	65.6 %
	yes	22	34.4 %
disc position (R)	no	47	73.4 %
	yes	17	26.6 %
disc position (L)	no	44	68.8 %
	yes	20	31.3 %

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

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.

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

descriptive statistics (n = 64; R = right; L = left)			
		Count	Column N %
arthrosis (R)	no	50	78.1 %
	yes	14	21.9 %
arthrosis (L)	no	42	65.6 %
	yes	22	34.4 %
lesion of myotendinous junction (R)	no	60	93.8 %
	yes	4	6.3 %
lesion of myotendinous junction (L)	no	60	93.8 %
	yes	4	6.3 %
muscle atrophy (R)	no	63	98.4 %
	yes	1	1.6 %
muscle atrophy (L)	no	63	98.4 %
	yes	1	1.6 %
muscle fibrosis (R)	no	64	100.0 %
	yes	0	0.0 %
muscle fibrosis (L)	no	64	100.0 %
	yes	0	0.0 %
tendon rupture (R)	no	63	98.4 %
	yes	1	1.6 %
tendon rupture (L)	no	63	98.4 %
	yes	1	1.6 %

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.

▶ **Fig. 2** Temporal muscle and tendon – fatty muscle fiber replacement

▶ **Fig. 3** Temporal muscle and tendon – peritendinous fluid collection

▶ **Fig. 4** Temporal muscle and tendon – partial rupture of the tendon

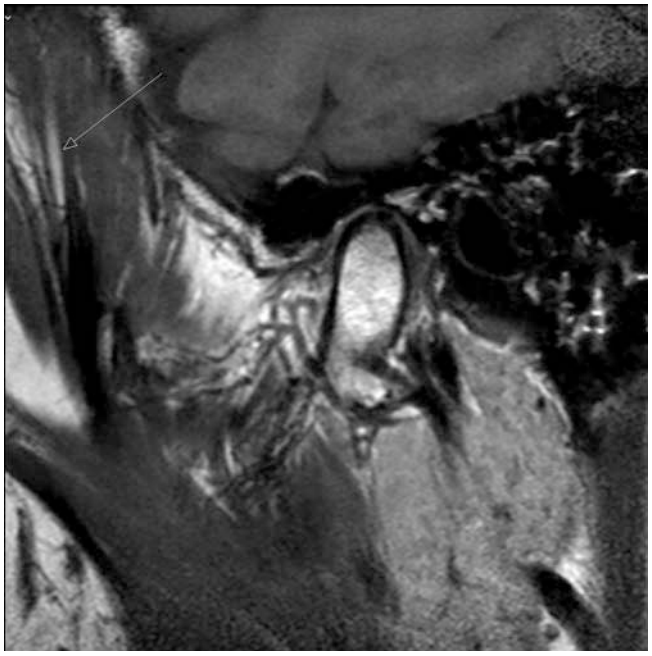
Moreover, temporal tendinitis cannot be regarded as a typical component of degenerative temporomandibular joint disease or the clinical CMD complex.

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.

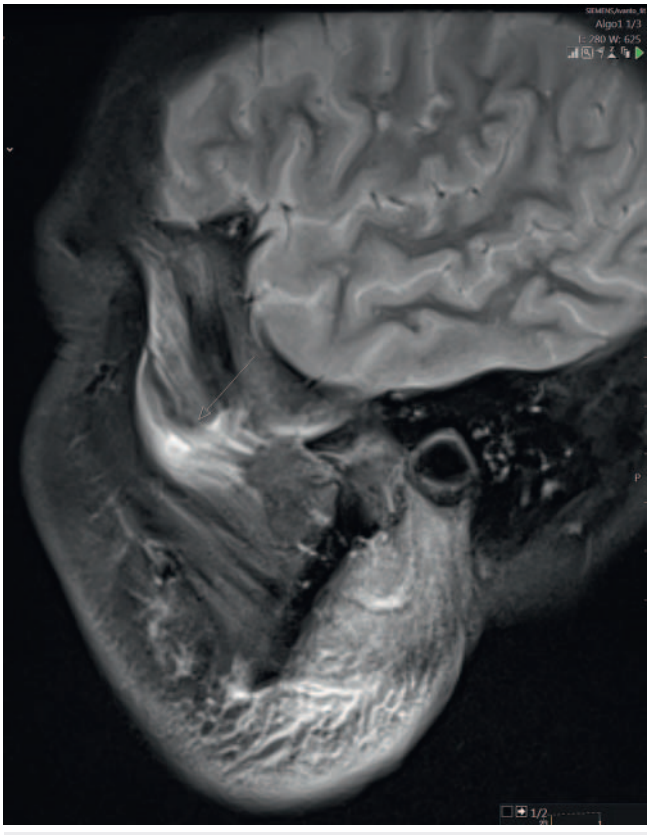
On detailed investigation of the **examined** temporal muscle parenchyma, our study **showed** only isolated cases of fatty replacement and no fibrosis. Obviously, the common symptom of movement restriction of the jaw is not caused by muscle fibrosis, rather, joint structures (disc or capsule) are likely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

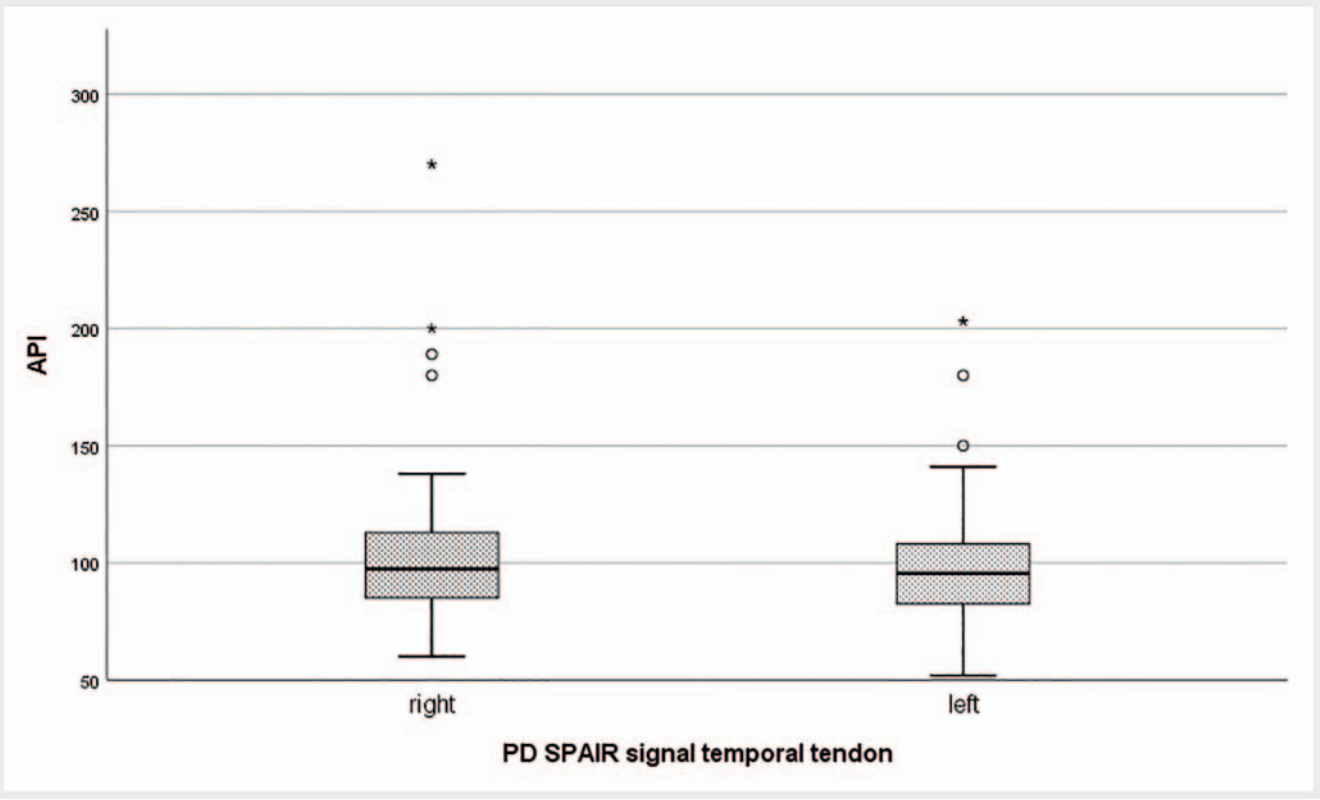


▶ **Fig. 2** Temporal muscle and tendon – fatty muscle fiber replacement. PD fat sat sagittal plane. Fatty replacement of muscle parenchyma (arrow), normal signal of temporal tendon.

▶ **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.



▶ **Fig. 5** Boxplot PDw-signal tendon: Distribution of API (average pixel intensity) in PD SPAIR images of temporal tendon. Small range of measured PD SPAIR signal in temporal tendon. Significantly deviating values suggesting tendon pathology were found only in a few cases. These patients showed disc dislocation, deformity of the capitulum, and asymmetric range of motion. In only two of these cases, discrete PD hyperintense signal alterations of the tendon were visually assessable.

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 show**ed** 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**).

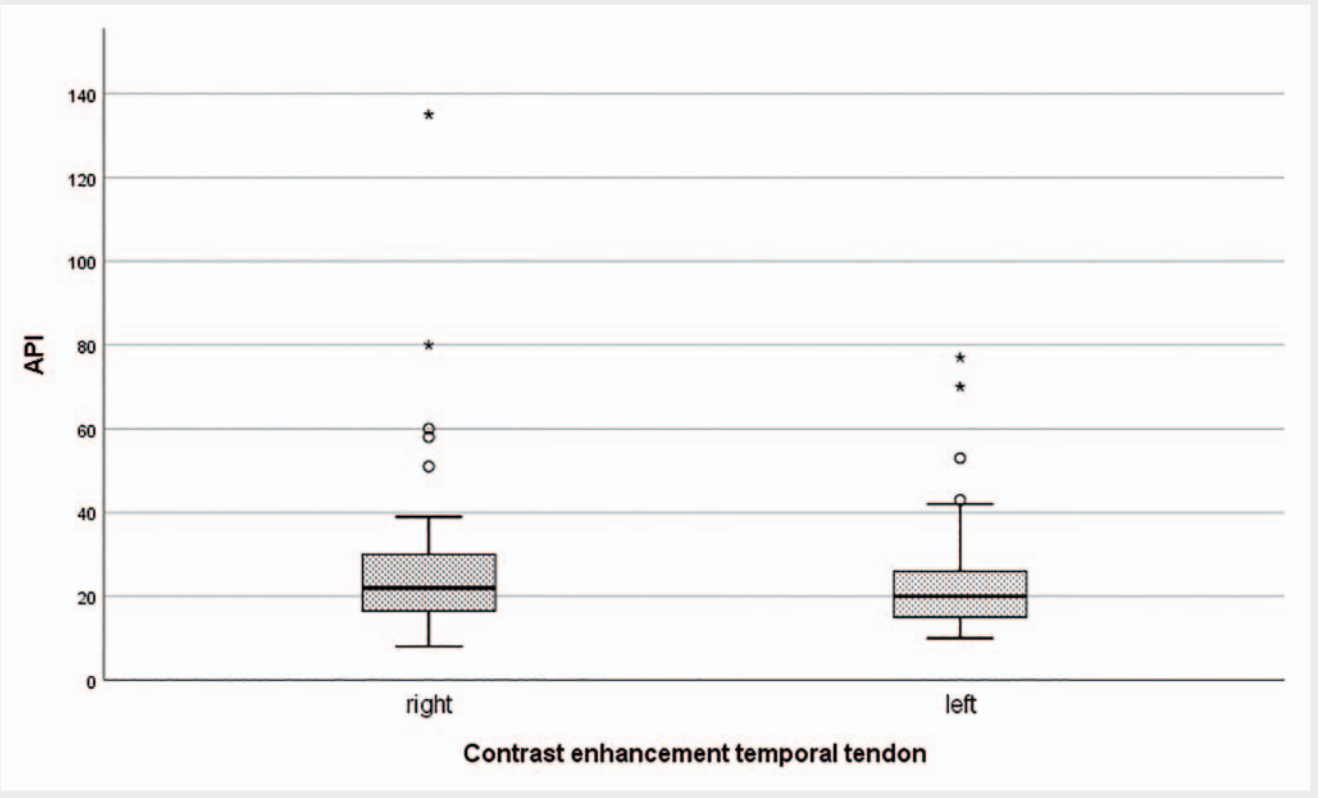
A clear tendency to a substantial elongation of the coronoid process was also not recognizable in our group of patients [22].

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].



▶ **Fig. 6** Boxplot contrast enhancement tendon: Distribution of API (average pixel intensity) on contrast-enhanced images of temporal tendon. Small range of measured contrast enhancement in temporal tendon. Outliers only in very few cases suggesting tendon lesion, in one case slightly pronounced enhancement was visually assessable. Four cases also showed simultaneously elevated API in PD-weighted images.

▶ **Table 4** Summary of results of Mann-Whitney U-Test: Distribution of temporal tendon lesions and TMJ arthrosis/disc dislocation. P-value 0.05. See text.

Grouping variable		Test variable	p-value
Arthrosis	Left y/n	PD SPAIR signal of left temporal tendon	0.159
	Right y/n	PD SPAIR signal of right temporal tendon	0.581
Arthrosis	Left y/n	Contrast enhancement of left temporal tendon	0.571
	Right y/n	Contrast enhancement of right temporal tendon	0.384
Arthrosis	Left y/n	left temporal tendon diameter	0.018
	Right y/n	right temporal tendon diameter	0.451
anterior disc dislocation	Left y/n	PD SPAIR signal of left temporal tendon	0.680
	Right y/n	PD SPAIR signal of right temporal tendon	0.215
anterior disc dislocation	Left y/n	Contrast enhancement of left temporal tendon	0.289
	Right y/n	Contrast enhancement of right temporal tendon	0.903
anterior disc dislocation	Left y/n	left temporal tendon diameter	0.502
	Right y/n	right temporal tendon diameter	0.391

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.

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