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ORIGINAL ARTICLE

Association of volume and voxel intensity of the articular disc and lateral pterygoid muscle in migraine patients: a study with magnetic resonance imaging

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ABSTRACT

Objectives: To compare the volume and voxel intensity of articular disc and lateral pterygoid muscle (LPM) between a group of migraine patients and a control group (those without history of headache) by using magnetic resonance imaging (MRI).

Patients and methods: MRI scans of 17 migraine patients and 15 healthy controls subjects were analysed and processed, using ITK-SNAP software, by a single investigator, for calculation of volumes and voxel intensity of articular disc and superior and inferior head of LPM.

Results: There were statistically significant differences between migraine patients and controls regarding the volume and voxel intensity of articular disc and inferior head of LPM, increasing in migraine patients. Intra-rater was highly consistent and reproducible (intra-class correlation coefficient [ICC] = 1).

Conclusions: Higher voxel intensity in disc and inferior head of LPM of MRI scans was linked to the increased volume of articular disc, inferior head of LPM and migraine.

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Migraine; temporomandibular joint; articular disc; headache; volumetric assessment

Introduction

Migraine is the most common disabling brain disorder [1]. Chronic migraine is a condition characterized by the experience of migrainous headache on at least 15 d per month. This condition is highly disabling as the pain affects the patient's productivity and quality of life [1,2], and thus many patients with chronic migraine also have medication overuse, which is defined as using a compound analgesic (opioid, triptan or ergot derivative) on at least 10 d per month. Migraine is characterized by intense headache, which usually manifests in a pulsating pain, unilateral or bilateral, with periodic occurrence. Nausea, vomiting, photophobia and phonophobia are the most frequent clinical symptoms. The migraine attacks can last from 4 to 72 h [2,3].

The most recent studies about the pathophysiology of migraine suggest that a primary neuronal dysfunction leads to a sequence of alterations intracranially and extracranially generating the migraine, comprising the four phases of premonitory symptoms, aura, headache and postdrome [3,4]. Strong familial involvement and genetic mutations, and early onset of migraine suggest that there is an important genetic component [5]. Trigeminal vascular model proposed by Moskowitz suggests the involvement of trigeminal nerve, as trigeminal activation results in the release of neuropeptides, therefore, generating neurogenic inflammation with amplified vascular permeability and dilation of blood vessels [5,6].

Temporomandibular disorders (TMDs) are comprised a group of varied or a series of clinical signs and symptoms that result in temporomandibular joint (TMJ) pain, masticatory muscle pain or both [7,8]. Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) Axis I [9] guidelines developed a subclassification of TMD consisting of three primary diagnostic categories [7]: I Muscle disorder subgroup (myofascial TMD, with or without limited jaw opening), II Disc displacement subgroup (reducing or non-reducing, with or without limited jaw opening) and III Arthralgia, osteoarthritis, osteoarthrosis subgroup (joint conditions).

In a retrospective analysis, migraine was reported as one of the most common diseases associated with TMD [10,11] and as well as it is more prevalent in women [12]. Some authors stated that TMD could act as a triggering factor for migraine, or *vice versa*, as their pathophysiology is closely linked to the caudate nucleus of the trigeminal nerve [11,13].

Some authors have found a connection between migraine and TMD, showing that TMD can increase the severity and frequency of migraine attacks [14,15]. Evidences showed that subjects with both migraine and TMD are predisposed to have hypertrophy of the lateral pterygoid muscle (LPM) and disc displacement [16,17].

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Considering the prevalence of migraine and TMD in the world population and the poorly understood relationship between these two comorbidities, the aim of this study was to evaluate the TMJ articular disc and LPM regarding volume and voxel intensity based on MRI examinations of migraine patients and healthy controls.

Patients and methods

Patient population

Approval from the local institutional review board was obtained according to protocol number 055/2005. Our sample consisted of a subset one used in a previous study of MRI of TMJ. We evaluated 110 MRIs of TMJ from the database of the Division of Radiology of a private hospital. Informed consent was obtained from all study subjects. The subjects were selected according to the following exclusion criteria: poor image quality and lack of some data in the patient file.

After applying the exclusion criteria, a total of 32 subjects were gathered and divided into two groups: one with patients who had migraine ($n = 17$) and the other with healthy controls ($n = 15$). The age of the subjects ranged from 16 to 71 years old, with a mean age of 38.91 ± 17.02 years. Every subject (patient or control) was investigated by neurologists according to criteria set by the 2004 International Classification of Headache Disorders.

The control group included 15 healthy volunteers without any type of headache. TMJs and masticatory muscles were clinically examined by a calibrated dentist.

All examinations were conducted after the subjects were enrolled in the study and before MRI scan.

MRI acquisition

All subjects underwent standard MRI for TMJ evaluation by using a 1.5-T MRI scanner (Sigma; General Electric, Milwaukee, WI) with a bilateral and dedicated circular polarized transmit-receive coil of 8 cm diameter. Images were obtained in closed mouth and open mouth positions, according to previous studies [16,18].

The MRI protocol used the following parameters:

- T1-weighted image (repetition time of 850 ms), echo time of 8.5 ms, section thickness of 5 mm, field of view of 180 × 180 mm and matrix of 256 × 256 pixels.
- T2-weighted fat-saturated image (repetition time of 1500 ms), echo time of 100.2 ms, section thickness of 3.0 mm, field of view 15 × 15 cm and matrix of 512 × 512 pixels.

Image analysis

All images were interpreted on a 17-inch LCD monitor in a quiet room under dim light conditions. By using an axial localizer image, it was possible to obtain parasagittal images (i.e. perpendicular to the condylar axis) for analysis of the disc-condyle relationship according to protocol proposed by Tasaki and Westesson [19], that is, in closed mouth (maximum habitual intercuspation) and open mouth positions. All images were individually evaluated by two dentomaxillofacial radiologists, both with at least 15 years of experience in MRI of TMJ, who used the Merge eFilm Workstation software, version 1.5 (Merge Healthcare, Chicago, IL). Analysis was conducted until a consensus was reached.

Measurement of volume and voxel intensity

Images were recorded in Digital Imaging and Communications in Medicine (DICOM) format and then exported to ITK-SNAP software version 3.4.0 (Cognitica, Philadelphia, PA), which calculates the volume of each segmented structure in cubic millimetres (mm^3) and voxel intensity.

One investigator, experienced in the use of ITK-SNAP, participated in this process. During image analysis, the investigator was blind to the patients' clinical information. T2-weighted fat-saturation images of each corresponding slice were processed in axial, sagittal and coronal planes to obtain the volume and voxel intensity of the disc and superior and inferior head of LPM of each TMJ. Through the segmentation process, it was possible to obtain volumetric data and T2 fat-saturation intensity. This methodology allowed determining an association between increased intensity areas in T2 fat-saturation images and increased fluid regions [20].

Right and left discs as well as superior and inferior head of LPMs in the right and left sides were segmented individually before being manually delineated by using the polygon mode tool, which uses different labels and colours to obtain the volume of each muscle. The values regarding volume and voxel intensity of the discs and LPMs were obtained by using the processing tools of ITK/SNAP similarly to other studies [20,21].

Volumetric image is a three-dimensional (3D) array of intensities in which each pixel in the image is referenced by three coordinates, namely X, Y and Z. Z indicates the slice number to which the pixel belongs, Y indicates the row in the slice and X indicates the column in the slice. The grey-level intensity of the voxel and the segmentation label corresponding to it are displayed in the tool options panel. An example of structure segmentation (i.e. articular disc and superior and inferior head of LPM) is shown in Figure 1. Each segmented structure has its own label indicating volume (mm^3), voxel count and intensity mean. After this process, the morphology of the segmented structure is also assessed in 3D.

Intra-rater accuracy was assessed by re-segmenting and reprocessing the images.

Statistical analysis

The groups were compared by using Fisher's exact, chi-square, Mann–Whitney or Kruskal–Wallis tests. The models of logistic regression were adjusted to estimate the probability of migraine in relation to the variation of each variable. The intra-class correlation coefficient (ICC) for intra-rater assessment at different moments was calculated. All data

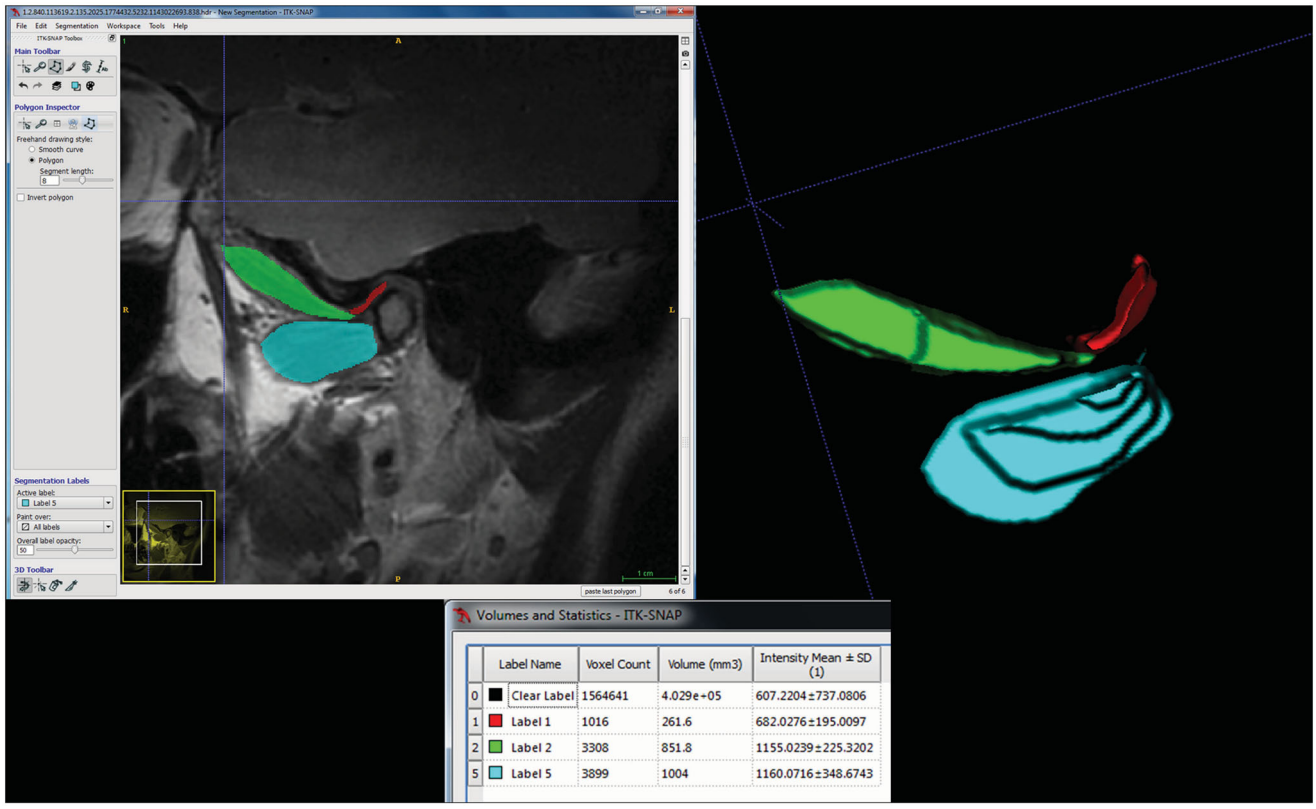

Figure 1. A parasagittal image of segmented temporomandibular joint (TMJ) components: articular disc, inferior head of lateral pterygoid muscle and superior head of lateral pterygoid muscle in ITK-SNAP software. After the segmentation process is concluded, statistics regarding voxel count, voxel intensity and volume of structures are displayed with its correspondent label. The morphologies of the segmented structures are also shown in the image.

were statistically analysed by using Minitab version 16 (Minitab, Inc., State College, PA) at the significance level of 5%.

Results

A total of 32 subjects participated in the study, of whom 25 (78.13%) were female and seven were male (21.87%). Their age ranged from 16 to 71 years old, with mean age of 38.91 and a standard deviation of 17.02 years. The total number of patients with migraine was 17 (53.13%). The female:male ratio in migraine patients was 3.25:1; the female:male ratio in controls was 4:1.

Table 1 shows the frequency and percentage of patients with migraine in each disc displacement category. In the right side, one can note that anterior disc displacement with reduction (ADDR) was observed in 52.94% of the patients with migraine and in 6.67% of the controls, whereas anterior disc displacement without reduction (ADDWR) was observed in 35.29% of the migraine patients and in none of the controls. In normal position (NP), there were 11.76% of patients with migraine and 93.33% of normal ones. Fisher's exact test ($p < .0001$) indicates that the categories ADDR and ADDWR are more associated with migraine. In the left side, one can note that ADDR was observed in 52.94% of the patients with migraine and in 20% of the controls, whereas ADDWR was observed in 5.88% of the patients with migraine and in none of the controls. In NP, there were 41.18% of patients with migraine and 80% of normal ones. Disregarding the ADDWR and considering the low frequency, chi-square test ($p = .0384$) showed that ADDR is more associated with migraine.

Table 1. Association of the disc position with headache.

	Right-side		Left-side	
Disc position	Migraine	Normal	Migraine	Normal
ADDR	9 (52.94%)	1 (6.67%)	9 (52.94%)	3 (20.00%)
ADDWR	6 (35.29%)	0 (0.00%)	1 (5.88%)	0 (0.00%)
ND	2 (11.76%)	14 (93.33%)	7 (41.18%)	12 (80.00%)

ADDR: anterior disc displacement with reduction; ADDWR: anterior disc displacement without reduction; ND: non-displaced disc.

Table 2 shows the descriptive measures of the numerical variables (i.e. volume and voxel intensity) per group of migraine patients. Significant differences were found between the groups regarding volume of right articular disc ($p < .0001$; volume was higher in migraine patients), voxel intensity of right articular disc ($p < .0001$; voxel intensity was higher in migraine patients), volume of inferior head of LPM in the right side ($p = .0127$; volume was higher in migraine patients), volume of inferior head of LPM in the left side ($p = .0005$; volume was higher in migraine patients), voxel intensity of inferior head of LPM in the right side ($p = .0127$; voxel intensity was higher in migraine students) and voxel intensity of inferior head of LPM in the left side ($p = .0005$; voxel intensity was higher in migraine patients).

The variables presented in Table 2 were assessed based on the simple logistic regression model, which is aimed at estimating the increase in the probability of migraine in relation to the increase in the values of these variables. The results are shown in chart form (Figure 2), with significant

Table 2. Comparison between the migraine groups in the numerical variables (Mann–Whitney test).

Variable	Group	N	Mean	Standard deviation	Minimum	Median	Maximum	p Value
VOL_Disc_RS	MIGRAINE	17	159	32	106	157	229	**<.0001**
	NORMAL	15	91	29	29	94	143	
VI_Disc_RS	MIGRAINE	17	618	120	443	608	889	**<.0001**
	NORMAL	15	352	114	112	366	556	
VOL_LPM_Sup_RS	MIGRAINE	17	1433	2123	324	1033	9455	.3262
	NORMAL	15	746	406	101	661	1655	
VI_LPM_Sup_RS	MIGRAINE	17	3648	1887	1258	4013	7927	.3647
	NORMAL	15	2898	1578	393	2568	6427	
VOL_LPM_Inf_RS	MIGRAINE	17	5014	7859	1022	3210	35,175	**.0127**
	NORMAL	15	2204	809	1335	1966	3701	
VI_LPM_Inf_RS	MIGRAINE	17	12,335	4590	3969	12,466	21,512	**.0127**
	NORMAL	15	8560	3140	5183	7636	14,375	
VOL_Disc_LS	MIGRAINE	17	126	44	28	146	182	.1216
	NORMAL	15	101	37	48	100	171	
VI_Disc_LS	MIGRAINE	17	492	167	108	568	706	.1309
	NORMAL	15	397	146	186	388	665	
VOL_LPM_Sup_LS	MIGRAINE	17	1794	3621	180	899	15,708	.2417
	NORMAL	15	711	328	155	622	1238	
VI_LPM_Sup_LS	MIGRAINE	17	3780	2102	698	3492	7544	.2417
	NORMAL	15	2762	1276	602	2414	4806	
VOL_LPM_Inf_LS	MIGRAINE	17	5433	8077	1332	3592	36,375	**.0005**
	NORMAL	15	2173	675	1149	2079	3224	
VI_LPM_Inf_LS	MIGRAINE	17	13,720	5039	5171	13,949	27,807	**.0005**
	NORMAL	15	8438	2620	4461	8073	12,522	

VI: voxel intensity; LPM: lateral pterygoid muscle; LS: left-side; RS: right-side; Sup: superior; Inf: inferior; VOL: volume; N: sample size.
The bold values signify $p < .05$.

odds ratio (OR) and 95% confidence intervals being presented in blue. The variables, whose 95% confidence intervals and OR were not significant for migraine are in grey.

By using the stepwise regression model for multiple multivariate analysis of all variables (Table 2), it was concluded that the voxel intensity of inferior head of LPM in the left side was more important to explain the migraine ($p = .0005$).

Table 3 presents the comparison of disc displacements with other TMJ measures on the same side. Significant differences were observed in disc volume ($p = .0009$; ADDR and ADDWR had higher volume than non-displaced disc [ND]), voxel intensity of disc ($p < .0001$; ADDR and ADDWR had higher voxel intensity than ND) and voxel intensity of superior head of LPM ($p = .0481$; ADDWR had was higher voxel intensity than ND).

For comparison of disc displacement in the left side (Table 4), the patient with unilateral ADDWR was disregarded. Significant differences were observed in disc volume ($p = .0005$; ADDR had higher volume than ND) and in voxel intensity of disc ($p < .0001$; ADDWR had higher intensity voxel than ADDR and ND) when comparing ADDR with ND.

Table 5 shows that the method has excellent reliability, as long as the ICC is 1 for all variables.

Discussion

The results regarding the association between anterior displacement of the articular disc and reduction in migraine as found in this study are reinforced by previous findings. For instance, Lopes et al. [16] assessed 20 patients with migraine by means of MRI, reporting anterior disc displacement in 70% of the patients. Other studies, such as those by Goncalves et al. [22] and Ballegaard et al. [15], confirmed the relationship of migraine with TMD and possible internal derangement, such as disc displacements with reduction. It was also emphasized that the relationship between tension-type headache and presence of TMD symptoms have been demonstrated, but investigations on the association between TMD and migraine still has some incongruities [14].

In our study, a higher percentage of disc displacements with reduction was found to be in association with migraine patients, which is in accordance with the literature [16]. It should be considered, however, that the evolution of disc displacement with reduction into a possible one without reduction is a fact to be considered in these patients, but this would only be possible through a longitudinal study.

The voxel intensity values of the articular disc and LPM reflect the greyscale mean for these structures, which in turn constitute the MRI signals emitted by their tissues. In this study, the T2-weighted sequence was chosen because it is the most used parameter in the evaluation of pathological changes [23,24]. The T2-weighted sequence reflects a region of tissue with fluid (e.g. effusion, oedema and hydropic degeneration), which would be the accumulation of water in the intracellular environment (i.e. cell hyperhydration) as a result of imbalances in the control of osmotic gradient at cytoplasmic membrane level and in the mechanisms of absorption, elimination of water and intracellular electrolytes [24]. An increase in T2 signal intensity reflects an increase in mean voxel intensity, which in turn would indicate a biological change in the structure under study [20]. It is noticeable that a normal articular disc has a hyposignal and, therefore, low voxel intensity, whereas a muscle has intermediate signal at T2 and median voxel intensity [25,26]. In this study, this methodology is pioneering in relation to this type of analysis.

Image evaluation of the LPM is important because its palpation is quite controversial [27] due to its location and difficult access. The findings of this study showed that the

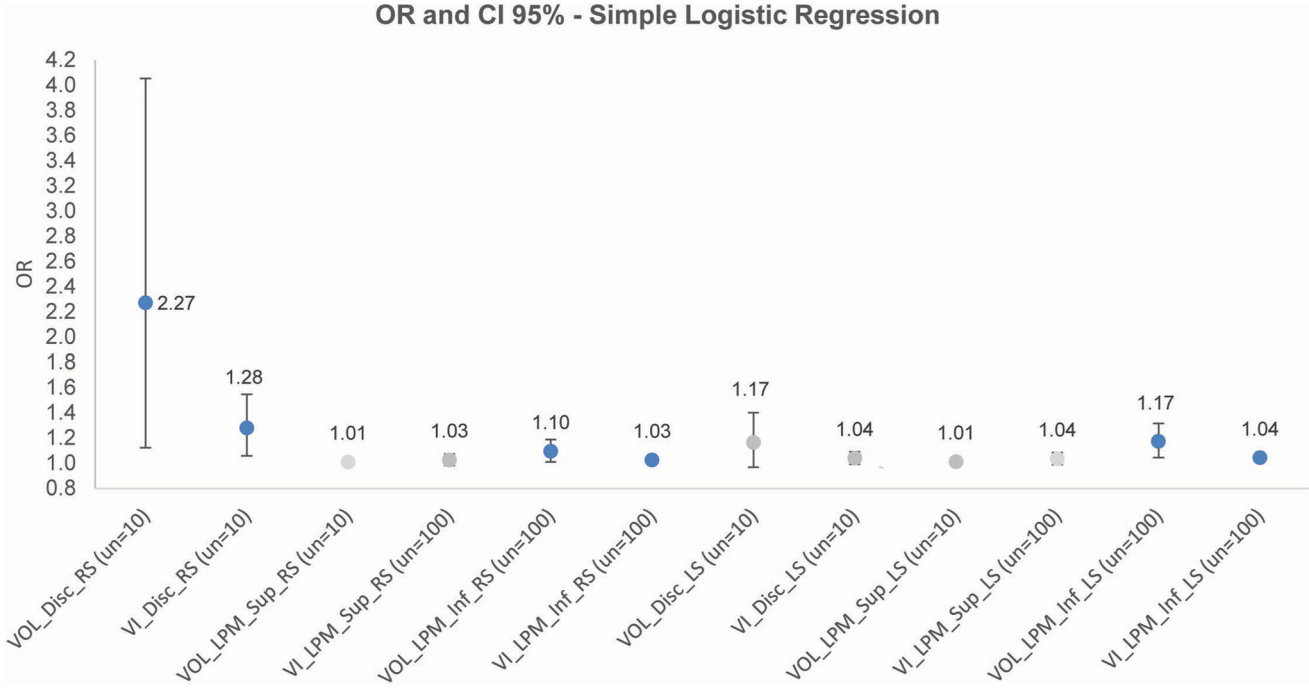

Figure 2. Variables presented in Table 2 evaluated through Simple Logistic Regression model. Odds ratio (OR) with 95% confidence intervals are significant and highlighted in blue colour (VOL_Disc_RS, VI_Disc_RS, VOL_LPM_Inf_RS, VI_LPM_Inf_RS, VOL_LPM_Inf_LS and VI_LPM_Inf_LS). The variables in which 95% confidence interval (CI) of the OR do not have a significant influence on the migraine and are in grey. VOL: volume; RS: right side; LS: left side; LPM: lateral pterygoid muscle; Inf: inferior; VI: voxel intensity.

Table 3. Comparison of the position of the disc on the right-side (Kruskal–Wallis test).

Variable	Disc position	*N*	Mean	Standard deviation	Minimum	Median	Maximum	*p* Value
VOL_Disc_RS	ADDR	10	150	20	124	149	187	**<.0001**
	ADDWR	6	186	25	154	182	229	
	ND	16	90	26	29	96	129	
VI_Disc_RS	ADDR	10	579	81	480	579	726	**<.0001**
	ADDWR	6	723	97	599	707	889	
	ND	16	354	106	112	375	500	
VOL_LPM_Sup_RS	ADDR	10	1614	2785	324	597	9455	.0755
	ADDWR	6	1280	450	732	1202	2041	
	ND	16	734	398	101	656	1655	
VI_LPM_Sup_RS	ADDR	10	3008	1641	1258	2317	5476	**.0481**
	ADDWR	6	4973	1748	2842	4670	7927	
	ND	16	2849	1547	393	2550	6427	
VOL_LPM_Inf_RS	ADDR	10	6309	10,228	1570	3230	35,175	.1438
	ADDWR	6	3003	1266	1022	3325	4207	
	ND	16	2324	801	1335	2181	3701	
VI_LPM_Inf_RS	ADDR	10	12,370	5219	6098	12,544	21,512	.1460
	ADDWR	6	11,663	4919	3969	12,913	16,337	
	ND	16	9027	3109	5183	8471	14,375	

ADDR: anterior disc displacement with reduction; ADDWR: anterior disc displacement without reduction; ND: non-displaced disc; VI: voxel intensity; LPM: lateral pterygoid muscle; Inf: inferior; Sup: superior; VOL: volume; RS: right side; N: sample size.
The bold values signify $p < .05$.

Table 4. Comparison of disc position on the left-side (Mann–Whitney test).

Variable	Disc position	*N*	Mean	Standard deviation	Minimum	Median	Maximum	*p* Value
VOL_Disc_LS	ADDR	12	154	19	110	156	177	**.0005**
	ND	19	86	27	28	91	121	
VI_Disc_LS	ADDR	12	597	72	426	608	689	**<.0001**
	ND	19	339	105	108	353	471	
VOL_LPM_Sup_LS	ADDR	12	731	282	324	657	1175	.5565
	ND	19	1603	3445	155	786	15,708	
VI_LPM_Sup_LS	ADDR	12	2840	1094	1257	2553	4564	.5565
	ND	19	3372	1943	602	3053	7511	
VOL_LPM_Inf_LS	ADDR	12	3137	1707	1297	3255	7160	.5840
	ND	19	4406	7785	1149	2637	36,375	
VI_LPM_Inf_LS	ADDR	12	12,183	6628	5038	12,642	27,807	.5296
	ND	19	10,508	3441	4461	10,239	17,912	

VI: voxel intensity; LPM: lateral pterygoid muscle; LS: left-side; RS: right-side; Sup: superior; Inf: inferior; VOL: volume; ADDR: anterior disc displacement with reduction; ND: non-displaced disc; N: sample size.
The bold values signify $p < .05$.

Table 5. Intra-class Correlation Coefficient (ICC) to assess the agreement between the measurements at different moments.

Variable	Measure	Mean	SD	Median	ICC (CI95%)
VOL_Disc_RS	1	127	46	127	1.000 (1.000–1.000)
	2	127	46	127	
VI_Disc_RS	1	493	178	487	1.000 (0.999–1.000)
	2	495	178	487	
VOL_LPM_Sup_RS	1	1111	1588	717	1.000 (1.000–1.000)
	2	1111	1588	717	
VI_LPM_Sup_RS	1	3297	1762	2785	1.000 (1.000–1.000)
	2	3300	1767	2786	
VOL_LPM_Inf_RS	1	3697	5848	2569	1.000 (1.000–1.000)
	2	3700	5849	2569	
VI_LPM_Inf_RS	1	10,566	4358	9976	1.000 (1.000–1.000)
	2	10,569	4355	9976	
VOL_Disc_LS	1	115	42	109	1.000 (1.000–1.000)
	2	168	165	121	
VI_Disc_LS	1	447	162	432	1.000 (1.000–1.000)
	2	448	162	432	
VOL_LPM_Sup_LS	1	1286	2668	768	1.000 (1.000–1.000)
	2	1286	2668	768	
VI_LPM_Sup_LS	1	3303	1812	2983	1.000 (1.000–1.000)
	2	3310	1812	2985	
VOL_LPM_Inf_LS	1	3905	6051	2853	1.000 (1.000–1.000)
	2	3908	6051	2853	
VI_LPM_Inf_LS	1	11,244	4835	11,081	1.000 (0.999–1.000)
	2	11,272	4819	11,081	

VI: voxel intensity; LPM: lateral pterygoid muscle; LS: left-side; RS: right-side; Sup: superior; Inf: inferior; VOL: volume; SD: standard deviation.

inferior head of LPM had an increase in both volume and voxel signal intensity in patients with migraine. Increased voxel signal intensity on T2-weighted MRI reflects changes in the muscle composition, since it usually has normal intermediate signal intensities [28]. Therefore, an increase in the mean voxel values suggests the presence of components contributing to this increase, as in the case of T2-weighted sequence. This would indicate the presence of fluid, leading us to infer that there was oedema or aqueous degeneration, which may result in an increase in the muscle volume, as observed in migraine patients. This finding was not observed in the superior head of LPM. Wang et al. [29] assessed the real role of the superior and inferior head of the LPM by using electromyography, reporting that the superior head is responsible for the stabilization of the articular disc and condyle during mandible movements, whereas the inferior one accounts for the mouth opening movements. We believe that the lack of alteration in signal intensity (and consequently in the muscle composition) and superior head volume of this muscle would indicate that inferior head is necessary to functionally stabilize the mandible position and degree of mouth opening as well as to keep the disc correctly positioned, which would reflect a greater percentage of disc displacements. That is, in patients with migraine, perhaps hyperactivity of the inferior head of the LPM is more important as it could cause the same changes shown here for the superior one in trying to stabilize the disc, thus resulting in a high frequency of displacement. This finding is quite interesting as it could explain the high incidence of disc displacement in migraine patients, as reported by other studies [16,30].

The articular disc of the TMJ is a fibrocartilaginous structure, which has average dimensions ranging from 1.0 to 1.5 cm in the mid-distal direction and from 0.7 to 0.8 cm in the anterior-posterior direction, being morphologically a biconcave structure with anterior and posterior bands and posterior thickening [31]. All this composition associated with the anatomical conformation of the disc is responsible for its correct congruence with the condyle and temporal structures (i.e. tubercle and articular fossa) during TMJ movements, allowing rotation and translation of the condylar and temporal surfaces and favouring that the disc is always positioned between them [23].

According to our results, an increase in the disc signal intensity in migraine patients corresponds to a change in its fibrocartilaginous components. This may result in hydropic degeneration and consequently change the conformation of the disc, culminating in an increase in its volume and affecting its correct function. As a result, the congruence between disc and articular components is made difficult, thus favouring the development of anterior disc displacement in patients with migraine. Higuchi et al. [32] evaluated the MRI scans of patients with joint pain and found alterations in the signal intensities of condylar medullary and bilaminar zone in patients with the presence of disc displacement. Visual analysis was performed instead of mathematical assessment of signal strength and volumetric value of the disc, as performed in our research. In this aspect, our study can be considered pioneering in this methodology, which makes comparison a difficult task.

This study has some limitations. The sample size was small. In addition, our study did not include patients with tension-type headache, who may be more relevant to TMD.

Conclusion

Pain is not an ever-present sign of disc displacement with reduction, since many patients with this type of derangement are asymptomatic. However, when TMJ structures are not correctly adapted to the new disc position, painful symptoms can occur. The presence of muscle pain rather than joint pain, as observed in our study, may be associated with the presence of migraine, which increases the frequency of episodes to daily or weekly. The inferior head of LPM had its volume and voxel intensity increased, which corroborates its tendency of hypertrophy in patients with TMD-associated migraine.

Disclosure statement

The authors discard any kind of conflict of interest about this study.

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References

[1] Weatherall MW. The diagnosis and treatment of chronic migraine. Ther Adv Chronic Dis. 2015;6(3):115–123.

[2] Lipton RB, Bigal ME, Steiner TJ, et al. Classification of primary headaches. Neurology. 2004;63(3):427–435.

[3] Bahra A. Primary headache disorders: focus on migraine. Rev Pain. 2011;5(4):2–11.

[4] Cutrer FM. Pathophysiology of migraine. Semin Neurol. 2006; 26(2):171–180.

[5] Peres MF, Masruha MR, Zukerman E, et al. Potential therapeutic use of melatonin in migraine and other headache disorders. Expert Opin Investig Drugs. 2006;15(4):367–375.

[6] Moskowitz MA. The neurobiology of vascular head pain. Ann Neurol. 1984;16(2):157–168.

[7] Cairns BE. Pathophysiology of TMD pain–basic mechanisms and their implications for pharmacotherapy. J Oral Rehabil. 2010;37(6): 391–410.

[8] Costa AL, D'Abreu A, Cendes F. Temporomandibular joint internal derangement: association with headache, joint effusion, bruxism, and joint pain. J Contemp Dent Pract. 2008;9(6):9–16.

[9] Look JO, Schiffman EL, Truelove EL, et al. Reliability and validity of Axis I of the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) with proposed revisions. J Oral Rehabil. 2010;37(10):744–759.

[10] Di Paolo C, D'Urso A, Papi P, et al. Temporomandibular disorders and headache: a retrospective analysis of 1198 patients. Pain Res Manag. 2017;2017:1.

[11] Franco AL, Goncalves DA, Castanharo SM, et al. Migraine is the most prevalent primary headache in individuals with temporomandibular disorders. J Orofac Pain. 2010;24(3):287–292.

[12] Goncalves MC, Florencio LL, Chaves TC, et al. Do women with migraine have higher prevalence of temporomandibular disorders? Braz J Phys Ther. 2013;17(1):64–68.

[13] Goncalves DA, Bigal ME, Jales LC, et al. Headache and symptoms of temporomandibular disorder: an epidemiological study. Headache. 2010;50(2):231–241.

[14] Florencio LL, de Oliveira AS, Carvalho GF, et al. Association between severity of temporomandibular disorders and the frequency of headache attacks in women with migraine: a cross-sectional study. J Manipulative Physiol Ther. 2017;40(4):250–254.

[15] Ballegaard V, Thede-Schmidt-Hansen P, Svensson P, et al. Are headache and temporomandibular disorders related? A blinded study. Cephalalgia. 2008;28(8):832–841.

[16] Lopes SL, Costa AL, Gamba Tde O, et al. Lateral pterygoid muscle volume and migraine in patients with temporomandibular disorders. Imaging Sci Dent. 2015;45(1):1–5.

[17] Gerwin R. Myofascial trigger points comorbidity in headache In: Giamberardino M., Martelletti P, editors. Comorbidities in headache disorders. Berlin (Germany): Springer; 2017. p. 197–212.

[18] Lopes SL, Costa AL, Cruz AD, et al. Clinical and MRI investigation of temporomandibular joint in major depressed patients. Dentomaxillofac Radiol. 2012;41(4):316–322.

[19] Tasaki MM, Westesson PL. Temporomandibular joint: diagnostic accuracy with sagittal and coronal MR imaging. Radiology. 1993; 186(3):723–729.

[20] Fan Z, Kocis K, Valley R, et al. High-pressure transvenous perfusion of the upper extremity in human muscular dystrophy: a safety study with 0.9% saline. Gene Ther. 2015;26(9):614–621.

[21] Fan Z, Kocis K, Valley R, et al. Safety and feasibility of high-pressure transvenous limb perfusion with 0.9% saline in human muscular dystrophy. Mol Ther. 2012;20(2):456–461.

[22] Goncalves DA, Camparis CM, Franco AL, et al. How to investigate and treat: migraine in patients with temporomandibular disorders. Curr Pain Headache Rep. 2012;16(4):359–364.

[23] Tomas X, Pomes J, Berenguer J, et al. MR imaging of temporomandibular joint dysfunction: a pictorial review. Radiographics. 2006;26(3):765–781.

[24] Schellhas KP, Wilkes CH. Temporomandibular joint inflammation: comparison of MR fast scanning with T1- and T2-weighted imaging techniques. AJR Am J Roentgenol. 1989;153(1):93–98.

[25] Orhan K, Nishiyama H, Tadashi S, et al. Comparison of altered signal intensity, position, and morphology of the TMJ disc in MR images corrected for variations in surface coil sensitivity. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006;101(4):515–522.

[26] Imanimoghaddam M, Madani AS, Hashemi EM. The evaluation of lateral pterygoid muscle pathologic changes and insertion patterns in temporomandibular joints with or without disc displacement using magnetic resonance imaging. Int J Oral Maxillofac Surg. 2013;42(9):1116–1120.

[27] Stelzenmueller W, Umstadt H, Weber D, et al. Evidence - the intraoral palpability of the lateral pterygoid muscle - a prospective study. Ann Anat. 2016;206:89–95.

[28] Kuroda M, Otonari-Yamamoto M, Araki K. Evaluation of lateral pterygoid muscles in painful temporomandibular joints by signal intensity on fluid-attenuated inversion recovery images. Oral Radiol. 2018;34(1):17–23.

[29] Wang MQ, Yan CY, Yuan YP. Is the superior belly of the lateral pterygoid primarily a stabilizer? An EMG study. J Oral Rehabil. 2001;28(6):507–510.

[30] Ashraf J, Zaproudina N, Suominen AL, et al. Association between temporomandibular disorders pain and migraine: results of the health 2000 survey. J Oral Facial Pain Headache. 2019. DOI:10.11607/ofph.2213

[31] Alomar X, Medrano J, Cabratosa J, et al. Anatomy of the temporomandibular joint. Semin Ultrasound CT MR. 2007;28(3): 170–183.

[32] Higuchi K, Chiba M, Sai Y, et al. Relationship between temporomandibular joint pain and magnetic resonance imaging findings in patients with temporomandibular joint disorders. Int J Oral Maxillofac Surg. 2019. DOI:10.1016/j.ijom.2019.06.028