Relationship of Other Joint Problems and Anterior Disc Position in Symptomatic TMD Patients and in Asymptomatic Volunteers

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Anterior disc position has been highly correlated with temporomandibular disorders (TMD). It was hypothesized that internal derangement of the temporomandibular joint may be a part of a joint phenotype that imparts an increased risk for joint disorders. If this hypothesis is true, an increased prevalence of joint disorders in individuals diagnosed with displaced discs should be expected. A total of 263 symptomatic TMD patients and 82 asymptomatic volunteers was examined. Asymptomatic volunteers with anteriorly displaced discs were twice as likely as asymptomatic volunteers without disc displacements to report pain/dysfunction in other joints. Symptomatic patients with and without displaced discs reported an increase in other joint problems three to four times greater than in asymptomatic subjects. Compared to symptomatic TMD patients without disc displacement, symptomatic TMD patients with anteriorly displaced discs were also twice as likely to report other family members as being affected by TMD. Familial aggregation of TMD and an increased prevalence of other joint problems in these individuals may represent more than a serendipitous occurrence.

J OROFACIAL PAIN 1996;10:15-20.

key words: temporomandibular joint, mandibular condyle, magnetic resonance imaging, joint instability, comparative study

The performant point disorders (TMD) is generally believed to be multifactorial in nature.¹ Frequently mentioned etiologic factors include occlusal disharmonies, psychologic profile, musculoskeletal injuries,^{2,3} parafunctional habits,^{4,5} distress, and connective tissue laxity.⁶⁻¹¹ Several studies have reported an association between generalized joint laxity and the signs and symptoms associated with TMD.^{11,12} Systemic joint laxity has been suggested to be significantly more prevalent in individuals with TMD than in asymptomatic control subjects.¹⁰ Consistent with this observation, patients with TMD and systemic joint laxity are also reported to have significantly more general musculoskeletal complaints than do asymptomatic control subjects.^{11,13} These findings suggest that joint laxity may be a significant risk factor for TMD.

One possible manifestation of joint laxity in the temporomandibular joint (TMJ) may be the occurrence of altered disc position. Disc anomalies in the absence of pain have been reported in other joints, including the knee, the cervical spine, and the lumbar spine.¹⁴⁻¹⁸ It is possible that disc abnormalities may represent

a joint phenotype that predisposes to joint dysfunction, including TMD. It is hypothesized that internal derangement of the TMJ (disc displacement) may be a manifestation of such joint laxity. Such changes in joint anatomy could result from heritable forms of altered collagen metabolism and may affect other joints in addition to the TMJ. Thus, abnormalities of collagen metabolism may serve as a predisposing factor for the development of internal derangement. If this hypothesis is true, a familial aggregation for internal joint derangement would be expected. Individuals from such families would be expected to have an increased prevalence of pain and dysfunction in joints other than the TMJ. Although the predisposition to joint derangement may be heritable and therefore show familial aggregation, the clinical expression (joint pain and dysfunction) may show sex-limited expression.

The purpose of this study was to evaluate evidence for a familial aggregation of TMJ derangement and to evaluate evidence for the occurrence of other joint problems in individuals with TMJ derangement. The hypothesis is that the presence of joint disorders is linked by a common etiologic mechanism at a genetic level.

Materials and Methods

Eighty-two asymptomatic volunteers and 263 symptomatic patients with TMD seeking treatment at the Eastman Dental Center Department of Temporomandibular Joint Disorders, Rochester, NY, were included in this study. Asymptomatic volunteers answered a solicitation for examination

Table 1Age, Sex, and MRI Class of StudyParticipants

	Sex		Age (years)		
MRI*		n	Mean	SD	
A/N	F	25	27.2	7.3	
A/N	M	30	26.5	9.0	
A/ADP	F	17	26.1	7.5	
A/ADP	M	10	25.5	8.4	
S/N	F	36	28.9	10.7	
S/N	M	6	30.5	15.1	
S/ADP	F	197	30.5	13.2	
S/ADP	М	24	28.0	12.0	

 $^*A =$ asymptomatic, no report of temporomandibular joint dysfunction; N = normal MRI, no disc displacement: S = symptomatic, temporomandibular joint dysfunction; ADP = anterior disc position, disc displacement on MRI.

- 1. A TMJ subjective questionnaire documenting the absence of jaw pain, TMJ noise, locking, and a positive history for TMD.
- Clinical TMJ and dental examination as described by Roberts et al¹⁹ for signs and symptoms commonly associated with TMD or internal derangement. All asymptomatic volunteers demonstrated an acceptable range of mandibular movements with maximal opening of at least 40 mm.

All study participants had bilateral high-resolution magnetic resonance imaging (MRI) scans in the sagittal (closed and opened) and coronal (closed) planes to evaluate the TMJs for the presence or absence of internal derangement.²⁰ On the basis of these findings, each study participant was then classified as having a normal MRI (no internal derangement) or abnormal MRI (anterior disc position), thereby dividing the study participants into the following four groups:

- 1. Asymptomatic subjects with no TMD; normal MRI (A/N)
- 2. Asymptomatic subjects with no TMD; anterior disc position on MRI (A/ADP)
- Symptomatic subjects with TMD; normal MRI (S/N)
- 4. Symptomatic subjects with TMD; anterior disc position on MRI (S/ADP)

Patients were also asked about the presence of joint pain and dysfunction in other joints. A positive response was noted only when the patients said they had sought consultation by their physician for evaluation of the joint(s).

Results

A total of 345 white subjects were examined for this study. The age, sex, and results of the bilateral MRI analysis are shown in Table 1. The mean age of the symptomatic patients was slightly greater than the mean age of the asymptomatic volunteers. However, this difference was not statistically significant. Eighty-nine percent (233 of 263) of the symptomatic patients were female and 51% (42 of 82) of the asymptomatic volunteers were female. Eighty-four percent (221 of 263) of the symptomatic patients and 32.9% (27 of 82) of the asymptomatic volunteers had ADP according to

MRI [†]	N	n	Gender			Age (years) [‡]	
			F	М	%	Mean	SD
A/N	55	7	3	4	12.7	31.4	8.3
A/ADP	27	3	1	2	11.1	26.0	12.1
S/N	42	4	3	1	9.5	31.7	20.8
S/ADP	221	52	43	7	23.5	28.4	13.1

Table 2 Subject	Reporting	Familial	Aggregation*
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*N = total number of individuals; n = number of individuals reporting other family members affected by TMD. tA = asymptomatic, no report of temporomandibular joint dysfunction; N = normal MRI, no disc displacement; S = symptomatic, temporomandibular joint dysfunction; ADP = anterior disc position, disc displacement on MRI. tAge of individuals reporting other family members affected by TMD.

Table 3 Subjects Reporting Other Joint Problems (Joint Pain and/or Dysfunction)*

		n	Gender		7936	Age (years)		Other	
MRI [†]	Ν		F	М	%	Mean	SD	affected joints [‡]	
A/N	55	5	1	4	9.1	30.8	11.4	Knee, spine, ankle, hand	
A/ADP	27	6	2	4	22.2	28.8	15.1	Knee	
S/N	42	15	13	2	35.7	34.6	14.0	Knee, cervical spine, hand, shoulder, ankle	
S/ADP	221	64	59	5	28.9	35.0	15.3	Knee, shoulder, hand, cervical spine, ankle, hip, lumbar spine	

*N = total number of individuals: n = number of individuals reporting other family members affected by TMD.

tA = asymptomatic, no report of temporomandibular joint dysfunction; N = normal MRI, no disc displacement; S = symptomatic, temporomandibular joint dysfunction; ADP = anterior disc position, disc displacement on MRI.

‡Joints reported in decreasing frequency.

the MRI. The mean age of the individuals with normal MRI was slightly more than the mean age of individuals with ADP. Forty percent (17 of 42) of the asymptomatic females and 25% (10 of 40) of the asymptomatic males were found to have ADP by MRI. Eighty-five percent (197 of 233) of the symptomatic females and 80% (24 of 30) of the symptomatic males were found to have ADP by MRI. There was no statistically significant difference in mean age between subjects with normal disc position and ADP in either the symptomatic or asymptomatic groups.

The study participants who reported other family members affected with TMD is shown in Table 2. Symptomatic individuals with anterior disc position were twice as likely to report additional family members with TMD. Unfortunately, the small numbers of individuals in other groups prevents the inference of anything more than speculation.

The number of individuals reporting other joint problems is presented in Table 3. There were no subjects in this study who had rheumatoid arthritis. "Other joint problems" refers to joint pain and dysfunction in nonTM joints. This did not include asymptomatic joint noise in nonTM joints (eg, popping, clicking, cracking). Approximately 9% (5 of 55) of the asymptomatic volunteers with normal disc position reported having other joint problems. The symptomatic patients reported other joint problems from 29% to 36% of the time. The asymptomatic volunteers with ADP reported having other joint problems 22% of the time. In all cases, the knee was the joint most frequently reported to be affected.

Discussion

Both host and environmental factors have been hypothesized to play a role in the etiology of TMD. Temporomandibular joint disorders has been noted with increased frequency in individuals with mitral valve prolapse, indicating a possible etiologic association with altered collagen

Morrow et al

metabolism. Altered collagen metabolism may be important in joint laxity. Higher ratios of collagen type III to type III + I have been reported in patients with TMI derangement and systemic joint laxity than in control subjects. 11,21,22 Genetic (inherited) predisposition to altered collagen metabolism may result in a morphologic (developmental) abnormality of any joint including the TMJ. These individuals may be at increased risk for developing TMD when also subjected to other etiologic factors. Systemic joint laxity has been reported to occur more frequently in female teenagers than in male teenagers. These findings may represent a sex-limited expression of joint laxity.23-25 Temporomandibular joint disorders is much more prevalent in females than in males. Scientific support for sex-limited joint physiology has been found in animal models. A direct link of estrogen to the development of osteoarthritis in rabbit knees has been suggested. Up-regulation of estrogen receptors in cartilage might initiate the osteoarthritic changes.23 Ben-Hur et al26 have suggested that estrogen acts directly on chondrocytes through an estrogen receptor-mediated mechanism. In a study of induced inflammatory arthritis in rodents, female mice demonstrated a greater ability to degrade cartilage than did male mice.27 A study of estrogen and progesterone receptors in the human TMJ concluded that the TMJ disc is, at least in some males and females, potentially a female sex hormone target tissue.28 Further investigations are necessary to elucidate the significance of estrogen receptors in the pathogenesis of joint destruction.

Recognition of a possible association between joint laxity and TMD is not new. Subsequent studies have also reported correlations between altered collagen ratios, joint laxity, and TMD phenotypes.^{11,21,22}

Disc displacement of the TMJ has been suggested as a possible predisposing factor for TMD. Unfortunately, disc position cannot be reliably determined by clinical examination alone. A better estimation of disc position may be determined by arthrographic and magnetic resonance imaging techniques. Both methods have been correlated with surgical²⁹ and autopsy findings³⁰⁻³² and have proven reliable in assessing the position and function of the articular disc. It is likely that clinical examination of the TMJ may underestimate the presence of soft tissue anomalies, some of which may be significant risk factors for the development of TMD. Anterior disc position has been previously reported to occur in 78% of individuals with symptomatic TMD.33 Anterior disc position has

also been reported in asymptomatic volunteers.^{34,35} The present study suggests that ADP occurs in approximately 33% of the time in asymptomatic volunteers.

Our findings show symptomatic patients with TMD and ADP were twice as likely to report other family members who have also been affected with TMD. The presence of ADP alone may not be sufficient to result in TMD but may increase the risk of developing TMD. If the propensity to develop ADP is inherited, then to that degree the risk to develop TMD will also be inherited. In such a situation, familial aggregation of TMD and an increase in the prevalence of other joint problems would be expected.

Subjects with anterior disc position (A/ADP and S/ADP) were three times as likely to report other joint problems. It may not be difficult to explain pain in subjects with ADP, but it is difficult to explain the four times increase in other joint problems in the symptomatic normal group as compared to the asymptomatic volunteers. Moskowitz et al³⁶ have suggested that peripheral joints shown to be architecturally normal can develop osteoarthritis later in life. They concluded that osteo-arthritis development is a result of a decrease in the durability of articular cartilage related to mutant protein collagen molecules. This may suggest that there are multiple collagen phenotypes that may impart differing capacities for breakdown over time. It is not known presently if the SN group can be associated with altered collagen metabolism.

Conclusions

The present study confirms the previously reported higher prevalence of ADP in symptomatic TMD subjects33 compared to asymptomatic volunteers.34,35 Familial aggregation for the development of TMD appears to be more likely in symptomatic patients with ADP. It is possible that this observation implies a genetic tendency. A genetic propensity for joint irregularities may affect more than one joint. Both groups of symptomatic patients and asymptomatic volunteers with anterior disc position reported an increased frequency of other joint problems when compared to asymptomatic volunteers with normal joints. Temporomandibular joint disorders probably represent a heterogeneous group of conditions that may be etiologically dissimilar. However, temporomandibular joint disorders may share a number of common and sometimes overlapping clinical signs and

symptoms with other joint disorders. Advances in the diagnosis and treatment of TMD depend on the ability to define these etiologically distinct subforms. To this end, recognition of a familial aggregation for TMD and identification of families segregating for similar forms of the condition may prove a valuable resource in the scientific investigation into the etiology of at least some forms of TMD. Prospective longitudinal studies will be required to define these relationships.

References

- Dibbets JMH, van der Weele LT. Prevalence of TMJ symptoms and x-ray findings. Eur J Orthod 1989;11: 31-36.
- Rugh JD, Solberg WK. Psychological implications in temporomandibular pain and dysfunction. Oral Sci Rev 1976;7:3–30.
- Storey AT. Neurophysiology of temporomandibular joint disorders. In: Carlson DS, McNamara JA Jr, Ribbens KA (eds). Developmental Aspects of TMJ Disorders, monograph 16, Craniofacial Growth Series. Ann Arbor, MI: University of Michigan, Center for Human Growth and Development, 1985:115–144.
- Riolo ML, Brandt D, Ten Have TR. Associations between occlusal characteristics and signs and symptoms of TMJ dysfunction in children and young adults. Am J Orthod 1987;92:467–477.
- Nilner M. Functional disturbances and diseases in the stomatognathic system among 7 to 18 year olds. J Craniomand Pract 1985;3:358-367.
- Chun DS, Koskinen-Moffett L. Distress, jaw habits and connective tissue laxity as predisposing factors to TMJ sounds in adolescents. J Craniomandib Disord Facial Oral Pain 1990;4:165–176.
- Bates RE Jr, Stewart CM, Atkinson WB. The relationship between internal derangements of the temporomandibular joint and systemic joint laxity. J Am Dent Assoc 1984; 109:446–447.
- Biro F, Gewanter HL, Baum J. The hypermobility syndrome. Pediatrics 1983;72:701-706.
- Helkimo E, Westling L. History, clinical findings and outcome of treatment of patients with anterior disk displacement. J Craniomand Pract 1987;5:269–276.
- Kim SM, Cohen SG, Douglas P. Relationship between internal derangement of TMJ and mitral valve prolapse [abstract 1877]. J Dent Res 1989;68(special issue):416.
- Westling L. Temporomandibular joint dysfunction and systemic joint laxity. Swed Dent J 1992;16(suppl 81):1–79.
- Hecht C, Adair S. Generalized joint hypermobility and TMJ signs and symptoms in children [abstract 513]. J Dent Res 1985;64:(special issue):232.
- Blasberg B, Chalmers A. Temporomandibular pain and dysfunction syndrome associated with generalized musculoskeletal pain: A retrospective study. J Rheumatol 1989;16(suppl 19):87-90.
- Boden SD, Davis DO, Dina TS, Stoller DW, Brown SD, Vailas JC, Labropoulos PA. A prospective and blinded investigation of magnetic resonance imaging of the knee. Abnormal findings in asymptomatic subjects. Clin Orthop 1992;282:177-185.

- Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg 1990;72:403–408.
- Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic resonance scans of the cervical spine in asymptomatic subjects. J Bone Joint Surg 1990;72:1178–1184.
- Negendank WG, Fernandez-Madrid FR, Heilbrun LK, Teitge RA. Magnetic resonance imaging of meniscal degeneration in asymptomatic knees. J Orthop Res 1990;8:311-320.
- Shellock FG, Morris E, Deutsch AL, Mink JH, Kerr R, Boden SD. Hematopoietic bone marrow hyperplasia: High prevalence on MR images of the knee in asymptomatic marathon runners. AJR 1992;158:335–338.
- Roberts CA, Tallents RH, Katzberg RW, Espeland MA, Handelman SL. Correlation of clinical parameters to the arthrographic depiction of TMJ internal derangements. Oral Surg Oral Med Oral Pathol 1988;66:32–36.
- Katzberg RW, Westesson P-L, Tallents RH, Anderson R, Kurita K, Manzione JV, Totterman S. Temporomandibular joint: Magnetic resonance assessment of rotational and sideways disc displacements. Radiology 1988;169:741–748.
- Kuivaniami H, Tromp G, Prockop DJ. Mutations of collagen genes: Causes of rare and some common diseases in humans. FASEB J 1991;5:2052–2060.
- Byers PH. Brittle bones—fragile molecules: Disorders of collagen gene structure and expression. Trends Genet 1990;6:293-300.
- Tsai CL, Liu TK. Up-regulation of estrogen receptors in rabbit osteoarthritic cartilage. Life Sci 1992;50:1727– 1735.
- Aufdemorte TB, Van Sickels JE, Dolwick MF, Sheridan PJ, Holt GR, Aragon SB, Gates GA. Estrogen receptors in the temporomandibular joint of the baboon (*Papio cynocephalus*): An autoradiographic study. Oral Surg Oral Med Oral Pathol 1986;61:307–314.
- Milam SB, Aufdemorte TB, Sheridan PJ, Triplett RG, Van Sickels JE, Holt GR. Sexual dimorphism in the distribution of estrogen receptors in the temporomandibular joint complex of the baboon. Oral Surg Oral Med Oral Pathol 1987;64:527–532.
- Ben-Hur H, Mor G, Blickstein I, Likhman I, Kohen F, Dgani R, et al. Localization of estrogen receptors in long bones and vertebrae of human fetuses. Calcif Tissue Int 1993;53:91-96.
- Da Silva JA, Larbre JP, Seed MP, Cutolo M, Villaggio B, Scott D, Willoughby DA. Sex differences in inflammation induced cartilage damage in rodents. The influence of sex steroids. J Rheumatol 1994;21:330–337.
- Abubaker AO, Raslan WF, Sotercanos GC. Estrogen and progesterone receptors in temporomandibular joint discs of symptomatic and asymptomatic persons: A preliminary study. J Oral Maxillofac Surg 1993;51:1096–1100.
- Bronstein SL, Tomasetti BJ, Ryan DE. Internal derangements of the temporomandibular joint: Correlation of arthrography with surgical findings. J Oral Surg 1981; 39:572-584.
- Westesson P-L, Rohlin M. Diagnostic accuracy of doublecontrast arthrotomography of the temporomandibular joint. Correlation between arthrography and morphology of autopsy specimens using dissection and cryosectioning. Swed Dent J 1983;11(suppl 13):1-21.

- Tasaki MM, Westesson P-L. Temporomandibular joint: Diagnostic accuracy with sagittal and coronal MR images. Radiology 1993;186:723–729.
- Brooks SL, Westesson P-L. Temporomandibular joint: Value of coronal MR images. Radiology 1993;188: 317-321.
- Paesani D, Westesson P-L, Hatala M, Tallents RH, Kureta R. Prevalence of temporomandibular joint internal derangement in patients with craniomandibular disorders. Am J Orthod Dentofacial Orthop 1992;101:41–47.
- Tallents RH, Hatala MA, Westesson P-L, Katzberg RW, Murphy W, Proskin H. Temporomandibular joint sounds in asymptomatic volunteers. J Prosthet Dent 1993; 69:298-304.
- Kircos LT, Ortendahl DA, Mark AS, Arakawa M. Magnetic resonance imaging of the TMJ disc in asymptomatic volunteers. J Oral Maxillofac Surg 1987; 45:852-854.
- Moskowitz RW, Punn Y, Haqqi TM. Genetics and osteoarthritis. Bull Rheum Dis 1992;41:4-6.

Resumen

La Relación de Otros Problemas de Articulación y Posición Anterior del Disco en Pacientes con Síntomas de Desórdenes Temporomandibulares y Voluntarios Asintomáticos

La posición anterior del disco ha sido altamente correlacionada con los desórdenes temporomandibulares (DTM). Se ha planteado la hipótesis que el malfuncionamiento interno de la articulación temporomandibular puede ser parte de un fenotipo articular que imparte un riesgo elevado de padecer desórdenes articulares. Si esta hipótesis es verdadera, se debería esperar un prevalencia aumentada de desórdenes articulares en individuos diagnosticados con discos desplazados. Se examinaron 263 pacientes con síntomas de DTM y 82 voluntarios asintomáticos. Los voluntarios asintomáticos con discos desplazados anteriormente tenían el doble de posibilidades de reportar dolor/disfuncion en otras articulaciones, en comparación con los voluntarios asintomáticos sin discos desplazados. Los pacientes sintomáticos con y sin discos desplazados mostraron un incremento en otros problemas articulares, tres o cuatro veces mayor en comparación con los pacientes asintomáticos. Los pacientes con síntomas de DTM y con discos desplazados anteriormente tenían el doble de posibilidades de informar que otros miembros de la familia sufrían de DTM en comparación con los pacientes con síntomas de DTM sin desplazamiento de disco. El agregado familiar de los DTM y una prevalencia elevada de otros problemas de articulación en estos individuos puede representar mas que una ocurrencia por casualidad.

Zusammenfassung

Beziehung zwischen anderen Gelenkproblemen und anteriorer Diskusposition bei symptomatischen MAP-Patienten und bei asymptomatischen Probanden

Eine anteriore Diskusposition wurde schon in einen engen Zusammenhang mit Myoarthropathien gebracht. Eine häufige Hypothese lautet, dass Diskusluxationen des Kiefergelenks Teil eines Gelenkphenotyps sein können, welcher mit einem erhöhten Risiko für Arthropathien belastet ist. Wenn diese Hypothese war ist, sollte bei Leuten mit verschobenem Diskus die Prävalenz für Erkrankungen anderer Gelenke erhöht sein. Es wurden 263 symptomatische MAP-Patienten und 82 asymptomatische Probanden untersucht. Asymptomatische Probanden mit anterior verlagertem Diskus berichteten doppelt so oft wie asymptomatische Probanden ohne Diskusverlagerung über Schmerzen und Störungen in anderen Gelenken. Symptomatische Patienten mit und ohne Diskusverlagerung berichteten über eine drei- bis viermal höhere Häufigkeit anderer Gelenkprobleme als asymptomatische Probanden. Verglichen mit symptomatischen MAP-Patienten ohne Diskusverlagerung, bestand bei symptomatischen MAP-Patienten mit anterior verlagertem Diskus eine doppelt so hohe Wahrscheinlichkeit, dass auch andere Familienmitglieder eine Myoarthropathie aufwiesen. Familiäre Häufung von MAP und eine erhöhte Prävalenz anderer Gelenkprobleme bei den untersuchten Personen könnten mehr als nur eine zufällige Koinzidenz darstellen.