Generalized Joint Hypermobility and Temporomandibular Disorders: Inherited Connective Tissue Disease as a Model with Maximum Expression

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Aims: *To study the relationship between generalized joint hypermobility (GJH) and temporomandibular disorders (TMD) by assessing prevalence and patient characteristics of TMD in a population of patients with maximum expression of GJH as a symptom of inherited connective tissue disease. In addition, diagnostic reliability of a series of clinical signs indicative of temporomandibular joint (TMJ) hypermobility was tested.* ***Methods:*** *The study sample consisted of 42 subjects with GJH, 24 with Marfan syndrome and 18 with Ehlers-Danlos syndrome. A subgroup of 27 individuals was selected by age (≥ 18 yrs) and was compared to 40 controls with TMD and normal peripheral joint mobility. TMD diagnoses were assigned to each subject according to the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).* ***Results:*** *In the GJH sample (n = 42), 71.4% of the subjects were symptomatic for TMD. Of those, 13.3% had sought treatment. A myofascial pain diagnosis was made in 69%, disc dislocation with reduction was diagnosed in 85.7%, and TMJ arthralgia in 61.9%. Multiple TMD diagnoses were assigned in 69% of the subjects; of these, 57% had 3 or more subgroup diagnoses. Joint noises (*$P < .01$*) and recurrent TMJ dislocations (*$P < .01$*) were a frequent finding in adult GJH subjects (n = 27) compared to controls, with symptomatic GJH subjects presenting more and more prolonged dislocation events than asymptomatic subjects (*$P < .001$*). TMJ hypermobility signs were expressed significantly more often in GJH compared to controls with TMD and normal joint mobility.* ***Conclusion:*** *This study indicates a positive relationship between GJH and TMD.* J OROFAC PAIN 2005;19:47–57

Key words: joint hypermobility, temporomandibular disorders, temporomandibular joint, temporomandibular joint dislocation

Generalized joint hypermobility (GJH) is a cardinal feature of inherited connective tissue disease. The term *inherited connective tissue disease* refers to a group of disorders considered to be the result of laxity of supporting ligaments. Failure of the structural components of these ligaments is caused by defective metabolism of collagen or fibrillin, which is caused by mutations in the genes coding for these 2 extracellular matrix proteins.[1] Joint laxity also occurs without involvement of other structures and has been suggested to predispose individuals to the development of temporomandibular disorders (TMD) and osteoarthritis. To assess conflicting evidence in the literature regarding the association

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between TMD and GJH, and to study the effects of structural joint component laxity on TMD, a model of a GJH disorder that has maximum expression should be evaluated.[2,3] This is true of both the hypermobility type and the classical type of Ehlers-Danlos syndrome (EDS) and of the Marfan syndrome (MFS). Joint laxity is a cardinal feature of both of these diseases.

EDS is a heterogeneous group of heritable connective tissue disorders characterized by articular hypermobility, skin extensibility, tissue fragility, and chronic joint and limb pain. The condition comprises 6 major types, each with different molecular basis, and is reflected in mainly structural aberrations of collagen. The most prevalent types of EDS are the hypermobility type (1:10,000) and the classical type (1:15,000 to 25,000). These conditions account for 85% to 90% of EDS syndromes, and share an autosomal dominant inheritance, skin hyperextensibility, and GJH as major diagnostic criteria. Easily bruised skin and generalized tissue fragility are characteristic of the classical type; joint hypermobility is the dominant clinical manifestation in the hypermobility type. Laboratory diagnosis includes detection of biochemical abnormalities of collagen type V and type I and genetic linkage to the genes encoding the proα1 or proα2 chains of these collagen types.[4] Instability and recurrent subluxation of the temporomandibular joint (TMJ) has been reported as a prevalent joint sign in hypermobility and classical EDS by various authors.[5–13] MFS is an autosomal dominant multisystem disorder with a variable phenotype, caused by mutation in FBN1 gene on 15q21 coding for fibrillin-1, an extracellular matrix protein associated with tissues displaying elastic properties. Multiple organ systems are affected, with most features being age-related. Most prominent are skeletal overgrowth, joint instability, subluxation of the eye lens, mitral valve prolapse, and dilatation and/or rupture of the ascending aorta. The prevalence is estimated at 1:5,000.[13]

The term *TMD* embraces a number of clinical problems that involve the masticatory musculature, the TMJ and associated structures, or both. The most frequently presenting symptom is pain. TMD pain is usually localized to the muscles of mastication, the preauricular area, and/or the TMJ. The classification of TMD is hampered by limited knowledge of the cause and progression of these disorders.[14–16] Clinical diagnoses of TMD apply criteria identifying abnormalities of structure and function of the muscles of mastication and/or the TMJs and are divided into 3 groups. Muscle disorders include both painful and nonpainful disorders. The common painful muscle disorders are myofascial pain (pain of muscle origin, including a complaint of pain as well as pain associated with localized areas of tenderness to palpation in muscle) with or without limited jaw opening (limited jaw movement and stiffness of the muscle during stretching). Disc displacements may occur with or without reduction (ie, limited jaw opening). A third group embraces arthralgia (pain and tenderness in the joint capsule and/or the synovial lining of the TMJ), osteoarthritis (an inflammatory condition within the joint resulting from a degenerative condition of the joint structures), and osteoarthrosis (a degenerative disorder of the joint in which joint form and structure are abnormal). To make diagnoses of these groups, it is first necessary to rule out some specific muscle conditions (muscle spasm, myositis, and contracture), as well as joint conditions (polyarthritis, acute traumatic injuries, and infections in the joint).[17,18]

Several studies have been performed to analyze the association between GJH and TMD.[2,3,19–28] The results of these studies are conflicting: Some have yielded an association between TMD and generalized joint laxity, while others could not demonstrate an interrelation. Furthermore, none of these studies analyzed the clinical signs and symptoms of TMJ hypermobility, nor did they address possible underlying connective tissue alterations that might account for elongation of the collateral TMJ ligaments or disc displacement.

The aim of this study was to assess the prevalence and characteristics of TMD in a population of patients with maximum expression of GJH as a symptom of inherited connective tissue disease. In addition, measurement reliability and diagnostic validity of a series of clinical signs indicative of TMJ hypermobility were tested.

Materials and Methods

The study group, or hypermobility group (HG), comprised 42 patients with inherited connective tissue disease (15 subjects with hypermobility-type EDS, 3 with classical EDS, and 24 with MFS). Mean ± SD age was 27.4 ± 15.5 years (range 6 to 61 years), and gender distribution was 26.2% male to 73.8% female. All individuals had been diagnosed clinically and biochemically at the Centre for Medical Genetics, Ghent University Hospital, according to the aforementioned criteria.[4,13] Since the pool of available control subjects seeking TMD treatment at the Ghent

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University Hospital exclusively consisted of adults, a subgroup of EDS and MFS patients (n = 27) at least 18 years of age was selected for comparison with the control group (CG); this subgroup comprised 12 patients with hypermobility-type EDS and 15 with MFS (mean age ± SD 36.5 ± 11.4 years; range 18 to 61 years; 20.6% male to 79.4% female). Forty subjects with normal peripheral joint mobility were individually matched to the adult HG subgroup (n = 27) for age and gender and were included as controls (mean age ± SD 36.4 ± 11.8 years; age range 19 to 68 years; 22.4% male to 77.6% female). To minimize confounding of facial pain assessment, individuals with a history of orofacial trauma, rheumatoid arthritis, or whiplash were excluded.

Joint mobility was assessed in each individual by determining the mobility score as proposed by Beighton et al.[29] The maneuvers used in this scoring system are as follows:

1. Passive dorsiflexion of the little fingers beyond 90 degrees (1 point for each hand), 2 points
2. Hyperextension of the elbows beyond 10 degrees (1 point for each elbow), 2 points
3. Passive apposition of the thumbs to the flexor aspect of the forearm (1 point for each thumb), 2 points
4. Hyperextension of the knees beyond 10 degrees (1 point for each knee), 2 points
5. Forward flexion of the trunk with the knees fully extended so that the palms of the hands rest flat on the floor, 1 point

Measurements were made by means of a protractor. The possible scores ranged from 0 to 9, with a higher score denoting greater joint laxity. A score of ≥ 3 indicated widespread hypermobility of the peripheral joints.[1,29]

The clinical examination and patient interview were based on the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD).[17,18] The RDC/TMD provide clinical researchers with a standardized system of methods for recording the history and the clinical signs of functional disturbances in the masticatory system. A dual-axis system with known reliability and validity of the applied examination methods is used.[30,31] Clinical physical findings are recorded with Axis I; behavioral, psychologic, and psychosocial status are recorded with Axis II. The diagnostic system as proposed in RDC/TMD is nonhierarchical and allows for the possibility of multiple diagnoses for a given subject. TMD diagnoses are divided into 3 groups:

Group I: Muscle diagnoses (muscle disorders)
- Ia. Myofascial pain
- Ib. Myofascial pain with limited opening

Group II: Disc displacements (disc disorders)
- IIa. Disc displacement with reduction
- IIb. Disc displacement without reduction, with limited opening
- IIc. Disc displacement without reduction, without limited opening

Group III: Arthralgia, arthritis, arthrosis (joint disorders)
- IIIa. Arthralgia
- IIIb. Osteoarthritis of TMJ
- IIIc. Osteoarthrosis of TMJ

A subject can be assigned at most 1 muscle (Group I) diagnosis. Each joint may be assigned at most 1 Group II diagnosis and 1 Group III diagnosis. This means that, in principle, a subject can be assigned from 0 to 5 diagnoses; however, cases with more than 3 diagnoses appear to be rare.[17]

The RDC/TMD patient interview was extended with questions on pain (somatic system involvement, pain zone, character, pattern frequency, onset, and modifying factors) and TMJ dislocation characteristics. The TMJ dislocation characteristics were frequency of dislocation events (once or more a day, once or more a week, less than once a week), factors that triggered the onset of dislocation (spontaneous onset, random mandibular movement, chewing, speaking, laughing), dislocation pattern (generally starting at the right side, at the left side, no pattern), duration of dislocations (very short, few seconds, more than 1 minute), reduction of dislocations (spontaneous, manipulative repositioning, assisted repositioning), and consequences of dislocation events (no consequence, stiffness, pain, stiffness and pain).

TMJ dislocation, also known as open lock or subluxation, was defined as a condition in which the condyle is positioned anterior to the articular eminence and is unable to return to a closed position. It is manifested clinically as an inability to close the mouth without a specific manipulative maneuver. There is usually a clinical history of excessive range of motion that is not painful, but pain may occur at the time of dislocation, and residual pain may follow the episode. Dislocation may be the result of a physical jamming of the disc-condyle complex beyond the articular eminence that is maintained by muscle activity or a true hyperextension of the disc-condyle complex beyond its normal translation position.[32] A capsular

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condition was assessed clinically by means of additional registration of reproducible incoordination and jumps during mandibular movement, evaluation of the quality of joint endfeel, joint play under distraction, and the presence of a preauricular depression at the end of mandibular opening. Joint endfeel, a means for assessing condylar function during the range of motion testing, was assessed during assisted maximal opening by noting the quality of the movement at the end of the assisted opening. Its quality was scored either as normal, hard, soft, or stiff and as with or without pain. To test the capsular ligaments, joint play was performed by applying caudal force on the joint. This permitted discrimination between joint and muscle as sources for restriction. The quality of the movement on caudal joint distraction was classified either as normal, hypomobile, or hypermobile and as with or without pain.[33] A preauricular depression was defined as the clinical presence of an extraoral depression in front of the external auditory meatus and situated at the lateral pole of the condyle, presenting at the end of the mandibular opening cycle, and was scored positive if assessed by combined observation and palpation.[28] Radiological assessment of condyle hypertranslation, ie, the condyle excessively passing the eminentia at the translation phase of mandibular opening,[21,32] was not performed due to the extent of the examined population.

Statistical analysis was performed with the Fisher exact test and chi-square test for comparison of proportions, the Mann-Whitney test (unpaired Wilcoxon test) to analyze the effect of qualitative factors on continuous variables, and rank correlation analysis to describe the relationship between 2 continuous variables. Receiver operating curve (ROC) analysis was used to compute sensitivity and specificity of a series of clinical signs of TMJ hypermobility. Differences at the $P < .05$ level were considered statistically significant.[34,35] Interexaminer agreement was tested with kappa statistics (Cohen's or κ for nominal and ordinal variables)[33] and intraclass correlation coefficients (ICCs) were computed for continuous variables.

The study design had previously been approved by the Ethical Committee of Ghent University Hospital. Informed consent was obtained from the subjects.

Results

Measurement Reproducibility

All kappa values guaranteed an acceptable level of interexaminer agreement. Nominal and ordinal variables tested included palpation for muscle (at 16 different sites; $\kappa = 0.60$) and joint tenderness (lateral and posterior pole; $\kappa = 0.78$), evaluation of the quality and occurrence of joint sounds on vertical opening ($\kappa = 0.69$) and with excursive movements ($\kappa = 0.65$), and registration of the jaw opening pattern ($\kappa = 0.78$). The ICCs were computed for continuous variables, such as linear measurements of mandibular border positions, and ranged from 0.92 to 0.99, indicating excellent reliability between the calibrated examiners. The values obtained for both methods to analyze reliability proved acceptable compared to previously reported values (Table 1).[30,33,36]

Clinical Assessment of Hypermobility of Peripheral Joints

Of the HG patients, 88.1% received a Beighton score ≥ 3; 73.8% of those patients received a score ≥ 5 (median ± SD 5.0 ± 2.4). In 11.9% of the HG patients, the Beighton criteria for GJH were not met (< 3) because of chronic joint pain, edema, or chronic stiffness. In the adult HG subgroup, a score ≥ 3 was recorded for 88.9% of the subjects (median ± SD 5.8 ± 2.7). No inter-relationships could be assessed between Beighton score and gender, age, or disease subtype. No individual in the CG had a Beighton score ≥ 3 (0%), which was indicative of normal peripheral joint mobility.

TMD Prevalence in Hypermobile Population

Sixty-nine percent of the subjects were assigned a diagnosis of myofascial pain (Group Ia), whereas unilateral and bilateral disc displacement with reduction (Group IIa) were diagnosed in 9.5% and 76.2% of the subjects, respectively. TMJ arthralgia (Group IIIa) was assigned unilaterally or bilaterally in 23.8% and 38.1% of HG subjects, respectively. When individual TMD diagnoses were analyzed (Table 2), 7.1% of the individuals were found to have no diagnosis, and 23.8% were assigned a single diagnosis (ie, 7.1% unilateral and 14.3% bilateral disc displacement with reduction, and 2.4% bilateral TMJ arthralgia). Multiple TMD diagnoses were assigned in 69.1% of the individuals, of whom 57.1% presented with 3 or more different RDC/TMD group diagnoses (Table 2). Thirty of

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Table 1 Interexaminer Reliability for Measurements of Clinical Signs of TMD

Clinical sign	Statistics*	Tested	Reported†
Vertical dimension			
Unassisted opening without pain (mm)	ICC	0.97	0.90–0.94
Maximum unassisted opening (mm)	ICC	0.99	0.96–0.98
Maximum assisted opening (mm)	ICC	0.99	0.94–0.98
Jaw opening pattern	K	0.78	0.56–0.70
Jaw excursions			
Lateral excursions (mm)	ICC	0.98	0.67–0.70
Protruded movement (mm)	ICC	0.92	0.30–0.68
Joint sounds on vertical opening (on palpation)	K	0.69	0.62–0.79
Joint sounds with excursive movements (on palpation)	K	0.65	0.37–0.75
Pain with function/movement (mean)	K	0.80	0.63–0.83
Pain on palpation			
Masticatory muscles (mean of 16 palpation sites)	K	0.60	0.52–0.86
TMJ (2 palpation sites)	K	0.78	0.52–0.84

*ICC = intraclass correlation coefficient (> 0.90, excellent; 0.80–0.89, good; 0.70–0.79, acceptable; < 0.70, not acceptable); K = Cohen's kappa (> 0.8, excellent; 0.6–0.8, good; 0.4–0.6, acceptable; < 0.4, not acceptable).
†Wahlund et al,[30] Dworkin et al,[33] and Goulet et al.[36]

the 42 cases were symptomatic (71.4%), ie, the subjects reported pain or tenderness of masticatory muscles and TMJ, joint sounds, and/or limitation or disturbance of mandibular movement, indicating a strong tendency for development of TMD in patients with GJH. Of these symptomatic subjects, 13.3% had sought treatment themselves.

In the adult HG subgroup, 70.4% of the subjects were assigned a diagnosis of myofascial pain (Ia), 96.3% were assigned a diagnosis of disc displacement with reduction (IIa) (14.8% unilaterally and 81.5% bilaterally), and 59.2% were assigned a diagnosis of TMJ arthralgia (IIIa) (18.5% unilaterally and 40.7% bilaterally). All subjects had TMD (100%); 92% were assigned multiple TMD diagnoses. In the CG (n = 40), 85% of the subjects had a muscle diagnosis (Group I); 35% had myofascial pain (Ia) and 50% had myofascial pain with limited jaw opening (Ib). Bilateral disc displacement diagnoses were assigned to 60% of CG subjects: 52.5% had disc displacement with reduction (IIa) and 7% had disc displacement without reduction and limited jaw opening (IIb). A TMJ arthralgia diagnosis (IIIa) was assigned in 37.5% of cases. Eighty-five percent of the CG subjects had multiple TMD diagnoses.

Pain History and Pain Characteristics

Diagnoses of myofascial pain alone (MP) were significantly less prevalent in the adult HG subgroup (11%) compared to CG (50%) (P = .001). Myofascial pain with unilateral or bilateral arthralgia (MPA) occurred in a higher rate in the adult HG subgroup (59% in the adult HG subgroup versus 35% in CG), but the difference was not statistically significant (P = .071) (Table 3).

Table 2 Individual TMD Subgroup Diagnoses in the Hypermobile Population (n = 42)

Individual diagnosis	n	Proportion (%)
No TMD diagnosis	3	7.1
Single TMD group diagnosis	10	23.8
Group Ia	0	0.0
Group IIa (uni- or bilateral)	9	21.4
Group IIIa (uni- or bilateral)	1	2.4
Multiple TMD group diagnosis	29	69.1
Group I + II	5	11.9
Group I + II + III	24	57.2

A subject could be assigned a maximum of one muscle diagnosis (Group I), whereas each joint could be assigned a maximum of one diagnosis from Group II and one diagnosis from Group III.[17,18]

The incidences of joint noises (96% in HG versus 60% in CG) and TMJ dislocations (100% in HG versus 0% in CG) were both significantly greater in the HG adult subgroup (P < .001 for both). Interpersonal factors (ie, social or family problems) and emotional status (ie, depression, anxiety) were assessed as contributing psychosocial factors in TMD etiology significantly more frequently in HG subjects than in CG subjects (P = .013 for interpersonal factors; P = .046 for emotional status).

There were no significant differences in pain character (ie localized, migrating, spreading, or

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Table 3 Individual TMD Pain Diagnoses in the Adult HG Subgroup and CG

	HG (n = 27)		CG (n = 40)		
	n	%	n	%	*P**
Myofascial pain	3	11	20	50	.001
Myofascial pain with arthralgia	16	59	14	35	.071

*Fisher's exact test; differences at the $P < .05$ level were considered significant.

Table 4 TMJ Dislocation Occurrence Among Symptomatic and Asymptomatic Hypermobile Individuals (n = 42)

	Asymptomatic (n = 12)		Symptomatic (n = 30)		
	n	%	n	%	*P**
Once or more a day	1	8	22	73	< .001
Once or more a week	7	58	5	17	
Less frequent	4	34	3	10	

*Chi-square tests (2 × 3 table); differences at the $P < .05$ level were considered significant.

irradiating) between the adult HG, CG, and TMD subgroups ($P = .455$ for myofascial pain; $P = .595$ for MPA). With regard to pain onset (ie, spontaneous, provoked, or triggered onset) and pain frequency no differences were established between the distinct groups ($P = .811$ for myofascial pain; $P = .333$ for MPA). Although a diurnal pain pattern was frequently reported in symptomatic HG patients (67% among MP and 75% among MPA), no significance differences were found compared to symptomatic controls ($P = .602$ for MP; $P = .540$ for MPA).

TMJ Dislocation Characteristics

When comparing symptomatic and asymptomatic subjects with GJH, ie, individuals with or without a report of pain or tenderness in the masticatory system, a significant difference was found in occurrence of TMJ dislocations: 73% of the symptomatic cases reported dislocations at a rate of once or more a day, whereas 58% of pain-free subjects reported an occurrence of once or more per week ($P < .001$) (Table 4). There was no significant difference concerning the factors provoking dislocations between symptomatic and asymptomatic subjects ($P = .777$). Duration of the dislocations was generally longer in symptomatic HG subjects ($P < .001$). No interrelation was found between these dislocation characteristics and other clinical observations (eg, hypermobility score, range of mouth opening) or pain history records (eg, system involvement, pain character, pain onset) among symptomatic or asymptomatic subjects. TMJ dislocation was not recorded in controls.

Evaluation of Clinical TMJ Hypermobility Signs

A series of clinical observations indicative of the character and the extent of condylar mobility was tested for measurement reliability and diagnostic validity. Although linear measurement of mandibular border positions is not considered a highly reliable method for assessing condylar mobility or hypermobility,[37–39] active range of opening movement (AROM) was significantly greater in both sexes in the HG (HG males 52.50 ± 1.56 mm versus male controls 41.4 ± 7.4 mm, $P < .001$; HG females 47.5 ± 1.10 mm versus female controls 36.9 ± 9.1 mm, $P < .001$). Unpaired Wilcoxon tests calculated AROM to be significantly influenced by gender in HG ($P < .001$), and by the presence of self-reported pain ($P < .001$), pain on excursion ($P < .001$), and pain on palpation ($P = .048$) (Table 5). Rank correlation analysis yielded no association between AROM and age (Spearman's coefficient $r_s = –0.113$, $P = .469$), body height ($r_s = .183$, $P = .183$), or hypermobility score ($r_s = 0.189$, $P = .225$).

Intraexaminer reliability was good to excellent for all variables (Table 6). Since there were no significant differences in the expression of these clinical signs or symptoms between adults and children in the HG, the data were pooled. Sensitivity and specificity and the positive likelihood ratio and negative likelihood ratio of the clinical observations are displayed in Table 7. A large endfeel distance in the absence of muscular pain (sensitivity 92.9; specificity 88.0), large linear measurements of lateral border positions (sensitivity 90.5; specificity 92.0), reproducible "jumps" during mandibular movement (sensitivity 100.0; specificity 82.0), a preauricular depression (sensitivity 95.2; specificity 84.0), and recurrent TMJ dislocations (sensitivity 97.6; specificity 90.0) seem to be useful as reliable clinical signs indicative of increased condyle mobility (as a result of structural capsular laxity). These variables had values for the area under the ROC curve (≥ 0.900), which is indicative of excellent discriminatory diagnostic capacities.[34,35]

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Table 5 Mann-Whitney test (unpaired Wilcoxon test) for Qualitative Factors Influencing the Active Range of Mandibular Opening in the HG (n = 42)

Dependent variable	Qualitative variable/subgroup	Mean	SD	*P**
Maximum opening (mm)	Gender			
	Male	52.50	1.56	< .001
	Female	47.50	1.10	
	Self-reported pain			
	Pain	48.04	1.23	< .001
	No pain	50.52	1.41	
	Pain on excursion			
	Pain	48.10	1.22	< .001
	No pain	50.39	1.50	
	Pain on movement			
	Pain	48.67	1.06	.048
	No pain	49.84	2.11	

*Differences at the $P < .05$ level were considered significant.

Table 6 Intraexaminer Reliability for Measurements and Clinical Observations Indicative of TMJ (Hyper)mobility

Clinical observation	Statistics*	Intraexaminer reliability
Maximal unassisted mandibular opening (mm)	ICC	0.96
Endfeel distance (mm)	ICC	0.92
Hyperelastic endfeel quality	K	0.90
Lateral excursions (mm)	ICC	0.89
Reproducible incoordination of mandibular movement	K	0.89
Reproducible jumps during mandibular excursions	K	0.88
Preauricular depression at end of opening cycle	K	0.83

*ICC = intraclass correlation coefficient (> 0.90, excellent; 0.80–0.89, good; 0.70–0.79, acceptable; < 0.70, not acceptable); K = Cohen's kappa (> 0.8, excellent; 0.6–0.8, good; 0.4–0.6, acceptable; < 0.4, not acceptable).

Table 7 Sensitivity and Specificity of TMJ (Hyper)mobility Clinical Signs and Symptoms (Hypermobile n = 42; Control n = 40)

Clinical observation	Cutoff	Sensitivity	Specificity	PLR	NLR
AROM					
Males	50.1 mm*	72.7	89.0	6.55	0.31
Females	45.0 mm*	71.0	87.1	5.50	0.33
Endfeel distance	3.6 mm*	92.9	88.0	7.74	0.08
Endfeel hyperelasticity	Present	97.6	38.0	1.57	0.06
Lateral excursions (sum; mm)	19.5 mm*	90.5	92.0	11.31	0.10
Reproducible incoordination of mandibular movement	> 3 trials	95.2	62.0	2.51	0.08
Reproducible jumps during mandibular excursions	> 3 trials	100.0	82.0	5.56	0.00
Preauricular depression at end of opening cycle	Present	95.2	84.0	5.95	0.06
Recurrent TMJ dislocations	> once a wk	97.6	90.0	9.76	0.03

Positive likelihood ratio (PLR) = true positive rate/false positive rate; negative likelihood ratio (NLR) = false negative rate/true negative rate.
*The cutoff values for mandibular border positions were calculated for the measurements obtained in this specific population (males and females and children were pooled, except for AROM).

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Discussion

Conflicting evidence exists in the literature on the role of GJH as a potential risk factor for TMD development.[2,3,19–28] An important aspect contributing to inconsistency in the literature is the problem of how to define and assess GJH. First, there is major controversy on the reported prevalence of GJH, which is entirely determined by the study population and reflects the dependence of hypermobility on age, gender, and family and ethnic background.[2] Previous studies have documented the incidence of signs and symptoms of TMD in heterogeneous populations with GJH[2,3,19–28] or assumed benign joint hypermobility syndrome (BJHS) disorders[24,39] where joint laxity occurs without a known underlying collagen or fibrillin defect. Both BJHS and GJH, as symptoms of connective tissue disease, are supposed to result from capsular laxity, which is generally caused by structural alterations in the supporting ligaments.[30] By definition, BJHS is not synonymous with GJH: BJHS is said to exist when hypermobility becomes symptomatic, and as a rule has more serious (polyarticular) joint involvement.[1,40] There is no agreement on whether the molecular cause of joint hypermobility is different between individuals having GJH and those developing BJHS. Little is known about the biochemical cause and the evolution of joint pathology in BJHS.[40–45] Therefore, BJHS should not be evaluated as a model of disease in order to study the effects of structural joint component laxity on TMD.

Generalized capsular laxity usually is assessed clinically using the Beighton scale.[1,29,46] The score is obtained by measurement of the mobility of 5 peripheral joints, with a score of 3 out of 9 or greater defining hypermobility.[29] Previous studies have set their cutoff value at scores of 3[19,23,38] or 4,[39] or have only assessed 1 or 2 joints on the dominant side,[47,48] which has produced confusing evidence. The cutoff value of ≥ 3 allows for refinement of GJH diagnosis in terms of poly- and pauci-articular (ie, less than 5 joints involved) varieties.[1,29] Epidemiologic studies have shown that joint hypermobility (depending on the population and the criteria used) is seen in 3% of Western populations[49] and up to 10% of Middle Eastern[50] and 25% of African populations,[51] with a rapid decrease in the first decade of life.[29,30] The majority of these studies looked at GJH, but it is known that pauci-articular hypermobility is more prevalent than the poly-articular variety.[45,49] The overall methodological quality of studies on GJH and TMD has also varied considerably, thus influencing the possible association between GJH and TMD.[2] Therefore, some authors have suggested that selection bias could be minimized by studying populations with inherited connective tissue disease as a model of disease with maximum expression of GJH.[2,3] As the TMJ ligaments and disc basically consist of a fibrous network of collagen types I, II, and V and elastin,[52] EDS and MFS were selected as models of disease with known molecular alterations in connective tissue.[3,12]

Second, assessment of GJH may be confounded by a number of factors, including pain of myofascial origin, joint edema, and stiffness, often manifesting in a circadian pattern. For these reasons, the Beighton sum score should be seen as a random indication of peripheral joint mobility standing for the moment of assessment. This explains the presence of a low GJH score in 11.9% of the study subjects.

Third, there is no substantial evidence to assess an interrelation between TMJ hypermobility and hypermobility of the 5 peripheral joints evaluated in the Beighton sum score.[2,3] Consequently, when potential etiologic factors for TMD are considered, restraint is called for assignment of GJH diagnosis in subjects not affected with an inherited connective tissue disorder.

The present study indicates a positive association between structural GJH and TMD. It also confirms reported associations of GJH with a variety of complaints of the general locomotor system, such as myalgia, arthralgia (up to 38% in HG), dislocation of major and peripheral joints (TMJ dislocations in 100% of subjects with GJH), and soft tissue lesions.[2] There were no significant differences in patient and pain characteristics among the symptomatic HG and CG subjects, except for psychosocial factors. The low incidence of treatment seeking in the symptomatic HG subjects (13.3% of all subjects reporting of pain) may be the result of major somatic problems crowding out TMD-related discomfort in the syndromes. Social problems and emotional instability may be the result of the debilitating nature of inherited connective tissue disorders and may substantially contribute to TMD development.

Fifty-nine percent of HG subjects were diagnosed with MPA, compared to 35% in the CG (Table 3), but this difference was not statistically significant. This finding is consistent with former reports of arthralgia being associated with joint instability.[38] The occurrence of joint noises (96% in HG versus 60% in CG) and dislocations (100% in HG versus 0% in CG) were statistically significant findings, confirming previous reports on interrelation between joint clicks and dislocations.[18,30]

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In the HG, AROM was significantly dependent on gender (greater in males; $P < .001$) and the presence of self-reported pain ($P < .001$), pain on excursion ($P < .001$), and pain on palpation, as was previously reported in healthy subjects.[38] The AROM in the HG, contrary to the expectations in GJH, was low compared to reports of AROM in healthy individuals[21–23,28,37,38] but this might be explained by their TMD.

Although a high occurrence of osteoarthrosis has been reported in hypermobile TMJs of healthy patients,[53] no degenerative disorders of this nature were found in the study group. Since degenerative TMJ disorders are known to occur with increasing age, the latter finding may be influenced by the age distribution in the sample group.

At present, there is no general agreement on the point at which the TMJ should be classified as hypermobile. Radiographically, it has been postulated that the TMJ is hypermobile when the condyle is excessively passing the articular eminence at the translation phase of mandibular opening.[21,53] Epidemiologic surveys, however, have yielded prevalences of radiographically assessed condylar hypertranslation up to 39%, contesting the diagnostic validity of this criterion.[54] Despite a considerable number of reports on the subject, there are not yet any validated criteria for clinical assessment of TMJ hypermobility. Since the range of mandibular movements is reported to be closely related to facial morphology,[55,56] linear measurement of mandibular border positions generally is not considered a highly reliable method for assessing condylar (hyper)mobility.[37–39] Only a few weak correlations were found between linear measurement of maximal mandibular opening capacity and peripheral joint mobility either at active or assisted range of motion.[23,38,57]

Recurrent TMJ dislocations were commonly recorded during the structured patient interview as a consequence of capsular laxity. This study also showed that the occurrence and duration of dislocations were respectively higher and longer in symptomatic GJH individuals compared to asymptomatic individuals. Further research is needed to elucidate the contribution of dislocations to the development of TMD. Table 7 displays a series of clinical signs or symptoms of TMJ hypermobility that were more expressed in adult GJH subjects compared to adult control subjects with TMD and normal joint mobility. A hyperelastic endfeel with large endfeel distance and a preauricular depression on maximum opening have been suggested as clinical indications of condyle hypertranslation,[28] but these signs lacked epidemiologic validation. Moreover, joint endfeel quality and a preauricular depression may be difficult to establish in the presence of pain. Two other frequent clinical observations in patients with capsular laxity, reproducible incoordination of mandibular movement (on 3 or more consecutive trials and occurring during both vertical and horizontal excursions), and "jumps" during these movements,[28] also yielded an acceptable diagnostic reliability in this study. However, since the cutoff values for clinical TMJ hypermobility measurements are entirely dependent on the population, it is recommended that a combination of these indicative signs be used in diagnosis rather than use cutoff values as absolute criteria for assignment of a condylar hypermobility diagnosis. This is certainly true in the linear measurement of AROM in the presence of myofascial pain. Future analysis of potential risk factors and confounders in larger hypermobile populations (children versus adults) may provide the clinician with odds ratios of risk factors and better insight into the role of structural capsular defects for TMD development.

The present study analyzed signs and symptoms of TMD in a population of patients with a clear clinical, biochemical, and genetical diagnosis of EDS or MFS. Both disorders are characterized by GJH, which is caused by aberrations in the biosynthesis of connective tissue components, leading to structural alterations of joint components. A recommendation to examine the peripheral joints in any TMD patients cannot be offered on the basis of this study. However, since a high score on the Beighton scale may indicate severe connective tissue involvement, an additional examination needs to be performed in any patient presenting with TMD and hypermobility characteristics. In such cases, the examiner should be suspicious of a connective tissue involvement.

Conclusion

The present study aimed to assess TMD in a population with an inherited connective tissue disorder as a model of disease with maximum expression of GJH. The data on prevalence and patient characteristics of TMD in this population indicate a positive association between GJH and TMD, the greater proportion presenting a combination of myofascial pain and disc displacement associated with uni- or bilateral TMJ arthralgia. A series of clinical signs indicative of condylar hypermobility was presented, together with computed values for sensitivity and specificity. Reliable discriminatory diagnostic capacities for assessment of endfeel

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distance in the absence of muscular pain, linear measurement of lateral border positions, reproducible "jumps" during mandibular movement, a preauricular depression at the end of the opening cycle, and recurrent TMJ dislocations were also shown. Linear measurement of maximal unassisted mouth opening was significantly greater in GJH subjects compared to controls. No association was found between this measurement and with body height or masticatory or TMJ pain symptoms, but it proved to be related to gender. The findings suggest that, as in healthy subjects, the vertical range of mandibular movement in GJH patients is not a reliable instrument for assessing condylar (hyper)mobility. Recurrent TMJ dislocations (once or more a day, with a duration of several seconds) were a frequent finding in symptomatic GJH individuals compared to asymptomatic individuals, but their contribution to TMD development remains elusive.

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