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ORIGINAL ARTICLE

Vural Kavuncu · Sezai Sahin · Ayhan Kamanli
Ayse Karan · Cihan Aksoy

The role of systemic hypermobility and condylar hypermobility in temporomandibular joint dysfunction syndrome

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Abstract *Objective*: To evaluate the risk of temporomandibular joint dysfunction (TMD), when both systemic joint hypermobility (SJH) and localized condylar hypermobility (LCH) exist. *Materials and methods*: Sixty-four consecutive outpatients with temporomandibular joint clicking or pain, and 77 sex- and age-matched control subjects, were recruited in the study. LCH was diagnosed when condylar subluxation was present, and SJL was diagnosed by using Beighton's method. The frequency of symptoms, mean mouth opening, and the frequency of subjects with SJL and LCH were the main outcome measures. *Results*: Out of the 64 patients, 16 patients were suffering from pain, 20 patients from joint-clicking and 28 patients from both. Both SJH and LCH were more frequently observed in those patients with TMD than in control subjects. The risk of TMD was higher if LCH and SJH existed jointly. *Conclusion*: Both systemic and localized hypermobility may have a role in the etiology of TMD.

V. Kavuncu
Department of Physical Medicine and Rehabilitation,
Medical Faculty, Afyon Kocatepe University,
Afyon, Turkey

S. Sahin
Physical Medicine and Rehabilitation Clinic,
Kahramanmaras Devlet Hastanesi,
Kahramanmaras, Turkey

A. Kamanli
Department of Physical Medicine and Rehabilitation,
Medical Faculty, Firat University, Elazig, Turkey

A. Karan · C. Aksoy
Department of Physical Medicine and Rehabilitation,
Istanbul Medical Faculty, Istanbul University,
Istanbul, Turkey

V. Kavuncu (✉)
AKU, ANS Uygulama ve Arastirma Hastanesi,
Fizik Tedavi Klinigi, İnonu Bulvari Afyon,
03200, Turkey
E-mail: kavuncu@aku.edu.tr
Tel.: +90-505-5618010
Fax: +90-272-2172029

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Introduction

Temporomandibular joint dysfunction (TMD), characterized by chronic facial pain, is a complex clinical condition involving either masticatory muscles or the temporomandibular joint (TMJ). Patients suffering from pain on muscle palpation and/or mandibular movements, joint sounds and limitation on the movements of mandibula are diagnosed as suffering from TMD, which is an umbrella term.

The correlation between TMD and systemic joint hypermobility (SJH) has been reported in some studies [1–4]. SJH is a systemic disorder which is characterized by excessive movement of the joints. A lax capsular or ligamentous structure predisposes to condylar hypermobility or TMJ disk displacement, which are associated with parafunctions, overwork, and trauma. Overwork is believed to be the result of altered occlusal relationship, joint pathology, and internal derangement which subsequently cause muscle spasm and pain.

The abnormal relationship between the TMJ capsule, the bony architecture, and the associated musculoligamentous structure could result in a localized condylar hypermobility (LCH). We do not know much about the role of LCH in the development of TMD when SJH is either present or absent. We investigate the risk of TMD when both SJH and LCH exist.

Materials and methods

The subjects in this study were selected from patients who applied to the outpatient clinic of a University Hospital for different reasons. Subjects were included in the study if they were between age 15 and 60 and willing to participate. They were excluded if they were pregnant

or if they had a systemic rheumatologic disease, a TMJ condylar growth deformity, a systemic or joint infection or a malignancy. Subjects who agreed to participate in the diagnostic survey underwent clinical examination conducted by a physician, and an informed consent was obtained from the patients. All clinical examinations were performed by the same examiner. The screening was based on the Research Diagnostic Criteria for Temporomandibular Disorders (RDC/TMD) [5]. This index classifies TMD into muscle disorders, disc displacements, and degenerative joint disease such as osteoarthritis.

The joints and masticatory muscles at 20 muscle sites were palpated for the existence of either pain or tenderness. Myofascial pain diagnosis was based on either self-reporting of pain at rest or tenderness to palpation of at least three muscles in the defined sites. On average, 1 kg digital pressure was applied on muscle sites. Mouth opening was assessed by edge-to-edge measuring from the upper to the lower incisors when a subject is asked to open his or her mouth. Mouth opening of about 40 mm or less was accepted as limited movement. During the palpation, the physician searched for joint clicking. Disc displacement was diagnosed in the presence of joint clicking. Non-reducible disc displacement was diagnosed if significant limitation exists in opening even in the absence of joint sounds. The terms arthralgia and osteoarthritis were used when pain or crepitus on the joint was recognized, or in the presence of clues of degenerative arthritis on X-rays.

The overall sample consisted of 64 patients diagnosed with TMD and 77 control subjects who were sex- and age-matched with no evidence of current or past TMJ symptoms. The diagnosis of LCH was based on the radiological evidence of condylar subluxation. Direct roentgenograms of TMJ in both the mouth opened and closed positions were obtained in a specific lateral position. An anterior displacement of condyle through the eminence of the temporal bone was accepted as subluxation. SJH was evaluated by Beighton's method, which involves a scoring scale of 9. An equal or a greater score than 4 was accepted as hypermobility.

Independent Student's-*t*, chi-square, Mantel-Haenszel chi-square and Pearson correlation tests were used for statistical analysis.

Results

Patients with TMD and the control group did not differ significantly in terms of age (the mean ages were 31.6 ± 10.2 and 34.8 ± 10.4 respectively) and sex (39% of the subjects with TMD and 45.4% of the control subjects were women).

Patients experiencing the symptoms of either pain or clicking related to TMJ or craniofascial muscles were classified according to the RDC/TMD index. Out of 64 patients, 16 patients (25%) were suffering from pain, 20 patients (31.2%) from joint-clicking, and 28 patients (43.7%) from both. Ten patients (15.6%) in the patient group and four subjects (5.7%) in the control group had limited mouth-opening distance (less than 40 mm). Forty-four patients (68.8%) were diagnosed as having muscle disorders, 48 patients (75%) as having disc displacement and 16 patients (25%) as having arthralgia or degenerative osteoarthritis. A subject was assigned to more than one diagnosis group in the event that he or she had symptoms relating to more than one condition.

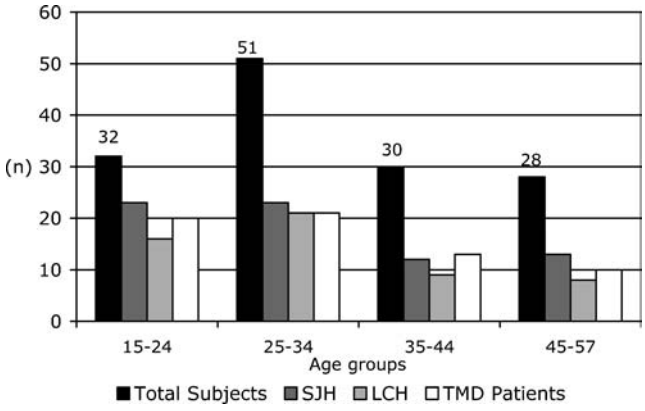

Fig. 1 The existence of systemic joint hypermobility, localized condylar hypermobility and TMD in different age groups. *LCH* localized condylar hypermobility, *SJH* systemic joint hypermobility

Since the age range of the study population was wide (15–57), the study population was divided into age subgroups (5–24, 25–34, 35–44, and 45–57) to specify differences between these groups in the population (Fig. 1). Proportionally, SJH, LCH, and TMD were mostly seen in subjects between ages 15 and 24.

Figure 2 displays the existence of SJH or LCH in both TMD patients and control subjects. Both systemic joint laxity and TMJ laxity were more frequently observed in those patients with TMD than in control subjects ($p < 0.001$). LCH was found in 38 subjects

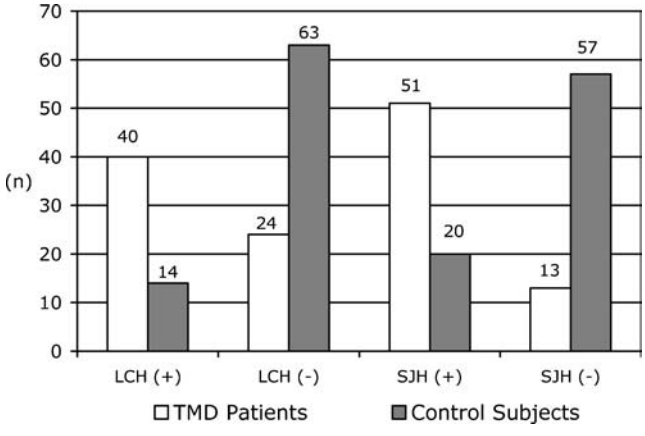

Fig. 2 The existence of localized condylar hypermobility and systemic joint hypermobility in TMD patients and control subjects. *LCH* localized condylar hypermobility, *SJH* systemic joint hypermobility

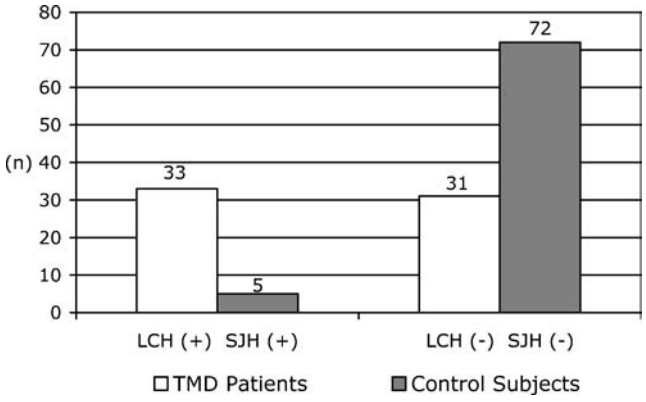

Fig. 3 The co-existence of localized condylar hypermobility and systemic joint hypermobility in TMD patients and control subjects. *LCH* localized condylar hypermobility, *SJH* systemic joint hypermobility

(53.5%) with SJH and in 16 subjects (22.8%) without SJH ($p < 0.001$).

LCH and SJH were present together in 38 subjects, and TMD was diagnosed in 33 (86.8%) of them. On the other hand, TMD was diagnosed only in 31 subjects out of 103 (30.1%) who had neither SJH nor LCH (Fig. 3). The odds ratio and 95% confidence intervals for hypermobility syndromes in TMD were as follows; for SJH 11.2 (5.1–24.7), for LCH 7.5 (3.5–16.2), and for SJH and LCH co-existence 15.3 (5.5–43). The risk of TMD was higher if LCH and SJH were present simultaneously (Mantel–Haenszel chi-square test, $p < 0.001$).

Discussion

In this study, the subjects consisted of both TMD patients and healthy subjects. TMD was diagnosed based on the data which were reported by the patients and on physical examination of the masticatory system and the joints. The majority of the patients were suffering from joint-clicking and jaw pain. TMJ pain mostly originated from the compression of the highly-vascularized and innervated posterior disk attachment, stretching the joint capsule, and joint distension due to TMJ effusions occurring in joints with disk displacement [6]. Disk displacement either with or without opening limitation is accepted as TMD. An anterior displaced disk is not always related to muscle pain or a limited range of motion. Therefore, the need for treatment for those patients who have only joint-clicking due to disk displacement is under debate. A previous study showed that most patients with clinically-detectable dysfunction without significant symptoms of TMJ do not seek treatment [7].

The current study suggests a positive correlation between SJL and TMD, which is consistent with some previous studies [8–10]. Joint laxity might be seen in healthy individuals without any symptoms, whilst it can cause chronic pain complaints in some individuals. It has been previously reported that oral parafunctions are correlated more strongly with the signs and symptoms of TMJ when SJL is present [11]. Clinical features related to the musculoskeletal system and visceral organ involvements in hypermobile subjects suggest a disorder of generalized collagen tissue. Skin biopsy revealed lower values of total collagen and a higher ratio of collagen type III to III + I in TMJ patients than in those of control subjects [12]. Clinical studies have also shown a close relation between LCH and TMD. Those patients with symptomatic LCH demonstrated some pathological changes such as hypertrophy, atrophy, and contracture in their lateral pterygoid muscle [13].

Impaired joint proprioception and excessive mobility could lead to ligament and joint-capsule trauma in hypermobile TMJ. When joint-clicking is present, mouth opening has been reported as larger [14]. Normal interincisal mouth opening has previously been defined as 48.9 ± 4.9 mm [15]. Mean measurement for both TMD and the control group were both within the normal range of motion for the mandible. Although we found mouth opening larger for the TMD patients, it was not statistically significant. Because the main cause of condylar subluxation is early translation rather than rotation movement of the jaw, excess mouth opening is not a prominent feature of LCH. Muscle spasm might prevent excess movement in TMD patients with hypermobility.

In the present study, a close relation was found between SJH and LCH. The TMD risk was higher in when both SCH and LCH were present than in any alternative situation. This suggests another, additive effect of both conditions in the etiopathogenesis of TMD. Because there is a close relationship between TMD and both systemic and localized laxity, these conditions have to be taken into account in clinical practices. Localized TMJ laxity could be a clinical indication of systemic joint laxity.

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