Correlation between Dento-Skeletal Characteristics and Craniomandibular Disorders in Growing Children and Adolescent Orthodontic Patients: Retrospective Case-Control Study

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SUMMARY

Purpose. The aim of this retrospective case-control study was to identify, in a group of growing children and adolescents affected by malocclusion, specific dento-skeletal characteristics which could be correlated to the onset, in the above-mentioned subjects, of craniomandibular disorders (CMD).

Materials and methods. Among the patients treated at the Paedodontics and Orthodontics department of Bari Dental School, we recruited a group of patients with malocclusion and symptoms of temporomandibular disorders, as an experimental group. We considered as controls those patients who, match-paired to their skeletal class depending on the ANB angle, did not show any CMD sign or symptom.

Results. Of the 128 examined patients, 15 showed signs and/or symptoms of CMD (11.7%). When compared to 15 patients non-affected by CMD, we could not detect statistically significant differences in both skeletal and occlusal characteristics. It is still interesting to notice how in CMD patients, characteristics of skeletal hyperdivergence are often to be found.

Conclusions. The present study seems to confirm that in growing children and adolescents, the presence of signs and/or symptoms of CMD is not associated to a specific vertical skeletal growth pattern or to other specific occlusal characteristics.

Key words: joint, orthodontic, pain, growth, occlusion.

Introduction

The American Academy of Orofacial Pain defines “Craniomandibular Disorders” (CMD) as a heterogeneous group of clinical pathological conditions affecting the structures of temporomandibular joint, masticatory muscles and articular disc (1).

CMD have a multifactorial etiology, with different risk factors interacting at the individual level (2). CMD can be sometime associated with oral diseases (3-11).

The role of malocclusions as skeletal predisposition to CMD disorders is a debated topic, still posing an unanswered question. Manfredini et al. in a systematic review of the literature on temporomandibular disorders and facial morphology (12) reported that only a single research group assessed the relation of specific cranio-facial
characteristics with CMD in adolescents. In young patients there are some difficult situations (13-15) which require imaging investigation (16).

The objective of the present retrospective case-control study was to identify, in a group of growing children and adolescents affected by malocclusion, specific dento-skeletal characteristics we could correlate to the onset of craniomandibular disorders (CMD).

### Materials and methods

The sample of this retrospective study included 128 patients (mean age: 12.7 years; 65 females, 63 males) treated at the Paedodontics and Orthognatodontics department of University of Bari Dental School. Among these patients we selected as experimental group 15 patients with type I, II, III skeletal class malocclusions (measured by the ANB angle), who showed, after a gnathological evaluation, signs or symptoms of temporomandibular disorders according with the RDC/TMD (Reaserch Diagnostic Criteria for TemporoMandibular Disorders) (17). We considered as controls 15 patients who did not manifest any signs and/or symptoms of craniofacial disorders, and affected by the same skeletal sagittal malocclusion, in accordance with the matching of the ANB angle (considered as a matching criteria).

All the data and information relative to each recruited patient were inferred from their clinical history, examining the patient’s clinical records, radiographs and dental casts.

The inclusion criteria were as follows: age under 18; no history of orthodontic treatment; diagnosis of CMD; lateral X-rays with excellent contrast. The exclusion criteria were: incomplete clinical documentation; presence of systemic pathologies and conditions characterised by facial and/or cervical algia different from CMD (for example, fibromyalgia); previous gnathological and/or orthodontic treatments and/or prosthetic rehabilitations; previous traumas and/or fractures.

We performed a clinical evaluation of craniofacial disorders, according with the European Academy of Craniofacial Disorders (E.A.C.D.) protocol, consisting in:

- an anamnestic questionnaire;
- a clinical examination recording the subjective perception of the pain with a Visual Analogue Scale (VAS) adequate to the patient’s age. The presence of vicious habits was also investigated;
- extent of the maximum opening. The presence of a limitation was recorded for maximum opening below 40 mm;
- with the same method (see above) we measured the distance between the upper and lower median line in laterality and protrusion movements. For laterality the normal value ranges from 8 and 12 mm, while for protrusion it is higher or equalling 6 mm;
- the opening deviation was evaluated considering the path of the median line of the mandible during maximum opening, it was reported as present or absent;
- evaluation of “endfeel”;
- execution of the “joint muscle test”;
- execution of the “joint play test”;
- execution of the “compression test”;
- the masseter, the anterior, median and posterior region of the temporal bone, the lateral and median pterygoid muscle, the muscles of the submandibular region, the neck and shoulders were digitally palpated, detecting the absence/presence of pain;
- spontaneous or induced pain in the ATM was determined with the digital palpation of the joint, both at rest and in dynamic phase, during mandibular movements (18).

Lateral cephalograms for the craniofacial skeletal evaluation were traced on acetate sheets. The linear and angular measures analysed are shown in Figure 1.

On dental casts we evaluated the extent of overjet and overbite, with a millimetre callipers, and the presence/absence of anterior and/or posterior crossbite (unilateral or bilateral) and scissor-bite. For both overbite and overjet we considered pathologic values below 0 and over 2 mm.
For each recruited patient we collected data, in anonymous form, on an Excel sheet; these data were analysed with Stata/MP11 software. For dichotomous and category variables we evaluated the absolute frequency, the proportions were compared with the chi-square test. In order to evaluate the association between variables, we calculated the odds ratio values, indicating the confidence intervals and using the z-score test. Continuous variables were described as mean, indicating the standard deviation and the median; medians were compared with the Wilcoxon-Mann-Whitney rank-sum test, analogue non-parametric of the t-student test for non-matched samples.

## Results

Of the 128 examined patients, 15 subjects (10 boys and 5 girls) showed signs and/or symptoms of CMD (articular and/or myofascial unilateral or bilateral pain, click, reduced mouth opening - locking or functional limitation - and deviation of the median line during mandibular movements); a percentage of 11.72%.

We selected our experimental sample after a gnathological evaluation, then we subdivided it according to the typology of symptoms and signs. Of the 15 patients, 10 complained myofascial and/or articular pain (at rest or during palpation), 7 showed articular noises (click) and 3 had limited mouth opening. Of these, 1 showed pain and click simultaneously, 2 had pain associated with reduced opening and 1 had all 3 symptoms; these data are shown in Figure 2. Furthermore, 3 subjects had deflexion during opening and 2 reported two accessory symptoms like cephalaea and otalgia.

The control group consisted of 15 patients (8 girls and 7 boys) chosen among those who did not show any sign and/or symptom of CMD, in accordance with the inclusion/exclusion criteria, were match-paired with the subjects of study for gender and age (approximately), and for skeletal class (precisely), depending on the correspondence of the cephalometric value of the ANB angle.

With the cephalometric measurement of the ANB...
angle, our matching factor, the sample was divided into the three skeletal classes: 6 class I subjects, 6 class II subjects and 3 class III subjects for each group, as shown in Table 1.

We radiographically analysed the skeletal characteristics of the entire sample and we evaluated the differences between the two groups calculating the non-parametric correlation coefficient (Table 2).

We measured the angular and linear measures of the cranial base (Ba-S-N, S-N, Ba-S Ba-N), as well as the gonial angle and the length of the mandibular branch and then we matched the two groups with the same methods, obtaining the same, non-significant, results.

In the same way we then matched the values of the craniomandibular and intermaxillary angle and the anterior vertical dimensions.

After this, we evaluated the divergence measuring the intermaxillary angle, observing that none of the patients showed the distinctive characteristics of hypodivergence, while 13 subjects resulted mesodivergent and 17 hyperdivergent; 9 of them belonged to the experimental group (Table 3).

The ratio between the anterior vertical dimensions (Sor-SNA e SNA-Me) was evaluated in relation to the age of each subject. It emerged that only 3 subjects out of 30 showed a skeletal normocuvitibite, while 14 had an open bite ratio and 13 a deepbite one (Table 4).

The dental patterns were evaluated on casts, calculating the extent of the overjet and the overbite and the potential presence crossbite and its typology (anterior, unilateral posterior, bilateral posterior, scissors-bite). Of 30 subjects, 7 had crossbite, of whom 4 unilateral posterior crossbite, 2

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Table 1 - Descriptive statistic of age and skeletal class of study groups.

<table>
<thead>
<tr>
<th></th>
<th>CDM group</th>
<th>Control group</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Average</td>
<td>10y10m</td>
<td>10y7m</td>
</tr>
<tr>
<td>Range</td>
<td>8y2m - 17y 9m</td>
<td>8y - 12y 11m</td>
</tr>
<tr>
<td><strong>Skeletal class</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Class I</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Class II</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Class III</td>
<td>3</td>
<td>3</td>
</tr>
</tbody>
</table>

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Figure 2
Distribution of symptoms in the experimental group.
scissors-bite and 1 anterior crossbite, with similar distribution in both groups. The overjet resulted to be normal in just one subject, whereas the vast majority showed an increased ratio (27 individuals); 2 patients had inverted overjet. In this latter case we could not detect significant differences among the two samples.

A more interesting result, although just below the significance threshold, was the one relative to pathological overbite: this characteristic was in fact found with a higher frequency in the study sample (P=0.068), with a prevalence of the deep

### Table 2 - Mean, standard deviation, median and coefficient of correlation between the two groups.

<table>
<thead>
<tr>
<th></th>
<th>CDM group</th>
<th>Control group</th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Std.dev</td>
<td>Median</td>
<td>Mean</td>
<td>Std.dev</td>
<td>Median</td>
<td>P</td>
<td></td>
</tr>
<tr>
<td>ANB</td>
<td>3.47</td>
<td>3.28</td>
<td>4.00</td>
<td>3.60</td>
<td>3.27</td>
<td>4.00</td>
<td>0.932</td>
<td></td>
</tr>
<tr>
<td>SNA</td>
<td>80.73</td>
<td>2.97</td>
<td>80.00</td>
<td>81.83</td>
<td>2.82</td>
<td>81.50</td>
<td>0.279</td>
<td></td>
</tr>
<tr>
<td>SNB</td>
<td>77.27</td>
<td>3.83</td>
<td>76.00</td>
<td>78.23</td>
<td>4.87</td>
<td>78.00</td>
<td>0.479</td>
<td></td>
</tr>
<tr>
<td>Craniomandibular angle</td>
<td>35.53</td>
<td>5.80</td>
<td>36.00</td>
<td>34.13</td>
<td>6.54</td>
<td>34.00</td>
<td>0.724</td>
<td></td>
</tr>
<tr>
<td>Intermaxillary angle</td>
<td>26.76</td>
<td>5.33</td>
<td>28.00</td>
<td>25.83</td>
<td>5.23</td>
<td>25.00</td>
<td>0.561</td>
<td></td>
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<tr>
<td>Sor-SNA</td>
<td>6.02</td>
<td>0.38</td>
<td>6.00</td>
<td>5.99</td>
<td>0.38</td>
<td>6.00</td>
<td>0.983</td>
<td></td>
</tr>
<tr>
<td>SNA-Me</td>
<td>6.29</td>
<td>0.47</td>
<td>6.30</td>
<td>6.51</td>
<td>0.51</td>
<td>6.30</td>
<td>0.297</td>
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</tr>
<tr>
<td>Ba-S-N</td>
<td>130.53</td>
<td>4.69</td>
<td>128.00</td>
<td>132.53</td>
<td>6.35</td>
<td>132.00</td>
<td>0.492</td>
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<tr>
<td>S-N</td>
<td>6.78</td>
<td>0.28</td>
<td>6.80</td>
<td>6.95</td>
<td>0.38</td>
<td>7.00</td>
<td>0.174</td>
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<tr>
<td>Ba-S</td>
<td>4.30</td>
<td>0.32</td>
<td>4.30</td>
<td>4.30</td>
<td>0.23</td>
<td>4.40</td>
<td>0.967</td>
<td></td>
</tr>
<tr>
<td>Ba-N</td>
<td>10.10</td>
<td>0.12</td>
<td>10.10</td>
<td>10.35</td>
<td>0.11</td>
<td>10.40</td>
<td>0.169</td>
<td></td>
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<tr>
<td>Mandibular branch</td>
<td>4.04</td>
<td>0.41</td>
<td>4.00</td>
<td>4.26</td>
<td>0.47</td>
<td>4.10</td>
<td>0.156</td>
<td></td>
</tr>
<tr>
<td>Pc-Go-Gn</td>
<td>127.70</td>
<td>4.92</td>
<td>128.00</td>
<td>125.50</td>
<td>5.79</td>
<td>126.00</td>
<td>0.211</td>
<td></td>
</tr>
</tbody>
</table>

### Table 3 - Test results of the chi-square relative to the divergence.

<table>
<thead>
<tr>
<th></th>
<th>CMD group</th>
<th>Control group</th>
<th>TOT</th>
<th></th>
<th>Odds ratio</th>
<th>95% IC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Divergence</td>
<td>N(%)</td>
<td>N(%)</td>
<td>N(%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mesodivergence</td>
<td>5 (33.3)</td>
<td>8 (53.3)</td>
<td>13 (43.3)</td>
<td>0.269</td>
<td>2.286</td>
<td>0.42-12.95</td>
</tr>
<tr>
<td>Hiperdivergent</td>
<td>10 (66.7)</td>
<td>7 (46.7)</td>
<td>17 (56.7)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Table 4 - Test results of the chi-square relative to the anterior vertical dimensions.

<table>
<thead>
<tr>
<th></th>
<th>CMD group</th>
<th>Control group</th>
<th>TOT</th>
<th></th>
<th>Odds ratio</th>
<th>95% IC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior vertical dimensions</td>
<td>N(%)</td>
<td>N(%)</td>
<td>N(%)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Normovertibite</td>
<td>1 (6.7)</td>
<td>2 (13.3)</td>
<td>3 (10)</td>
<td>0.543</td>
<td>2.154</td>
<td>0.098-135.985</td>
</tr>
<tr>
<td>Openbite</td>
<td>7 (46.6)</td>
<td>7 (46.7)</td>
<td>14 (46.7)</td>
<td>0.599</td>
<td>2</td>
<td>1.081-134.579</td>
</tr>
<tr>
<td>Deepbite</td>
<td>7 (46.6)</td>
<td>6 (40)</td>
<td>13 (43.3)</td>
<td>0.522</td>
<td>2.33</td>
<td>0.093-156.989</td>
</tr>
</tbody>
</table>
Studies carried out on subjects of the same age range, randomly selected in the population. Magnusson (19) estimated the prevalence of CMD in a population of children and adolescents around 14.10% between 12 and 15 years of age, and 5.11% between 5 and 11 years of age.

In our sample the presence of malocclusions does not seem to be a predisposing factor for the onset of temporomandibular disorders, contrary to what was strongly held in the past, at least in the age range we considered. The reason of this uniformity of prevalence between CMD in patients with malocclusions and healthy subjects, could be explained by the young age of the selected subjects, thus confirming the theory according to which CMD symptoms emerge when the articular structure adaptation threshold is overcome (12). The temporomandibular joint has in fact a significant capacity for adaptation to masticatory micro-traumas (20); however, this capacity wears off in time since some predisposing factors like ligament laxity, incorrect posture, extreme psychic irritability or sensitivity, parafuncions and, of course, malocclusions or traumas concur to alter the existing balance and engender the pathology at issue. The adaptation mechanisms are more efficient in young subjects because of a higher elasticity of articular structures able to better stand mechanical stress. These mechanisms reduce their efficiency in time, because of modifications affecting structural components caused by ageing. It is in fact to be seen a decrease in blood perfusion and, consequently, in the nutrient supply, together with an increase in rigidity and, in predisposed subjects, progressive tissue modifications due to chronic inflammation mediated by enzymes, bone morphogenetic proteins, free radicals and proinflammatory and nociceptive neuropeptides.

In our study, we evaluated the skeletal and dental characteristics of the examined subjects. Several recent studies (21-25), investigated the relation between dento-skeletal characteristics and temporomandibular disorders, based on the hypothesis that some morphostructural variables can predispose or increase the risk to develop articular disorders.

For what concerns the cephalometric data, we could not find any significant difference between the two samples. These results are consistent with the high reliability of skeletal data, which seem not to be influenced by age or any other specific demographic factor.
with those obtained by Gidarakou et al. (22). These Authors compared two groups of patients with normal articular morphology and correct position of the disc, having pain as the only discriminating factor. The fact that in this analysis there were not any discrepancies between the dento-skeletal characteristics of symptomatic and asymptomatic subjects, suggests that the algic symptomatology of CMD can onset independently from ATM pathological alterations, which was confirmed by our study. This confers a higher importance to other variables composing the multifactorial etiopathogenesis of these disorders, like those related to the subjective perception of pain (genetic, hormonal, central mediation mechanisms).

Different results were obtained by the same Authors (21, 23, 25) when the examined samples were composed of subjects affected by unilateral or bilateral disc dislocation, with or without reduction, compared to healthy subjects. In these studies significant differences were found in the dimensions of the cranial base; linear dimensions were reduced in relation to controls, while the cranial base angle (Ba-S-N) was more obtuse. The experimental group also showed complete maxillary retrognathism (evidenced by SNA and SNB values lower the norm). The analysis of these results led us to ask if retromaxillism and retromandibulism, perhaps due to alterations in the cranial base, could predispose to the development of CMD or if, vice versa, these morphological modifications could be caused by a disc dislocation affecting the skeletal growth direction and type. The last hypothesis seems to be the most accredited, although the mechanisms producing the aforementioned modifications have not been clarified yet.

A difference was found in relation to the craniomandibular angle (between S-N and Go-Gn), being higher in subjects with disc dislocation, together with a more obtuse gonial angle and a shorter mandibular branch, with consequent increase in the vertical dimension of the face third inferior. These data are consistent with the studies reported in literature in the last years: the most recent tendency describes in fact the hyperdivergent facial typology as the most frequently associated with articular pathologies, perhaps because the retruded and post-rotated position of mandible could contribute to the determination of disc dislocation (26). Our study is consistent with this assumption since, although any significant difference between the experimental group and the control one were to be seen, it is still interesting to point out that any patient affected by CMD had hypodivergence, while 10 subject out of 15 (66.67%) had an increased intermaxillary angle and the typical facial features of hyperdivergent subjects.

These observations are in contrast with the past when the decrease in inferior vertical dimension and deepbite were believed to be the main factors responsible for craniomandibular disorders (27). Once again, it is evident that opinions vary much in time and in the type of study and depending on the samples; our knowledge in these matters is not sufficient yet, and it should born in mind that there are several unclear aspects about the relation between occlusion, skeletal characteristics and CMD.

For what concerns the dental patterns, in our study the overjet resulted increased in nearly all patients, in fact the 86.67% of the experimental group (13 subjects out of 15), showed such sagittal discrepancy between the superior frontal and inferior group. It is possible to correlate this data with the loss of the anterior guide, a factor which was reported by several authors in association with CMD as a cause of occlusal instability with consequent functional overload on articular structures. However, it is true that that this characteristics were also common in controls, evidence explained by the fact that our investigation was done on patients with orthodontic problems who could therefore be more likely to show alteration of dental relations.

Although the role of malocclusions in the aetipathogenesis of craniomandibular disorders has been considerably scaled down, the evidence persists, confirmed by several studies, that a correlation (even slight) between these two conditions exists. In recent literature, this fact is explained with the development of an articular or-
thopaedic instability secondary to the occlusal instability due to peculiar alterations in dental patterns. This is believed to be a risk factor for the anterior disc dislocation, as claimed by Manfredini (28, 29). It has been recently assumed that in predisposed subjects a “occlusal hypervigilance” mechanism is present, making these subjects more sensitive to occlusal modifications and inevitably leading them to discomfort, pain and dysfunction. The occlusal hypervigilance theory can be envisaged in the wider hypothesis of the General Hypervigilance, according to which it is the perceptive habit that determines the subjective amplification of a variety of unpleasant sensations, not just the painful ones, thus suggesting the involvement of central sensitisation mechanisms responsible for hyperalgesia (30).

The occlusal factor which is currently mostly associated with the onset of temporomandibular disorders is the unilateral posterior crossbite, consistently with the theories of Michelotti and Tecco (31, 32). This type of crossbite seems to alter the occlusal balance necessary to keep a physiological relation between the different articular structures, this justifies the orthodontic correction of this malocclusion to prevent the onset of CMD (33). In our sample, however, crossbite was equally distributed in both groups.

The only factor which resulted poorly correlated, even though it did not overcome the significance threshold, was the pathologic overbite, found with higher frequency in the study sample (P=0.068), with prevalence of the deep typology (P=0.099), compared to the control group. This could suggest a mild association between this characteristic and CMD, highlighting the need for studies on larger samples, as to increase the test sensitivity; this could show a clearer correlation between dental deep-bite and articular disorders. Interestingly, in our samples we often found the morphological characteristics of hyperdivergence in parallel with dental deep-bite. The simultaneous presence of these two requisites in the same subject is not common. We could therefore assume that the very association between these two elements could be the risk factor for temporomandibular disorders. The concurrence of these two factors could be determined by alterations in mandibular morphology, potentially predisposing the development of an orthopaedic instability of the articular structures. This fact should be evaluated more in depth with further testing, nevertheless it suggests the need for further researches on ampler samples in order to identify other possible associations.

The limit of our study was the limited sample size which prevented us from obtaining more significant results. This problem is due to the low incidence of craniomandibular disorders in young children and adolescent population and, consequently, to the limited number of affected subjects we could study.

Our future aim will be to widen the sample to examine and to study the simultaneous presence of different morphological characteristics, whose association may be more significantly correlated to CMD.

References

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