Clinical manifestations of somatic pathology in patients with temporomandibular joint disorders

Volovar O., Malanchuk V., Lytovchenko N.*, Kostiuk T.

Bogomolets National Medical University, Ukraine, Kiev

DOI: 10.5455/jrmds.2017525

ABSTRACT

Clinical examination results of 248 patients with temporomandibular joint disorders (mean age – 26.0±7.4 years) are shown. Clinical manifestations of cardiovascular diseases, tonsils condition (in particular, nasopharynx microbial flora), indicators of the acute phase reactions are studied. In a number of patients moderate, diffuse, myocardial metabolic alterations, rhythm disturbances (sinus arrhythmia, tachycardia, bradycardia, extrasystoles, His bundle branch block), mitral valve disease were revealed, which are the signs of connective tissue dysplasia and also severe tonsils hypertrophy, chronic tonsillitis, nasopharyngeal streptococcal and staphylococcal infection.

Keywords: temporomandibular joint, circulatory system, connective tissue, dysplasia, diagnosis, treatment.

INTRODUCTION

Temporomandibular joint disorders (TMJD) play a special part in dental disorders, typical for nearly 65% of population. In recent years such disorder becomes an increasingly frequent tendency in young patients [1]. Necessity for diagnostics of the overall condition of internal organs and, in particular, condition of the circulatory system (CS) and determination of genetically determined condition of connective tissue (CT) in patients with TMJD, is due to high prevalence of degenerative/dystrophic and destructive inflammatory joint diseases, and lack of generally accepted approach, when it comes to etiology and pathogenesis [2, 3].

CT disease genetic heterogeneity defines a wide range of clinical options – starting with widely known genetic syndromes (Marfan, Ehlers-Danlos syndromes), ending with numerous undifferentiated (nonsyndromic) forms with multifactorial developmental mechanisms. CT dysplasia is manifested by external phenotypic traits and clinically significant one or more organs or systems dysfunction. External traits comprise various dysembyogenic stigma, among which the most famous are: “Gothic” palate, “tower-shaped” skull, hypermobility syndrome, multiple pigmented spots, flatfoot, sandal gap. Such a micro-traits amount to several hundred, in sum [9].

Systemic lesion is often related to the wide spread concretely of CT, which composes skeleton and whole organs stroma, in the body. The nature of lesions is determined mostly by parenchymal-stromal ratio. Tissue and organs morphological alterations are nonspecific and manifest in a similar way, varying by the rate of severity in case of various dysplasia. They already figure while antenatal period and worsen during whole human life time. Genetically prognosed defects of various CT components determine the reduction in its stability, fragility, strength, leading to formation of clinical manifestations in those organs and tissues, where development, integrity and functional role of CT are of maximal significance. Undifferentiated CT dysplasia is certainly not a single nosology, but genetically heterogeneous group, being a basis for development of various chronic diseases [3, 4].

Circulatory system alterations are often observed in case of CT dysplasia. In 1990 heart
CT dysplasia was detached as a separate syndrome – disease which comprises isolated cardiac skeleton lesions as valves prolapse, interatrial septum aneurysm, false chordae, pulmonal artery aneurysm, pulpillary muscles dystonia, aortic root dilatation, arterial and venous angiodyplasia (visceral arteries and aorta tears and ruptures), valvular regurgitation of the lower extremities veins, in the absence of other phenotypic traits of CT syndrome [3, 4, 5, 6].

Heart CT dysplasia problem is currently central due its severe course and high prevalence in population. Thus, mitral valve prolapse is reported to occur in 4-15% of population, in 77% of which it’s combined with other valves prolapse. Additionally, heart CT dysplasia syndrome can cause such severe complications as infective endocarditis, thromboembolism, arrhythmia, potentially leading to sudden cardiac death [8]. The most extensively studied and common manifestation of undifferentiated CT dysplasia is mitral valve prolapse, which is considered to be the most prevailing valve abnormality in young people, as well as in females of reproductive age. Reliable correlation was revealed between the presence of atrial fibrillation and the severity of clinical manifestations of CT dysplasia. Arrhythmic syndrome belongs to the group of disorders that lead to death in patients with CT dysplasia. Some authors consider the early repolarization syndrome as one of the cardiac signs of CT dysplasia [7].

The most unfavorable variants of heart diseases accompanying by CT dysplasia are: valves and chordae myxomatous transformation, arrhythmia, and heart chambers dilatation. They are accompanied by severe disorders of cardiac hemodynamics, myocardial contractility, decrease in pump function. Typically, hemodynamics alterations tend to form simultaneously with CT metabolic alterations [8].

Cardiac histo-embryonic features are a basis for development of the disease caused by genetically determined imperfection of connective tissue substrate (heart development takes its onset on the 3rd week of gestation from several embryonic rudiments: mesenchyme – endocardium and vessels, visceral mesoderm - myocardium and epicardium, neuroectoderm – neural ganglia and fibers) [9, 10]. Type I collagen forms cardiac muscle connective tissue basis, type III collagen forms predominantly cardiac valves, and type II, to a lesser extent. Type I collagen (adventitia, media thick fibers) and type III (intima thin fibers and those closely attached to elastic fibers in media) compose aorta in equal proportions. Type I collagen, predominantly, composes TMJ ligaments, type II collagen (50% of dry weight) composes articular cartilage, type II (up to 40%) and IX (8-10%) collagen – articular disc (formed by fibrocartilage); type I and V – ligaments and bones (Sulimov A. F. et al., 2004). In particular, different types of collagen in CS and TMJ tissues determine the diversity and high frequency of disease manifestations for these organs in patients with CT dysplasia [10, 11, 12]. Lower limb and pelvic varices are common clinical manifestations of CT dysplasia. Valvular heart disease is statistically diagnosed in 50% of female subjects. Valvular deficiency can also be traced in gastrointestinal tract appearing as cardiopyloric valves deficiency which leads to gastroesophageal reflux disease and duodenogastric reflux [13]. Pathogens can act as one of TMJD etiologic factors, including streptococcus, which indirectly influences by means of cross-sensitization to CS and joint tissue.

**Study objective** – to explore CS and tonsils pathology manifestations via levels of C-reactive protein (CRP), antistreptolysin O (ASO), rheumatoid factor (RF), fibrinogen, leukocytes (WBC), erythrocyte sedimentation rate (ESR), lymphocytes (LYM), monocytes (MON), and to identify nasopharyngeal microbial flora in patients with TMJD aiming generation of complex approach in diagnostics and treatment of these patients.

**Study subject and methods**
The study seizes 248 patients (52 males, 196 females; mean age – 26.0 ± 7.4 years), receiving counseling and treatment at the Dental Center of A. A. Bogomolets National Medical University during 2005-2011. Controls/Control group included 26 subjects (13 males and 13 females; mean age – 25.7 ± 6.8 years) without somatic diseases and signs of TMJD, but with physiologic occlusion. The core group included 222 patients with TMJD (39 males, 183 females; mean age – 26.3 ± 8.0 years), varying by the degree of structural/conformational changes in TMJ, detected while X-ray, CT scan or MRI of the joint. Patients examination was carried out in compliance with standard examination guidelines/methodology/techniques for subjects with TMJD, iris (iridobiomicroscopy) was examined additionally. All patients were
referred to cardiologist and ECG. Clinical records were provided by subjects for the following anamnesis morbi and vitae reviewing. CRP, ASO, RF, fibrinogen levels were measured in patients. In case of CRP and RF levels deviation, seromucoid levels were additionally investigated, which can indicate on rheumatoid arthritis and rheumatism, when evaluated in combination with above-mentioned values [14].

Tonsillar or nasal, in case of tonsillectomy, swabs were taken in patients for the purpose of detection of the pathogenic microbial flora, and identification of body responsiveness to antimicrobial therapy. Tonsillar hypertrophy, their doughtiness, hyperemia and engorgement were taken into consideration and dynamics of pathological changes in tonsils (ENT specialist consultation) was estimated.

According to the illustration of projection areas of the human body by B. Jensen, heart is projecting in the sector 8:50 – 9:50 of the right iris, but signs related to the left iris are more typical and fully reflect heart condition. Therefore, studying changes in the heart we considered only left iris changes.

Tonsils projection area is located on the right iris in 1:50 – 2:20 region and on the left – 9:40 – 10:10. According to most illustrations of projection areas of the human body, lower limbs are projecting on both irises 5:50 – 6:10. Detection of fibrous, thinned vessels of identical diameter without loops and glomera adjacent to projection areas of lower limb allows to predict the tendency for veins diseases [16].

Digital data, obtained due to research, were processed on PC using such applications: Microsoft Excel 2007, Statistica 7.0 and SPSS 17.0 (USA) standard version. Arithmetic mean (AM), standard deviation (SD), standard error (SE), number of variants (n), trait proportion in % (P), indicating the standard error of proportion (Pm), significance point (p) were determined. During analysis of the difference of the nominal or ordinal scale related values, tables of joint traits distribution were created and χ2 Pearson test was used [15, 16].

### Table 1. ECG results in patients of the core group n, (P±Pm)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Females</th>
<th>Males</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Moderate metabolic myocardial alterations</td>
<td>7 (3.2±1.2)</td>
<td>3 (1.4±0.8)</td>
<td>10 (4.5±1.4)</td>
</tr>
<tr>
<td>Moderate alterations of myocardium</td>
<td>52 (23.4±2.8)</td>
<td>5 (2.3±1.0)</td>
<td>57 (25.7±2.9)</td>
</tr>
<tr>
<td>Moderate diffuse myocardial alterations</td>
<td>22 (9.9±2.0)</td>
<td>6 (2.7±1.1)</td>
<td>28 (12.6±2.2)</td>
</tr>
<tr>
<td>Diffuse myocardial alterations</td>
<td>8 (3.6±1.3)</td>
<td>1 (0.5±0.5)</td>
<td>9 (4.1±1.3)</td>
</tr>
<tr>
<td>Sinus tachycardia</td>
<td>9 (4.1±1.3)</td>
<td>2 (0.9±0.6)</td>
<td>11 (5.0±1.5)</td>
</tr>
<tr>
<td>Sinus arhythmia</td>
<td>15 (6.8±1.7)</td>
<td>3 (1.4±0.8)</td>
<td>18 (8.1±1.8)</td>
</tr>
<tr>
<td>Sinus bradycardia</td>
<td>5 (2.3±1.0)</td>
<td>0 (0±0)</td>
<td>5 (2.3±1.0)</td>
</tr>
<tr>
<td>Extrasystoles</td>
<td>4 (1.8±0.9)</td>
<td>0 (0±0)</td>
<td>4 (1.8±0.9)</td>
</tr>
<tr>
<td>His bundle block</td>
<td>4 (1.8±0.9)</td>
<td>0 (0±0)</td>
<td>4 (1.8±0.9)</td>
</tr>
</tbody>
</table>

### Table 2. CS diseases in patients of the core group n, (P±Pm)

<table>
<thead>
<tr>
<th>Disease</th>
<th>Females</th>
<th>Males</th>
<th>Both</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital defect of the mitral valve</td>
<td>3 (1.4±0.8)</td>
<td>1 (0.5±0.5)</td>
<td>4 (1.8±0.9)</td>
</tr>
<tr>
<td>Mitral valve prolapse</td>
<td>13 (5.9±1.6)</td>
<td>3 (1.4±0.8)</td>
<td>16 (7.2±1.7)</td>
</tr>
<tr>
<td>Tricuspid valve prolapse</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Myocardidystrophy</td>
<td>3 (1.4±0.8)</td>
<td>0 (0±0)</td>
<td>3 (1.4±0.8)</td>
</tr>
<tr>
<td>Metabolic cardiomyopathy</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Essential hypertension, stage II</td>
<td>2 (0.9±0.6)</td>
<td>0 (0±0)</td>
<td>2 (0.9±0.6)</td>
</tr>
<tr>
<td>Acute myocarditis</td>
<td>2 (0.9±0.6)</td>
<td>0 (0±0)</td>
<td>2 (0.9±0.6)</td>
</tr>
<tr>
<td>Paroxysmal tachycardia</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Rheumatism in childhood</td>
<td>3 (1.4±0.8)</td>
<td>1 (0.5±0.5)</td>
<td>4 (1.8±0.9)</td>
</tr>
<tr>
<td>Rheumatic heart disease</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Lower limb varices</td>
<td>35 (15.8±2.4)</td>
<td>9 (4.1±1.3)</td>
<td>44 (19.8±2.7)</td>
</tr>
<tr>
<td>Lower limb thrombophlebitis</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>Pelvic varices</td>
<td>1 (0.5±0.5)</td>
<td>0 (0±0)</td>
<td>1 (0.5±0.5)</td>
</tr>
<tr>
<td>chronic venous insufficiency, stage I-II</td>
<td>2 (0.9±0.6)</td>
<td>0 (0±0)</td>
<td>2 (0.9±0.6)</td>
</tr>
<tr>
<td>Phlebectomy</td>
<td>2 (0.9±0.6)</td>
<td>0 (0±0)</td>
<td>2 (0.9±0.6)</td>
</tr>
</tbody>
</table>
RESULTS AND DISCUSSION

Metabolic, moderate, diffuse myocardial alterations, abnormal heart rhythm and intracardiac conduction and His bundle branch block were revealed on ECG data analysis in patients of the core group (table 1).

Mitral and tricuspid valves defect or prolapse is diagnosed in 21 (9.5±2.0%) patients, myocardiodystrophy – in 3 (1.4±0.8%) patients, essential hypertension – in 2 (0.9±1.9%) subjects. Metabolic cardiomyopathy, coronary heart disease, paroxysmal tachycardia and rheumatic heart disease were reported to occur in rare cases in patients from the core group. Rheumatic heart disease was diagnosed in 4 (1.8±0.9) patients and acute myocarditis – in 2 (0.9±0.6%) patients in childhood. Thus, CS abnormalities were observed in 36 (16.2±2.5%) patients of the core group (table 2).

CS related changes were not reported in patients from the control group. In patients of the core group from the side of ENT-organs, most frequently occurred such diseases: tonsils hypertrophy, III degree – in 59 (26.6±3.0%) subjects, chronic tonsillitis – in 112 (50.5±3.4%) patients, adenoid vegetations – in 3 (1.4±0.8%) patients. In childhood, chronic tonsillitis took place in 59 (26.6±3.0%) patients, tonsillectomy – in 19 (8.6±1.9%), and adenectomy – in 12 (5.4±1.5%) patients (table 3).

Culturing of nasopharyngeal mucus demonstrated sufficient diversity of microbial flora in upper respiratory tract. I and II growth degrees prove/witness that the patient is a carrier of a certain flora, III and IV – etiological significance of the microorganism. High titers of tonsillar or nasopharyngeal microbial flora were detected in 72 (52.2±4.3%) patients.

Regarding the acute phase reactions, following results are obtained: elevated CRP level >6 mg/L was detected in 8 (5.8±2.0%) patients among 138, quantitative CRP value was WNL in 130 (94.2±2.0%) patients; ASO level elevation >200 IU/ml – in 14 (10.1±2.6%) patients, this indicator was 200 IU/ml in 7 (5.1±1.9%) subjects.

Of note/worth sharing is that in patients with CRP, ASO elevation and high titers of pathogenic microbial flora, seromucoid blood levels remained WNL (±5 CU). Positive RF value of 32 IU / ml (normal <8 IU / ml) was detected in 2 (1.4±1.0%) females, who were referred to rheumatologist consultation aiming confirmation of existing rheumatic process. Thus, in majority of patients with TMJD, rheumatic or rheumatoid inflammation evidences weren’t detected.

Fibrinogen level >2,4 g/L was measured in 8 (5.8±2.0%) patients, elevated ESR – in 6 (4.3±1.7%), leukocytosis – in 3 (2.2±1.2%), lymphocytosis – in 24 (17.4±3.2%) patients, lymphopenia – in 4 (2.9±1.4%) patients, monocytosis – in 14 (10.1±2.6%), monocytopenia – in 11 (8±2.3%) patients. Analysis of lymphocyte, monocyte counts in 52 (37.7±4.1%) subjects of the core group revealed immunodisfunction, indicating the microbial flora toxic effect on the human body.

Complete examination allowed to exclude active inflammation in TMJ.

Tonsils hypertrophy wasn’t diagnosed in patients of the control group, pathogenic microbial flora wasn’t identified.

Lacunes, defibrated and crimped stroma fibers, autonomous ring local bulging phenomena, toxic radiance, adaptational rings, toxic pigmented spots, wastes and sodium ring were detected after iridobiomicroscopy data analysis of heart projection area (Fig. 1, 2) Current changes were observed in 122 (55.0±3.3%) patients.
This can be interpreted as an evidence for genetic heart weakness, which manifested itself as a defect or mitral valve prolapse, changes in heart rate and regularity (sinus arrhythmia, bradycardia, tachycardia, diffuse myocardial metabolic alterations and etc.). The most common signs of CT weakness in tonsillar area were more or less significant structural traits: crimped, pale fibers, iris stroma defibration, lacunes. Toxico-dystrophic changes in the form of lymphatic rosary indicated on reduction in drainage function of lymphatic system and potential tonsils hyperplasia.

Stroma weakness, defibration or lacunes, crimped white trabecules, autonomous ring bulging symptom, lymphatic rosary, toxic radiance were diagnosed in lower limb projection area in 189 (85%) patients. Blurred stroma pattern on iris periphery and lymphatic rosary appearance indicate on reduction in lymphatic drainage function, tendency to engorgement and lower limb edema, veins valvular insufficiency. Patients, experiencing toxic radiance in this projection area, complained on occasional cramps in lower limb muscles. Dark, crimped streaks, stroma defibration closer to iris periphery, blurred indistinct iris fibers pattern, slaggy lymphatic rosary were detected in 44 (19.8±2.7%) patients with varices of lower limb in corresponding iris area. Structural and toxico-dystrophic signs in heart projection areas were observed in 5 (19.2±7.7%) patients of the control group, in tonsils areas – in 8 (30.8±9.1%), in lower limb areas – in 7 (26.9±8.7%) patients, which was reliably validated by χ² Pearson criterion. TMJD treatment, taking into account minimal drug effect on CS organs, for not provoking relapse, is recommended for patients of the core group with CS diseases or chronic tonsillitis. An important aspect is taking into consideration cardiologist and ENT-specialist recommendations. Tonsils were treated with iodine solution, and daily UV-treatment of the oropharynx was carried out in patients after pathogenic microbial flora (streptococci, staphylococci) detection. Patients were consulted by ENT-specialist and then they received recommended treatment, if required, in case of tonsils moderate hypertrophy and engorgement.
It is known that chronic tonsillitis is caused by beta-hemolytic and other streptococci and staphylococci, inhabiting the tonsillar lacunes and being highly pathogenic, with toxins capable of CT lesions. Cardiac valves prolapse, arrhythmias were common manifestations in patients with chronic tonsillitis. Chronic tonsillitis is a risk factor for heart diseases development in children with CT dysplasia (Nagornaya N.V. et al., 2005). Manifestations of CS abnormalities (36 (16.2±2.5%) patients), inactive chronic tonsillitis in 112 (50.5±3.4%) patients with TMJD, and signs of iris changes in corresponding areas, indicative of systemic CT defect, were identified in our study. However, in patients with TMJD, rheumatic or rheumatoid processes in the body were not detected, but X-ray revealed TMJ arthrosis or dysplasia. Moreover, complete examination, carried out, made it possible to exclude active inflammation of CT tissues and heart. Considering all above mentioned, chronic infection foci sanitation, treatment of potential associated oral, ENT-organs diseases, local treatment of tonsils, physiotherapy, targeting the immune system are meaningful to be added to complete conservative treatment of patients with TMJD. Elimination of tonsillar or nasopharyngeal infection is an important condition of lymphoid apparatus sanitation, health improvement and body strengthening in general.

Thus, CS, in general, and valvular apparatus of the heart, lower limb veins and pelvis in particular, being a system of organs, originating from mesoderm, rich in collagen, are involved in pathological process in CT dysplasia, which was revealed in patients with TMJD. Patients clinical examination results, mentioned above, are indicative of CT dysplastic processes key role in development of pathological changes of TMJ.

CONCLUSION

1. Cardiac disorders on ECG: moderate myocardial alterations in 25.7% of patients, moderate diffuse alterations in 12.6%, as well as sinus tachycardia or bradycardia, extrasystoles, His bundle branch block are diagnosed in majority of patients with TMJD.
2. Core group patients demonstrated prolapse, cardiac valves defects (9.5%), myocardiodystrophy (1.4%), metabolic cardiomyopathy, coronary heart disease, essential hypertension, acute myocarditis and so on.
3. Chronic tonsillitis (50.5%), chronic tonsillitis in childhood (26.6%), tonsillectomy (8.6%), adenectomy (5.4%), adenoid vegetations are typical for patients with TMJD.
4. Streptococcal infection and chronic inflammation of nasopharynx in patients with TMJD can lead to changes of CS, provoke development of cardiac or articular pathological conditions (rheumatism) and suppress immune system.
5. Structural local, chromatic, reflex iris stroma changes in heart projection area (55.0%), tonsils projection area (83.3%), lower limb projection area (85.1%) in core group patients indicate CT congenital weakness of CS, tonsils and predisposition to development of associated diseases.
6. Based on study results and literature review it is fair to assume that dysplastic systemic CT alterations, manifesting with pathological changes of CS, tonsils are the basis for degenerative/dystrophic and destructive inflammatory TMJ disorders.
7. If CS and tonsils diseases take place, patients should be referred on ECG; CRP, ASO, RF should be measured; complete blood count should be conducted. Patients should be examined by cardiologist and ENT-specialist if CS and tonsils pathological changes are detected. Standard medical examination of these patients by the dental surgeon is required for screening of TMJ and body, in general.

Acknowledgements

This paper is derived from the residency dissertation of Oksana Volovar.

Authors contribution

Oksana Volovar developed the study concept and design, interpretations of data, and drafting of the manuscript. Vladyslav Malanchuk, Nataliia Lytvchenco, Tetiana Kostiuk developed the protocol, analysis of data and drafting of the manuscript.

Conflict of interest

The authors have no conflict of interest to disclose.

REFERENCES


