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PECULIARITIES OF THE INFLUENCE OF MULTIMORBID PATHOLOGY AND THEIR TREATMENT ON THE DENTAL STATUS (LITERARY REVIEW)

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Abstract:

Recently, there has been a growing concern among dentists about comorbidities in patients seeking dental care, as this figure, according to some authors, reaches 60-80%. Studies by I.S. Valias et al. (2016) showed that at the dental appointment associated pathology, such as, arterial hypertension occurs in (52%)people, diabetes (31.3%) and cardiovascular disease (27.5%) people.

Keywords: Multimorbid Pathology, Dental Status

In Romania, 92% of cases of comorbidities occur at the outpatient dental appointment. At the same time, there are 1.35 types of systemic pathology per patient, indicating their polymorbidity in populations. Iranian researchers K.M. Siddiqi et al. (2016) among 12960 patients at dental appointments identified comorbidities in 47% of people over the age of 40, and they were more common in women (51%). According to the same authors, a single systemic pathology was identified in 83.2% of patients, while como- and polymorbid status occurred in 16.8% of those examined. Arterial hypertension (28%), diabetes mellitus (20%), and cardiomyopathy (15%) were more common in the patients examined. A study by P.G. Serov (2010) revealed that in Russia, comorbidities in combination with dental pathology occurred in 82.9% of people, of whom 25% of patients were found to be polymorbid. Herewith, the frequency of ischemic heart disease was 64.5%, in 23.7% - diabetes mellitus, in 21.1% - atherosclerotic changes in blood vessels, in 21.1% - respiratory diseases, in 17.1% - allergies, in 15.8% - obesity, in 14.5% - gastrointestinal diseases. As we know, atherosclerosis is a systemic inflammatory disease that develops in the large arteries and is responsible for coronary heart disease, stroke, and peripheral arterial disease. Evidence has been published, based on epidemiological studies, on the relationship between periodontitis and vascular atherosclerosis (KozarovE.V., etal., 2005; LockhartP.B., etal., 2012; Papapanou P.N., 2013; van der Bijl P., 2014).

M.J. Lakhani et al. (2013) and E.N. Anisimova et al. (2012) also found a prevalence of arterial hypertension (50% and 92% respectively) in the incidence of comorbidities in dental practice. Bulkina et al. (2012) showed that chronic generalized periodontitis occurs in 68% of patients with cardiovascular diseases, of which hypertension accounts for 26%, coronary heart disease - 10.5%, neurocirculatory dystonia - 68%. Hedstrom L., etal., (2015) in their studies showed that patients with arterial hypertension often have varicose sublingual veins in the oral cavity, which is a reliable indicator of the existing disease. According to Borovsky EV, (1981); LivadaR., ShiloahJ., (2014) oral mucosa in patients with arterial hypertension undergoes atrophy and hyperplasia, which is probably associated with impaired microcirculatory bed and the intake of calcium antagonists. In studies (PatilP.M., etal., 2015) it is shown that oral sarcoidosis can be the first signs of the development of bronchopulmonary pathology. At the same time, xerostomia is considered as the most significant clinical symptom of sarcoidosis (KolokotronisA.E., etal., 2009; Borovskaya A.B., 2014). Studies (OhtsukaS., etal., 2001) have shown that in the active phase of the disease salivary secretion rate decreases significantly, along with a decrease in salivary a-amylase activity and kallikrein content.

At the same time, according to (PatilP.M., etal., 2015). D.G. James and O.P. Sharma (2000) against the background of salivary gland dysfunction, patients complain of dysgeusia, burning sensation in the mouth and increased size of parotid salivary glands. The authors of these studies believe that bilateral parotid salivary gland enlargement may be the first sign of sarcoidosis in a patient without other symptoms or clinical abnormalities typical of the disease.

Numerous studies have established the relationship

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between periodontitis and COPD (PrasannaS.J., 2011; ShenT.C., etal., 2015; BhavsarN.V., etal., 2016; ChungJ.H., etal., 2016).Thus, chronic obstructive pulmonary disease (COPD) is recognized as potentially related to general inflammatory processes among a series of associated pathologies. Research by Gikoshvili, H.I., (2009) revealed that in patients with COPD, respiratory failure and pulmonary heart disease are major factors in the progression of periodontal disease. There was evidence of a clinical association between the need to hospitalize a patient with COPD and the degree of periodontitis aggravation (ShenT.C., etal., 2015; ZengX.T., etal., 2012; BansalM., etal., 2013; WhiteP., etal., 2014;LingM.R., etal., 2015). As the authors point out, the mechanism of the relationship between COPD and periodontitis is the aspiration of bacterial contents of the oral cavity into the respiratory tract or back. At the same time, patients with COPD have intensive formation of hard supra- and sub-gingival deposits, pronounced gingival recession without signs of periodontal inflammation, atypical vascular response to cold testing and periodontal bone resorption (E.D. Shikhnabieva, 2007; E.V. Kosova, 2009; Kh.I. Gikoshvili, 2009; ChungJ.H., etal., 2016). Studies Dubov L.V., 2010; Shkurova T.A. et al., 2013, 2014) have shown the presence of inflammatory processes in the periodontium in patients with bronchial asthma (BA), caused by suppression of local immunity in the tissues of the oral cavity.

Recently it has been proved that changes in the oral cavity can reveal such nosological forms of gastrointestinal diseases as celiac disease, gastroesophageal reflux or intestinal inflammation (Tsimbalistov A.V. et al., 2013). Macroscopic and histological characteristics of manifestations in the oral cavity are similar to those found in the tissues of the gastrointestinal tract (Kvetnoy I.M. et al., 2009; Tytyuk S.Yu. et al., 2016; LankaraniK.B., etal., 2013).

Studies (RashidM., etal., 2011) have shown that dental enamel defects, dental caries and aphthous stomatitis are more common in patients with celiac disease and regress on a gluten-free diet. A consequence of gastroesophageal reflux disease is tooth enamel erosion, which is combined with dental caries in 25-83% of patients, more often in children (Dahshan A., etal., 2010; SmithC.H., etal., 2015).

In works Tytyuk S.Yu. et al., 2019; Tytyuk S.Yu., Iordanishvili A.K., 2019; MulicA., etal., 2013; BartlettD.W., etal., 2013, it is shown that in inflammatory bowel diseases there is an increase in caries and non-carious dental lesions in 50% of people of young and middle age from 18 to 35 years.

According to Glavnov P.V. et al., 2015; CrippaR., etal., 2016; (GheorgheC., etal., 2004; Hovde 0., MoumB.A, 2012) Crohn's disease prevalence is 150 people per 100000 inhabitants of Europe and America. At the same time, pathological processes in the oral cavity in Crohn's disease are a consequence of reduced levels of micronutrients and macronutrients in the blood serum due to malabsorption disorders in the intestine, or local immune reactions to oral antigens (Raikov B.S., 2018; HusseyS., etal., 2011; BoirivantM., CossuA., 2012). These changes are accompanied by dryness in the oral cavity and halitosis, externally manifested as typical and pathognomonic changes in the form of "cobblestone" hyperplasia of oral mucosa, stomatitis, gingivitis, periodontitis, cheilitis, geographic glossitis (Mdinaradze G.N, 2006; Robakidze N.S. et al., 2017; Kolomiets S.V. et al., 2017; Mamaeva M.I., 2017; KolhoK.L., etal., 2011; BoirivantM., CossuA., 2012; KatsanosK.H., etal.,2015).

StudiesRobakidze N.S., Pikhur O.L., 2015; Tytyuk S.Yu., Iordanishvili A.K., 2019, show that hard dental tissues in Crohn's disease are affected by multiple carious and non-carious defects Functional disorders of the oral cavity in patients with Crohn's disease during eating and communication are accompanied by painful sensations and cause psychosocial stress .

Unlike Crohn's disease, nonspecific ulcerative colitis occurs in a more severe form (Akhrieva H.M. et al., 2017). In NSCLC oral changes occur in 5-20% of cases, and pyostoma with vegetations is considered to be specific for it. Recurrent aphthae, gingivitis, ulcers similar to gangrenous pyoderma of the skin, ulcers with fibrinous plaque formed in patients with poorly functioning stoma, hemorrhagic ulcers with active disease course, glossitis, dryness in the mouth can be found in the oral cavityMdinaradze G.N, 2006; Trukhan D.I., Victorova I.A., 2010; Livzan M.A. et al., 2016; Robakidze N.S., Baranovsky A.Yu., 2016; Spitsina M.S. et al., 2018).

Gastroesophageal reflux (GERD) is a physiological movement of gastric contents into the esophagus and oropharynx, resulting from relaxation of the lower esophageal sphincter. It can become pathological when spontaneous frequently recurrent belching, nausea, heartburn, cough occur, and therefore it is defined as GERD (Maev I.V. et al., 2000, 2013). According to VandenplasY., (2009), oral manifestations in GERD correlate with tooth enamel erosion and its prevalence among adults and children with GERD reaches from 47.5% to 83.3%.

Tolstova O.O. et al., 2013; DahshanA., etal., 2002, 2010. Lukina G.I. (2011) found that among all pathologies of the gastrointestinal tract in GERD disease, the lowest salivary pH values (pH=5.2) are determined in the oral cavity. GERD may also be the cause of pathologies in the soft tissues of the mouth and salivary gland dysfunction, considers Uspensky Y.P. et al., 2015; SilvaM.A., etal., 2001). O.DiFede et al. (2008).

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R.A. Aivazova et al. (2017) and N.V. Kostina et al. (2013) reported a significant association of GERD with a burning sensation in the mouth, xerostomia, subjective halitosis and erythema of the mucosa of the soft palate and uvula.

Among all diseases of the gastrointestinal tract, chronic gastritis accounts for 70 to 80% (SipponenP., MaaroosH.-I., 2015). (Olshevsky V.A., 2001; Arutyunov S.D. et al., 2004; Kao, C.-Y., etal., 2016; Afanasenkova T.E. et al., 2018). H. Miusbachi et al. (2000) first established the relationship between gastritis caused by H. pylori infection and colonization of pathogens in the oral cavity.

Lukina G.I. (2011) in her study found that H.pylori contamination of the tongue surface was detected only in chronic gastritis and gastric and duodenal ulcer disease. It was revealed that plaque and saliva in the oral cavity act as a secondary reservoir for H. Pylori (E.A. Bazikyan et al., 2008; SoutoR., ColomboA.P., 2008; Janushevich O.O. et al., 2013).

Pashkova G.S., 2010; and Janushevich O.O. et al., 2014) there was a clear parallel between the presence of H. pylori in the oral cavity and the development of periodontitis Against the background of hyperacidic state in the mouth revealed increased salivation, pallor and swelling of oral mucosa, stomatitis phenomena, bleeding gums, halitosis, gum tissue edema, enamel erosions, wedge-shaped lesions of teeth. According to (Pogurets Y.K. et al., 2017; Kaisina T.N. et al., 2017; Abakumova M.A., Konysheva A.K., 2017; Kulumbegova I.R., Khubulov S.A., 2019) with hypoacid gastritis, in contrast, salivary secretion rate decreases, which coincides with the formation of angular cheilitis and white-yellow plaque on the back of the tongue. Combined inflammation of the mucous membrane of the pyloric zone of the stomach and 12 fistulas in clinical practice is diagnosed as gastroduodenitis. In this case motor function of the stomach and peristalsis of the duodenum are disturbed that leads to delay of chyme in initial parts of intestine (I.V. Maev, A.A. Samsonov, 2005).

Lukina G.I. (2011) in her research has shown that with pathology of esophagogastroduodenal region the common clinical signs of oral mucosa are pastosity (in 58,6% of cases), dryness (in 43% of cases) and plaque on a tongue back (in 50,9% of cases). These patients complain of bad breath, bitterness, sourness and burning, and the incidence of gingivitis, angular cheilitis, geographic tongue, enamel hypoplasia, dental caries is higher compared with healthy people (A.V. Tsimbalistov, N.S. Robakidze, 2005; O.O. Janushevich et al., 2014; A.Yu. Shcherbakova et al., 2014).

It is known that UFDD significantly changes the acid-base balance in the oral cavity, which leads to the development of multiple caries, enamel erosion and inflammatory processes in periodontal tissues (Khaykin M.B. et al, 2006; Moiseeva M.V., Belova E.V., 2011; Yanushevich O.O. et al., 2013; Kosoyuga S.Y., Varvanina S.E., 2015; Nerobeev A.S. et al., 2018), changes in the saliva ratio of various organic acids (Perevoshchikova O.A., 2011).

Recently, numerous authors have monitored the therapeutic effect of systemic drugs on the indicators of the oral cavity. At the same time, studies of saliva, as a rule, are conducted using mass spectrometry with an emphasis on liquid chromatography, because of the small sample volume and low concentrations of the substances to be determined. Data show that the pharmacokinetics of drugs in saliva are more complex than in plasma. The detection of a drug in saliva depends on a number of factors such as dose, frequency of administration, specificity, and sensitivity of analyte detection(DrummerO.H., 2006).

BoskerW.M.,HuestisM.A., 2009; AravindhaBabuN., etal., 2014, after oral or intramuscular administration of sulfapyridine drug, intramuscular injection of penicillin benzyl and streptomycin, intravenous injections of chloramphenicol or chlortetracycline low concentrations were detected in saliva

There have been described results of determining salivary concentrations of a number of systemic drugs: carbamazepine(VasudevA., etal., 2002), digoxin(DrobitchR.K., SvenssonC.K., 1992), topiramate(MilesM.V., etal., 2003) methadone(MoolchanE.T, etal, 2001), disopyramides(SagawaK., etal., 1997), docetaxel and paclitaxel(MortierK.A., etal., 2005), pregabalin, azithromycin, paracetamol(IdkaidekN., etal., 2017, 2018), cortisol, dehydroepiandrosterone and 17-hydroxyprogesterone(GallagherP., etal.,2006).

It should be noted that the drugs for the treatment of tuberculosis ethambutol, amikacin and pyrazinamide are not determined in saliva, and isoniazid, rifampicin, moxifloxacin, ofloxacin, clarithromycin, doripenem and amoxiclav give a wide range of obtained values and extremely low concentration in saliva, while the drugs - gatifloxacin and linezolid well determined in saliva (vandenElsens. H.J., etal., 2018).

H. Clement et al. (2017) showed that the drugs venaflaxine, citalopram, quetiapine, aripirazole, and methylphendiate, except for apirazole and its metabolite, are determined in saliva and correlate with their plasma concentrations.

Studies on healthy volunteers found that the drugs sitagliptin, cinalcet, metformin, tolterodine, hydrochlorothiazide, azithromycin, rosuvastatin and cloxacillin were easily detected in saliva, whereas the other drugs montelukasta, lornoxicam, diacylhein, losartan and tamsulosin were not detected in

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saliva.IdkaidekN., ArafatT., (2012, 2014). N. Lee et al. (2014) in experiments on mice showed that the drug metformin accumulates in the salivary glands and is excreted in saliva.

More than 400 drug names have inhibitory effects on salivary gland secretory function (YingJoannaN.D., ThomsonW.M., 2015; YeohS.-C., etal., 2018; TeohL., etal., 2019).

There are various mechanisms by which certain drugs cause xerostomia. For example, anticholinergic, adrenergic, and psychotic drugs affect sympathetic, and antihistamines and antimotropic drugs affect parasympathetic innervation of salivary glands (Miranda-RiusJ., etal., 2015).

The following groups of drug groups are also responsible for suppression of secretory function of salivary glands - hypotensive and antiparkinsonian drugs, diuretics, sleeping pills, bronchodilators, muscle relaxants, beta-adrenoblockers, laxatives, narcotic drugs, alpha 2-adrenostimulants (Gaisina E.F. et al, 2018; Ozhegina A.L., 2019; JayakaranT.G., 2014; TeohL., etal., 2019).

Multiple carious dental defects, fungal and bacterial infections of the oral mucosa, red squamous lichen, cheilitis, glossitis are revealed in the oral tissues of xerostomia (Pozdnyakova A.A. et al., 2013; Gaisina E.F. et al., 2018;VillaA., AbatiS., 2011).

Sialorrhea causes exposure to heavy metals (mercury, thallium), taking acetylcholinesterase inhibitors and other drugs (Freudenreich O., 2005). Profuse salivation can lead to social isolation and sleep disturbance in patients (Zalyalova Z.A., 2017). In addition, hypersalivation causes interstitial swelling of salivary glands, parotitis, skin irritation around the lips and chin, aspiration pneumonia (Miranda-RiusJ., etal., 2015).

Hyperplasia is accompanied by swelling and redness of the gum, causing the patient to have problems with speech, function, and aesthetics (HatahiraH., etal., 2017). The interdental papillae swell or overgrow, covering the vestibular part of the tooth in the cervical and proximal areas. This creates an obstacle for the patient to perform individual oral hygiene, resulting in the formation of abundant plaque (HeasmanP.A., HughesF.J., 2014). Gingival inflammation and bleeding develops.

Gingival hyperplasia was also noted in patients who were prescribed immunosuppressant cyclosporine for the prevention of transplant rejection and treatment of autoimmune diseases (Chukhray I.G. et al., 2012; Kiladzhieva E.B., Gaidarova A.A., 2016.

Cyclosporine causes gingival hyperplasia in 25-30% in adults and > 70% in children (PaperI., 2004) in 1-4 months after intakeHachiraH., etal.,(2016). At the same time a positive correlation between the concentration of cyclosporine in blood plasma and the probability of developing gingival hyperplasia was revealed (HallmonW.W., RossmannJ.A., 1999).It was found that hyperplastic changes in the gingival tissues are caused by calcium channel blockers, which are prescribed to correct hypertension, arrhythmia and angina pectoris. Gingival hyperplasia has been shown to develop from 2 to 14 months after initiation of calcium channel blockers (HatahiraH., etal., 2017).

The action of the drugs usually ends with metabolism and/or subsequent excretion with the urine, to a lesser extent with the bile or directly through the intestinal wall. A secondary excretory role is played by sweat, tears, breast milk, and saliva (Kambhampati, S.R.P., etal., 2000). Saliva excretion is not really the way a drug is excreted because the drug is usually swallowed and reabsorbed, thus it is a form of "salivary recirculation" (WashingtonN., etal., 2001).

Free concentrations of drugs and their metabolites have been found to be detected in saliva (PatsalosP.N., BerryD.J., 2013). Most water-soluble drugs pass from blood plasma into saliva by passive transport (ApsJ.K., MartensL.C., 2005).

It is recognized that the salivary concentration of fat-soluble drugs is a reflection of their plasma concentrations in the free form. At the same time, the concentration of drug compounds such as iodide and spiramycin is higher in saliva than in blood plasma. A number of drugs - caffeine, phenytoin and theophylline - are excreted in saliva only in complex with proteins.

A bitter taste in the patient's mouth after taking the medication is a major indication of the excreted drug. The concentration of a drug in saliva is affected by its ability to bind to proteins, degree of dissociation, lipophilicity and chemical stability in saliva, salivary pH, salivary secretion rate, oral tissue condition, and the performance of the oral bicarbonate buffer system (Aps

J.K., Martens L.C., 2005; Raju K.S., et al., 2013).

In their studies, I.P. Sapos et al. (2019) established the effects of angiotensin II receptor blocker (drug losartan) and P-adrenoreceptor antagonist (drug isoproterenol) on salivary gland morphology and secretion in rats. It was found that losartan, affecting the mRNA of the renin-angiotensin system, reduced the secretory activity of acinar cells, blocked the Na+/K+pump, but did not change cell morphology. In contrast, isoproterenol influenced the gene expression of acinar cells of parotid and submandibular salivary glands, thus causing their hypertrophy, without disturbing the secretory activity.

Based on the above, mixed saliva samples are easy to obtain and can be an excellent alternative to blood samples when studying pharmacokinetics or monitoring drug action. However, it should be noted

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that drug concentrations in mixed saliva can vary. Lack of oral hygiene, gingival inflammation, and missing teeth (and thus lack of gingival fluid) are factors that can affect drug concentrations in saliva. Thus, co-morbidities contribute to the development of pathological conditions in the oral tissues, and against this background, there are various medications to correct them. Statistics show that in 2-4% of cases people are hospitalized because of the reaction caused by taking medications (JayakaranT.G., 2014).

Currently, the list of side effects from taking medications has been expanded by the description of pathological reactions in the oral cavity and perioral area (TopelL.A., etal., 2004). Patterns of dental disease have been identified that can help not only dentists, but also general practitioners, to determine the causal relationship to a particular drug or group of drugs.

The mechanism of a drug reaction is not always known and predictable, because in addition to known data on its pharmacodynamics and/or pharmacokinetics, various interacting variables can enter into the reaction, which can lead to unfortunate results. The gerontological population, which is a major consumer of medications, should not be overlooked. Often these patients self-administer over-the-counter medications to improve their health status, making it difficult to diagnose the disease (CiancioS.G., 2004).

It should be noted that oral tissue reactions caused by systemic medications clinically manifest as xerostomia, edema, nonspecific ulceration, bullous or ulcerative gingivitis, mucosal pigmentation, gingival edema, halitosis, dysgeusia, dental discoloritis, etc. In this connection, this literature review is aimed at expanding the professional knowledge of dentists for successful work with patients with concomitant diseases with the prospect of early diagnosis, improving the quality of dental care and preventing the development of pathologies.

Thus, all data on revealing the dental status of patients with concomitant system diseases are reduced either to the general reaction of the body to the disease itself, or in the oral cavity as an open system, which provoked its development. Many examples are given that the role participants of the pathological process in the oral cavity are epithelial cells and nonspecific immunity, microflora and its metabolites, the protective systems of the oral cavity, whose markers reflect the emerging imbalance in the organs and systems of the body. In this case there is a "bouquet" of diseases, which requires a versatile examination and comprehensive treatment. Therefore, our review of the literature emphasized the need for a comprehensive dental and system-wide approach, the implementation of which, organizes the coordinated work between the signaling pathways of cellular metabolism and organ function.

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