

DENTAL FOCAL DISEASE. ETIOPATHOGENESIS AND DIAGNOSIS

ADRIANA SĂCELEANU¹

¹County Clinical Emergency Hospital Sibiu

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Abstract: Focal disease is a pathological condition characterized by a large variety of functional disorders and organic tissue alterations due to chronic focal infections, which, episodically and through the blood, nervous or digestive systems, various microbes, microbial toxins, toxic products of tissue septic disintegration, endo and exogenous allergens are disseminated into the organism, which generate a vast array of dysfunctional and lesional events.

Cuvinte cheie: boala de focar, focar dentar

Rezumat: Boala de focar este o stare patologică caracterizată printr-o mare diversitate de tulburări funcționale și alterări organice tisulare, datorită unor focare cronice de infecție, din care episodic se diseminează în organism pe cale sanguină, nervoasă sau digestivă diverși microbi, toxine microbiene, produși toxici de dezagregare septică tisulară, alergeni endo și exogeni, care generează un tablou vast de manifestări disfuncționale și lezionale.

The real scientific research in this area began with Rosenow, who succeeded in demonstrating experimentally the pathological, anatomical and bacteriological correlations and the continuous or intermittent sustained flooding of streptococci in bloodstream. In our country, Hatieganu and Goia were preoccupied with the concept of focal disease and showed that this is a disease of the whole organism, comprising two pathological entities:

- **presence of chronic focal infections**, consisting of microbial masses and compounds of tissue septic degradation encapsulated in a fibrous connective membrane with chronic, latent evolution or oligo- or locally asymptomatic representing the primary manifestation.
- **dysfunctional or lesional manifestations at distance**, which consist of functional disorders and tissue damage in predisposed parenchymal organs, with rich and varied symptoms, forming metastases.

Diverse chronic focal infections, with varied local and general etiology can represent the focal infection with remote dysfunctional manifestations affecting mainly some organs such as, the musculoskeletal system (acute and chronic deforming arthritis, polyarticular rheumatic), the eye (retinitis, iridocyclitis, choroiditis), the digestive tract (colitis, gastritis, ulcers), the cardiovascular system (endocarditis, myocarditis, Raynaud's disease, acrocyanosis), the urinary system (nephritis), the nervous system (sciatica, back pain, polyneuritis).

The damage produced to any organ or system largely depends on individual responsiveness to constitutional factors, the overall condition of the body, the number of focal infections, their content and virulence of microorganisms. Any chronic inflammatory process, wherever enclosed in the body, is a source of infection. The most common locations for the development of chronic focal infections are the teeth, tonsils, facial sinuses (maxillary), middle ear, gallbladder, gastrointestinal tract, urogenital tract.

Regarding the spread, in percentage terms:

- 90% of the focal infections within the body are found in the cephalic region,
- 10% in the rest of the body.

From a **quantitative** perspective, general focal infections are classified according to their percentage frequency and location, according to the table below:

Table no. 1. Location and percentage frequency of general focal infections

Cephalic focal infections 90%	Extracranial focal infections 10%
Periodontal infections 72 %	Adnexitis 2-7 %
Tonsils infections 18 %	Bronchial 0, 8 %
Sinus infections < 18 %	Intestinal 0, 5 %
Middle ear < 18 %	Extrahepatic biliary tract 0, 5 %

Qualitatively speaking, the most aggressive are:

- amygdaline forms
- periodontal infections

According to **aggression**, the chronic focal infections can be differentiated in:

- active
- passive

Most times, one cannot make a chronological distinction between them, since a passive focal infection can activate, and an active one can become passive.

Classifications of dental focal infections

In the past century, Slauck showed that 93% of the active focal infections are provided by teeth. The anatomical conformation of the teeth creates special conditions for chronic infections because the enamel and dentin do not benefit from

¹Corresponding author: Adriana Săceleanu, Str. Bălea, Nr. 1A, Ap. 33, Sc. C, Sibiu, România, E-mail: vicentiu.saceleanu@gmail.com, Tel: +40740 022931

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defence capacities, being inaccessible to the tissue and humoral defence elements.

Today, in order to detect the focal infections, we first examine the teeth and the tonsils and afterwards the other organs.

Focal infections in the oral cavity can be:

- **odontal** (at teeth level)
- **periodontal** (at periodontium level)

Odontal focal infections are developed at the dental pulp level encapsulated in the endodontic cavities (pulp chamber and root canals) and the **periodontal** ones develop at the marginal and apical periodontium level.

In terms of topography and manner of occurrence, they can be classified into:

- **primary**
- **secondary**

The primary focal infections can be: intradental, extradental and residual.

1. **Intradental focal infections** come from the pulp damage, as a result of the untreated decay or of a therapeutic failure. They can develop from *chronic pulpitis, direct and indirect failed pulp capping, vital and devital pulpectomies, simple gangrene, residual pulpitis*.

Potential dental focal infections are considered to be those teeth which suffered accidental injuries, those with pathological abrasion, which can generate chronic pulpopathies, old and large obturations especially of amalgam, exaggerated vital teeth grinding for prosthetic purpose exaggerated, very destroyed teeth.

2. Extradental focal infections:

- Ø **Periapical** focal infections are the most common, being represented by complications of chronic pulpitis and gangrene, such as fibrous apical periodontitis, cyst epithelial granuloma, diffuse progressive chronic periodontitis.

Incorrect root fillings may cause periapical focal infections, especially in pluriradicular teeth.

- Ø **Marginal focal infections** are due to the formation and occurrence of **periodontal pockets**. Their secretion is only partially drained, even under favourable anatomical conditions of wide opening of the pocket, thus constituting a powerful source of infection, especially when it is serpinous and with retention tendency.

In all periodontal pockets, there is also a tissue aggression manifested by tissue enzymes, microbial flora, endo- and exotoxins, whose virulence is differentiated by the action of various local and general factors.

For all these reasons, the marginal infections are similar with the apical and intradental infections.

Besides periodontal pockets, frequent pericoronitis can be mentioned at the wisdom tooth level, that through its chronic state, can turn in primary focal infection.

Marginal chronic irritations caused by dental plaque or by the prosthetic dentures incorrectly marginally adjusted can cause chronic marginal periodontal inflammatory reactions that may occur in the genesis of the focal disease.

Included teeth, whose follicular sac may become infected by oral communication or through blood, may become focal infections.

3. **Residual focal infections** – are rare and among which, the following can be mentioned: chronic periapical infectious in the depth of the alveolar bone, when after tooth extraction a proper alveolar curettage has not been performed (periapical osteitis processes, radicular cysts). These can become focal dental infections and are detected only radiologically.

Secondary focal infections are called as such because their occurrence is related to the existence of a primary focal infection, from which the infection migrates to the neighbouring or at the remote anatomical regions.

Thus, there may be: lymphadenopathies, adenitis, maxillary sinusitis, odontogenic chronic thrombophlebitis. Some secondary focal infections may disappear after the removal of the primary infection, others require medical or surgical treatment and some may persist as such (regional lymphadenopathy after tooth extraction in focus).

Etiopathogenesis of the focal disease

The etiopathogenetic mechanism of the focal disease requires a review of several theories:

- Ø **Etiopathological microbial theory** - considers that the focal disease is caused by the dissemination of the bacteria from the focal infection through blood and lymph, either under the form of Lexter septicaemia with massive disseminations in the general flow, or under the form of attenuated disseminations.
- Ø **Allergic theory** (Berger, Rossle, Goia) – is the most widely accepted theory of today, according to which remote disorders are allergic, representing the result of the interaction of antigens that enter the form the infection into the organism and the antibodies generated by the previous penetration of antigens that sensitized the body.
- Ø **Theory of toxicity** (Slauk) – argues that the focal disease is due to the irritation of the anterior medullary horns and certain nerve centres by the toxins in the focal disease, which migrate through the endo- and perineural lymph towards the large cerebral lymphatic tanks, directly acting on the CNS centres and nerve cells.
- Ø **Vascular theory** (Riker) – excitations of the focal infection act upon the central and peripheral neuro-vegetative system. As a response to these excitations, histamine, acetylcholine and adrenaline release increases, vascular tone is changed, ischemia processes occur, as well as vasodilatation, followed by stasis, both damaging the blood tissue irrigation.
- Ø **Adaptation syndrome theory** (Selye) – includes all non-specific defence reactions that occur in the body in response to various harmful factors (stress). The toxins from the cell and microbial disintegration irritate the diencephalon, either directly or by way of peripheral nerves, leading to the stimulation of ACTH secretion, followed by the hypersecretion of the adrenal cortical hormones. This theory considers the focal infection as an excessive defence reaction of the organism.
- Ø **Psychosomatic theory** (Mathis, Winckler) - old, nerve-based conception, originating from Pavlov's research, arguing that the focal disease is due to disorders of CNS and the peripheral organs.

It is difficult to specify which of these mechanisms are responsible for various pathological manifestations of the focal disease, as they are frequently associated, acting in parallel or by overlapping.

Nowadays, it is known that:

- Ø **pathogenically**, chronic focal infections are bounded by a fibro-conjunctive membrane, which actually, is a barrier, however imperfect in their septic content made up of toxic microorganisms and tissue degradation products that can disseminate in the body through blood or lymph.
- Ø decreased body resistance, exacerbation of microbial virulence, tissue hyperemia or loco-regional trauma are the **main dissemination factors**.
- Ø during the periods of **inactivity**, chronic focal infections are well defined and controlled by the body through the

cell, humoral and immune barrier. Through cell and tissue polymerization, fibroblastic collagenization and hyaline metamorphosis, a sealing processes sealing of the fibro-conjunctive barrier occurs, as well as an increase of the body defence means. and potentiation of the means of defence of the body.

- Ø During **activity**, there is a cell and tissue depolymerization process that leads to an increase of the capillary walls permeability allowing the diffusion of microbial endo and exogenous toxins in the body, causing diverse symptoms in some receptive tissues.
- Ø immuno-chemical barrier fails in some circumstances (flu and other infections of the body), which may lead to the activation of a focal disease.
- Ø active focal infections are wrongly considered only those radiographically visible focal diseases, that are large and extended; many of them can be inactive, just as in the case of the intrapulpar invisible focal infections, which can be very active (e.g., pulp gangrene without radiographically apical visible changes).

Active focal infectious may play a dominant role in case of RAAS, poststreptococcal glomerulonephritis or modelling role in rheumatoid arthritis, sero-negative arthritis, such as ankylosing spondylitis, Reiter's syndrome, Crohn's disease, Behcet's syndrome, and some collagenoses.

General **clinical signs** are not specific but may alert the doctor when they are accompanied by other disorders too. The most common signs are: fatigue, sleep disturbances, headaches, palpitations, angina events, vasomotor disturbances, multiple pains in the whole body, athralgia, low grade fever, cross localized pain, such as shoulder-knee, bloating dyspeptic disorders, cardio disorders, hyperthyroidism. Characteristic is that the general signs potentiate and extend after effort and especially to weather changes.

Local signs consist of myocardial disorders, coronary disorders, joint inflammation, anemia, rhinitis, laryngotracheitis, iritis, iridocyclitis, phlebitis, enterocolitis.

Focal infection diagnosis is a very important and difficult issue for the medical practice because one has to specify whether the pathological manifestations are due to an independent disease or to the existence of chronic focal infection. They must be by no means detected and drained by the team made up of the internist in collaboration with other specialists: dentist, urologist, ophthalmologist, neurologist, gynecologist, surgeon, otolaryngologist; the internist physician should do a complete and thorough exam, eliminating other diseases with similar symptoms (TB, cancer, hemopathies etc.) that can evolve with headache, fatigue, loss of appetite, low grade fever, palpitations, sweating etc.

In setting the diagnosis, certain laboratory tests, radiological, biopsy and other various tests are required. The most important biochemical reactions in the focal infection are: ESR, blood count, serum gamma, transaminases, ASO.

The most important focal diseases specific tests are:

- histamine test (Remky)
- atrial muscle test (Slauk)
- march sample (Bircher, BÖTTNER)
- salicylic acid test (Heisen)
- salicylate test (Blumenkron)
- tourniquet test (Borbely, Gotshal)

Periodontal focal infections diagnosis within a focal disease

It is necessary for the dentist to have at hand a series of clinical and radiological signs with high diagnostic value for detecting the primary odonto-periodontal focal infections.

Anamnesis includes: identification data (name, age,

occupation, address, living and working conditions), the reason for consultation, current medical history, family history, personal pathological history.

Clinical examination

- **subjective examination** that contains data related to the patient about the nature of pain: chewing discomfort, pain upon the action of fluids, chemicals.
- **physical examination** - which contains data obtained by doctor through:

a) Inspection that should highlight:

- *exobucal*: facial symmetry (vertically, laterally), profile (convex, concave, right) profile, skin integrity, ATM examining (static and dynamic).

- *endobucal*: examination of lips, jugal mucosa, Stenon's duct orifice, palate, tongue, sublingual region, mouth, arches exam, teeth cavities, bulky crowns, presence of fistulas and scars, pathological pockets and purulent secretions .

b) Palpation outlines the lips, tongue, gums, tooth mobility and pathological migrations etc.

c) Percussion identifying axial and transverse pathological changes at odontal level.

Complementary examinations consist in accomplishing the vitality proof, diaphanoscopy, thermometry, radiological and specific tests.

A. Radiological examination needs to be performed for all the teeth and edentate spaces in order to detect any residual focal infections.

B. Specific tests

a. Physical agents provocation tests

- Ø mastication test (**Lukomski**)
- Ø vibration test (**Wannenmacher**)
- Ø ultra rays test (**Gutzeit-Kuchlin**)
- Ø high frequency voltage test (diathermal)
- Ø submento-submandibular ganglion massage test (**Wilde**)
- Ø X rays test (**Driak**)

b. Biological agents provocation tests

- Ø penicillin test(**Fenner**)
- Ø specific antigen test (Bottyan-Kallay)
- Ø histamine test (Erkes)
- Ø pyrifera- pyrogen preparation test with nonpathogenic microbial sources.

c. Cancellation test (Huneke)

- Ø Testul cu impletol

d. Cancellation-provocation test (Adler)

Generally, the tests do not have pathognomonic value, but in conjunction with clinical, radiological examination and various complementary examinations allow the establishment of a certain diagnosis.

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