

EARLY DIAGNOSIS AND CLASSIFICATION OF PROFESSIONAL FLUOROSIS

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SUMMARY: On the basis of evaluating the clinical course and supplementary method of examination carried out in 132 patients suffering of professional fluorosis and in 200 workers in the premorbid state the author proposes criteria of early diagnosis. Considering Zislin's classification of professional fluorosis, literature data and own findings the author introduces 2 new criteria in the classification, namely, content of fluorine in the urine and length of work in this profession.

Diagnosis of professional fluorosis (PF) and its classification have been poorly studied. After development of PF classification by D. M. Zislin and E. Ya. Girskaia [3], new study methods have been introduced in clinical practice which have allowed some indications of the disease to be revealed [2, 5, 6, 8]. However, this classification does not take etiology into account. There are no parameters for fluorine in the human biological environment. Both domestic and foreign authors have confirmed the existing correlation between concentration of fluoride in the air of a manufacturing facility and its accumulation in the organism and excretion with urine ($r=0.86$) [5, 7]. Urine fluorides are an adequate indicator of the fluoride effect, and they correlate with the time of their impact on the organism. That is why it is important to include the length of professional employment in a classification of PF. Earlier PF classifications were based on the diagnosis of bone changes [1]. However, with PF the development of hepatitis, erosive gastritis, and ulcerous disease prior to bone changes is possible. Some authors separate out the period of functional disturbances in the 1st stage of PF which is unreasonable because it is impossible to imagine the existence of function without structure; they are viewed as a close union.

Our classification of PF is a further development of the classification of D. M. Zislin and co-authors [3], taking into consideration parameters of fluoride in urine and length of professional employment. It more fully represents damage to internal organs (hepatitis, gastritis, etc.). Results of testing of 132 patients with PF and 230 workers with premorbid condition are presented. X-ray testing of bones and joints, lungs, stomach, and gall bladder has been performed; radiological and ultrasound studies of liver, gall bladder, pancreas; and fibrogastroscopy examination of the esophagus, stomach, and duodenum. Concentration of fluoride in urine, bile, and stomach juices was studied in more than 400 samples [7]. Fluoride concentration in the air of industrial areas exceeded MAC [maximal allowable concentration] twofold and more on average. Its correlation with clinical signs and fluoride concentration in urine in workers from Dnepropetrovsk and Krasnoyarsk aluminum factories (1978-1988) has been performed.

Here are the criteria for diagnosis of the PF stage. During stage I (initial) the length of professional employment, as a rule 7-15 years, concentration of fluoride in urine 1-3 days after cessation of contact with fluoride in most cases is 6 times higher ($5.1 \cdot 10^{-5} \pm 0.42 \cdot 10^{-5}$ mol/L) compared to control ($1.26 \cdot 10^{-5} \pm 0.17 \cdot 10^{-5}$ mol/L).

Primary syndromes and symptoms. Musculoskeletal system. Arthralgia, ossalgia, myalgia, muscle spasms. X-ray either does not reveal changes or reveals initial hyperostosis (periostosis), more often symmetrical in sites of attachment of ligaments and muscles. There is an increased density of primary mandible and tubular bones on densitometry. Oral cavity: impairment of teeth enamel (loss of shine, abrasion, chalk-like stripes, or spots involving more than 1/3 of the tooth surface), stomatitis, gingivitis.

Gastroduodenal system. Superficial antral and erosive gastritis with increase or normal secretory function of the stomach; duodenal ulcer, seldom of antral part of the stomach with increased or normal

secretory function; superficial and erosive duodenitis. In small doses fluoride stimulated adenylate cyclase [4, 6]. This leads to an increase of organ function (features of acidism, increased secretory function of the stomach, and erosions and ulcers of the stomach and duodenal mucosa). A triad of gastroduodenal system impairment with PF can include one of the listed conditions, but more common are erosive gastritis and ulcerous disease.

Hepatobiliary system. A latent course of persistent hepatitis with normal or insignificantly altered functional hepatic tests – protein forming with decreased amount of albumins in serum, glycogen forming (increased hyperglycemic coefficient), less often a pigment test with an increased level of unconjugated bilirubin. Hepatitis is diagnosed primarily during scanning and ultrasound testing of the liver.

Upper respiratory tract. Rhinitis, laryngitis, pharyngitis, more often hypertrophic or subatrophic. Visual organs: chronic conjunctivitis.

Neurocirculatory dystonia (NCD), and asthenic-neurotic syndrome are also possible, and less often vegetosensory polyneuritis, hyperfunction of the adrenal cortex, small foci of dystrophy in the premacular zone, and impairment of visual function – decrease of light adaptation up to 10 optical units.

As shown by retrospective analysis, early diagnosis of PF is complicated due to insufficient competency of company physicians and professional pathologists of employee health departments; lack of modern methods of evaluation in most outpatient clinics and professional pathology departments, allowing performance of ultrasound testing, immunological evaluation, etc. Presence of fluoride in human biological environments is not studied.

During stage II of PF (full clinical picture) the length of professional employment is, as a rule, 10-20 years and concentration of fluoride in urine 1-3 days after cessation of contact with it is 7-8 times greater ($9.3 \cdot 10^{-5} \pm 0.26 \cdot 10^{-5}$ mol/L) compared to control group.

Musculoskeletal system. Deforming osteoarthritis with impairment of the joint function degree I; moderate hyperostosis mainly in tubular bones, pelvic bones, ribs, and vertebra (periostosis, endostosis, enlargement of cortical layer, narrowing of bone marrow canal, and partial calcification of sites of attachment of ligaments and muscles).

Oral cavity. Chalk-like spots occupy more than ½ of tooth surface, alternating with dark-yellow or brown pigmentation sections; abrasion and fragility of teeth; subatrophic and atrophic stomatitis, and gingivitis.

Gastroduodenal system. Superficial or moderately expressed primarily antral gastritis with increased or normal, less often decreased secretory function of the stomach. Erosive gastritis, duodenal ulcer with increased, normal or less often decreased secretory function. Erosive, superficial, and moderately expressed duodenitis.

Hepatobiliary system. Persistent hepatitis with normal, insignificantly altered, less often moderately altered functional hepatic.

Upper respiratory tract. Subatrophic and atrophic rhinitis, laryngitis, and pharyngitis. Visual organs: chronic conjunctivitis.

Other organs and systems can be affected as well: NCD, seldom myocardial dystrophy, asthenic-neurotic syndrome, vegetosensory polyneuritis; auditory neuritis, decreased function of adrenal cortex and sex glands, increased retinal degeneration and impairment of visual function; and cochlear neuritis of toxic origin. Decrease of hearing can originate in the development of otosclerosis.

Stage III of PF (expressed osteosclerosis) is rarely seen (in 7% of workers), length of employment is, as a rule, 20 years and more and fluoride concentration in urine is 7-8 times greater than in control group.

Leading syndromes and symptoms common for stage III. Musculoskeletal system: expressed and systemic hyperostosis (diffuse marble shadow of the bone, no details of its composition are visualized,

significant calcification of ligaments and tendons which leads to impaired mobility of the chest, vertebra, joints). Deforming osteoarthritis with impaired joint function degree II-III.

Oral cavity. Abrasion and fragility of teeth are more expressed. Atrophic gingivitis and paradontosis.

Gastroduodenal system. Superficial or moderately expressed gastritis with normal, less often decreased secretory stomach function; seldom expressed atrophic gastritis. Erosive gastritis, ulcer disease with the same localization of the ulcer with increased, normal, less often decreased secretory function of the stomach; erosive, superficial and moderately expressed duodenitis. Hepatobiliary system: persistent gastritis with insignificant or moderately altered functional testing of the liver.

Upper respiratory tract. Rhinitis, laryngitis, pharyngitis, more often atrophic. Visual organs: chronic conjunctivitis. Changes of other organs and systems are possible, like in stage II.

Involvement of bronchopulmonary system is seen in $\frac{1}{3}$ of patients with stage I and in $\frac{1}{2}$ of patients with stage II fluorosis. Insignificant expression of symptoms was characteristic of clinical implications in the pulmonary system. In some patients professional bronchial asthma and chronic bronchitis with bronchospastic syndrome was diagnosed. X-ray examination of the thoracic organs revealed diffuse pneumosclerosis. In the origin of chronic bronchitis, a main role is assigned to the combined effects of mixed alumina-containing dust and vaporous toxic substances.

Here is the formulation of stage II PF diagnosis: deforming osteoarthritis of knee and elbow joints with degree I impairment of joint function, moderate hyperostosis of tubular bones, erosive antral gastritis with increased secretory function of stomach, persistent hepatitis, and subatrophic pharyngitis.

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