

DIAGNOSIS AND TREATMENT OF DEFORMING OSTEOARTHRITIS IN SUBJECTS IN CONTACT WITH FLUORIDE COMPOUNDS

V. N. MEDVEDEVA, Doctor of Medical Sciences
Zaporozhsky Institute of Continuing Education for Physicians

SUMMARY: A comparative study is presented of 378 workers with osteoarthritis deformans (OD) contacting with fluorine compounds and in 106 patients with primary OD. The diagnostic criteria in these two categories of patients coincided. But the pain syndrome in the joints in professional fluorosis (PF) was associated with ossalgias (85%), tenderness of the bones (50.1%) and in 100% by symmetric hyperostosis. OD in PF was accompanied by a more pronounced density, was not accompanied by formation of nodes, and secondary synovitis was 4 times frequent. In OD plus PF males prevailed (96.7 e), and were averagely 10 years younger. In OD+PF treatment diet, calcium agents, glutamic acid should be used that bind and excrete fluorine from the body.

Deforming osteoarthritis (DOA) has the highest prevalence rate among joint diseases [1, 3-9]. There is little information about the development of DOA in subjects in contact with fluoride compounds [2].

We have studied the characteristics of the diagnosis and treatment of DOA with patients who had previous contact with fluoride compounds in comparison to primary DOA. 484 patients have been examined, of them 106 had primary DOA, and 378 were workers in the main professions of electrolysis departments, and the department of dust control and preparation of fluorides at Krasnoyarsk and Dneprovsk aluminum factories; where the main unfavorable factors are fluoride compounds, which for many years have had a concentration 1.2-6 times exceeding maximally allowed concentrations (MAC). Besides a general clinical examination, X-ray of joints and bones has been performed with the step-type aluminum standard and concentration of fluorine in the biological environment of the organism (urine, bile, gastric juice) has been determined with a fluoride-selective electrode. The method's sensitivity is high: 0.05 – 1 µg/mL [10].

Morbidity analysis has revealed that, according to the number of cases and days of disability per 100 workers, diseases of bone-joint system are the most common chronic diseases. All examined subjects have been divided into three groups: first group – 241 workers without professional fluorosis (PF), second – 65 with stage I PF, third – 72 subjects with stage II PF. In the first group, the average age was (36.7 ± 0.7) years, length of employment (8.8 ± 0.7) years; in the second – (43.2 ± 0.4) and (14.8 ± 0.4) years; and in the third – (48.5 ± 1.2) and (19.5 ± 0.6) years, respectively.

Rate and degree of the joint syndrome increased depending on the length of employment and the stage of PF. The most common symptom was arthralgia which developed over 3–14 years with on average (9.7 ± 0.37) years of employment at the factory. Elbow joint involvement was seen in 39.1%, knee joint in 30.3%, and shoulder joint in 25.2% of patients. Most rare were pains in the small joints of hands (6.2%). Joint pain was more intense after prolonged standing, at night, and when descending stairs. The intensity of pain would increase by the end of the day. Degree of arthralgia increased with a higher PF stage. Painfulness upon palpation of joints and active and passive movements was rarer but also increased with longer employment and higher PF stage. Crackling of joints was a common complaint amongst patients: 34.0, 76.9, and 98.6%, respectively in each group. No swelling was seen in the joints of the first group, in 1.5% of patients in second group, and in 6.9% of patients in the third group. No deformation or impairment of joint function was seen in the first group, in the second these were detected in 6.2% and 3.1%, in the third in 16.3% and 16.2%, respectively.

X-ray examination showed the following changes in joints: in the first group acanthoid growths of bone tissue – in 16.3%; in the second and third groups – osteophytes in almost all subjects (84.6% and 87.5%, respectively); narrowing of the joint space in 24.6 and 26.4%; joint deformation in 12.3 and 20.8%; ossification of sites of attachment of ligaments, muscles and joint capsules in 6.2 and 12.5%; impairment of joint functions in 6.2 and 20.8%, respectively in each group.

The results of clinical urinalysis, concentration of sialic acids, C-reactive protein, mucoproteins, and ratio of protein fractions of blood were within normal limits in all cases without secondary sinovitis. Clinical, laboratory, and X-ray assessments confirmed that the joint lesion took the course of secondary DOA.

Most common concomitant diseases in all patients were chronic gastritis (88.7, 91.6, and 98.4%), including erosive (25, 21.3, and 30.1%) and chronic persistent hepatitis (32.8, 66.2, and 73.6%). Ulcer disease with localization of the duodenal ulcer was seen in 33.9, 30.1, and 42.3%; duodenitis in 50.8, 38.6, and 58.2%, respectively in each group. Often concomitant diseases included chronic cholecystitis, chronic bronchitis, and inflammatory diseases of the upper respiratory tract.

Diagnostic criteria of DOA with PF and DOA were mostly the same. However, DOA with PF had some specific symptoms. Pain syndrome in joints during PF was often followed by ossalgia (85.2%), and pain on percussion of wrists (50.1%) in 100% of patients – with symmetrical hyperostosis of bones in sites of attachment of ligaments and muscles, more often in bones of forearms and shins, rarely in pelvic bones, ribs, and vertebral bones (periostosis, endostosis, enlargement of cortical layer, narrowing of bone marrow canal, calcification of sites of attachments of ligaments, and muscles and joint capsules). DOA during PF was seen with more pronounced thickening of the cartilage structure and was not followed by the formation of Heberden's nodes; secondary synovitis was diagnosed 4 times less with PF. Among patients with DOA developed against a background of PF, males were dominant (96.7%). With primary DOA, 21.4% of patients were male.

For differential diagnosis, most significant are length of professional employment, concentration of fluoride at the work site, and biological environment of the organism, which in our studies were 3-10 times greater than the MAC. Thus, fluoride concentration in urine 1-3 days after cessation of contact in subjects of the first group was $5.22 \cdot 10^{-5} \pm 0.41 \cdot 10^{-5}$ mol/l, second – $7.51 \cdot 10^{-5} \pm 0.59 \cdot 10^{-5}$ mol/l, third group – $9.4 \cdot 10^{-5} \pm 0.27 \cdot 10^{-5}$ mol/l and in control group – $1.4 \cdot 10^{-5} \pm 0.017 \cdot 10^{-5}$ mol/L. Fluoride concentration in urine was significantly higher in all the groups compared to control, and was significantly different between the first and second groups and the second and third groups. Significant increase of fluoride concentration in all the groups compared to control was also discovered in bile and gastric juice.

In the origin of DOA in patients with PF and those in contact with fluoride compounds, it is possible that fluoride binding with calcium is deposited not only in bones but also in cartilage and ligaments of joints, disrupting metabolic processes in the cartilage and altering resistance even to a normal physical load. A similar process is probably significant in the development of synovitis as well as gout and chondrocalcinosis. One can assume that crystals of calcium fluoride enter the synovial fluid, are phagocytosed by neutrophil granulocytes of synovial fluid and synoviocytes, as a result of which lysosomal enzymes are released and activated, which cause an inflammatory reaction.

Basic principles of treatment of patients with fluoride-induced DOA are the same as with primary DOA. However, etiological factors must be considered and actions directed towards decreasing the amount of fluoride entering the human organism (following safety measures), and its binding and excretion from the organism must be taken. Fluoride compounds enter the organism mainly via the respiratory tract and mucous membranes of the stomach and intestines. It is reasonable to prescribe food substances that bind and excrete it through the intestines and kidneys. This is achieved by an increased portion of milk and calcinated cottage cheese, smaller portions, and sufficient amounts of food pectin substances and cellulose, watermelons, and grapes, which have a diuretic effect like milk. Considering that a significant amount of fluorine, according to our data, is excreted with bile, food must contain

sufficient amounts of vegetable oils which have a choleric effect. During work hours, one should limit the intake of fat, which favors ingestion of fluorine into the intestines. Monitoring concentration of fluoride in water is also necessary. If it is greater than 1.5 mg/L, defluorination of water is indicated.

For treatment of patients with DOA which develops as a result of fluoride compounds, calcium products and glutamic acid should be used.

Hyperbaric oxygenation was used in 15 patients; course of treatment – 10-15 sessions. Positive effects were seen in all patients, especially in people with pain syndrome in muscles; less effective was the treatment for pain in joints and bones.

References

1. M .G. Astapenko, K. V. Bayatova, V. M. Chepoi, etc. // *Rheumatology*. – 1986. – No.4. – P. 10-14.
2. M. Vykidal, *Pharmacotherapy and rheumatology*. – M. : Medicine, 1976. – P. 216.
3. D. M. Zislin, T. B. Tulayeva, L. I. Tikhoretckaya, T. K. Semennikova, // *Rheumatology*. – 1984. – No. 3. – P. 32-34.
4. *Clinical rheumatology* / under Kh. L. F. Karreya: Trans. From Eng. – M. : Medicine, 1990. – P. 446.
5. V. A. Nasonova, M. G. Astapenko, *Clinical rheumatology*. – M. : Medicine, 1989. – P. 591.
6. V. M. Chepoi, “Diagnostics and treatment of joint diseases” – M. : Medicine, 1990. – P. 303.
7. I. V. Schumada, O. Ya. Suslova, V. I. Stetcula, etc. “Diagnostics and treatment of degenerative-dystrophic lesions of joints.” – K. : *Health*, 1990. – P. 200.
8. Otte P. // *Z. Rheum.* – 1983. – Bd. 42, N 4. – S. 242-248.
9. Simon L., *Rheumatologie*. – Paris : Masson, 1984. – P. 592.
10. Faustino P. M. A., Ballintani S. A. // *Anais*. 24. – Rio de Janeiro, 1986. – P. 385-388.