

CHARACTERIZATION OF NERVOUS SYSTEM INTOXICATION IN OCCUPATIONAL FLUOROSIS

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SUMMARY: Neurological symptomatology in the form of the syndrome of vegetative-vascular dysfunction, or the asthenovegetative syndrome with polyneuritic (sensory and vegetative) disorders was detected in 78.8 per cent of patients with occupational fluorosis in preosteal and osteal stages. Clinical and physiological investigations of the nervous system (psycho-physiological procedures, electroencephalography, chronaximetry) showed patients with fluorosis to exhibit disturbed nervous activity and dysfunction of subcortical-axial nonspecific structures of the brain.

Fluorine is a poison with a broad spectrum of action affecting the metabolic processes, tissue respiration, and neuroendocrine regulation. For this reason, even before affecting the bones, occupational fluorosis manifests itself clinically as a generalized disease with syndromes affecting a number of important systems and organs, including the nervous system.

The neurotoxic effect of fluorine compounds has been proven by numerous experimental studies (A. F. Aksyuk and G. V. Bulychev; S. K. Bikhullina and E. I. Panycheva; R. D. Gabovich and M. M. Epshtein; V. A. Knizhnikov et al.; M. S. Sadilova et al.). However, the clinical picture of fluorine intoxication's effect on the nervous system has not been adequately researched. Workers with prolonged exposure to fluorine compounds are known to exhibit symptoms of vascular autonomic dysfunction (N. B. Alperina and M. I. Sokolsky; M. A. Akhmetov and T. T. Tagbergenov; M. T. Berdykhodzhin; V. A. Vasilieva et al.), and vascular autonomic dysfunction has been recognized as typical in occupational fluorosis (E. Ya. Girskaia). Some published articles (D. M. Zislin and E. Ya. Girskaia; V. R. Ovechkin) point out the possibility of a primary effect on the peripheral nervous system in occupational fluorosis.

In order to obtain a more accurate picture of neurological symptomatology, we surveyed the clinical picture of the disease in 80 patients diagnosed with occupational fluorosis, including 36 with stage 1 fluorosis (per D. M. Zislin and E. Ya. Girskaia) and 44 with stage 2 or 3 fluorosis. The population predominantly included patients 40 to 50 years of age with an occupational exposure to fluorine compounds in excess of 10 years. 45 patients were engaged in the main production process at cryolite factories (as operators of autoclaves, reactors, dryers, etc.) and exposed only to fluorine compounds at concentrations significantly exceeding MAC values. 35 patients were employed in aluminum production (electrolytic cell operators, anode makers, etc.) where the exposure to hydrogen fluoride is merely one of a number of occupational hazards.

Neurological symptomatology was present in 63 patients (78.8%), including 24 diagnosed with fluorosis without bone affection (stage 1) and 34 with advanced stages of fluorosis. We were able to distinguish two clinical variants of nervous system affection in the disease: vascular autonomic dysfunction syndrome and asthenic autonomic syndrome in combination with polyneuritis-type disorders (both sensory and autonomic).

Vascular autonomic dysfunction syndrome has been diagnosed in 37 patients (46.3%), including 16 stage 1 fluorosis patients and 21 stage 2-3 patients. Patients' complaints were typical of occupational fluorosis and included nearly permanent pains in the extremities without precise

localization, aggravating at rest and at night, frequent headaches, transient vestibular dysfunctions, hyperhidrosis, and chills in the extremities. On examination, the patients exhibited persistent red dermatographism and hyperactive Golgi tendon and deep tendon reflexes.

Asthenic autonomic syndrome with polyneuritis-type dysfunctions has been diagnosed in 26 patients (32.5%), including 8 stage 1 fluorosis patients and 18 stage 2-3 patients. In these patients, pains and paresthesias in the extremities, painful muscle cramps, fatigue, headache, and other health problems were more pronounced. On examination, they exhibited persistent red dermatographism, acrocyanosis, palm and foot hyperhidrosis, decreased finger skin temperature, and hyperactive Golgi tendon and deep tendon reflexes. Skin hypoesthesia in the extremities, whether of polyneuritic (“gloves” and “socks”) or “spotty” (skin areas around elbow and knee joints) type, manifested as well.

Some patients were subjected to clinical neurophysiological testing. Psychophysiological tests (reflexometry, word association experiment, and Bourdon test) revealed an excessive and varying latency in visual–motor and word–word response and a slower tempo and lower scores on the Bourdon test in 23 out of 34 patients examined. Introducing additional complexity in the test by requiring differentiation did not produce a greater effect on the test results than in the control group. These features of higher nervous activity may be assessed as neurodynamic disturbances in the functional subsystems of the brain (per P. K. Anokhin) responsible for the conditioned responses being tested.

An electroencephalographic examination at rest and under photic stimulation was performed on 26 patients. Frontal, central, parietal, occipital, and temporal EEG leads were recorded using uni- and bipolar electrodes.

5 patients exhibited a normal EEG. 8 patients exhibited unstable bioelectric activity with α -wave domination (α -wave strongly modulated by frequency and amplitude, spontaneous changes in activity type, and paroxysmal activity), pathological waveforms, and disturbed bioelectric response to photic stimulation. 3 patients exhibited a pronounced bioelectric desynchronization (a dysrhythmic EEG) in the anterior lobes with unchanged α -waves in the temporal and occipital lobes. 7 patients exhibited a dysrhythmic EEG in all leads. In 2 patients, the bioelectric desynchronization was manifested as a “flat” EEG. Low-amplitude, slow-rhythm activity prevailed in one patient. These features of cortical bioelectric activity are most likely caused by the cortical waves being malaffected by the nonspecific subcortical and truncal structures of the brain, reflecting a dysfunction in these structures (L. P. Latash).

Chronaximetry in 25 patients revealed an increased rheobase in cutaneous receptors of the fingers and an increased chronaxy of the sensory and flexor motor nerves in the extremities. A significant segment of the patient population exhibited disturbances in the chronaxy ratio within antagonistic muscle pairs and their respective motor nerves. In particular, nearly-equal chronaxy was recorded for finger flexors and extensors (9 patients), radial and median nerves (9 patients), and tibial and fibular nerves (11 patients). Typically, chronaxy values were equalized due to a chronaxy increase in the flexors to the level of the extensors. 5 patients exhibited a strong chronaxy imbalance with antagonistic pairs. These chronaximetry data reveal the changes in cutaneous receptors of the extremities and disturbed subordination of the neuromuscular apparatus to the nervous system which occur in many fluorosis patients.

The above results of neurological and neurophysiological examinations of fluorosis patients demonstrate that the neurotoxic effect of fluorine compounds manifests as a number of clinical and clinico-physiological symptoms of functional disorders of the central nervous system. Despite not being specific to fluorosis, these symptoms should nevertheless be taken into consideration along with

other clinical, laboratory, and hygienic data for the purpose of establishing a diagnosis of fluorosis, assessing the severity of the pathological process, and providing patient treatment.

Conclusions. 1. The clinical picture of occupational fluorosis characteristically includes the neurological symptomatology in the form of vascular autonomic dysfunction syndrome or asthenic autonomic syndrome combined with polyneuritis-type disorders.

2. According to the data of clinical neurophysiological examinations, occupational fluorosis patients frequently exhibit higher nervous activity disturbances and dysfunctions of nonspecific subcortical and truncal structures of the brain.

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