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Acute Fluoride Poisoning

Ostre zatrucie związkami fluoru

Utilizing fluoride compounds in dentistry has been a significant milestone in dental caries prevention and treatment. Nowadays, using filling materials without fluoride content is considered malpractice, and fluoride prophylaxis, especially exogenous one, has become an indispensable element of hard dental tissues care. However, one must not forget that, despite their huge medical potential, fluoride compounds may cause very serious or even fatal acute poisoning, as well as teeth and bones fluorosis tantamount to chronic poisoning.

Most cases of acute fluoride poisoning results from accidental consumption of a substance containing fluoride, professional exposure to fluoride, and in some cases the poisoning may be the result of a suicide attempt [5,10,11,13,14]. It should be stressed that there also occurred cases of mass fluoride poisoning caused for instance by consumption of roach poison powder accidently added to scrambled eggs instead of powdered milk or after consumption of drinking water in which, for unexplained reasons, the concentration of fluoride was on the level of 150 ppm [8,12].

Several cases of child mortality have also been described in literature. These deaths were the consequence of consumption of fluoride containing dental caries prevention agents. Dukes described a case of a 27-month-old boy who died after swallowing a small number of fluoride tablets (the exact number of tablets is not known but it was not higher than 100). The surprising aspect of this case is that death occurred 5 days after the poisoning [6]. Another example is death of a 3-year old boy who died 3 hours after swallowing mouth rinse containing 4% stannous fluoride [4]. While examining the amount of fluoride compounds swallowed by 7 and 11-years old children after brushing teeth using gel containing 1.25% F, Borysewicz–Lewicka et al. found out that, although it is within the acceptable limits, it may be twice higher than the acceptable level of daily fluoride consumption combined with diet and rinses [3].

Data concerning fatal dose of fluoride differ among authors. At the beginning of 1940s it was considered to be 100 mg F/kg body weight. At the end of the 20th century and the beginning of the 21st century the views have changed. On the basis of scientific data and reports concerning child mortality as the result of consumption of fluoride containing substances, Whitford concluded that there is a high probability of child's death in case of a dose of 15 mg F/kg body weight. One should

also keep in mind that even a dose as low as 5 mg F/kg body weight is treated as probably toxic and may constitute immense risk for child's life warranting immediate therapeutic intervention [18]. Currently, the minimum toxic dose is considered to be 1.8 mg F/kg body weight. In case of providing fluoride compounds from various sources, a single dose should not exceed 0.1-0.8 mg F/kg body weight. Other authors claim that safely tolerated dose is within a range of 8-18 mg/kg body weight [quoted after 17]. According to the calculation performed by Szczepańska and Pawłowska, in case of a child weighing 19 kg, swallowing 7.6 g of gel containing 12.5 mg fluoride compounds per 1 g gel may result in acute poisoning. For a person weighing 50 kg, the toxic dose is 20 g. In case of fluid containing 10000 ppm of fluoride, symptoms of acute poisoning in a child weighing 19 kg may appear after consumption of 9.5 ml of the fluid, and for a person weighing 50 kg the toxic amount is 25 ml [17].

Pathological mechanism of fluoride compounds toxicity is, to a certain degree, based on the formation of insoluble calcium fluoride, which is the consequence of high affinity of these compounds to calcium ions. Thus, there occurs a significant decrease of calcium level in blood and a simultaneous increase of potassium level. Proudfoot reports that, in case of poisoning with fluoroacetate, the compound combines with coenzyme A (CoA-SH) to form fluoroacetyl CoA, which can substitute for acetyl CoA in the tricarboxylic acid cycle and reacts with citrate synthase to produce fluorocitrate, a metabolite which then binds very tightly to aconitase, thereby halting the cycle [quoted after 14].

It is believed that one of the predictive factors of acute fluoride poisoning is the peak value of fluoride concentration in blood serum. Symptoms of acute fluoride poisoning depend on the dose and substance pH but also on the mode of administration, fluoride compounds concentration, patient's age, acid-base balance and the degree of absorption and climate. It is also believed that highly soluble compounds are more dangerous than poorly soluble substances [7,10]. In most cases, the symptoms of acute fluoride poisoning occur shortly after poisonous substance consumption. Hodge distinguishes four groups of events occurring with acute fluoride poisoning: 1 – enzymatic processes inhibition; 2 – calcium complexes formation; 3 – shock; 4 – organ damage [9].

Initially patients suffer from nausea (hematemesis may even occur in some patients), then a strong, disseminated pain within the abdomen develops. These symptoms may be accompanied by various unspecific effects, such as persistent salivation, lacrimation, nose discharge, fatigue, cyanosis, moist and cold skin, dyspnea, diarrhea and headache. After entering the organism, fluoride compounds quickly cause hypocalcaemia which results in tetanic convulsions, heart rhythm disorders or ventricular fibrillation as well as mucous membranes bleeding as a consequence of homeostasis disorders. Increase of blood serum potassium level is also observed, which, in turn, has a generalized toxic effect on the proper function of mucous membranes. Experimental studies performed on animals showed that fluorides cause acute inflammation of mucous membranes, cell atrophy, necrosis, cell layer thinning as well as intramucosal and submucosal edema. In course of poisoning progression the symptoms become more severe, contractions of strong extremities or even tetany may occur. Blood pressure decreases, pulse becomes thread or even impalpable. Arrhythmia, respiratory and metabolic acidosis and finally respiratory depression and heart arrest develop. In the course of acute fluoride poisoning, mixed acidosis, both respiratory and metabolic, worsens, the pH gradients between environments separated by mucous membranes decrease, which, in turn, leads to

fluorides or rather hydrofluoric acid entering the cells and disrupting their physiological functions. Due to the existence of this type of mechanism in acute fluoride poisoning, it is necessary to treat acidosis or even incite some degree of alkalosis. Most frequently the occurrence of large degree of disorientation and coma precede death. Taking into account that fluoride poisoning may be a life threatening condition, treatment must be initiated immediately. The treatment goal is above all elimination of the toxic fluoride compounds from the organism as well as reduction of the degree of their absorption from the alimentary tract [2,7,10,15,17,18].

Except for using calcium salts, the treatment of fluoride poisoning is non-specific. The first step in the therapeutic procedure is provoking vomiting by administering emetics, although, in most cases of fluoride poisoning, vomiting occurs spontaneously. After inducing vomiting, calcium salts are administered in the form of 1% calcium chloride or calcium gluconate. Drinking od milk is also advised. Inducing vomiting is counter-indicated in case of poisoning with hydrofluoric acid or silicofluoric acid as well as in case of patient's passing out, occurrence of convulsions and undetectable pharyngeal reflex. In the latter cases, patients should be intubated and stomach rinsing should be performed using substances containing calcium salts or activated carbon. Following these emergency procedures the patient must be hospitalized. In the hospital, analgesic and shock-controlling treatment should be initiated. The patient's condition may also require oxygen therapy and assisted breathing. Sometimes hemodialysis and electrocardioversion are necessary. Patient's monitoring and appropriate therapy should be continued until the results of biochemical tests are within the reference range and the vital activities and mental alertness go back to normal [1,2,7,10,15,18].

Describing acute fluoride poisoning one cannot omit volatile fluorides (the acceptable concentration of fluoride compounds in the air is 0.05 mg/m3). They can cause cough, dyspnea and shivers lasting for 24 hours after exposure. In some cases the symptoms include breath sounds and cyanosis, and in some other, there is a delayed pulmonary edema. In case of fluoride contact with skin, especially in the form of hydrofluoride (the only form of fluoride absorbed through skin), there occur chemical burnings, abscesses and sometimes necrosis. The result of contact between fluoride compounds and moist mucous membrane surface is the formation of hydrofluoride. The acid causes mucous membranes ulceration, hemoptysis and sometimes symptoms and discomfort from the gastrointestinal tract [1,2,5,7,15,16,18].

Taking into account that acute fluoride poisoning is a life-threatening condition, it is absolutely essential to prevent children from any unsupervised contact with fluoride preparations. Parents and guardians should be educated and hygienists and doctors should be convinced about the necessity of informing patients about fluoride compounds toxicity. At the same time, the manufacturers of fluoride-containing substances should use special protections making it impossible for children to get access to the preparations (child-resistant packing). Summing up, on the one hand, fluoride compounds help keep teeth healthy but, on the other hand, their improper or excessive usage may be significantly dangerous for human organism.

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SUMMARY

Utilizing fluoride compounds in dentistry has been a significant milestone in dental caries prevention and treatment. However, one must not forget that, despite their huge medical potential, fluoride compounds may cause very serious or even fatal acute poisoning, as well as teeth and bones fluorosis tantamount to chronic poisoning. In the article the causes, manifestations, methods of treatment of the acute fluoride poisoning were presented. It was stated in conclusions that taking into account that acute fluoride poisoning is a life-threatening condition, it is absolutely essential to prevent children from any unsupervised contact with fluoride preparations. Parents and guardians should be educated and hygienists and doctors should be convinced about the necessity of informing patients about fluoride compounds toxicity.

Keywords: fluoride, acute fluoride poisoning

STRESZCZENIE

Zastosowanie związków fluoru w stomatologii stanowi milowy krok w zapobieganiu i leczeniu choroby próchnicowej. Należy jednak pamiętać, iż związki fluoru mimo tego, iż charakteryzują się niezwykłym potencjałem prozdrowotnym mogą wywoływać, prowadzące nawet do zgonu ostre zatrucia, jak również fluorozę zębów i kości, a zatem zatrucie przewlekłe. W artykule przedstawiono przyczyny, objawy i metody leczenia ostrego zatrucia związkami fluoru. We wnioskach stwierdzono, że Biorąc pod uwagę fakt, iż ostre zatrucie związkami fluoru jest stanem zagrażającym życiu konieczne jest uniemożliwianie dzieciom samodzielnego, nie nadzorowanego kontaktu z preparatami zawierającymi fluorki. Wskazana jest edukacja prozdrowotna rodziców i opiekunów, ale także przekonanie higienistek i lekarzy stomatologów o konieczności informowania pacjentów o toksyczności związków fluoru.

Słowa kluczowe: związki fluoru, ostre zatrucie związkami fluoru