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ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

Медицинские новости Грузии

საქართველოს სამედიცინო სიახლენი

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თანამშრომლობითა და მისი პატრონაჟით

ЕЖЕМЕСЯЧНЫЙ НАУЧНЫЙ ЖУРНАЛ

ТБИЛИСИ - НЬЮ-ЙОРК

GMN: Georgian Medical News is peer-reviewed, published monthly journal committed to promoting the science and art of medicine and the betterment of public health, published by the GMN Editorial Board and The International Academy of Sciences, Education, Industry and Arts (U.S.A.) since 1994. **GMN** carries original scientific articles on medicine, biology and pharmacy, which are of experimental, theoretical and practical character; publishes original research, reviews, commentaries, editorials, essays, medical news, and correspondence in English and Russian.

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GMN: Медицинские новости Грузии - ежемесячный рецензируемый научный журнал, издаётся Редакционной коллегией и Международной академией наук, образования, искусств и естествознания (IASEIA) США с 1994 года на русском и английском языках в целях поддержки медицинской науки и улучшения здравоохранения. В журнале публикуются оригинальные научные статьи в области медицины, биологии и фармации, статьи обзорного характера, научные сообщения, новости медицины и здравоохранения.

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GMN: Georgian Medical News – საქართველოს სამედიცინო სიახლენი – არის ყოველთვიური სამეცნიერო სამედიცინო რეცენზირებადი ჟურნალი, გამოიცემა 1994 წლიდან, წარმოადგენს სარედაქციო კოლეგიისა და აშშ-ის მეცნიერების, განათლების, ინდუსტრიის, ხელოვნებისა და ბუნებისმეტყველების საერთაშორისო აკადემიის ერთობლივ გამოცემას. GMN-ში რუსულ და ინგლისურ ენებზე ქვეყნდება ექსპერიმენტული, თეორიული და პრაქტიკული ხასიათის ორიგინალური სამეცნიერო სტატიები მედიცინის, ბიოლოგიისა და ფარმაციის სფეროში, მიმოხილვითი ხასიათის სტატიები.

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При представлении в печать научных экспериментальных работ авторы должны указывать вид и количество экспериментальных животных, применявшиеся методы обезболивания и усыпления (в ходе острых опытов).

4. К статье должны быть приложены краткое (на полстраницы) резюме на английском, русском и грузинском языках (включающее следующие разделы: цель исследования, материал и методы, результаты и заключение) и список ключевых слов (key words).

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2. Size of the article, including index and resume in English, Russian and Georgian languages must be at least 10 pages and not exceed the limit of 20 pages of typed or computer-printed text.

3. Submitted material must include a coverage of a topical subject, research methods, results, and review.

Authors of the scientific-research works must indicate the number of experimental biological species drawn in, list the employed methods of anesthetization and soporific means used during acute tests.

4. Articles must have a short (half page) abstract in English, Russian and Georgian (including the following sections: aim of study, material and methods, results and conclusions) and a list of key words.

5. Tables must be presented in an original typed or computer-printed form, instead of a photocopied version. **Numbers, totals, percentile data on the tables must coincide with those in the texts of the articles.** Tables and graphs must be headed.

6. Photographs are required to be contrasted and must be submitted with doubles. Please number each photograph with a pencil on its back, indicate author's name, title of the article (short version), and mark out its top and bottom parts. Drawings must be accurate, drafts and diagrams drawn in Indian ink (or black ink). Photocopies of the X-ray photographs must be presented in a positive image in **tiff format**.

Accurately numbered subtitles for each illustration must be listed on a separate sheet of paper. In the subtitles for the microphotographs please indicate the ocular and objective lens magnification power, method of coloring or impregnation of the microscopic sections (preparations).

7. Please indicate last names, first and middle initials of the native authors, present names and initials of the foreign authors in the transcription of the original language, enclose in parenthesis corresponding number under which the author is listed in the reference materials.

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In GMN style for each work cited in the text, a bibliographic reference is given, and this is located at the end of the article under the title "References". All references cited in the text must be listed. The list of references should be arranged alphabetically and then numbered. References are numbered in the text [numbers in square brackets] and in the reference list and numbers are repeated throughout the text as needed. The bibliographic description is given in the language of publication (citations in Georgian script are followed by Cyrillic and Latin).

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ავტორთა საყურადღებოდ!

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1. სტატია უნდა წარმოადგინოთ 2 ცალად, რუსულ ან ინგლისურ ენებზე,დაბეჭდილი სტანდარტული ფურცლის 1 გვერდზე, 3 სმ სიგანის მარცხენა ველისა და სტრიქონებს შორის 1,5 ინტერვალის დაცვით. გამოყენებული კომპიუტერული შრიფტი რუსულ და ინგლისურენოვან ტექსტებში - **Times New Roman (Кириллица)**, ხოლო ქართულენოვან ტექსტში საჭიროა გამოვიყენოთ **AcadNusx.** შრიფტის ზომა – 12. სტატიას თან უნდა ახლდეს CD სტატიით.

2. სტატიის მოცულობა არ უნდა შეადგენდეს 10 გვერდზე ნაკლებს და 20 გვერდზე მეტს ლიტერატურის სიის და რეზიუმეების (ინგლისურ, რუსულ და ქართულ ენებზე) ჩათვლით.

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6. ფოტოსურათები უნდა იყოს კონტრასტული; სურათები, ნახაზები, დიაგრამები - დასათაურებული, დანომრილი და სათანადო ადგილას ჩასმული. რენტგენოგრამების ფოტოასლები წარმოადგინეთ პოზიტიური გამოსახულებით **tiff** ფორმატში. მიკროფოტოსურათების წარწერებში საჭიროა მიუთითოთ ოკულარის ან ობიექტივის საშუალებით გადიდების ხარისხი, ანათალების შეღებვის ან იმპრეგნაციის მეთოდი და აღნიშნოთ სურათის ზედა და ქვედა ნაწილები.

7. სამამულო ავტორების გვარები სტატიაში აღინიშნება ინიციალების თანდართვით, უცხოურისა – უცხოური ტრანსკრიპციით.

8. სტატიას თან უნდა ახლდეს ავტორის მიერ გამოყენებული სამამულო და უცხოური შრომების ბიბლიოგრაფიული სია (ბოლო 5-8 წლის სიღრმით). ანბანური წყობით წარმოდგენილ ბიბლიოგრაფიულ სიაში მიუთითეთ ჯერ სამამულო, შემდეგ უცხოელი ავტორები (გვარი, ინიციალები, სტატიის სათაური, ჟურნალის დასახელება, გამოცემის ადგილი, წელი, ჟურნალის №, პირველი და ბოლო გვერდები). მონოგრაფიის შემთხვევაში მიუთითეთ გამოცემის წელი, ადგილი და გვერდების საერთო რაოდენობა. ტექსტში კვადრატულ ფჩხილებში უნდა მიუთითოთ ავტორის შესაბამისი N ლიტერატურის სიის მიხედვით. მიზანშეწონილია, რომ ციტირებული წყაროების უმეტესი ნაწილი იყოს 5-6 წლის სიღრმის.

9. სტატიას თან უნდა ახლდეს: ა) დაწესებულების ან სამეცნიერო ხელმძღვანელის წარდგინება, დამოწმებული ხელმოწერითა და ბეჭდით; ბ) დარგის სპეციალისტის დამოწმებული რეცენზია, რომელშიც მითითებული იქნება საკითხის აქტუალობა, მასალის საკმაობა, მეთოდის სანდოობა, შედეგების სამეცნიერო-პრაქტიკული მნიშვნელობა.

10. სტატიის ბოლოს საჭიროა ყველა ავტორის ხელმოწერა, რომელთა რაოდენობა არ უნდა აღემატებოდეს 5-ს.

11. რედაქცია იტოვებს უფლებას შეასწოროს სტატია. ტექსტზე მუშაობა და შეჯერება ხდება საავტორო ორიგინალის მიხედვით.

12. დაუშვებელია რედაქციაში ისეთი სტატიის წარდგენა, რომელიც დასაბეჭდად წარდგენილი იყო სხვა რედაქციაში ან გამოქვეყნებული იყო სხვა გამოცემებში.

აღნიშნული წესების დარღვევის შემთხვევაში სტატიები არ განიხილება.

Содержание:

FATAL ACUTE ALIMINIUM PHOSPHIDE POISONING - CASE REPORT AND LITERATURE REVIEW WITH REFERENCE TO CURRENT TREATMENT PROTOCOLS AND OUTCOME

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Aluminum phosphide (AlP) is a highly toxic fumigant pesticide used to kill rodents and insects, commonly used for grain preservation. Its use and abuse is growing, especially in developing countries because of its low price and market availability, but also because of its properties, which are considered almost ideal [1]. After deliberate oral ingestion AlP reacts with hydrochloric acid in the stomach and releases phosphine gas (PH_3) which is rapidly absorbed from the gastrointestinal tract and inhibits mitochondrial cytochrome oxidase and the supply of cells with oxygen, leading to circulatory failure [2]. The number of suicide attempts with AlP has been progressively increasing, especially in India and other eastern countries. Mortality after deliberate AlP poisoning is particularly high and 95% of patients die within the first 24 hours of poisoning [3]. Mortality rate can reach more than 60% even in experienced and well-equipped hospitals [1]. Due to its high human toxicity and mortality, it is banned in many countries. Although AlP is on the list of allowed pesticides in our country and has been on the market since 2009 under the trademark Quickphos Pellets 56 GE (according to data from the phytosanitary information system) so far no AlP poisoning has been reported in our poison control center. We report the first case treated in our institution as severe intentional poisoning with AlP, purchased in a neighboring country, which unfortunately had a fatal outcome. Mechanisms of toxicity, prognostic factors for lethal outcome and therapeutic options are also targeted.

Case report. A 35-year-old woman with a previous history of epilepsy was admitted to the University Clinic for Toxicology due to a suicidal attempt with ingestion of one 3-gram phostoxin tablet (containing 56% of AlP) two hours earlier. Vomiting and abdominal pain were the first symptoms. On admission she was conscious and oriented, with 16 breaths per minute, pale skin, blood pressure of 160/80 mmHg, pulse of 77 beats/min, oxygen saturation of 97% on ambient air, and epigastric pain sensitivity. The remaining status by systems was without significant changes. The ECG showed sinus rhythm with inverse T-waves in D3, AVF and left precordial leads. Early laboratory analyses showed leukocytosis (15.3 x 10^9/L), prolonged prothrombin time of 18.4 sec, and normal degradation products and enzyme status. Despite early medical treatment with single dose activated charcoal and isotonic crystalloid fluids, arterial blood pressure started to drop. So, bicarbonates, MgSo4, H2 blockers, electrolytes, dopamine, and hydrocortisone were added. The patient's condition worsened with an increase in respiratory rate and a drop in blood pressure. She was transferred to the intensive care unit (ICU), still conscious, oriented, contactable, with blood pressure 80/40 mmHg and pulse rate of 120/min. A chest X-ray did not show any abnormalities. Laboratory findings showed signs of hepatic lesion, rhabdomyolysis and renal failure (AST 2267.42 U/L, ALT 2102.26 U/L, CPK 1334.81U/L, blood urea nitrogen 23.03 mmol/L, creatinine 211.9 μmol/L, total protein 39 g/L, albumin 26.19 g/L). Arterial blood gas analyses showed metabolic acidosis with marked base excess (pO2 9.6 kPa, pCO2 4.14 kPa, pH 7.15, bicarbonate 11 mmol/L, BE -15). She was intubated and placed on mechanical ventilation for the next 24 hours, after which she was extubated and placed on an oxygen mask. An arterial line was set up for invasive blood pressure monitoring. Hemodynamic instability was treated with isotonic solutions and inotropic support (norepinephrine, dopamine and adrenaline). The treatment also included: antibiotics, analgesics, antithrombotic therapy, freshly frozen plasma, and parenteral 8.4% sodium bicarbonate adjusted for gas analysis results and acid-base status. Dopamine was replaced with phenylephrine, and furosemide was added. One day later, due to respiratory evasion, she was again intubated and placed on mechanical ventilation with sedation. Despite intensive treatment, the patient was still hemodynamically unstable, with severe metabolic acidosis and anuria. On the third day, cardiopulmonary insufficiency occurred several times. The patient responded well to parenteral treatment (atropine sulphate 2 mg, adrenaline 2 mg, digoxin 0.25 mg, furosemide 40 mg, sodium bicarbonate 150 ml, calcium gluconate 10 ml) and other cardiopulmonary resuscitation measures the first two times. The third time, cardiopulmonary resuscitation measures did not work and the patient had a lethal outcome.

Aluminum phosphide characteristics. AlP is a solid fumigant pesticide, effective in destroying harmful insects and rodents that damage the grain. It is cheap and easy to use, and hence widely used in agriculture as well as for other non-agricultural purposes. Its properties make it almost ideal for use: it does not affect the viability of the seeds, it does not contain toxic residues and leaves little allowed residue on the grains [1]. It can be found under various trade names (Celphos, Phosfume, Phostoxin, Quickphos and others) most commonly formulated as tablets, pellet, granules or powder [4]. AlP is not toxic per se, but active pesticide component-the toxic gas phosphine PH_3, which is formed in contact with water, acids or moisture, is responsible for its toxicity [5]. It is a colorless highly toxic, extremely flammable gas with a specific garlic odor and has a short half-life of 5-24 hours.

Mechanisms of toxicity. Although the exact mechanism of action of phosphine is not well known, it is thought to inhibit mitochondrial cytochrome oxidase and oxidative phosphorylation [2]. This ability to inhibit cytochrome c oxidase is more pronounced in vitro, whereas in vivo phosphine shows much lower activity. This results in a 70% reduction in oxidative respiration and a decrease in mitochondrial membrane potential [6,7].

Other mechanisms are also involved in the occurrence of the disruption of cellular respiration. The production of highly reactive hydroxyl radicals in the presence of phosphine along with the inhibition of the enzymes catalase and peroxidase lead to subsequent cell damage through lipid peroxidation [7].

Clinical manifestations. Clinical signs and symptoms occur shortly after ingestion of AlP. The main characteristic of AlP poisoning is the presence of systemic toxicity, but the first and most common signs are vomiting and abdominal pain. Cardiovascular involvement is due to the direct toxicity of phosphine to myocytes and adrenal glands, as well as fluid loss [8]. Marked hypotension, tachycardia, tachypnea, several ECG changes, metabolic acidosis, acute renal failure and irreversible shock are often described [8,3,7]. Although rare, ECG changes suggestive of myocardial infarction have also been reported in association with acute AlP poisoning [9,10]. Pulmonary edema, either cardiogenic or non-cardiogenic, may also be present [7,8].

Other less common manifestations of ALP poisoning are: hepatic necrosis, disseminated intravascular coagulation and acute tubular necrosis. There is conflicting evidence for magnesium levels disorder [7]. Phosphines and phosphides also have direct corrosive effects on tissues [11]. Although rare, local thermal injuries such as gastrointestinal ulcers, hemorrhages, and external skin burns have been reported in patients with lethal outcome, presumably caused by the use of potassium permanganate [12]. Such changes are internal and difficult to observe, therefore, they could often remain unrecognized and undiagnosed, contributing to the unfavorable outcome of AlP poisoning [13].

Treatment. Since there is no antidote for AlP poisoning, therapy mainly consists of supportive measures which should start as early as possible. Treatment protocols also vary from center to center. Current protocols recommend the use of potassium permanganate solution for gastric lavage because it oxidizes phosphine to non-toxic phosphate, preventing further absorption, which is followed by application of activated charcoal through a nasogastric tube. In order to reduce stomach pain, acidity and further release of phosphine, the use of antacids, proton pump inhibitors and H2 blockers is also recommended [3,14]. According to recent and contrary to previous studies, the use of activated charcoal and potassium permanganate is useless and even harmful to AlP poisoning. Some authors believe that the use of water-based solutions such as activated charcoal and potassium permanganate leads to greater release of phosphine-gas from the AlP [15]. In addition, AlP has a molecular weight of only 58 Daltons, which is much less than the absorption power of activated charcoal [16]. Cases of hemolysis and methemoglobinemia have also been reported in patients treated with potassium permanganate, which is considered to be a strong oxidizing agent [17]. Newer treatment protocols prefer the use of vegetable oils and liquid paraffin rather than water-soluble substances, since they prevent greater fumigation of phosphine and provide better motility of the gastrointestinal tract. This has been confirmed by some in vitro studies, as well as some case reports showing reduced acute AlP toxicity following the use of vegetable oils [16].

The results of experimental and clinical studies suggest the use of many other potential antidotes such as: glutathione, N-acetylcysteine, vitamins C and E, boric acid, magnesium sulfate, melatonin, L-carnitine and others, which are thought to reduce harmful oxidative properties of AlP, but for most of them additional assessment is required [18].

Management of severe hypotension is still a current issue for clinicians as it is a major factor affecting the severity and outcome of poisoning. A retrospective study conducted in Ethiopia reported a lower mortality rate of about 31% in hypotensive patients treated according to a protocol containing isotonic crystalloid solutions, dopamine, magnesium sulfate, hydrocortisone, and calcium gluconate in addition to gastric lavage [19]. Other authors believe that the usual treatment with high doses of crystalloid isotonic solutions gives unsatisfactory results. Vasoactive amines (dopamine, norepinephrine and phenylephrine) also give limited success in shock resuscitation [20]. According to Marashi et al., the poor response to massive crystalloid administration and vasoactive substances is due to insufficiency of the vessel wall integrity and their increased permeability. They believe that congestion of the organs and fluid transduction in the serous cavities is due to increased permeability and is not associated with heart failure [21]. Having this in mind, they suggest the use of high-molecular-weight colloidal solutions as volume expanders and resuscitation liquids. With this treatment they have successfully treated a patient with acute AlP poisoning [22]. Corticosteroids are still used in the treatment of AlP poisoning to reduce the doses of vasoactive substances, to potentiate the body's response to endogenous and exogenous catecholamines and to prevent the development of acute respiratory distress syndrome [23]. In order to find a solution for hemodynamic instability in patients with AlP intoxication, a single-centric randomized controlled trial was performed for the use of intralipid emulsion as an adjuvant therapy. Half of the patients received a continuous 20% intralipid emulsion (ILE) along with supportive treatment, and the rest did not. The benefits of ILE have been based on the known liposolubility of phosphine (phosphorus trihydride) with the assumption that ILE can counteract its toxic effects. The results of the study showed that intralipid emulsion was an effective and safe therapy, but the differences in mortality and mean systolic pressure between the groups were insignificant, while the need for intubation and mechanical ventilation were significantly smaller, confirming its therapeutic effect [24,25].

The second major problem faced by clinical toxicologists *is the management of severe metabolic acidosis.* Many authors use intravenous sodium bicarbonate to overcome acidosis. Some of those who corrected acidosis by intravenous administration of sodium bicarbonate, guided by basal excess, did not find a significant difference in pH and serum bicarbonate values between surviving and non-surviving patients [26]. There are also authors who believe that intravenous sodium bicarbonate cannot resolve metabolic acidosis, as $HCO3^-$ remains in the extracellular compartment, while intracellular acidosis worsens. According to them, generalized hypoperfusion is a major cause of severe metabolic acidosis and suggest the focus of treatment to be on management of hypotension with limited use of intravenous bicarbonate only in cases with pH less than 7 [27].

There are also conflicting opinions regarding cardiac toxicity. Some authors consider myocardial injury to be the most likely mechanism for cardiovascular toxicity and suggest digoxin, an intra-aortic balloon pump, and trimetazidine to support cardiac function [28], while others consider tissue hypoperfusion and intracellular acidosis responsible for reduced cardiac function. More recently, the application of extracorporeal membrane oxygenation (ECMO) has been considered the most promising technique for providing temporary cardiorespiratory support [29, 30]. Some authors believe that high-dose insulin improves carbohydrate energy utilization and may improve myocardial contractility. They report that four out of five patients treated with this hyperinsulinemia-euglycemic approach survived [31].

Another important part of treatment is the *tracking and treatment of electrolyte imbalances*, which may be responsible for cardiac dysfunction. Hypomagnesemia is often observed in patients acutely intoxicated with AlP, especially in those with hypotension and arrhythmia [32], but the therapeutic effects of magnesium sulfate supplementation are contradictory. Some studies have reported significantly fewer complications and reduced mortality in patients substituted for magnesium sulfate [33,34]. Contrary to this, in another study, no hypomagnesemia was observed, nor a significant difference in mortality between patients treated with and without magnesium sulfate was detected [35]. Therefore, most authors believe that magnesium sulfate should not be administered routinely, but only in patients with proven low levels [36]. Ca gluconate due to its mild membrane-stabilizing effects is given together with magnesium sulfate for the treatment of ECG changes in patients acutely poisoned with ALP [33,36]. A wide range of changes in potassium and calcium levels can be expected. Their correction is strongly recommended together with the treatment of acidosis (for pH <7) that may contribute to overcoming conduction disturbances [16].

Prognostic markers. It is generally accepted that mortality depends on the ingested dose, the severity of the poisoning, the duration and irreversibility of the shock despite the application of resuscitation measures. Although it is known that a dose of 150-500 mg AlP is potentially lethal and that one tablet is sufficient for fatal outcome [37], a rare case of survival after AlP poisoning with the same dose, the same formulation (Phostoxin), the same time to hospitalization and the same supportive treatment as ours has been described [38].

Numerous clinical and laboratory findings may serve as predictors of mortality in patients with AlP poisoning. According to the results of one Iranian study that used multiple regression analysis, systolic blood pressure, Glasgow coma score, urinary output and serum bicarbonate had the highest predictive value [39]. Variables such as systolic blood pressure, Glasgow coma score, leukocyte number, glucose levels, urea, blood pH, electrocardiogram changes, and number of ingested AlP tablets have been shown to be useful prognostic parameters in another study [40]. But the severity of the poisoning also depends on the form, i.e., the activity of the consumed preparation that contains AlP. Tablet consumption is associated with severe metabolic acidosis and high mortality, because they contain a fresh and active compound. Broken tablets or granular forms contain less active compound and therefore cause milder hypotension, milder metabolic acidosis and correspondingly lower mortality. Powdered forms of tablets do not cause systemic effects because they are inactive [3]. Arrhythmia is the most common cause of death in the first 24 hours, while in the following days death is usually due to the presence of shock, acidosis, acute respiratory distress syndrome and arrhythmia [32].

In this paper we presented the first case of AlP poisoning treated in our Clinic after ingestion of a potentially fatal dose of one tablet of phostoxin (containing 3 grams of AlP) by a young woman. The diagnosis was made on the basis of medical history and clinical presentation, while a confirmation test with silver nitrate was not available at our institution. The patient received a single dose of about 50 grams of medical charcoal orally, as it is recommended up to 4 hours after poisoning with solid preparations of AlP in toxic doses, but in our case it proved to be insufficient to absorb this toxic substance. According to some authors, this may be due to the low molecular weight of AlP [16]. Gastric lavage was not performed because the patient vomited multiple times and because of the possibility of increased AlP disintegration and increased toxicity with solutions composed of water [32]. Since we had no experience with such poisonings, neither potassium permanganate nor liquid paraffin was prescribed for decontamination. Although stable on admission, she soon became hypotensive and did not respond to standard supportive treatment, most likely due to the severe metabolic acidosis she developed. After she was transferred to the intensive care unit, she developed signs of multiorgan involvement, pulmonary edema, and respiratory failure despite all resuscitation measures. Signs of severe poisoning and prognostic factors for lethal outcome were present in this patient soon after admission to the hospital, and the lethal outcome occurred on the fourth day. The failure of conventional treatment protocols in our and in many other cases, and, in general, the high mortality from deliberate poisoning with AlP preparations were our motive to conduct a literature review and discover other therapeutic modalities that would yield better results.

It could be concluded that after many years each piece of the puzzle called acute AlP poisoning remains unsolved, including the main mechanism of toxicity. It is obvious that the management of acute oral poisoning with AlP is a challenging problem that clinicians still encounter. New knowledge is needed to answer the exact toxokinetic and toxodynamic mechanisms, as well as additional randomized trials on the effectiveness of newer therapeutic modalities. In the absence of antidote and consensus on treatment, until newer and more successful therapeutic strategies are developed, the key to treatment is rapid decontamination and prompt resuscitation and initiation of supportive measures. In the meantime, to reduce mortality in general, it is advisable for governments to restrict the easy availability of this pesticide.

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SUMMARY

FATAL ACUTE ALIMINIUM PHOSPHIDE POISONING - CASE REPORT AND LITERATURE REVIEW WITH REFERENCE TO CURRENT TREATMENT PROTOCOLS AND OUTCOME

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Aluminum phosphide (AlP) has been known for more than 80 years as an effective pesticide for grain protection, but also as a suicidal agent used for human self-poisoning. Phosphine gas released in contact with stomach acid after oral ingestion of AlP is responsible for its toxicity. The poison affects all systems, so the mortality rate is quite high, especially after deliberate ingestions. We report the first case of severe AlP poisoning seen in our institution with a fatal outcome and furthermore, we present literature review on existing and newer treatment options.

A 35-year-old woman with a history of epilepsy was admitted to the University Clinic for Toxicology in Skopje two hours after deliberate ingestion of one tablet of phostoxin (AlP). The first signs of poisoning were vomiting and abdominal pain, leukocytosis, prolonged PT, as well as inverted T waves in D3, AVF and left precordial leads on ECG. After developing respiratory failure and hypotension she was transferred to the intensive care unit (ICU). Her blood pressure was 80/40 mmHg, pulse rate 120/min. Laboratory findings showed signs of hepatic lesion, rhabdomyolysis and renal failure (AST 2267.42 U/L, ALT 2102.26 U/L, CPK 1334.81U/L, blood urea nitrogen 23.03 mmol/L, creatinine 211.9 μmol/L). Arterial blood gas analyses showed metabolic acidosis (pO_2 9.6 kPa, pCO_2 4.14 kPa, pH 7.15, bicarbonate 11 mmol/L, BE -15). The patient was placed on mechanical ventilation. Despite fluid supplementation, intensive therapy and inotropic support, hemodynamic instability worsened and cardiopulmonary resuscitation was performed three times. Un-

fortunately, the patient had a fatal outcome on the fourth day of intoxication.

Solid formulations of AlP are very toxic. One tablet of phostoxin containing 3 grams of AlP is sufficient for the progression of life-threatening symptoms and fatal outcome. In the absence of antidote and elucidated mechanisms of toxicity, the key to treatment is rapid decontamination and initiation of resuscitation measures.

Keywords: aluminum phosphide, phosphine, toxicity, treatment protocols, outcome.

РЕЗЮМЕ

СМЕРТЕЛЬНОЕ ОСТРОЕ ОТРАВЛЕНИЕ ФОСФИДОМ АЛЮМИНИЯ - ОТЧЕТ О СЛУЧАЕ И ОБЗОР ЛИТЕРАТУРЫ СО ССЫЛКОЙ НА ТЕКУЩИЕ ПРОТОКОЛЫ ЛЕЧЕНИЯ И РЕЗУЛЬТАТЫ

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Фосфид алюминия (AlP) известен не только как эффективный пестицид для защиты зерна, но и как способ самоубийства. Газообразный фосфин, выделяющийся при контакте с желудочной кислотой после перорального приема AlP, ответственен за его токсичность. Яд поражает все системы, поэтому смертность довольно высокая, особенно после приема внутрь. Сообщается о случае тяжелого отравления AlP со смертельным исходом; представлен обзор литературы и способы лечения.

35-летняя женщина с эпилепсией в анамнезе была госпитализирована в Университетскую клинику токсикологии в Скопье через два часа после приема одной таблетки фостоксина (AlP) с целью самоубийства. Первыми признаками отравления были рвота и боли в животе, лейкоцитоз, длительная ПТ, а также инвертированные волны Т в D3, AVF и левых прекордиальных отведениях на ЭКГ. После развития дыхательной недостаточности и гипотензии пациентку перевели в отделение интенсивной терапии. Давление - 80/40 мм рт.ст., частота пульса - 120 в минуту. Лабораторные данные показали признаки поражения печени, сопровождаемого рабдомиолизом и почечной недостаточностью (АСТ 2267,42 Ед/л, АЛТ 2102,26 Ед/л, КФК 1334,81 Ед/л, азот мочевины крови 23,03 ммоль/л, креатинин 211,9 мкмоль/л). Анализ газов артериальной крови показал метаболический ацидоз (PO_2 9,6 кПа, pCO_2 4,14 кПа, pH 7,15, бикарбонат 11 ммоль/л, BE - 15). Пациент переведен на искусственную вентиляцию легких. Сердечно-легочная реанимация проведена трижды. Несмотря на добавление жидкости, интенсивную терапию и инотропную поддержку, гемодинамическая нестабильность усилилась. К сожалению, у пациента на четвертый день интоксикации наступил летальный исход.

Твердые составы AlP весьма токсичны. Одной таблетки фостоксина, содержащей 3 грамма AlP, достаточно для прогрессирования опасных для жизни симптомов и летального исхода. В отсутствие противоядия и выясненных механизмов токсичности ключом к лечению является быстрая дезактивация и начало реанимационных мероприятий.

რეზიუმე

ალუმინის ფოსფიდით მწვავე სასიკვდილო მოწამვლა - შემთხვევა პრაქტიკიდან და ლიტერატურის მიმოხილვა მკურნალობის შედეგების გათვალისწინებით

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ალუმინის ფოსფიდი (AlP) ცნობილია როგორც არამარტო ეფექტური პესტიციდი მარცვლეულის დასაცავად, არამედ როგორც სუიციდის საშუალებაც. მის ტოქსიკურობას განსაზღვრავს გაზოვანი ფოსფინი, რომელიც გამოიყოფა კუჭის მჟავასთან AlP-ის კონტაქტის შედეგად მისი პერორალური მიღების შემდეგ. საწამლავი აზიანებს ყველა სისტემას, ამიტომაც, სიკვდილობა საკმაოდ მაღალია. აღწერილია AlP-ით მძიმე სასიკვდილო მოწამვლის შემთხვევა, მოცემულია ლიტერატურის მიმოხილვა და მკურნალობის საშუალებები.

35 წლის ქალი, ანამნეზში ეპილეფსიით, ჰოსპიტალიზებული იყო სკოპიეს ტოქსიკოლოგიის საუნივერსიტეტო კლინიკაში სუიციდის მიზნით ფოსტოქსინის (AlP) ერთი ტაბლეტის მიღებიდან ორი საათის შემდეგ. მოწამვლის პირველი ნიშნები იყო ღებინება და ტკივილი მუცელში, ლეიკოციტოზი, ხანგრძლივი პტ, ასევე, ინვერსირებული T ტალღები D3-ში, AVF-სა და მარცხენა პრეკარდიულ განხრებში ეკგ-ზე. სუნთქვის უკმარისობის და ჰიპოტენზიის განვითარების შემდეგ პაციენტი გადაყვანილი იყო ინტენსიური თერაპიის განყოფილებაში; არტერიული წნევა – 80/40 mmHg, პულსის სიხშირე წუთში – 120. ლაბორატორიულმა მონაცემებმა აჩვენა ღვიძლის დაზიანება, რაბდომიოლიზით და თირკმლის უკმარისობით (ასპარტატამინოტრანსფერაზა - 2267,42 ერთ/ლ, ალანინამინოტრანსფერაზა - 2102,26 ერთ/ლ, კრეატინკინაზა - 1334,81 ერთ/ლ, შარდოვანას აზოტი - 23,03 მმოლ/ლ, კრეატინინი - 211,9 მკმოლ/ლ). არტერიული სისხლის გაზების ანალიზმა აჩვენა მეტაბოლური აციდოზი (PO_2 - 9,6 კპ, pCO2 4,14 - კპ, pH 7.15, ბიკარბონატი - 11 მმოლ/ლ, BE -15). პაციენტი გადაყვანილი იყო ხელოვნურ ვენტილაციაზე. გულ-ფილტვის რეანიმაცია ჩატარდა სამჯერ. სითხის დამატების, ინტენსიური თერაპიის და ინოტროპული მხარდაჭერის მიუხედავად, ჰემოდინამიკური არასტაბილურობა გაძლიერდა. სამწუხაროდ, ინტოქსიკაციის მეოთხე დღეს დადგა ლეტალური გამოსავალი.

AlP-ის მყარი შემადგენლობა ძალიან ტოქსიკურია. ფოსტოქსინის ერთი ტაბლეტი, რომელიც შეიცავს AlP-ს 3 გრამს, საკმარისია სიცოცხლისათვის საშიში სიმპტომების პროგრესირებისა და ლეტალური გამოსავლისათვის. ანტიდოტის და ტოქსიკურობის დაზუსტებული მექანიზმების არარსებობის პირობებში მკურნალობის საშუალებას წარმოადგენს სწრაფი დეზაქტივაცია და რეანიმაციული ღონისძიებების დაწყება.