Following Accidental Low Dose Sodium Azide Ingestion - Case Report

Yanlışlıkla Düşük Doz Sodyum Azid Alımını Takiben Nöbet - Olgu Sunumu

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Abstract

Sodium azide is used as a preservative in many laboratory settings. Clinical experience with treatment of sodium azide intoxication is very limited. Some publications reported severe side effects such as seizure, coma, respiratory depression, metabolic acidosis, arrhythmia and asystole. Major side effects were observed following doses of 10 mg/kg or over, the minimal lethal dosage.

A 25 years old female dentist accidentally ingested approximately 5 ml of a diluted solution of (10 mg) sodium azide. Five minutes after ingestion, she became drowsy and had a generalized tonic-clonic seizure which continued for two minutes and was followed by a postictal period.

Critical toxicity symptoms may develop with exposure to low doses of sodium azide. The risk of exposure to sodium azide should be considered especially when there are mental status changes, tremor, tachycardia, temporary vision loss and shortness of breath in the presence of unexplained seizures.

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Key words: Sodium azide, ingestion, toxicity

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Özet

Sodyum azid bazı laboratuarlarda koruyucu madde olarak kullanılmaktadır. Fakat sodyum azid zehirlenmesinin tedavisiyle ilgili deneyim son derece sınırlıdır. En küçük ölümcül doz olan 10 mg/kg ve üstü dozlarda; nöbet, koma, solunum depresyonu, metabolik asidoz, aritmi ve asistol gibi ciddi etkilerin izlendiğini gösteren yayınlar mevcuttur.

Yirmibeş yaşında diş hekimi olan bir bayan hasta, yaklaşık 10 mg sodyum azid içeren 5 ml'lik seyreltilmiş çözeltiden yanlışlıkla içmişti. Alımdan 5 dakika kadar sonra uykuya eğilimi arttı ve 2 dakika kadar süren ve postiktal dönemin eşlik ettiği jeneralize tonik - klonik nöbet geçirdi.

Sodyum azidin düşük dozlarında da ciddi zehirlenme semptomları gelişebilir. Özellikle bilinç durumu değişiklikleri, tremor, taşikardi, temporal görme kaybı ve nefes darlığıyla birlikte olan açıklanamayan nöbetlerde sodyum azid maruziyeti düşünülmelidir. *(JAEM 2011; 10: 41-2)*

Anahtar kelimeler: Sodyum azid, alım, toksisite

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Introduction

Sodium azide (NaN_3) is a white to colorless, crystalline powder, which is highly water soluble, tasteless and odorless. Increased production and disposal of azides have increased the potential for human exposure and contamination of the environment (1). Also it could be used by terrorists for terrorist attacks (2). Sodium azide used mainly as a preservative in aqueous laboratory reagents and biologic fluids and as a fuel in automobile airbag gas generators, has caused deaths for decades. Access to sodium azide is usually easy for people working in such environments. Its exposure potential for the general population increases with the increased use of airbags . The most commonly reported health effect from azide exposure is hypotension, almost independent of route of exposure. Most industrial exposures are by inhalation. Most laboratory exposures or suicide attempts are by ingestion (1).

Some publications reported severe health effects such as seizure, coma, respiratory depression, pulmonary edema, flaccidity, metabolic acidosis, arrhythmia and asystole, which were observed following doses of only 10 mg/kg or above, the minimal lethal dosage (3). We present a patient with generalized tonic-clonic seizure activity associated with accidental low dose (10 mg) sodium azide ingestion.

Case Report

A 25 years old female dentist accidentally ingested approximately 5 ml of diluted solution of (10 mg) sodium azide. 5 minutes after ingestion she became drowsy and had a generalized tonic-clonic seizure activity which continued for two minutes and was followed by a postictal period. She was transferred to our emergency department by ambulance and the paramedics witnessed her seizure activity. When she was admitted to our department, she was

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conscious but suffered from nausea, vomiting and severe headache. Her physical examination was unremarkable. Her vital signs were normal. Initial arterial blood gas analysis revealed pH: 7.39, pCO_2: 34.3 mm Hg, pO_2: 97.5 mm Hg, HCO_3: 20.6 mEq/L, base excess: 4,2 and O_2 saturation 97.3%. Other laboratory values, including complete blood count, creatinine, amylase and liver enzymes were normal, her ß-hCG test was negative. In her ECG, normal sinus rhythm and sinus tachycardia was seen. Antero-posterior chest X-Ray was normal. Her emergent cranial tomography scans were reported normal. Orogastric lavage was performed with 2000 cc water. It was clear with minimal gastric contents. There was no sign of gastric bleeding. Then emergency esophagogastroscopy was performed and reported as normal. She was monitorized and followed up for 24 hours in the emergency department. No complication was observed and she was discharged as healthy with recommendations. She was followed for three months by telephone. No adverse effect was reported and she had had no health problem.

Discussion

The human health effects of sodium azide were first reported in 1927, but to date, the mechanism of its toxic effects has not yet been fully explained. Sodium azide poisonings occur very rarely. The basic physiological effect of sodium azide is vasodilatation, resembling nitroglycerine and other nitrites. The stimulation of carotid chemoreceptors can explain the hypotension. In part, sodium azide may have a vasodilator action similar to nitric oxide. Some authors argue that lethality is due to the formation of nitric oxide after conversion of sodium azide (4).

Based on the clinical literature, sodium azide mostly affects adults (99% of the cases). Intentional, as well as accidental, ingestions of sodium azide are the main causes of acute sodium azide intoxications (1). It can be ingested, inhaled, or absorbed transcutaneously. At lower doses it produces transient headaches, hypotension, and nausea (3). Early signs and symptoms of sodium azide exposure include palpitation, tachycardia, dyspnea, decreased mental status, collapse, weakness, unsteadiness, dizziness, sweating, hyperthermia, paleness, diarrhea, and transient vision loss within minutes of exposure. More severe and fatal toxic effects have later onsets within a range of an hour to several days. Some publications reported severe health effects such as seizure, coma, pulmonary edema, flaccidity, metabolic acidosis, cardiopulmonary failure, pulmonary edema, electrocardiogram changes, arrhythmia, bradycardia, and asystole were observed only following doses of 10 mg/kg or above, the minimal lethal dosage (1). Clinical experience with treatment of sodium azide intoxication is very limited. In the industrial workplace, episodes of diarrhea and mild lowering of blood pressure have been documented after prolonged dermal or inhalation exposure. In fact, this agent has been studied in clinical trials as a treatment for hypertension. No adverse effects were noted at low doses (5). Emergency measures generally applied for detoxification, such as gastric lavage, have not been helpful, probably because of the rapid absorption of sodium azide from the gastrointestinal and respiratory tracts before the initiation of treatment. Some publications reported trials of specific antidotes, attempts to remove the toxin, and treatment of specific health complications. Specific poisoning treatments, including amyl nitrite, sodium nitrite, and sodium thiosulfate, aimed to induce formation of methemoglobinemia and they were unsuccessful (6). Follow-up of these patients is unclear, but symptoms and full recovery time may redirect the clinician. Because no specific treatment is available for sodium azide intoxication, the best strategy is to avoid sodium azide toxicity by identifying the populations at risk and implementing preventive measures. People working in laboratories that use sodium azide or people involved in health care settings are at highest risk of fatal sodium azide intoxication (1).

Critical toxicity symptoms may develop with exposure to low doses of sodium azide. Especially, mental status changes, tremor, tachycardia, temporary vision loss and shortness of breath in the presence of unexplained seizures should alert the medical personnel to the possible risk of exposure to sodium azide.

Conflict of Interest

No conflict of interest is declared by the authors.

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