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Case Report

Cyanide intoxication with encephalitis clinic: a case report

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Abstract. Cyanide intoxication is one of the most dangerous poisonings and may occur by oral, respiratuary and dermal routes. Central nervous system is the most susceptible region to acute cyanide intoxication. A 3.5-years-old-girl was brought to our pediatric emergency polyclinic with complaints of fever, dullness and convulsion. On physical examination, lethargy and agitation, increased deep tendon reflexes and bilateral extensor plantar response were determined. On brain magnetic resonance imaging, increased signal intensity and decreased diffusion in bilateral basal ganglia on T2-fluid-attenuated inversion-recovery sequences were determined. The patient diagnosed as intoxication or encephalitis was taken to intensive care unit. Gastric lavage was performed and activated charcoal and acyclovir were given. After these managements, it was learnt that she had eaten a lot of apricot, plum and seed of apricot before the complaints. Therefore, she was diagnosed as cyanide intoxication and hydroxycobalamin treatment was given for five days. Our aim is to emphasize that in patients who were brought with symptoms and signs of encephalitis, intoxications also should be thought in differential diagnosis; therefore, we want to underline once again that taking detailed anamnesis is very important.

Key words: Cyanide intoxication; encephalitis; children

1. Introduction

Cyanide is one of the most rapidly acting poisons. Cyanide intoxications are the most dangerous poisonings and may occur by oral, respiratuary and dermal routes. Oral cyanide poisoning occurs after ingestion of various foods containing cyanogenic glycosides (1). Cyanide derivatives that present naturally at bitter almond and apricot seed, cherry seed and leaf, daphne tree, have many usage fields with chemical compounds like hydrogen cyanide, potassium cyanide, sodium cyanide, mercury cyanide, zinc cyanide, silver cyanide, magnesium cyanide, potassium nitroprusside, potassium thiocyanide (2). In this report, a 3.5 – years – old-girl with cyanide intoxication who was brought to our hospital with complaints of fever, convulsion, blurred consciousness, and who was misdiagnosed as encephalitis is presented.

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2. Case Report

Three and half-years-old-girl which was wholly healthy before, was brought to our pediatric emergency polyclinic with fever and dullness. It was learnt that the fever had begun 4 hours before admission, lasted for two hours, and then it had become normal spontaneously, and, dullness and convulsion complaints had started one hour before admission. After that, the patient had stayed unconscious and she had spurt style vomiting. There was no feature in her own history. There was second-degree relationship between the parent and the patient had four healthy sisters.

On physical examination, both body weight and height were between 3 and 10 percentile, blood pressure, respiration rate, pulse, body temperature were 90/55 mmHg, 30 /minute, 100 /minute, 36.5°C, respectively. Her general status was bad, and on neurological examination lethargy and

agitation, increased deep tendon reflexes and bilateral extensor plantar response were determined. Other systems' physical examinations were normal.

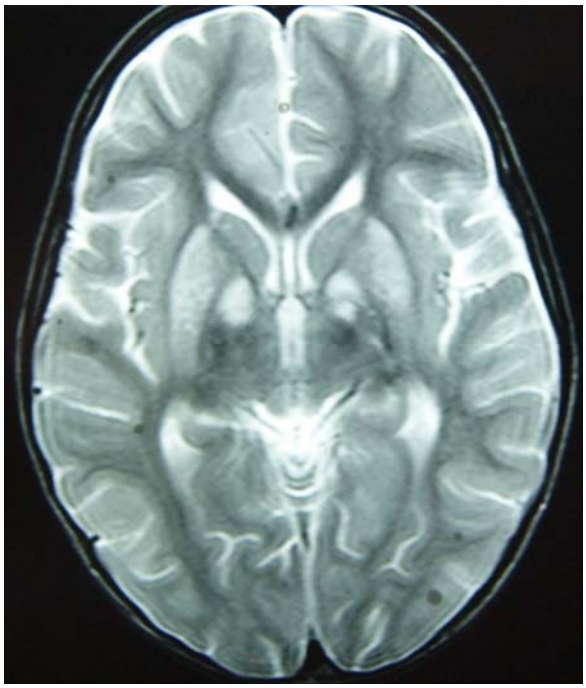

Figure 1. Brain magnetic resonance imaging shows the increased signal intensity in bilateral basal ganglia on T2 sequences

On laboratory examinations, complete blood count, liver, renal function tests and serum electrolytes, arterial blood gas analysis, prothrombin time and active partial thromboplastin time were in normal range except creatine kinase (190 U/L) (normal range: 26-270 U/L) , creatine kinase-MB (75 U/L) (normal range: 0-24 U/L). On cerebrospinal fluid examinations, glucose, protein, chloride and simultaneously serum glucose were 53 mg/dL, 17.2 mg/dL, 127 mEq/L, 110 mg/dL, respectively; microscopic examination was normal. Antistreptolysin O, salmonella and brucella serologies were negative, but C-reactive protein was 12.3 mg/L. At urine analysis, protein was 30 mg/dL, pH was 6.5; on microscopic examination, 43 leucocytes at every area were detected. Urine and blood cultures were negative; urine analyses were normal four days after hospitalization. Electroencephalographic examination and computerized tomography were normal on admission. Brain magnetic resonance imaging was performed five days after hospitalization, increased signal intensity and decreased diffusion were determined in bilateral basal ganglia on T2-fluid-attenuated inversion-recovery (FLAIR) sequences and diffusion-weighted MRI, respectively. Also increased signal intensities were seen in the parietal cortex beside the interhemispheric fissures on FLAIR sequences (Figure 1, 2).

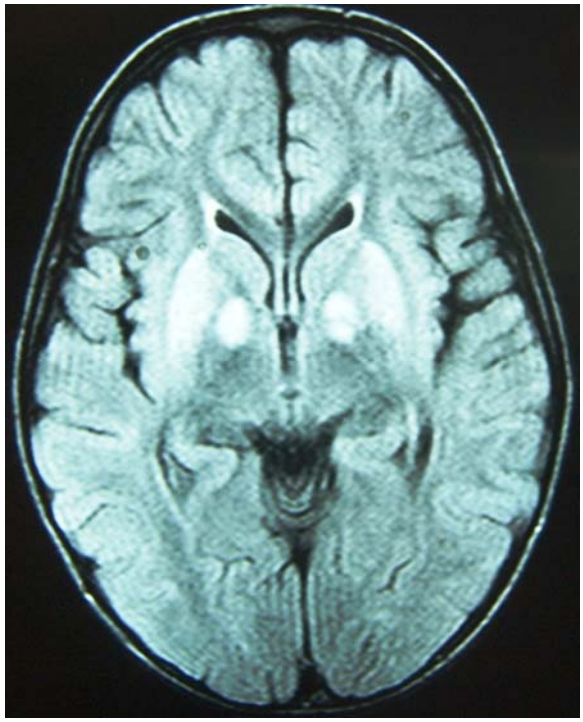

Figure 2. FLAIR sequences shows increased signal intensities in bilateral parietal cortex beside the interhemispheric fissures.

The patient, who was diagnosed as intoxication or encephalitis, was taken to our intensive care unit. Gastric lavage was performed and active charcoal (1 g/kg/dose, six doses in a day) was given. During follow-up, phenytoin, phenobarbital, midazolam, lidocaine treatments were given gradually due to refractor seizures. However, the convulsions were not under control until pentothal was started. In diagnosis, firstly, the entity of encephalitis was thought because of the combination of refractory convulsion and fever. Therefore, acyclovir was given intravenously to the patient until EEG was performed. On the third day of admission acyclovir treatment was discontinued because of normal EEG examinations. Then, it was learnt that she had eaten many apricots, plums and seeds of apricots before her complaints. Therefore, she was diagnosed as cyanide intoxication and hydroxycobalamin treatment was given for five days. After the hydroxycobalamin treatment, convulsions were controlled only by phenytoin. On follow-up, when general state was getting better, choreoathetoid movements began.

She was discharged on the 21st day of hospitalization. Two months later, she was controlled again and a markedly decrement in choreoathetoid movements was observed. The patient is still followed-up without convulsion.

3. Discussion

Cyanides are one of the rapidly acting toxic agents (1). Cyanides present naturally as sodium and potassium cyanide, in gas form they present as hydrogen cyanide (2). Particularly some plants like apple, apricot and peach' seeds and cores contains important degree more amounts cyanogen glycosides (2). With aspect to cyanide, shock and so death can be seen in minutes (2). Our patient had eaten many apricots and apricot cores before the complaints had started.

Amygdaline (D-mandelonitrile-B-D-glucoside) is a cyanogenic compound in apricot core. If cores of apricot have swallowed completely cyanide release lesser; but if had eaten with chewing, toxicity becomes more due to emulsion release from lysosomes. Apricots' cores are more toxic due to including excess cyanogen amount and can release hydrogen cyanide easily (2). Because of cyanide aspect to humans or animals by any route, releasing cyanide ion quickly binds to proteins and iron containing enzymes and causes loss of their functions. Cyanide causes enzyme inhibition with binding metallic cofactor of metalloenzymes (2).

Central nervous system is most susceptible region to acute cyanide intoxication and death can be seen due to respiratuary control center inhibition (2).

Cyanide contents of apricot cores changes from 4.09 to 112 mg/g, but it has been accepted as 2.92 mg/g in last publications (2). Lethal dose for humans changes from 50-60 mg to 1.52 mg/kg in last publications; determined lethal dose has been accepted as 56 mg/kg (2). Minimum fatal oral dose is 0.56 mg/kg (2). Cyanide level could not be measured in our patient because of technical insufficiency.

Respiratory disorders can arise after oral intake of cyanide. After oral intake, wheezy, deep and rapid respiration, dyspnea, acute respiratuary distress syndrome may be seen (2). Superficial filiform pulse, deeply and hardly heard heart sounds, congestive heart failure, hypotension, widened heart view were reported at comatose patients (2). In these situations, increment at blood pressure and pulse rate after gastric lavage and glucose infusion was reported (3). After acute oral intoxications, cases that goes to surgical exploration due to vomiting, gastrointestinal spasms, excess necrosis had been reported (3). Muscular rigidity, rabdomyolisis, increment levels of serum creatine and serum creatine kinase, albuminuria, thyroid toxicity are other toxic signs (2). Macular degeneration and optic atrophy are among signs of chronic toxicity (2). Metabolic acidosis is seen 67 % of oral toxic patients as acute (2). Headaches, loss of consciousness, convulsions, positive Babinsky sign, hemiparalysis, dysphonia, parkinsonism, coma, death can be seen because of neurological system affection after oral intake of cyanide (2). In addition, sharp almond smell in breath and clear pink color on skin can be seen in physical examination (2). Central nervous system signs were the major entities in our patient as consistent with literature. Uncontrolled convulsions and changes in consciousness were the first signs at the beginning. Cardiovascular complications were not seen in our case. Only vomiting was seen as a gastrointestinal complication. Our patient, who was entubated before starting Pentothal infusion due to uncontrolled convulsions, did not have dyspnea or acute respiratory distress syndrome.

At cyanide intoxication, changes in globus pallidus, posterior putamen and substantia nigra subthalamic nucleus, temporal and occipital cortex, cerebellum, caudate nucleus were reported in literature (4). In our patient bilateral globus pallidus and putamen involvement was present as consistent with literature.

In treatment, sodium thiosulphate, hydroxycobalamin like cyanide antagonists have been used (2). Other antidotes are amyl nitrite, sodium nitrite, hydroxylamine, p aminopropiophenone, 4-dimethylaminophenol, primakin like compounds. Antidote treatment will be continued until serum parameters (blood oxygen and serum pH) show no cyanide compound present, which effect mitochondria. Calcium channel blockers, antioxidants, anti-inflammatory drugs have been tested in aim of treatment (2). Our patient displayed a dramatically response to hydroxycobalamin treatment which supports our diagnosis.

4. Conclusion

As a conclusion, in patients with signs and symptoms of encephalitis, intoxications should be thought in the differential diagnosis, so we want to underline once again that getting detailed anamnesis is crucial.

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