Changes in Some Hematology Parameters in poisoning with Rice Tablet (Aluminum Phosphide)

Farshid Fayyaz (PhD)
Department of Legal Medicine, AJA University of Medical Sciences, Tehran, Iran

Corresponding Author: Farshid Fayyaz

Email : dr.farshid.fayyaz@gmail.com

Tel : +989123432060

Address: Department of Legal Medicine, AJA University of Medical Sciences, Tehran, Iran

Received: 03 Feb 2015
Revised: 01 Mar 2015
Accepted: 07 Mar 2015

ABSTRACT

Background and Objective: Aluminum Phosphide (ALP) is a solid non-organic phosphide with dark gray or dark yellow crystals. It reacts with stomach acid after ingestion and causes phosphine gas to be released. It is thought that phosphine causes toxicity from enzymatic interference and may even lead to cell death. This study aimed to investigate the effects of poisoning with rice tablet on levels of platelets, hemoglobin, white blood cells.

Methods: The clinical records of 67 cases of acute oral toxicity with aluminum phosphide admitted to Baharloo hospital and 28 forensic autopsy cases in Kahrizak forensic research center were studied. Recorded information included vital signs, demographic characteristics, numerous laboratory and clinical findings, complications and all pathologic findings.

Results: All patients had received standard symptomatic and supportive treatments. Among the tested subjects, 30 of 67 patients (44.8%) were male. The mean hemoglobin level of recovered and deceased individuals was 12.26 and 11.72 g/dl, respectively. There was a significant relationship between patients' WBC counts and mortality where the mean level of WBC in the deceased was more than that of the recovered (P=0.001). Mean SBP in the deceased and the recovered individuals was 79.67 ± 12.89 and 102.46 ± 22.57 mmHg, respectively.

Conclusion: Consumption of rice tablets results in blood pressure alteration, hemoglobin levels, platelets and leukocyte count. Tracking these alterations can reduce the side effects and mortality rate in the cases of rice tablet poisoning.

Keywords: Aluminum Phosphide, White Blood Cells, Hemoglobins, Blood Pressure, Patients.

This paper should be cited as: Fayyaz, F. [Changes in some Hematology Parameters in Poisoning with Rice Tablet (Aluminum Phosphide)]. mljgoums. 2015; 9(4):43-46

INTRODUCTION

Pesticide use or abuse causes 100,000 deaths annually around the world(1). The majority of reported pesticide-associated deaths in hospitals are due to suicide attempts (2). The most common causes of suicide include family issues, alcohol or drugs addiction, emotional disorders (most common cause of suicide in young people)(3), depression, medical illness, loneliness, ostracism or financial problems (1).
Most people in industrialized countries commit suicide by drugs while in developing countries the most common way of suicide is through agricultural pesticide consumption. In addition, the prevalence of suicide attempts in men is two to three folds higher than that in women in industrialized countries (3). In a 10-year epidemiologic study from 1983 to 2003 on cases of hydrogen phosphide exposure in the poison center of Mainz, Germany, it was demonstrated that among the 183 cases of aluminum phosphide poisoning, 65% were accidental, 28% were suicidal, 5% were job-related and 2% did not have a specified reason (4). In a study in India, the majority of poisonings in young subjects were suicidal and belonging to lower socioeconomic part of the society (mostly rural) with the frequency of upto two-fold suicide attempts among men compared to women. In addition, 50% of cases were reported to be aluminum phosphide (ALP) poisoning (5). Pure Phosphine gas is odorless (6). The garlic smell is due to impurities such as diphosphine, methane, arsine, hydrogen, and phosphine substituents (7,8). Phosphine gas is heavier than the air and explodes in concentrations more than 1.8% in the air, being spontaneously flammable at 38°C (9). This gas has anticholinesterase-like effects on the central nervous system and exposure limit for humans is 0.3 ppm (10). Aluminum phosphide is a solid inorganic phosphide with dark gray or dark yellow crystals with a melting point of more than 1000 ^{o}C. Being heavier than water, ALP is not water soluble and reacts with it instead (9,11,12).Each 3-g tablet releases one gram phosphine gas (7,13). Aluminum phosphide was used as a source of phosphine gas by Degesch Company in Germany for the first time and its tablet was implemented as a pesticide in the United States in 1985 (14, 15). According to evaluations, this tablet does not have teratogenic, mutagenic, and carcinogenic effects (12). After oral consumption of aluminum phosphide, t he phosphine gas is released in stomach which results in preliminary effects by affecting the circulatory system. Sometimes it is absorbed and emergence of poisoning symptoms due to slow release of phosphine gas. The aim of this study is to analyze the clinical findings related to consumption of ALP also known as the rice tablet in order to identify its symptoms and determination of effective treatment approaches.metabolized in liver which delays the

MATERIAL AND METHODS

This was a case study which included all the rice tablet (aluminum phosphide) poisoning patients who had been receipted and hospitalized in the poison center of Baharloo hospital in 2011. The biennial census records of poisoning patients in the Baharloo hospital and Kahrizak forensic medical research center were analyzed by the author and the required data was recorded in data collection forms.
According to the data types and parameters, Chi-square, ANOVA, and T-test were performed using SPSS statistical software (version 16) to evaluate the correlations. In this research, the required parameters were collected through scrutinizing the patients' information from the moment of administration until the complete recovery while their personal profiles remain unpublished.

RESULTS

The mean hemoglobin levels in men and women were 13.56 and 10.85 grams per deciliter respectively while this amount was 12.26 for the recovered and 11.72 for the deceased. Among all the patients, 15.9% (10 subjects) had anemia with an Hb level of lower than 10 from which 8 subjects were women and 2 were men. The low Hb level of <10 was present in two female cases in the early examinations and the rest of subjects (6 women and 2 men) developed this condition during the poisoning. Thirty percent of anemic patients were deceased (all were female) and two subjects faced hemoglobin drop and one had low hemoglobin level in the first day. Among patients with >10 hemoglobin levels, 39.6% were deceased. Nine subjects (13.4%) faced a hemoglobin drop of more than 2 units during the poisoning from which 7 were recovered and 2 had died. One male who encountered anemia during the disease had used the dissolved tablet in water for suicide and the rest of anemic patients had consumed fresh tablets. There was no relationship between hemoglobin levels and

the time between consumption and treatment, the number of consumed tablets, the type of tablets and the patients' age (P>0.05). In addition, there was no relationship between hemoglobin levels and mortality. No WBC leukopenia was observed below 4000. Among all the studied patients, 33.33% had leukocytosis (WBC>15000). This was observed in patients who had consumed at least a quarter of a fresh tablet and 68% of patients with leukocytosis and 23% of subjects with normal WBC deceased eventually. The mean levels of WBC were 13.15 and 12.73 g/dl in men and women respectively. There was no significant relationship between WBC levels and the delay between consumption and treatment, the number of administered tablets and age (P>0.05). There was a significant association between the type of the tablet and WBC count (P=0.037) with higher WBC levels in patients who consumed fresh tablets. The mean WBC levels were 9.81±2.96 and 13.43±4.94 respectively, among cases who consumed old and fresh tablets .The mean WBC count of recovered patients was 11.43 and 15.32 g/dl for the deceased. The WBC count showed a significant correlation with mortality (P=0.001). Furthermore, the mortality risk increased 6.4 folds when WBC count exceeded 15000 (compared to normal WBC). Thrombocytopenia (PLT < 1.4×10^5) was observed in 15.87% of patients and one subject suffered from thrombocytosis (PLT of 6.09×10^5). Thrombocytopenia was associated with consumption of at least one tablet. Among all the thrombocytopenia patients 50% and 35.9% of patients with normal PLT levels died eventually. One patient had used powdered tablet and the rest had consumed fresh ones. The mean PLT was found to be 205.923±90.73 and 194.083±76.13 ul in recovered and deceased subjects, respectively. The time between administration and treatment, the number of tablets, the type of tablets, and the age showed significant relation with neither PLT nor mortality (P>0.05). Furthermore, there were no relation between PLT levels and mortality (P=0.59).

DISCUSSION

The reduction in red blood cells has been observed in CBC tests of aluminum phosphide poisoning patients (7). In this study, the mean level of hemoglobin was 12.06±2.42 and a range of 5.4±17.9. In similar studies, erythrocyte damage (16) has been reported And 15.9% of patients in our research had anemia (80% were women and 20% were men). In several studies, the drop in Hb levels in response to Disseminated Intravascular Coagulation -derived microangiopatic hemolytic anemia and intravascular hemolysis was reported in aluminum phosphide poisoning cases (17-20).

Consumption of dissolved tablets in water also resulted in anemia, which could be due to the detrimental effects of phosphine on erythrocyte membrane even at low doses. The results of this study suggest that the drop in hemoglobin did not affect the mortality rate if the appropriate treatment is applied in time. Leukopenia is associated with rice tablet poisoning (7) and has been considered as a symptom of acute poisoning (13). However, in our study, there was no leukopenia incidence with the WBC of < 4000 while, 33.33% of patients had leukocytosis with WBC of > 15000 which was observed even in cases of a quarter of a tablet consumption. The mean WBC level was 12.91±4.84 with a range of 4000-24000. Leukocytosis and neutrophilia can be the results of infections, inflammation, stress, thrill, cardiac infarction, leukocyte adhesion deficiency and metabolic disorders, acute renal failure, acute poisoning and acute hemorrhage or hemolysis, etc. (21). The mean WBC was 11.43 in the recovered and 15.32 in the deceased patients having a significant relationship with mortality rate. These results were consistent with Louriz et al, (22) and Idrissi (23) where a mean WBC of 11.8±7.2 with a range of 2.2-30 (WBC of 8.5 in the recovered and 15.6 forand the deceased patients) has been reported with 32% leukocytosis incidence among the tested subjects. In this study, the type of consumed tablet had a stronger correlation with WBC count which suggests the effect of aluminum phosphide on leukocytes through one the above mentioned mechanisms (21). The mean PLt was found 201.41±85.03 with a range of 38-609 in our study and there was no significant association between this factor and mortality. Thrombocytopenia has also been considered as one of the late symptoms of poisoning in the previous studies(7).

CONCLUSION

Consumption of rice tablets results in blood pressure alteration, hemoglobin levels, platelets and leukocyte count. Tracking these alterations can reduce the side effects and mortality rate in the cases of rice tablet poisoning.

ACKNOWLEDGEMENTS

We would like to thank all those who helped us during this research, especially the AJA Medical Science University.

CONFLICT OF INTEREST

The authors declare no conflict of interest between them.

REFERENCES

1. Gunnell D, Eddleston M. *Suicide by intentional ingestion of pesticides: a continuing tragedy in developing countries.* International Journal of Epidemiology. 2003; 32(6): 902-909.

2. Lauterbach M, Solak E, Kaes J, Wiechelt J, Von Mach MA, Weilemann LS. *Epidemiology of hydrogen phosphide exposures in humans reported to the poison center in Mainz, Germany, 1983-2003.* Clinical Toxicology. 2005; 43(6): 575-581.

3. Siwach S, Gupta A. *The profile of acute poisonings in Harayana-Rohtak Study.* J Assoc Physicians India. 1995; 43(11): 756-759.

4. Organization, WH. *Phosphine and selected metal phosphides.* World Health Organization. 1988.

5. Mehrpoor O, Shadnia SH, Soltani Nejad K, Yaghmaei A. *Evaluation of electrolytes and blood glucose level in aluminum phosphide poisoning.* IJFM. 2009; 15(1): 49-53.

6. Sullivan JB, Krieger GR. *Clinical environmental health and toxic exposures.* Lippincott Williams & Wilkins. 2001.

7. Inchem I. *International Programme on Chemical Safety.* Chemical Safety Information from Intergovernmental Organizations. Isoniazid. 1999.

8. Rossoff IS. *Encyclopedia of Clinical Toxicology: A Comprehensive Guide to the Toxicology of Prescription and OTC Drugs, Chemical, Herbals, Plants, Fungi, Marine Life, Reptile and Insect Venoms, Food Ingredients, Clothing and Environmental Toxins.* 2001.

9. Anand R, Binukumar K, Gill KD. *Aluminum phosphide poisoning: an unsolved riddle*. *Journal of Applied Toxicology.* 2011; 31(6): 499-505. doi: 10.1002/jat.1692.

10. Hotchkiss BE. *EXTOXNET: extension toxicology network.* 1989: Cooperative Extension Service, Cornell University.

11. Wahab A, Wahab S, Khan RA. *Acute aluminium phosphide poisoning: an update.* Hong Kong J Emerg Med. 2008. 15(3): 152-5.

12. Troitzsch JH. *Overview of flame retardants.* Chemistry today. 1998; 16.

13. Cardis E, Gilbert ES, Carpenter L, Howe G, Kato I, Armstrong BK, et al. *Effects of Low Doses and Low Dose Rates of External Ionizing Radiation: Cancer Mortality among Nuclear Industry Workers in Three Countries*. *Radiation Research* . 1995; 142(2): 117-132.

14. Mehrpour O, Alfred S, Shadnia S, Keyler DE, Soltaninejad K, Chalaki N, et al. *Hyperglycemia in acute aluminum phosphide poisoning as a potential prognostic factor.* Human & experimental toxicology. 2008; 27(7): 591-595.(Persian)

15. Aggarwal P, Handa R, Wig N, Biswas A, Saxena R, Wali JP. *Intravascular hemolysis in aluminium phosphide poisoning.* The American journal of emergency medicine. 1999; 17(5): 488-489.

16. Sood AK, Mahajan A, Dua A. *Intravascular haemolysis after aluminium phosphide ingestion.* Journal of the Royal Society of Medicine, 1997; 90(1): 47.-48

17. Srinivas R, Agarwal R, Jairam A, Sakhuja V. *Intravascular haemolysis due to glucose-6-phosphate dehydrogenase deficiency in a patient with aluminium phosphide poisoning.* Emergency medicine journal: EMJ. 2007; 24(1): 67-68.

18. Khurana V, Gambhir I, Kishore D. *Microangiopathic hemolytic anemia following disseminated intravascular coagulation in aluminum phosphide poisoning.* Indian journal of medical sciences. 2009; 63(6): 257-9. doi: 10.4103/0019-5359.53396.

19. Fauci AS. *Harrison's principles of internal medicine*. McGraw-Hill Medical New York. 2008: Vol 2.

20. Louriz M, Dendane T, Abidi K, Madani N, Abouqal R, Zeggwagh AA. *Prognostic factors of acute aluminum phosphide poisoning.* Indian journal of medical sciences, 2009. 63(6): p. 227.

21. Hajouji IM, Oualili L, Abidi K, Abouqal R, Kerkeb O, Zeggwagh AA. *Severity factors of aluminium phosphide poisoning (Phostoxin))*. in Annales francaises d'anesthesie et de reanimation. 2006; 25(4): 382-5.

22. Jaiswal S, Verma R, Tewari N. *Aluminum phosphide poisoning: Effect of correction of severe metabolic acidosis on patient outcome.* Indian journal of critical care medicine: 2009; 13(1): 21.

23. Mostafazadeh B, Pajoumand A, Farzaneh E, Aghabiklooei A, Rasouli MR. *Blood levels of methemoglobin in patients with aluminum phosphide poisoning and its correlation with patient's outcome.* Journal of Medical Toxicolog. 2011; 7(1): 40-43.