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Neutropenia Due to Acute Exposure with Aluminium Phosphide

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***Dear Editor*;**

Aluminium phosphide (AIP) is a highly toxic insecticide that widely used for preserving foods in the form of incense(1, 2). AIP has been banned in some countries due to high toxicity and causing to death. Main pathological effect of AIP is mitochondrial dysfunction(1). A short time after exposure, it causes epigastric pain, nausea and diarrhea. Previously several complications, some of them may be fatal, such as metabolic acidosis, cardiac arrhythmias, respiratory distress syndrome, shock, gastroduodenitis, hepatitis, tubular necrosis, esophageal structure, adrenal insufficiency, hemolysis and methemoglobinemia have been reported as a result of AIP toxicity (1-3). AIP induced leukopenia has been reported previously (1) but AIP induced isolated neutropenia was not observed in literature. Here in we present a severe neutropenia case due to vocational AIP toxicity.

A 37 year old male patient was admitted to hematology clinic with complaints of fatigue, abdominal pain, diarrhea and high fever. He denied any chronic diseases and medications. It was learned that four days ago he has been exposed to occupational AIP in the form of incense. Also he reported a second exposure to AIP one month ago with similar complaints. But he did not came to the hospital. On physical examination: fever: 38.4 C, blood pressure: 110/60 mm/Hg, pulse 98/min. Lymphadenopathy or organomegaly was not observed. In laboratory findings, Hb: 13.8 g/dl, WBC: 3.8x109/L, Neu:0.4x109/L, Lym: 2.7x109/L, PLT: 248x109/L. Peripheral blood smear confirmed neutropenia without blastic or atypical cells. Bone marrow biopsy showed normal myeloid/erythroid ratio, myeloid maturation defect and minimal erythroid dysplasia and these findings were reported as compatible with toxic effects. The patient was hospitalized and antibiotics was started in febrile neutropenia protocol without G-CSF. On the 4th day of admission neutrophil values increased to 2.2x109/L spontaneously. Viral parameters, brucella, biochemical and hormonal tests were normal. The lack of other findings explaining the neutropenia and rapidly increment of neutrophil counts suggested that the patient had acute AIP toxicity.

AIP toxicity studies are mostly animal studies in the literature (3-6). In humans, mostly acute poisoning cases is seen and the majority of these cases are agricultural workers in Asian countries. Our case is also a patient engaged in grain fumigation because of his job. In the light of the available data neutropenia is not an expected laboratory finding of AIP toxicity. In fact leukocyte count is expected to be higher and in a study high leukocyte count has been reported to be associated with poor prognosis. Detailed medical history and profession should be questioned carefully in patients presenting with neutropenia. As in this case, it must be keep in mind that not only of drugs taken orally, other forms of exposure as well as inhaled medications should also be questioned.

REFERENCES

1. *Ntelios D, Mandros C, Potolidis E, Fanourgiakis P. Aluminium phosphide-induced leukopenia. BMJ Case Reports 2013;30:2013.*
2. *Mehrpour O, Jafarzadeh M, Abdollahi M. A systematic review of aluminium phosphide poisoning. Arh Hig Rada Toksikol 2012;63:61-73.*
3. *Turkez H, Togar B. Aluminum phosphide-induced genetic and oxidative damages in rats: attenuation by Laurus nobilis leaf extract. Toxicol Ind Health 2013;29:579-83.*
4. *Okolie NP, Aligbe JU, Osakue EE. Phostoxin-induced biochemical andpathomorphological changes in rabbits. Indian J Experimen Biol 2004;42:1096-9.*
5. *Dua R, Gill KD. Aluminium phosphide exposure: implications on rat brain lipid peroxidation and antioxidant defence system. Pharmacol Toxicol 2001;89:315-9.*
6. *Kligerman AD, Bishop JB, Erexson GL, et al. Cytogenetic and germ cell effects of phosphine inhalation by rodents: II. Subacute exposures to rats and mice. Environ Mol Mutagen 1994;24:301-6.*

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