www.nephropathol.com DOI: 10.34172/jnp.2022.17227 J Nephropathol. 2022;11(2):e17227

Journal of Nephropathology

An unusual cause of acute kidney injury "inhalation of diazinon"

Mohammadreza Rahimi Majd[1], Maliheh Yarmohamadi[1*], Mahmoud Parvin[2], Soheila Mirjan[1]

[1]Department of Internal Medicine, Faculty of Medicine, Semnan University of Medical Sciences, Semnan, Iran
[2]Department of Pathology, Shahid Labbafinejad Medical Center, Shahid Beheshti University of Medical Sciences Tehran, Iran

ARTICLE INFO

Article type:
Case Report

Article history:
Received: 22 May 2021
Accepted: 23 October 2021
Published online: 12 January 2022

ABSTRACT

Here, we present a 74-year-old man with acute kidney injury (AKI) due to diazinon 60% (accidental poisoning) which was successfully treated by hemodialysis.

Keywords: Organophosphate, Diazinon, Acute kidney injury

Case Report

Implication for health policy/practice/research/medical education:
Organophosphate poisoning usually presents with typical signs and symptoms of cholinergic excess and rarely can cause acute renal failure.

Please cite this paper as: Rahimi Majd M, Yarmohamadi M, Parvin M, Mirjan S. An unusual cause of acute kidney injury "inhalation of diazinon". J Nephropathol. 2022;11(2):e17227. DOI: 10.34172/jnp.2022.17227.

Introduction

Organophosphate compounds are widely used as insecticide in agriculture and veterinary practice (1). Organophosphate insecticides (such as diazinon) act by damaging acetylcholine esterase enzyme in the body. Organophosphate poisoning is a common phenomenon that presents classically with signs of cholinergic excess (2). We present a 74-year-old man with acute kidney injury (AKI) due to diazinon 60% (accidental poisoning) which was successfully treated by hemodialysis.

Case Presentation

A 74-year-old man was admitted to hospital because of fever, chills and urinary incontinence. The patient was not in alerted mental status, he had bizarre behaviors and transverse mandible vibration was noted and sweeting. He suffered from diabetes mellitus and he had a past medical history of replacement of heart valve and seizure. The patient's vital signs at admission were as follows; temperature; 39°C, heart rate; 72/min, respiratory rate; 25/min, blood pressure; 189/90 mm Hg and other physical exams were normal. The results of paraclinical tests were as follows; BUN = 98 mg/dL, Cr = 8 mg/dL, K = 4.5 mEq/L, Ca = 8.2 mg/dL, Na = 133 mEq/L, P = 6 mg/dL, WBC = 11.37×10^3/µL, hemoglobin = 10.7 g/dL, MCV = 90 fl, PLT = 103×10^3/µL. The patient was admitted to the nephrology unit, underwent five sessions of hemodialysis, and received 500 mg methylprednisolone pulse intravenously for three days. The patient underwent the kidney biopsy, which the result showed tubule-interstitial nephritis and crystal material in tubules (Figure 1A-D). The fifth day of hospitalization, the patient revealed the use of a huge amount of diazinon 60% as a pesticide one week before admission. During admission, the patient received supportive treatment and hemodialysis. He recovered completely after five sessions of hemodialysis. The patient's dieresis gradually improved and he was discharged after seven days with a plasma creatinine and BUN of 3.8 mg/dL and 56 mg/dL respectively. At follow-up two weeks later, creatinine dropped to 2.2 mg/dL.

Dissuasion

Diazinon is an organophosphate insecticide commonly used to control agricultural insects and livestock ectoparasites. WHO classifies diazinon as a "moderately hazardous pesticide" or "slightly toxic" (3). Research has shown that diazinon can be absorbed through the digestive

*Corresponding author: Maliheh Yarmohammadi,
Email: malihehyarmohamadi@yahoo.com

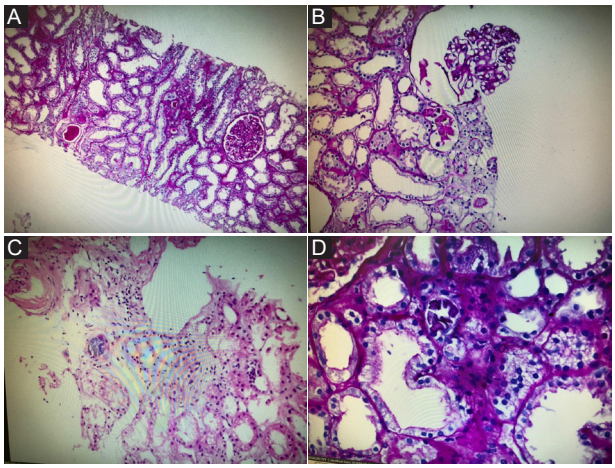

Figure 1. (A) A normal glomerulus and intra-tubular cellular debris (PAS ×40). **(B)** A normal glomerulus and intra-tubular cellular debris(PAS ×100). **(C)** Intra-tubular crystals obliterating the lumen (H&E ×100). **(D)** Intra-tubular crystals obliterating the lumen (PAS ×400).

system, skin or via respiratory mucosa when inhaled. In addition to its inhibitory effects on angiotensin-converting enzyme, it can increase formation of free radicals, thereby inducing oxidative stress and tissue lipid peroxidation in mammals and other organisms (4).

Renal injury is a rare and underestimated complication of organophosphate poisoning and very few reports discuss this complication (5). The clinical process of acute organophosphate poisoning can be characterized by three phases; acute cholinergic crisis, intermediate syndrome (IMS) and organophosphate-induced delayed neuropathy (6-8).

Persistent decline of renal function without recovery can lead to end-stage renal disease and increase the risk of death (9-11). The mechanism of AKI, induced by organophosphate poisoning is not clearly known. Dehydration, hypotension and bradycardia that induce renal hypoperfusion, direct toxic effects on renal parenchyma, endothelial cell damage, activation of immune and inflammatory responses, formation of free radicals, convulsive seizure and muscular fasciculation related rhabdomyolysis are all contributed to the decline of renal function (5).

We present a case of organophosphate poisoning causing ARF (acute renal failure). The diagnosis was confirmed by biopsy, which showed tubulointerstitial nephritis and acute tubular necrosis and crystal material in tubules. The patient recovered completely after hemodialysis.

Conclusion

Organophosphate poisoning usually presents with typical signs and symptoms of cholinergic excess and rarely can cause acute renal failure. Timely diagnosis and management of this complication confer a favorable prognosis to the patient.

Authors' contribution

MRM, MY and MP were the principal investigators of the study. MY and MP were included in preparing the concept and design. MRM, MY and SM revisited the manuscript and critically evaluated the intellectual contents. All authors participated in preparing the final draft of the manuscript, revised the manuscript and critically evaluated the intellectual contents. All authors have read and approved the content of the manuscript and confirmed the accuracy or integrity of any part of the work.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) were completely observed by the authors. Written informed consent was obtained from the patient to use the contents of patient's medical records.

Funding/Support

None.

References

1. Baydin A, Erenler AK, Yardan T, Kati C, Duran L, Dilek A. Acute organophosphate poisoning in adults: a 10-year analysis. HealthMED: J SocDevelop New Net Environ BH. 2014;8:151-60.
2. Zafar R, Munawar K, Nasrullah A, Haq S, Ghazanfar H, Sheikh AB, et al. Acute Renal Failure due to Organophosphate Poisoning: A Case Report. Cureus. 2017;9:e1523. doi: 10.7759/cureus.1523.
3. Harper B LB, Gervais JA, Buhl K, Stone D. Diazinon General Fact Sheet. National Pesticide Information Center: Oregon State University Extension Services; 2009 [accessed 5 April 2019]. Available from: http://npic.orst.edu/factsheets/Diazgen.html.
4. Kalender S, Ogutcu A, Uzunhisarcikli M, Açikgoz F, Durak D, Ulusoy Y, et al. Diazinon-induced hepatotoxicity and protective effect of vitamin E on some biochemical indices and ultrastructural changes. Toxicology. 2005;211:197-206. doi: 10.1016/j.tox.2005.03.007.
5. Lee FY, Chen WK, Lin CL, Lai CY, Wu YS, Lin IC, et al. Organophosphate Poisoning and Subsequent Acute Kidney Injury Risk: A Nationwide Population-Based Cohort Study. Medicine (Baltimore). 2015;94:e2107. doi: 10.1097/md.0000000000002107.
6. Agostini M, Bianchin A. Acute renal failure from organophospate poisoning: a case of success with haemofiltration. Hum Exp Toxicol. 2003;22:165-7. doi: 10.1191/0960327103ht343cr.
7. Cavari Y, Landau D, Sofer S, Leibson T, Lazar I. Organophosphate poisoning-induced acute renal failure. Pediatr Emerg Care. 2013;29:646-7. doi: 10.1097/

PEC.0b013e31828e9e45.
8. Faiz MS, Mughal S, Memon AQ. Acute and late complications of organophosphate poisoning. J Coll Physicians Surg Pak. 2011;21:288-90. PMID: 21575537.
9. Liangos O, Wald R, O'Bell JW, Price L, Pereira BJ, Jaber BL. Epidemiology and outcomes of acute renal failure in hospitalized patients: a national survey. Clin J Am Soc Nephrol. 2006;1:43-51. doi: 10.2215/cjn.00220605.
10. Pôncio L, Balbi AL, Rocha É P, Dias DB, Ponce D. The long-term outcome after acute kidney injury: a narrative review. J Bras Nefrol. 2015;37:115-20. doi: 10.5935/0101-2800.20150016.
11. Srisawat N, Kellum JA. Acute kidney injury: definition, epidemiology, and outcome. Curr Opin Crit Care. 2011;17:548-55. doi: 10.1097/MCC.0b013e32834cd349.