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Research Paper: Assessing the Clinical Features and Blood Biochemistries of Acute Organophosphorus Chemical Warfare Agents in Iranian Veterans

Khosrow Agin[1*], **Babak Mostafazadeh**[2]

1. Loghman-e Hakim Hospital, Shahid Beheshti Unuversity of Medical Sciences, Tehran, Iran.
2. Toxicological Research Center, Loghman-e Hakim Hospital, Shahid Beheshti Unuversity of Medical Sciences, Tehran, Iran.

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ABSTRACT

Background: Nerve Agents (NAs) are a chemical weapons, and their use is prohibited. They were used for numerous-times in the warfare of Iraq against Iran. The present study aimed to assess the clinical features and laboratory findings of the acute poisoning manifestations of organophosphorus chemical warfare agents (NAs) in Iranian veterans early after exposure.

Methods: A total of 25 male NAs-exposed cases were enrolled in the present study. We used the medical records of early combat in 1990 for data collection. The data of the exposed-victims were unavailable for a long time. Clinical features were evaluated and fasting blood glucose, sodium, potassium, insulin level, and protein electrophoresis were measured on the first day of exposure.

Results: Potassium, sodium, fasting blood glucose, and protein electrophoresis were abnormally noticeable on the first day post-exposure. The clinical features of the studied cases were manifested in the muscarinic and nicotinic systems, and Central Nervous Systems (CNS). Miosis, cramp, restlessness, and respiratory symptoms were dominant features in the study subjects.

Conclusion: The investigated exposed-victims of NAs indicated hypokalemia, hyperkalemia, hypoglycemia, normal insulin levels, and abnormal protein electrophoresis. The clinical features of the cholinergic crisis were markedly significant in three levels of the muscarinic, nicotinic, and CNS manifestations. Our findings suggested that the exposed cases were against a significant dose of NAs.

[*] **Corresponding Author:**
Agin Khosrow, MD.
Address: *Loghman-e Hakim Hospital, Shahid Beheshti Unuversity of Medical Sciences, Tehran, Iran.*
Tel: *+98 (21) 55413424*
E-mail: *agin@sbmu.ac.ir; khosrow.agin@yahoo.com*

1. Introduction

Toxic gas inhalation syndrome addresses the effects of exposed- humans inhaling chemical gases. The Chemical Warfare Gasses (CWGs) are a set of different chemical compounds, i.e., associated with induced toxicity in human biology. They are employed as weapon agents and have be changed to a gas state in certain physical conditions. They are classified based on the target organs, such as nerve, vesicant, lung-damaging, blood, incapacitating psychochemical, lacrimators, and vomiting agents [1]. Its effects on human biology depend on either the nature of the gas or its exposed volume. There are a great number of experiences of the outcomes and complications of chemical exposure worldwide as mass destructive agents; they play the most significant role as battlefield weapons in warfare and terrorism [2, 3]. The world's first experience of the CWG exposure was related to the first world war (1914) and using chlorine gas, i.e., considerably accompanied by casualties and mortalities. A gas arms race has been developed in the past century, with great diversity. Aside, it was used by Iraq's and Iran's combatants against civilians and the army on large scales in the modern era (1980-88 years) [4-6].

Research about the manufacture of synthetic insecticides began in the Nazi German government in 1934. Two years later, the production line of new organophosphate compounds began by chemist Gerhard Schrade in 1936. The first products being tabon and sarin, and later soman. These war gases are called G-nerve weapon agents because of their biological function [7]. Producing these compounds, i.e., known as the substances of mass murder or mass destruction was high and their stockpiles were estimated to be tabon 9000, tones, sarin1300 tones, and soman 20 tones until 1945 [8].

Sarin and Tabun were used as CWG for the first time during world war I in 1939 and the Iraq-Iran conflicts in 1980-88 [9]. They are an organophosphorus Nerve Agent (NA) that binds with the active site of the cholinesterase enzyme in the synaptic junction of the nervous system. The NAs accumulate neurotransmitter acetylcholine and globally lead to the overstimulation of nervous system functions in the exposed victims. Our initial knowledge and experience about the use of NAs are related to the Iraq-Iran war; this information was focused on the clinical features of intoxication, such as the overstimulation of cholinergic system manifestations.

The present study aimed to assess the clinical features and laboratory findings of the acute poisoning manifestations of organophosphorus chemical warfare agents (NAs) in Iranian veterans early after exposure.

2. Materials and Methods

The present cross-sectional and descriptive study was conducted at Imam Hossein general teaching Hospital affiliated to Shahid Beheshti University of Medical Sciences, Tehran City, Iran.

The sample study enrolled those who were coming from the Soman conflict area in 1990. They were a small group of chemical-exposed combatants, i.e., immediately transferred in a few hours from the soman (Kordestan Province) to Imam Hossein Hospital in Tehran by the airport force. It was a territory center for the chemical treatment of Iraq-Iran combatants in 1987. This center supported the war victims, especially chemical injury cases. This research was conducted during the initial chemical attacks of Iraq against Iran. The present study information was missed after collecting data in wartime for a long-time and was unavailable from 1987 to 2018. Fortunately, a part of the information was available during the past year. The type of used NAs was not exactly known. In those years, diagnostic capabilities were very insignificant; however, it was presumed soman or tabun. Evidence of the use of NAs in the Persian Gulf war has also been considered [10-12].

Fighters were educated for preventing the effects of exposed-chemical hazards before being registered for the war. They were popular volunteer forces who had received military training for the war. They were not interested in using wearing protective cloth, gas masks, and antidotes as auto-injection atropine. The triage of chemical-exposed veterans has encompassed admission at a special ward, fulfill the history of exposure events, with management based on gas war types. The most popular WCG was the nerve and mustard gases in those days that were used as the mass destructive agents against Iranian forces. Laboratory data were used in the study related to the first day-visit at the hospital admission.

The clinical features of the NAs were discovered by physicians and perhaps fighters; they were documented by the chemical detoxification teams at the conflict area. All chemical victims underwent the standard laboratory examination as CBC diff, Fasting Blood Sugar (FBS), electrolytes, kidney, and liver function test, and so on. Chest imaging was also conducted on them. The low FBS level in serum of exposed victims led to further

evaluation. We had not much experience concerning chemical exposures. Therefore, To understand its occurrence, we had additionally measured insulin levels, repeated the other laboratory data, and protein electrophoresis by the one extraordinary laboratory, and followed it up with the veterans for several days.

The data were analyzed by the SPSS v. 28. The study variables were summarised by Mean±SD as well as frequency and percentage. The normality of the data was evaluated using Kolmogorok-Smirnov test (P=0.2). Comparing mean scores was performed by one-way Analysis of Variance (ANOVA) and Independent Samples t-test. The tables and charts were created applying Excell software. The statistical significant value was set as P<0.05.

3. Results

The Mean±SD White Blood Cell (WBC) profile was as follows: 9.1±5.2×10^3 µL, poly: 59.5±15.2%, lymphocytes: 36.2±1.5%, and eosinophil: 2.4±1.2%. The Mean±SD Red Blood Cell (RBC) profile consisted of RBC: 5.7±1.1^6/µL, hemoglobin: 16.1±1.2 g/dl, hematocrit: 4±3.4%, and platelet:151.8±65.410^3/µL.

Table 1 discloses the characteristic of sodium, potassium, and FBS levels in three consequent days of the onset of exposure to Organophosphorus Chemical Weapon Agents (OPCWAs). Abnormal FBS, potassium, and sodium levels were detected in the serum of 5(2.8%), 8(33%), and 1(4%) cases, respectively on the first day of exposure. Figure 1 shows the frequency of electrolyte and FBS abnormalities in the exposed veterans. Potassium abnormalities included hyperkalemia in 21% and hypokalemia in 4% of the study cases. Potassium abnormalities were marked among exposed victims, especially on the second day. FBS abnormalities were found in the hypoglycemia range. The insulin levels were in a healthy range. There was no statistically significant difference between insulin level and serum sodium, potassium, and FBS levels in the consequent days (P>0.05).

Table 2 presents the resulting protein electrophoresis assay among the exposed victims. Protein electrophoresis reflected an abnormality in the bands of alpha1, alpha 2, beta, and gamma. Figure 2 illustrates the distribution of frequency protein electrophoresis abnormalities in different bands. The bands of alpha 2, beta, and gamma were raised in the value and alpha1 was declined.

Figure 3 reveals the frequency distribution of peripheral muscarinic signs in the veterans. Figure 4 presents the frequency of peripheral nicotinic signs in the veterans. Figure 5 demonstrates the Central Nervous System (CNS) signs in the veterans.

4. Discussion

NA are toxic chemical compounds, used as mass destruction weapons. NA are divided into two groups of G series and V and include tabun GA (1937), sarin GB

Table 1. The characteristics of sodium, potassium, and FBS levels in three consequently days of the onset exposure to OPCWAs

Laboratory Data	Mean±SD	Median	Mode	Rang	Min	Max	P
Fasting blood glucose/First day	69.6±13.6	68.6	70	40	50	90	0.6
Fasting blood glucose/Second day	93.9±2.0	95	95	98	60	158	0.003
Fasting blood glucose/Third day	97.9±11.8	86	76	40	72	112	0.001
Serum sodium	140.7±0.5	140.4	140	13	135	148	0.001
Serum potasium/ First day	4.2±0.5	4.2	4.2	2.4	3	5.1	0.04
Serum potasium/Second day	4.5±0.6	4.4	4.2	2.4	3.5	5.9	0.001
Serum potasium/Third day	4.3±0.4	4.2	4	1.7	3.6	5.3	0.001
Serum insulin level	10.1±3.0	10	7.5	14	5	19	0.001

Table 2. Profile resulting protein electrophoresis assay among exposed victims

Protein Electrophoresis	Mean±SD (mg/dl)	Median	Mode	Range	Min	Max	P
Albomin	59.7±4.0	60	56.5	13	51	65	0.2
Alpha 1	3.9±0.6	3.8	3.6	2.3	3.1	5.4	0.001
Alpha 2	7.7±1.4	7.6	7.1	7	5	12	0.001
Beta	11.4±1.8	11.8	10.7	8.9	5	13	0.001
Gamma	16.8±2.5	16.8	16.8	8.4	12	21	0.003

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(1938), soman GD (1944), and cyclosarin GD. Except for sarin (cyanide, o-isopropyl methyphosphonofluoridate), all of them are organophosphorus compounds [10]. The chemical properties of the G-type gases easily change from liquid to gas (more volatile agents), colorless, tasteless, odorless, and extremely neurotoxic. The Lethal Dose (LD50) was 38 µg/kg for soman and 41 µg/kg for sarin [11].

The main mechanism of NA is applied through irreversible covalent inhibitor binding with Acetylcholinesterase (AchE) in the central and peripheral synaptic junctions of the nervous system in the mammalian. AchE regulates the neurotransmitter of acetylcholine [12]. Exposure to NAs inhibits AchE action and increases the acetylcholine levels at the synaptic junctions of the nervous system at the toxic threshold level.

The production and use of war gases were banned in the convention of chemical weapons in 1993. However, war gas was used again in the role of urban terrorism in the Matssomato, Tokyo subway [13]; ultimately, they opened a new chapter to the threat attacks in the world. Sarin was used as mass destruction in the Halabja (Kordistan, Irag 1988), Ghouta, and Khan Shakhunin of Syrian in 2013 and 2017, respectively. The causality rate was estimated to be 1400 civilians [11].

The data gathering of exposed-subjects was objective during wartime; thus, we had inadequate knowledge about chemical attacks in 1988. The tabun and soman were identified by the odors of fruity and camphor, respectively [14]. The strategy against exposure intoxication included decontamination, detoxification, and transfer to the territory centers management. The principal managements used atropine (a cholinergic agonist) as auto-injection, benzodiazepine (for seizure prevention), and oxime antidote (cholinesterase reactivator) [15].

The clinical manifestations of the exposed victims are directly related to the level of intoxication and decontamination protocols. The exposed victims present acute

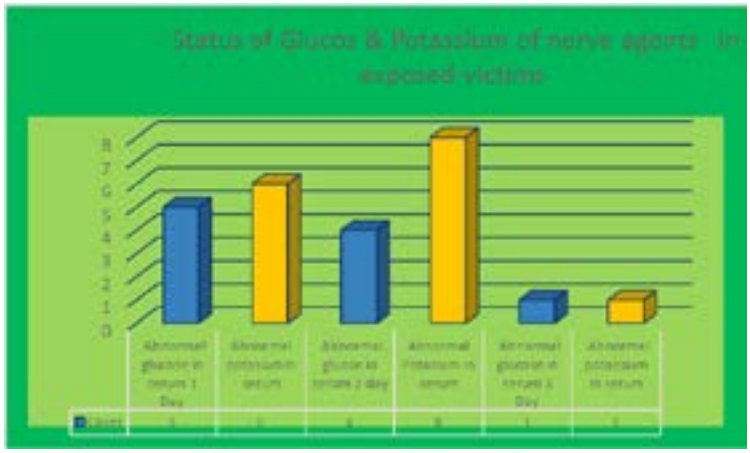

Figure 1. The status of glucose and potassium of NAs in the exposed cases

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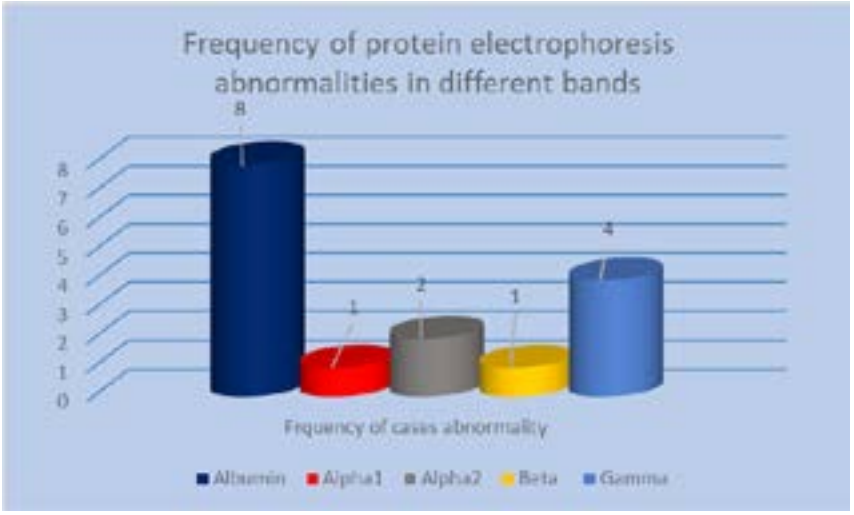

Figure 2. The distribution of frequency protein electrophoresis abnormalties in different bands

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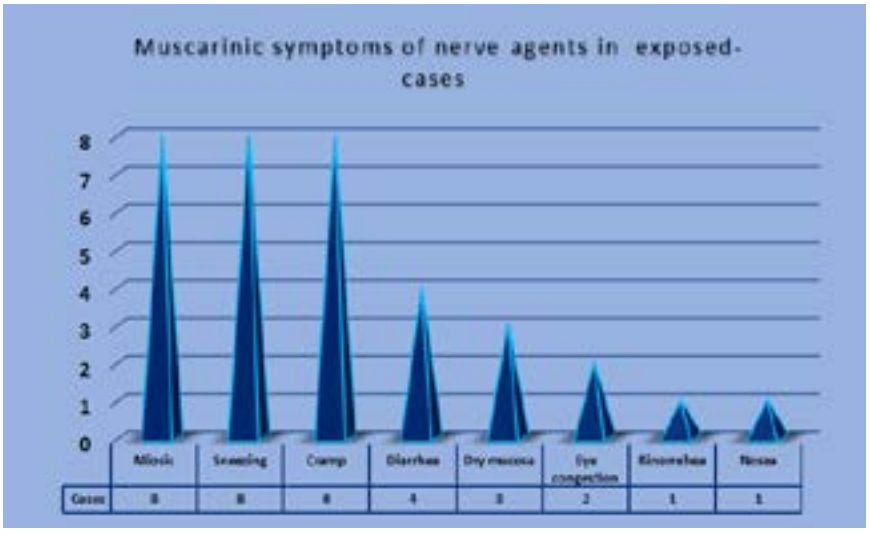

Figure 3. The muscarinic symptoms of NAs in the exposedcases

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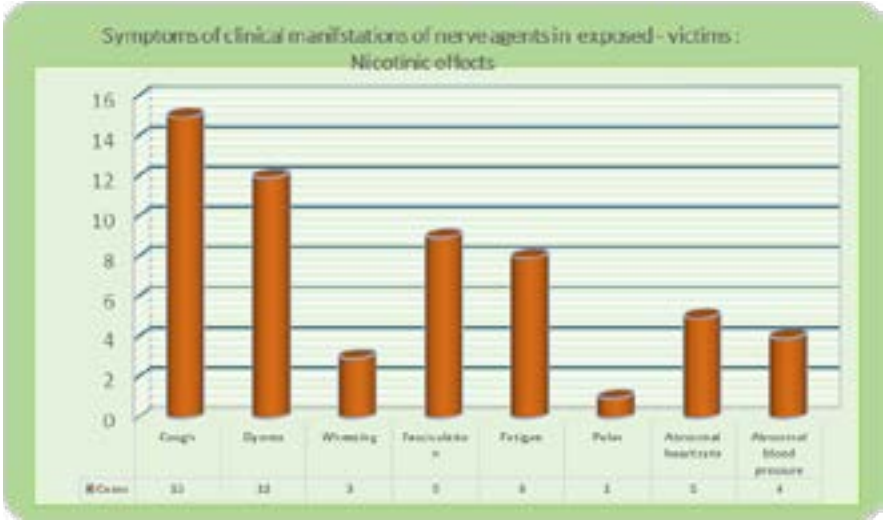

Figure 4. The status of the frequency peripheral nicotinic signs in the exposed veterans.

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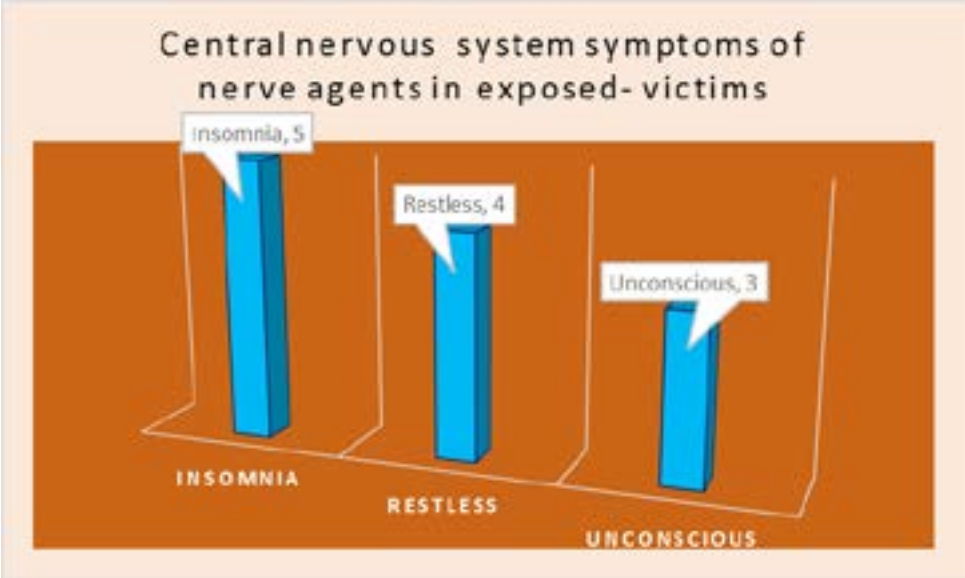

Figure 5. The status of the CNS signs in the exposed veterans

clinical features of muscarinic, nicotinic receptors stimulation, and CNS effects [16, 17].

The sample population was transferred immediately after exposure. Therefore, they presented considerable laboratory data abnormalities and established clinical features.

The main route of NA action is the respiratory system and its clinical features are based on the accumulation of acetylcholine in the CNS and peripheral nervous synaptic junctions [18]. NA is very neurotoxic and causes seizures, respiratory failure, and death. Besides, the peripheral signs are associated with the overstimulation of the muscarinic and nicotinic receptors (Cholinergic crisis) [18]. Besides, sarin has indirect effects on human health as chronic neurotoxicity, endocrine toxicity, immunotoxicity, and organophosphorus ester-induced delay neurotoxicity [18, 19].

Our results indicated hypoglycemia in the cases. The neuroendocrine effects of soman were evaluated in the animal lab; they indicated that the endocrine effects are dose-dependent and associated with the increased levels of glucose, corticosterone, norepinephrine, epinephrine, glucagon, Adrenocortical Hormone (ACTH), and declined insulin levels [20-22]. Hypoglycemia may occur due to the combined effect of hormonal endocrine abnormal secretions and decrease hypothalamus AchE activity [20, 21]. It may be concluded from the recent studies that the explored veterans were exposed to moderate and even to high doses of NAs.

Evaluating protein electrophoresis assay was the new concept in investigating NAs. Our results indicated that the alpha 1 value was decreased and the other parameters were increased to the standard values. It may be due to the acute stress of NAs-exposed victims, acute inflammation, and interference with acute-phase reactants. No clear interpretation of gamma-band abnormality is available.

Electrolyte abnormalities were a rare topic, i.e., reported in the NAs cases. Our results indicated that hypokalemia and hyperkalemia were detected among exposed cases. Hypokalemia was found among Matsumoto exposed cases [23]. Transient hypokalemia has improved in a few days and was a prominent feature among exposed victims. Hyperkalemia was a new concept in the endpoint of the study. We have no comment on this finding and no record was found in the recent studies (anima & human). The laboratory abnormalities may be affected by the causal effects of systematic involvement.

The muscarinic impact of NAs is related to the intensity of cholinergic activity (cholinergic crisis). They included miosis, blurred eye vision, tearing, copious respiratory secretions, bronchorrhea, bronchospasm, nausea, vomiting, diarrhea, abdominal cramp, bradycardia, frequent urination, and incontinence [24]. The first symptoms of NAs were reported in exposed cases among the citizen of Matsumoto, Japan; sneezing, copious salivation, and rhinorrhea, gradually became associated with blurred vision and impaired consciousness over time. Several clinical features of NAs are more stable and last longer in the victims. Miosis, fasciculation, abnormal rhythm, and blood pressure abnormality were detected over 24

hours of the onset of exposure [25]. Besides, miosis is the first sign of sarin exposure [26]. The present study outcomes have also improved with the recent report of human exposure symptoms.

The nicotinic clinical features of NAs are in the three levels; sympathetic system, striated muscles, and respiratory system. They consist of transient vasoconstriction associated with raising blood pressure, followed by hypotension, pallor, twitching, cramp, fatigue, respiratory failure, and death [27, 28]. Respiratory symptoms were markedly considerable in our cases. It included cough, dyspnea, and wheezing symptoms and may suggest the severity of exposure to the NAs.

Organophosphorus compounds have four distinct neurotoxicities. They include acute cholinergic crisis, intermediate syndrome, organophosphorus-induced delayed neuropathy, and chronic neurotoxicity [29]. Serious CNS system manifestations are incorporated into the suppression of respiratory center activity and circulatory collapse. Late-Life effects may appear with behavior and cognitive abnormalities. Neurobehavioral function abnormality was reported by NAs [30]. Our case finding was compatible with an acute cholinergic crisis. The absorption of NAs rapidly occurs through the respiratory system, eyes, and percutaneous with delay [31]. The acute symptoms of CNS are dose-response. Insomnia, restless or agitation, and unconsciousness symptoms were the significant observations of the current study. All evidence reflected using a considerable dose of NAs among Iranian victims.

5. Conclusion

Our NAs-exposed victims indicated hypokalemia, hyperkalemia, hypoglycemia, normal insulin levels, and abnormal protein electrophoresis. The clinical features of the cholinergic crisis were markedly significant in three levels of the muscarinic, nicotinic, and CNS manifestations. Our study findings may suggest that the exposed cases were against a significant dose of NAs.

Ethical Considerations

Compliance with ethical guidelines

All ethical principles are considered in this article. The participants were informed about the purpose of the research and its implementation stages. They were also assured about the confidentiality of their information and were free to leave the study whenever they wished, and if desired, the research results would be available to them.

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Author's contributions

Data collection, designing research, writing: Khosrow Agin; Editing and writing – review & editing: Babak Mostafazadeh.

Conflict of interest

The author declared no conflicts of interest.

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