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RESEARCH

Factors Affecting Thrombocytopenia in Patients with Pesticide Intoxication

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Abstract

Objective This study aimed to investigate the factors affecting the occurrence of thrombocytopenia in patients due to pesticide intoxication.

Methods A cross-sectional study was conducted from 2016 to 2021. Data were collected from patients admitted with acute pesticide poisoning, focusing on demographics, intoxication features, clinical outcomes, and laboratory findings. Patients were classified into three groups based on the day of platelet reduction: first day, second day, and third day onwards. Severity of platelet reduction was measured. Statistical analysis included descriptive statistics, comparative analysis, and logistic regression [odd ratio (OR), 95% confidence interval (CI)].

Results Of the 235 patients included, the majority were male (76.4%), with insecticides being the most frequent pesticide encountered. All patients had reduced platelet counts, but at different days and severity levels. Higher mortality rates and longer hospital stays were observed in the group with the presentation of thrombocytopenia on the third day or later. When comparing the third day to the first day, the administration of vitamin C medication [OR (95% CI), 7.664 (1.093–53.747), $p=0.04$] and the use of pantoprazole medication [OR (95% CI), 3.08 (1.257–7.543), $p=0.014$] were found to impact thrombocytopenia occurrence. Patients who were admitted to the intensive care unit had a 2.6-fold higher probability of developing thrombocytopenia [OR (95% CI), 2.635 (1.11–6.25), $p=0.02$]. There was no significant difference in the odds of more severe thrombocytopenia based on onset timing.

Conclusion This study sheds light on factors associated with the occurrence of thrombocytopenia, emphasizing the need for targeted interventions and further research to improve patient outcomes.

Keywords Pesticide · Thrombocytopenia · Mortality · Intoxication

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Introduction

Acute poisonings refer to harmful health effects that occure immediately after exposure to dangerous substances. This can include pharmaceuticals, excessive drug use, misuse of drugs, contact with chemicals, exposure to occupational and environmental toxins, biological agents, and envenomation [1, 2]. Intoxications are significant public health concerns that are often preventable, leading individuals to seek assistance from emergency departments [3]. Recent investigations have revealed that intoxications constitute a notable proportion of emergency department visits [4–6]. Pesticides are a prevalent cause of intoxication globally, including in Iran [7, 8].

Pesticides, whether chemical or natural, are widely used in agriculture to manage pests such as insects, birds, or microorganisms that harm plant diseases or impede weed growth [9, 10]. It is important to note that pesticides are also commonly used outside of agriculture to control pests in homes, combat disease-carrying insects, and maintain gardens. All pesticides have inherent toxicity and pose significant risks to human health and the environment, particularly when used extensively without proper safety measures [11]. The increase in pesticide production and use is driven by the need to improve agricultural output [12, 13]. The World Health Organization (WHO) has identified pesticide poisoning as a leading cause of severe toxicity and fatalities from acute poisoning, resulting in over 300,000 deaths annually [14]. Farmers are often exposed to pesticides through skin contact, inhalation, and ingestion, potentially leading to intoxication [15]. Due to high toxicity, pesticides cab be used in suicide attempts, increasing mortality and morbidity rates [16]. Pesticides have various harmful effects on human health, affecting the nervous system, skin, lungs, eyes, immune system, and reproductive system and increasing the risk of cancer [17]. Various studies have indicated that pesticide intoxication can adversely affect hematological parameters, leading to changes in blood cell counts, red blood cell parameters, and inflammation factors [18]. One potential effect of pesticide intoxication is thrombocytopenia, although research in this area is limited and there is a lack of studies examining the impact of thrombocytopenia on patient outcomes [19, 20]. Therefore, this study aims to investigate the factors influencing the occurrence of thrombocytopenia in patients, focusing on clinical demographics and its relationship to treatment outcomes.

Methodology

This cross-sectional study investigated the etiology of thrombocytopenia in patients with acute pesticide poisoning. The study was conducted at Khorshid Hospital, affiliated with Isfahan University of Medical Sciences, from 2016 to 2021. The study population comprised patients admitted to the poison ward or intensive care unit (ICU) of Khorshid Hospital. Ethical approval was obtained from the Ethics Committee of Isfahan University of Medical Sciences, and the study was reported in accordance with the STROBE statement.

All patients meeting the eligibility criteria during the study period were included. The inclusion criteria were as follows: patients with acute pesticide poisoning, including organophosphate, paraquat, zinc phosphide, and aluminum exposure; age over 18 years; no history of platelet disorders; and no history of antiplatelet drugs. The exclusion criterion was the lack of daily platelet reports in patients' clinical records.

Data collection in this retrospective study utilized a checklist comprising nine main sections. The first section assessed demographic variables such as age and sex. The second section evaluated intoxication features, including poison type, amount ingested, time of ingestion, time of admission, and route of exposure. Subsequent sections investigated past medical history, clinical outcomes, and clinical and laboratory findings at admission. Laboratory investigations encompassed hematological, hepatological, and nephrological profiles. Vital signs ion admission including blood pressure, heart rate respiratory rate, and O2 saturation were measured. Therapeutic measures, including gastric lavage, activated carbon administration, hemodialysis, medications, oil gavage, antibiotics, and anticoagulants, were also documented. Intoxication severity was assessed based on clinical symptoms, categorizing patients into latent, mild, moderate, or severe intoxication groups. Thrombocytopenia was defined as a platelet count less than 150×10^3 per microliter at admission and during hospitalization. Thrombocytopenia was classified based on its severity into three groups: mild (platelet: 100,000–150,000), moderate (platelet: 50,000–100,000), and severe (platelet < 50,000) [21].

Patients were stratified into three groups based on the day of platelet reduction: first day, second day, and third day onwards. Concurrent intoxications and pre-hospitalization medical interventions were examined. Data were extracted from patients' clinical records and entered into Excel.

Statistical analysis was performed using SPSS v26. Categorical variables were described using frequency and rate, while continuous variables were summarized using mean and standard deviation. A post hoc Bonferroni correction was employed to facilitate comparisons among groups. Additionally, multinomial logistic regression analysis was undertaken to assess the impact of various factors on the incidence of thrombocytopenia. A 95% confidence interval was assumed for all statistical tests.

Additionally, to specifically compare platelet dynamics at the time of thrombocytopenia onset across Day 1, Day 2, and Day 3 + groups, we fitted a linear mixed-effects model by restricted maximum likelihood (REML) with random intercepts for each subject and fixed effects for onset-day and time. Estimated marginal mean platelet counts at onset were extracted, and Sidak-adjusted post hoc pairwise comparisons tested Day 1 vs. Day 2, Day 1 vs. Day 3 +, and Day 2 vs. Day 3 + differences. For severity analyses—categorized by nadir platelet count into mild, moderate, and severe—we first used a chi-square test of independence to assess distributional differences across onset-day groups. We then employed a cumulative-link mixed-effects (ordinal logistic) model, incorporating patient-specific random intercepts and onset-day as a fixed effect, to estimate odds ratios for increasing thrombocytopenia severity among the three groups.

Results

A total of 235 patients were identified during the enrollment period. Demographically, the majority of patients were male (76.4%). The most frequently encountered pesticides among patients were insecticides, herbicides, and rodenticides, respectively. The mean age of the patients was 36.23 ± 16.83 years. All patients exhibited reduced platelet counts, with the majority experiencing this reduction on the first day of hospitalization.

Comparisons among these groups revealed that the mean age of patients was higher in the third day and subsequent groups. Predominantly male and married individuals were observed across all groups, with a high incidence of intoxication during the spring season. Additionally, most patients resided in suburban areas of Isfahan. A significant difference was observed in terms of pesticide type based on the days when thrombocytopenia occurred ($p = 0.009$). Insecticides were most prevalent in the first- and second-day groups, while herbicides predominated in the third-day group. Clinical outcomes analysis revealed a significantly higher mortality rate among patients in the third-day and more groups. Moreover, patients in the third-day group experienced significantly longer hospital stays. However, no significant association was found between toxicity severity and the day of platelet reduction. Further details can be found in Table 1.

Table 1 Comparative analysis of demographic, toxicological factors, length of stay and outcomes across different days of thrombocytopenia occurrence

Variable		Total	First day	Second day	Third day and more	*p*-value
Age			35.55 ± 15.56	35.47 ± 15.73	37.54 ± 19.03	
Sex	Male	180 (76.6%)	75 (42%)	39 (22%)	66 (36%)	0.349
	Female	55 (23.4%)	29 (52.7%)	10 (17.5%)	16 (28%)	
Pesticide type	Insecticide	100 (42.6%)	52 (52%)	19 (19%)	29 (%29)	0.009
	Herbicide	57 (24.3%)	14 (24.6%)	11 (19.3%)	32 (56.1%)	
	Rodenticide	66 (28.1%)	32 (48.5)	16 (24.2%)	18 (27.3%)	
	Other	12 (5.1%)	6 (50%)	3 (25%)	3 (25%)	
Marriage status	Married	162 (68.9%)	73 (45.1%)	37 (22.8%)	52 (32.1%)	0.135
	Single	70 (29.8%)	31 (44.3%)	12 (17.1%)	27 (38.6%)	
	Divorced	3 (1.3%)	0	0	3 (100%)	
Job status	Farmer	10 (4.3%)	3 (30%)	2 (20%)	5 (50%)	0.733
	Factory or service jobs	156 (66.4%)	67 (42.9%)	32 (20.5%)	57 (36.5%)	
	Housekeeper	49 (20.9%)	25 (51%)	9 (18.4%)	15 (30.6%)	
	Jobless or student	20 (8.5%)	9 (45%)	6 (30%)	5 (25%)	
Season of poisoning	Spring	74 (31.5%)	32 (43.2%)	20 (27%)	22 (29.7%)	0.462
	Summer	53 (22.6%)	26 (49.1%)	11 (20.8%)	16 (30.2%)	
	Fall	55 (23.4%)	25 (45.5%)	7 (12.7%)	23 (41.8%)	
	Winter	53 (22.6%)	21 (39.6%)	11 (20.8%)	21 (39.6%)	
Toxicity severity	Latent or mild	168 (71.5%)	76 (45.2%)	37 (22%)	55 (37.2%)	0.813
	Moderate	29 (12.3%)	12 (41.4%)	6 (20.7%)	11 (37.9%)	
	Severe	38 (16.2%)	16 (42.1%)	6 (15.8%)	16 (42.1%)	
Outcome	Death	58 (25.2%)	21 (36.2%)	9 (15.5%)	28 (48.3%)	0.021
	Recovery with no complications	172 (74.8%)	83 (48.3%)	40 (23.3%)	49 (28.5%)	
Length of stay(days)			2.88 ± 6.13^{a}	6.02 ± 7.93^{b}	11.6 ± 13.2^{c}	< 0.001
Length of stay(h)			71.21 ± 147.21^{a}	145.10 ± 191.81^{b}	276.47 ± 317.02^{c}	< 0.001

Different superscript letters represent significant differences in reported values

Variations in clinical laboratory parameters were noted with the only significant change being observed in the WBC and RBC count. Indeed, a prominent reduction in count was observed on the first day. Mean ± SD of WBC was 8.77 ± 3.68, 11.02 ± 4.98, and 12.52 ± 5.6 on the first day, second day, and third day and more, respectively (p value = 0.000). Vital signs remained unchanged across patients. Regarding therapeutic intervention, patients in the third-day group exhibited a significantly higher requirement for treatments and medications (NAC, vitamin C, vitamin E, corticosteroid therapy, pantoprazole, intubation, dialysis, and ICU admission). Comparative analysis of various variables is summarized in Table 2.

A logistic regression analysis was conducted to assess the influence of various therapeutic and toxicological factors on the occurrence of thrombocytopenia on the second and third days compared to the first day. Notably, in the analysis comparing the second day to the first day, the use of pantoprazole medication exhibited a significant effect. Specifically, the regression analysis revealed that patients administered pantoprazole had a threefold increased likelihood of developing thrombocytopenia compared to patients not receiving pantoprazole medication [OR (95% CI), 3.004 (1.193–7.567) $p = 0.02$].

Similarly, in the analysis comparing the third day to the first day, several factors including ICU hospitalization, administration of vitamin C medication [OR (95% CI), 7.664 (1.093–53.747), $p = 0.04$], and use of pantoprazole medication [OR (95% CI), 3.08 (1.257–7.543), $p = 0.014$] were found to impact thrombocytopenia occurrence. The regression analysis indicated that patients hospitalized in the ICU were associated with a 2.6-fold higher probability of developing thrombocytopenia [OR (95% CI), 2.635 (1.111–6.25), $p = 0.028$]. Details are available in Table 3.

Analysis of platelet count dynamics and thrombocytopenia severity by onset day was performed. Using a linear mixed-effects model fitted by REML with random intercepts for subjects and fixed effects of onset-day (Day 1, Day 2, Day 3 +) × time, the estimated marginal mean platelet counts at the day of thrombocytopenia onset were $118.4 \pm 6.5 \times 10^3/\mu L$ for Day 1, $132.7 \pm 7.1 \times 10^3/\mu L$ for Day 2, and $146.3 \pm 5.8 \times 10^3/\mu L$ for Day 3 +, with a significant interaction term ($p = 0.002$). Post hoc Sidak-adjusted pairwise tests revealed that Day 1 counts were significantly lower than Day 2 (mean difference $- 14.3 \times 10^3/\mu L$, $p = 0.031$) and Day 3 + ($-27.9 \times 10^3/\mu L$, $p < 0.001$), and that Day 2 was lower than Day 3 + ($- 13.6 \times 10^3/\mu L$, $p = 0.019$). These findings confirm that patients with earlier-onset thrombocytopenia experience more severe platelet depletion.

Among the 241 patients (6 missing severity), 104 (43.2%) had thrombocytopenia onset on Day 1, 49 (20.3%) on Day 2, and 82 (34.0%) on Day 3 or later. When classified by nadir platelet count into mild ($100–149 \times 10^3/\mu L$), moderate ($50–99 \times 10^3/\mu L$), or severe ($< 50 \times 10^3/\mu L$), the Day 1 group included 58/104 (55.8%) mild, 33/104 (31.7%) moderate, and 13/104 (12.5%) severe cases; Day 2 had 28/49 (57.1%) mild, 15/49 (30.6%) moderate, and 6/49 (12.2%) severe; and Day 3 + had 47/82 (57.3%) mild, 23/82 (28.0%) moderate, and 12/82 (14.6%) severe. A chi-square test of independence revealed no significant difference in the distribution of thrombocytopenia severity across the three onset-day groups ($\chi 2(4) = 0.25$, $p = 0.99$). In a cumulative-link mixed-effects (ordinal logistic) model with the patient as a random intercept and onset-day group as a fixed effect, there was no significant difference in the odds of more severe thrombocytopenia by onset timing: Day 2 vs. Day 1 OR = 0.98 (95% CI 0.59–1.63, $p = 0.94$) and Day 3 + vs. Day 1 OR = 1.22 (95% CI 0.67–2.21, $p = 0.52$). Thus, although the raw counts show

Table 2 Comparative analysis of therapeutic intervention across different days of thrombocytopenia occurrence

Variable	First day	Second day	Third day and more	p-value
Charcoal therapy	71 (48.6%)	26 (17.8%)	49 (33.6%)	0.167
Gastric lavage	63 (45.3%)	27 (19.4%)	49 (35.3%)	0.806
Gavage paraffin	8 (47.1%)	6 (35.3%)	3 (17.6%)	0.180
Pralidoxime	22 (33.3%)	16 (24.2%)	28 (42.4%)	0.107
Atropine	25 (33.8%)	17 (23%)	32 (43.2%)	0.079
NAC	33 (27.5%)	31 (25.8%)	56 (46.7%)	< 0.001
Vitamin C	23 (23%)	26 (26%)	51 (51%)	< 0.001
Vitamin E	21 (21.9%)	25 (26%)	50 (52.1%)	< 0.001
Corticosteroid therapy	33 (28.4%)	26 (22.4%)	57 (49.1%)	< 0.001
Pantoprazole	52 (31.7%)	40 (24.4%)	72 (43.9%)	< 0.001
Intubation	31 (29.8%)	22 (21.2%)	51 (49%)	< 0.001
Dialysis	10 (18.9%)	11 (20.8%)	32 (60.4%)	< 0.001
ICU admission	30 (25.4%)	26 (22%)	62 (52.5%)	< 0.001

NAC N-acetyl cysteine, *ICU* intensive care unit

Table 3 Logistic regression analysis of factors associated with thrombocytopenia occurrence on subsequent days compared to first day

Variables			OR (CI %95)	*p*-value
Second day	Pesticide type	insecticide	0.618(0.117–3.273)	0.571
		herbicide	0.287(0.035–2.379)	0.247
		rodenticide	0.118(0.012–1.118)	0.063
		other	1	
	Severity	Latent or mild	2.022(0.509–8.031)	0.317
		Moderate	1.568(0.332–7.396)	0.57
		Severe	1	
	Dialysis	Yes	0.296(0.037–2.403)	0.255
		No	1	
	ICU	Yes	1.287(0.507–3.269)	0.596
		No	1	
	Intubation	Yes	1.286(0.434–3.81)	0.65
		No	1	
	Atropine	Yes	1.97(0.717–5.412)	0.189
		No	1	
	NAC	Yes	3.536(0.669–18.692)	0.137
		No	1	
	Vitamin C	Yes	3.247(0.535–19.701)	0.201
		No	1	
	Vitamin E	Yes	1.432(0.292–7.033)	0.658
		No	1	
	Corticosteroid therapy	Yes	1.105(0.405–3.021)	0.845
		No	1	
	Pantoprazole	Yes	3.004(1.193–7.567)	0.02
		No	1	
	Intubation	Yes	1.286(0.434–3.81)	0.65
		No	1	
	Dialysis	Yes	0.296(0.037–2.403)	0.255
		No	1	
	ICU	Yes	1.287(0.507–3.269)	0.596
		No	1	
Third day and more	Pesticide type	insecticide	0.926(0.147–5.833)	0.935
		herbicide	0.503(0.059–4.275)	0.529
		rodenticide	0.145(0.014–1.526)	0.108
		other	1	
	Severity	Latent or mild	1.263(0.39–4.085)	0.697
		Moderate	1.171(0.312–4.403)	0.815
		Severe	1	
	Atropine	Yes	2.29(0.891–5.886)	0.085
		No	1	

Table 3 (continued)

Variables		OR (CI %95)	p-value
NAC	Yes	1.642(0.316–8.533)	0.555
	No	1	
Vitamin C	Yes	7.664(1.093–53.747)	0.04
	No	1	
Vitamin E	Yes	1.182(0.231–6.031)	0.841
	No	1	
Corticosteroid therapy	Yes	1.569(0.625–3.938)	0.337
	No	1	
Pantoprazole	Yes	3.08(1.257–7.543)	0.014
	No	1	
Intubation	Yes	1.431(0.532–3.851)	0.478
	No	1	
Dialysis	Yes	0.443(0.065–3.019)	0.406
	No	1	
ICU	Yes	2.635(1.111–6.25)	0.028
	No	1	

OR odd ratio, *CI* confidence interval, *NAC* N-acetyl cysteine, *ICU* intensive care unit

very similar severity proportions across groups, the longitudinal mixed-effects analysis confirms that onset-day does not independently predict thrombocytopenia severity.

Discussion

Pesticides encompass a diverse array of chemical compounds used for managing and eliminating various insect, fungal, herbaceous, and other pest species. These chemical agents have the potential to impact human health both acutely and chronically [22]. Human exposure to pesticides has long been recognized as a major public health issue, with approximately one million unintentional poisonings resulting in 20,000 deaths yearly as documented by the World Health Organization task force in 1990. An additional two million cases were expected due to intentional self-harm, particularly affecting individuals in developing countries where many cases are likely unreported, including an estimated 25 million occupational poisonings per year, the majority of which go undocumented [7].

The mean age of the patients in the current study was 36.23, which is higher than in a previous study conducted between 2016 and 2021 [23]. In terms of sex distribution, most of the patients were male, which was similar to past literature [19]. Men are predominant in both self-harm and unintentional poisoning cases, possibly because of their frequent exposure to pesticides, increasing their risk of occupational poisonings and self-harm due to their easy access to toxic substances [24]. The most frequent pesticide was insecticides, which was similar to a past study [23]; however, another national study in Malaysia showed that herbicides were the most frequent cause of poisoning. This difference could be explained by different geographical and agricultural statuses [25]. The mean time of hospitalization was 6.57 days, which was similar to the previous literature [26].

Previous studies show that pesticide exposure causes different conditions such as leukemia and multiple myeloma [26, 27]. Also, long-term exposure could alter blood cells and increase the number of RBCs, WBCs, and platelets [28]. But there is evidence supporting that pesticides could cause thrombocytopenia [29]. Acute thrombocytopenia is a consequence of pesticide acute intoxication with unknown pathophysiology. However, there is supporting evidence that certain types of treatment can affect this phenomenon [20]. Also, it is worth noting that there is limited evidence regarding thrombocytopenia's interaction with outcomes and the biological panel of patients with pesticide intoxication. Our study supports that a late reduction of platelets is associated with a higher rate of death. Prior investigations have shown that thrombocytopenia is a prognostic factor for death among ICU patients [30–32]. This phenomenon could be explained by a higher risk of cardiovascular mortality in patients with thrombocytopenia. Also, platelet count may have attributions to different prognostic factors like VEGF, which influences fibroblast and different vascular repair processes [33].

Additionally, our findings confirm that patients with earlier-onset thrombocytopenia experience more severe platelet depletion. This is consistent with previous results that have shown that, in pesticide intoxication, if thrombocytopenia progresses rapidly after exposure, it often indicated a more severe response. This could be due to the pesticide's direct toxic effects on the bone marrow or its ability to indue rapid platelet destruction [34].

Different studies have shown the impact of thrombocytopenia on ICU mortality. Also, our results showed an association between thrombocytopenia and ICU need in patients. The comparison of different laboratory findings of patients also showed no significant difference between the three groups. However, RBC and WBC count comparisons between groups showed a statistically significant difference, offering a probable interaction of these markers during intoxication.

We also estimated the predictive value of different clinical measurements on thrombocytopenia occurrence. The results of logistic regression showed that pantoprazole, ICU admission, and vitamin C are factors associated with thrombocytopenia.

There is supporting literature regarding pantoprazole-induced thrombocytopenia [35–38] [39]. The pathophysiology behind this phenomenon is not completely clear, but some research has proposed the effect of drug-induced antibodies and also direct toxicity of the drug as explanations for thrombocytopenia [38].

Mechanisms of PPI-induced cytopenia are unclear. The two most commonly accepted theories are the immune-mediated mechanism and toxic mechanism [38]. A case report hypothesized that this adverse effect may be immune mediated [39]. However, another case report presents a non-immune mechanism due to unresponsiveness to corticosteroid therapy [35].

The other factor was ICU admission. Thrombocytopenia is a common phenomenon among ICU patients, and there are different pathophysiological explanations for it. Infection and sepsis, different drugs, and different immunological processes such as immune-mediated platelet destruction and hemophagocytosis may affect the platelet count in patients [40–42].

Our results also showed the association of vitamin C on thrombocytopenia. Previous literature showed that vitamin C administration may cause platelet aggregation with no significant effect on PT or PTT [43–45].

While vitamin C at normal physiological concentrations is critical for PLT function, there is virtually no information on the impact of high concentrations of vitamin C on platelet function. Exposure of platelets to high doses of vitamin C alters endogenous production of lipid mediators by PLTs. These mediators could have unappreciated, yet far reaching impacts on not just PLTs function but on the entire circulatory system [43].

Furthermore, it is advisable to consider abstaining from administering clinical measurements that may potentially induce thrombocytopenia in patients exhibiting declining platelet trends, given the potential impact of such trends on patient mortality [32]. However, this assertion warrants further investigation through empirical studies.

Also, this is not without limitations. The monocentric nature of the study may bias the results, and a limited sample size could be suggested as a limitation of the current study.

Conclusion

This study sheds light on factors associated with the occurrence of thrombocytopenia in patients with pesticide intoxication, emphasizing the need for targeted interventions and further research to improve patient outcomes and mitigate the burden of pesticide-related poisonings.

Abbreviation WHO: World Health Organization

Author Contribution Author contributions Farzad Gheshlaghi: Conceptualization, Project administration, Funding acquisition, Data curation, Supervision, Writing- Reviewing and Editing Nastaran Izadi-Mood: Writing- Reviewing and Editing, Data curation Awat Feizi: Methodology, Software, Data Curation, Validation, Writing- Reviewing and Editing Rokhsareh Meamar: Conceptualization, Project administration, Data curation, Resources, Supervision. Visualization, Writing- Reviewing and Editing Shadi Hadad, Sima Heydari, Hosein Shafiee: Data collection, Data gathering, Investigation, All authors approved the final version of the manuscript.

NIM: writing—reviewing and editing, data curation;

AF: methodology, software, data curation, validation, writing—reviewing and editing;

RM: conceptualization, project administration, data curation, resources, supervision, visualization, writing—reviewing and editing;

ShH, SH, HSh: data collection, data gathering, investigation.

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Data Availability No datasets were generated or analysed during the current study.

Code Availability Not applicable.

Declarations

Ethics Approval and Consent to Participate This research has been performed in accordance with the Declaration of Helsinki and has been approved by the ethics committee of the Isfahan University of Medical Sciences (Ethics code: IR.MUI.MED.REC.1400.381).

Consent for Publication All authors approved the final version of the manuscript.

Competing interests The authors declare no competing interests.

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