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Prevalence and Associated Factors of Vasovagal Syncope among Medical Trainees in Jordan: A Cross-Sectional Study

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ABSTRACT

Background

Vasovagal Syncope (VVS) is common, yet its pathophysiology is incompletely understood. This study investigated the hypothesis that smoking, through its sympathomimetic effects, might be associated with a lower prevalence of VVS among medical trainees

Methods

A cross-sectional study was conducted using a self-administered online questionnaire distributed to Jordanian clinical medical students, interns, and junior residents. From 525 initial responses, 456 participants met the inclusion criteria. VVS was defined operationally based on self-report of episodes and typical triggers.

Keywords: Syncope; Vaso Vagal Syncope; Smoking; Pathogenesis; Autonomic Dysfunction; Smoking Effects; Cross-Sectional Study

INTRODUCTION

Syncope is defined as a self-limited, transient loss of consciousness [1]. Vasovagal syncope (VVS), also known previously as neurocardiogenic syncope, is the most common type, with 60-70% of all syncopes [2]. Though accurate pathophysiology remains poorly understood, the most documented mechanism is the autonomic dysfunction, sympathetic activity withdrawal, and increased parasympathetic tone, which leads to vasodilation, bradycardia, and decreased peripheral resistance, which eventually leads to cerebral hypo perfusion [3,4]. The trigger behind this initiation of autonomic dysfunction is still debated; it is said to be a defense mechanism against the brain stem shutting down by the higher centers or ventricular mechanoreceptors' paradoxical firing [5,6]. However, other sources debate it as a psychological issue, like anxiety that activates sympathetic stimulation [7]. Also, Family history and sex of the subject are important predictors of vasovagal syncope in offspring [8]. The prevalence of syncope for a lifetime of 70 years is 42%, with an annual rate of 6%. Moreover, the incidence ranges from 18.1 to 39.7 per 1,000 patients [9].

Medical students experience significant stress-related morbidity, with studies showing 40.6% suffer from nonrefreshing sleep, 31.1% from moderate-severe sleep disturbances, and 38.4% from insomnia - conditions strongly associated with stress (29.6%), anxiety (41.3%), and depression (44.6%)[10]. This population demonstrates concerning lifestyle habits, including a 49.5% smoking prevalence in Georgian medical students [11]. Vasovagal syncope (VVS) affects this group substantially, with a 2009 study of 630 clinical students reporting 12% prevalence (77 cases), showing marked female predominance (88%) and a mean age of 23 years (range 20-45).

Affected students included 22% graduate and 78% undergraduate candidates, with 57% (44/77) planning surgical careers, 9% (7/77) of whom were discouraged by their episodes. Primary triggers were hot environments (79%, n=61), prolonged standing (73%, n=56), surgical masks (47%, n=36), and diathermy smell (23%, n=18), while preventive measures included pre-theater nutrition (61%, n=47) and leg movement (18%, n=14) [12].

The pathophysiology involves NO-mediated vasodilation during VVS episodes (220% urinary cGMP increase versus 67% decrease in controls)[4], while smoking appears to modulate autonomic responses by reducing baseline vagal tone while enhancing sympathetic vasoconstriction, potentially offering cerebral perfusion protection during arrhythmias [13]. Familial studies reveal important predispositions, demonstrating 32% VVS prevalence among 62 medical students and 228 relatives, with a median first faint age of 14 years. Transmission patterns show stronger maternal inheritance (affecting both sexes) while paternal inheritance significantly impacts only sons.

Despite this background, the specific relationship between smoking and VVS risk remains unexplored in medical trainees.

This study aimed to:

1) determine the prevalence of VVS among Jordanian clinical medical students, interns, and junior residents;

2) test the hypothesis that smoking is associated with a lower prevalence of VVS due to its sympathomimetic effects;

3) identify other associated lifestyle and familial factors.

METHODS

Study Design and Sample Size

This cross-sectional study examined the association between syncope episodes and smoking propensity among clinical medical students, interns, and junior residents - a population particularly vulnerable to medical training stressors. Existing literature indicates stress prevalence of 31.7% among medical students and 73.0% among interns [14,15].

Sample size calculation was performed using G*Power 3.1 and EpiInfo software. For a Chi-square test with a 95% confidence level, 5% margin of error, and a 50% effect size, a minimum of 385 participants was required. We collected 525 responses. After applying exclusion criteria (cardiovascular diseases, anemia, diabetes mellitus, seizures, other syncope types, or incomplete questionnaires), the final analytical sample comprised 456 participants. Data were collected through an online self-administered English questionnaire using convenience sampling. The data were collected from 10th January 2025 to 16th Feb 2025.

Data Collection Instrument

The 30-item survey was organized into four domains: sociodemographic characteristics (age, gender, academic year), medical history (cardiac, neurologic, and related comorbidities), lifestyle factors (smoking habits, physical activity, family history), and vasovagal syncope characteristics (triggers, symptoms, duration, relieving factors). Question formats included yes/no, multiple-choice, checkbox, and limited open-ended items.

Questionnaire Development and Validation

Due to the absence of validated questionnaires simultaneously assessing vasovagal syncope and smoking, we developed a comprehensive instrument by: adapting items from validated questionnaires (identified through PubMed and Scopus searches) with authors' permission [9,16], incorporating established syncope assessment tools (e.g., Calgary Syncope Score) [17].The questionnaire underwent rigorous validation: expert review by impartial analysts and a consultant cardiologist to: eliminate double-barreled, ambiguous, or misleading questions, ensure scientific accuracy, and establish content and face validity. Then, pilot testing (n>30) to evaluate was conducted to evaluate readability and comprehension, construct validity, criterion validity, and internal consistency (reliability).

Operational Definitions

- Vasovagal syncope (VVS): Was defined operationally based on self-report of a transient loss of consciousness and/or typical pre-syncopal symptoms in response to common VVS triggers (e.g., fear, pain, prolonged standing, sight of blood) as per the questionnaire items with free medical history.

- Smoking status: Participants were classified as 'smokers' if they reported current use of cigarettes, vapes, or shisha.

Statistical Analysis

All analyses were performed in SPSS 26.0 (IBM). After excluding students aged > 30 years or with cardiovascular conditions (arrhythmias, orthostatic hypotension, first-degree AV block, hypertension, valvular heart disease), neurological disorders (epilepsy, other seizures), anemia or diabetes while retaining those with primary headaches 456 students remained for analysis; cases with any missing values were removed. Descriptive statistics were generated for every demographic and behavioral variable, and Pearson's χ^2 tests evaluated their individual associations with vasovagal syncope. A full-model(forced entry) binary logistic regression was then fitted with vasovagal syncope (0 = did not experience vasovagal syncope, 1 = experience vasovagal syncope) as the dependent variable, and age, weight, nightly sleep hours, smoking status, sex, caffeine use, gum consumption, family history of vasovagal syncope, number and type of smoking products, perceived stress, changes in smoking habits and physical-activity frequency entered simultaneously as predictors, Variable inclusion was based on theoretical relevance and prior findings in the literature.. Results are reported as B coefficients, standard errors, Wald χ^2 statistics, p-values, adjusted odds ratios (Exp B) and their 95 % confidence intervals. Logistic-regression assumptions were checked: model fit was adequate (Pearson χ^2 = 459.8, df = 425, p = 0.12; deviance χ^2 = 384.8, df = 425, p = 0.92) and all variance-inflation factors were < 2.5, indicating no multicollinearity.

ETHICAL CONSIDERATIONS

The study received ethical approval from the University of Jordan Institutional Review Board (IRB Code: 1667/2025/67). Key ethical provisions included: obtaining informed consent from all participants, ensuring participant anonymity and data confidentiality, guaranteeing voluntary participation with the right to withdraw, and emphasizing that there is no provision of short-term benefits or rewards.

RESULTS

The study analyzed data from 525 initially collected responses, with 456 participants meeting inclusion criteria after excluding those with anemia, diabetes, or seizure history. The final sample comprised 56.8% females and 43.2% males, with a mean age of 23.76 years (SD=1.97). Most participants were fourth-year medical students and interns, predominantly single (72.3%) and with normal BMI (58.1%) (Table 1). A statistically significant association was observed between gender and vasovagal syncope (VVS) occurrence (p=0.008), while BMI showed no significant relationship (p=0.393). The overall prevalence of self-reported VVS was 20.6% (n=94). Of the 121 smokers (26.5%), 22 (18.2%) reported VVS, compared to 72 of 335 nonsmokers (21.5%). No significant association was found between smoking status and VVS (χ^2=0.112, p=0.738). In multivariable analysis, significant independent predictors of VVS were female gender (Adjusted Odds Ratio [AOR]=1.92, p=0.012), positive family history (AOR=8.66, p<0.001), and engaging in weekly physical activity (AOR=0.30, p=0.004).

Smoking characteristics revealed that 26.5% (n=121) of participants were current smokers, among whom 18.2% (n=22) reported VVS episodes. Comparatively, 21.5% (n=72) of non-smokers (n=335) experienced VVS.

Multiple logistic regression analysis demonstrated no significant association between smoking status and VVS (Wald χ^2=1.729, p=0.188), though vaping showed borderline statistical significance (p=0.056) that may warrant further investigation with larger samples (Table 2). Cross-tabulation of VVS cases by smoking status revealed no meaningful distributional differences (Pearson χ^2=0.112, p=0.738), as illustrated in Figure 1.

Among the 94 confirmed VVS cases (population prevalence=20.6%), only 8.5% (n=8) had received a formal medical diagnosis, including 2 cases among smokers (Table 3).

To examine the effect of smoking status on the number of VVS episodes among the 94 students who had experienced syncope, a Poisson model was first estimated, but showed substantial over-dispersion (Pearson χ^2/df = 5.17); consequently, a negative-binomial model was applied. The negative-binomial model demonstrated good fit (Pearson χ^2/df = 0.983). In that model, non-smokers exhibited a 33 % lower episode rate than smokers (Exp B = 0.670, 95 % CI 0.408–1.100), although the difference was not statistically significant (p = 0.113).

Adjusted logistic regression controlling for gender, age, and comorbidities identified family history as the strongest predictor of VVS (p<0.001), with affected individuals demonstrating 8.7-fold increased odds (OR=8.658). Of the 42 participants reporting familial VVS, nearly half (47.6%, n=20) experienced personal episodes. Physical activity patterns showed a complex relationship, with weekly and occasional exercisers exhibiting 70.5% and 54.3% reduced VVS odds, respectively (p=0.004 and p=0.029), while extreme exercise patterns showed no association (p=0.581) (Table 4).

No significant associations emerged between VVS and sleep duration (OR=0.873/hour, p=0.107), caffeine consumption (OR=0.855, p=0.639), or gum chewing habits (OR=1.175, p=0.548). However, notable trends suggested potential protective effects of increased sleep (12.7% lower odds/hour) and caffeine abstinence (14.5% reduced odds), while non-gum chewers showed 17.5% elevated odds. Stress analysis proved inconclusive due to the absence of zero-stress respondents, though non-significant trends suggested higher stress levels might correlate with reduced VVS occurrence (Level 1 vs Level 3 stress: OR=8.567, p=0.144; Level 2 vs Level 3: OR=5.249, p=0.249) (Table 4).

Variable	Category	Count	Percentage
Gender	Females	259	56.80%
	Males	197	43.20%
Medical Year	4th year	111	24.30%
	5th year	41	9.00%
	6th year	82	18.00%
	Internship	163	35.70%
	Junior residency	59	12.90%
Nationality	Jordanian	434	95.20%
	Non-Jordanian	22	4.80%
	Jordan University Hospital	118	25.90%
	Al-Basheer	36	7.90%
	King-Abdullah University Hospital (KAUH)	46	10.10%

Training Hospital	King Hussein Cancer Center (KHCC)	6	1.30%
	King Hussein Medical Center (KHMC)	33	7.20%
	Al-Karak Hospital	124	27.20%
	Prince Hamzeh Hospital	39	8.60%
	Private hospitals	54	11.80%
BMI	< 18.5	22	4.80%
	18.5 – 24.9	230	50.40%
	25 – 29.9	160	35.10%
	30 – 34.9	36	7.90%
	35 – 39.9	6	1.30%
	≥ 40	2	0.40%
Marital Status	Single	432	94.70%
	Engaged	13	2.90%
	Married	11	2.40%

Table 1: Sociodemographic characteristics of the participants

Smoking Type / Habit	Count (% Yes)	p-Value for Syncope
Cigarettes (1 = smoke)	62 (13.6%)	0.135
Vapes (1 = vape)	63 (13.8%)	0.056
Shisha (1 = hookah)	52 (11.4%)	0.273
No Change in Habit	44 (9.6%)	-
Decreased Smoking	29 (6.4%)	0.273
Increased Smoking	45 (9.9%)	0.525

Table 2: Descriptive table for smoking types and habits among the participants

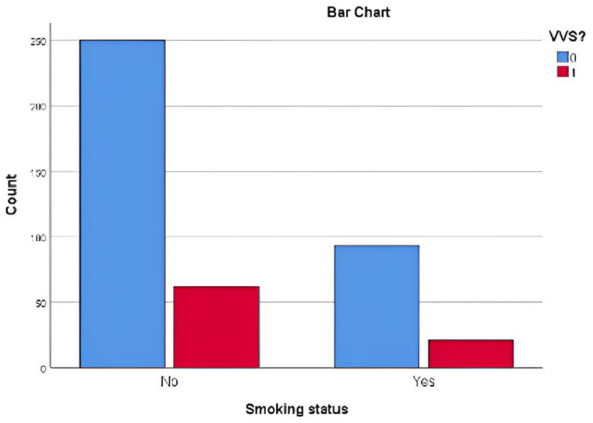

Figure 1: Clustered bar count of VVS by smoking status

Proportion Test Results

- Chi-square (Pearson): 0.112
- p-value from chi-square: 0.738

No significant association was found between smoking status and VVS. Further investigation in larger samples may still be warranted to confirm the trend.

S.no	Characteristic	Category	Count (%)
1	Clinically Diagnosed	Yes	8 (1.8%)
		No	448 (98.2%)
2	Number of VVS Attacks in a whole life	0	362
			-79.40%
		1–5	78 (17.1%)

		5–10	12 (2.6%)
		10–15	1 (0.2%)
		15–20	2 (0.4%)
		>20	1 (0.2%)
3	Pre-syncope Symptoms	Lightheadedness	74 (16.2%)
		Nausea	42 (9.2%)
		Sweating	35 (7.6%)
		Palpitations	35 (7.6%)
		Vision abnormalities	51 (11.1%)
		Hearing abnormalities	31 (6.8%)
		Pallor	40 (8.8%)
		Rising sensation from abdomen	15 (3.3%)
4	Triggers	Supine position	19 (4.2%)
		Sitting	12 (2.6%)
		Standing for some period	43 (9.4%)
		Fear, pain, instrumentation	24 (5.3%)
		During physical exercise	9 (1.9%)
		After cessation of exercise	5 (1.1%)
		During fever	6 (1.3%)
		Heat, warmth, hot bath	21 (4.6%)
		Sight of blood	15 (3.3%)
5	Investigations (n=8)	CBC	5 (62.5%)
		Glucose level	5 (62.5%)
		24-ECG	1 (12.5%)
		EEG	0 (0%)
		Electrolytes	2 (25%)
		Urinalysis	0 (0%)
		Echocardiogram	0 (0%)
		Tilt table	0 (0%)
		EPS	0 (0%)
		Exercise test	0 (0%)

Note: 448 who are not clinically diagnosed include those who don't suffer from vvs, one participant may have more than one trigger, pre-episode symptom, and investigation. Investigations are among the 8 who were clinically diagnosed. CBC: complete blood count, 24-ECG: electrocardiogram, EEG: electroencephalogram, EPS: electrophysiological study

Table 3: Descriptive table for vasovagal syncope

Variable	Category (Reference)	Count (%)	p-Value (Sig.)	Odds Ratio (Exp(B))	Chi-Square (Wald)
Sleep Hours	– (continuous)	–	0.107	0.873	2.597
Caffeine	Yes (Reference)	364 (79.8%)	–	–	–
	No	88 (19.3%)	0.639	0.855	0.219
Gums	Yes (Reference)	184 (40.4%)	–	–	–
	No	267 (58.6%)	0.548	1.175	0.36
Stress Rate	3 (Reference)	150 (32.9%)	–	–	–
	1	88 (19.3%)	0.144	8.567	2.14

	2	214 (46.9%)	0.249	5.249	1.327
Physical Activity	Daily (Reference)	90 (19.7%)	–	–	–
	Never	52 (11.4%)	0.581	0.782	0.304
	Occasionally	196 (43.0%)	0.029	0.457	4.744
	Weekly	110 (24.1%)	0.004	0.295	8.189
Family Hx (VVS)	No (Reference)	407 (89.3%)	–	–	–
	Yes	42 (9.2%)	< 0.001	8.658	29.921

Table 4: Multivariable logistic regression analysis of factors associated with Vasovagal Syncope (VVS)

DISCUSSION

In this cross-sectional study conducted on 456 medical students, residents, and interns across various hospitals in Jordan, we studied the hypothesis that smoking may decrease the incidence of vasovagal syncope (VVS) due to its sympathomimetic effects, but after statistical analysis was carried out, no significant difference was found. However, when vaping was isolated, it showed a borderline trend (p=0.056). Some significant predictors of VVS were found, including the female gender (p=0.008), family history (p<0.001), and physical activity (weekly exercise p=0.004; and occasional exercise: p=0.029).

Of the 121 smokers surveyed, 22 reported symptoms of VVS (18.2%) compared to 72 of the 335 non-smokers (21.5%) (p=0.188). However, we do believe that the borderline trend noticed with vaping (p=0.056) warrants further investigation on a larger population as the autonomic effects of nicotine delivery methods may differ substantially, with vaping producing more abrupt sympathetic activation than cigarettes [18], with previous studies highlighting the provocative effects of vaping on the autonomic nervous system, leading to symptoms of autonomic dysregulation like tachycardia [12].

Previously documented increased incidence of VVS among females compared to males was also confirmed in our data (p=0.008), which may be attributable to many physiological differences, including the increased vagal tone in females due to higher levels of estrogen or the lower volume of circulating blood that decreases the preload of the heart chambers [3,9]. Recent work has also suggested that progesterone may also influence sympathetic withdrawal thresholds [19]. Notably, the relationship between exercise and the occurrence of VVS showed a U-shaped association. Exercising daily and never exercising both showed no significant effect on the occurrence of VVS, whereas occasional and weekly exercise seemed to have a protective effect (p=0.004 and p=0.029, respectively). This could be explained by the improved orthostatic tolerance caused by moderate exercise, as reported by Sheldon, and Fu and Levine [17,20]. Finally, family history emerged as the strongest predictor for the occurrence of VVS; our study concluded that family history increased the chance of VVS occurrence by 8.7-fold (p <0.001), aligning with Serletis who found parental syncope doubled offspring risk [10]. This highlights the relevance of genetic predisposition, with emerging genome-wide evidence suggesting the involvement of multiple interacting loci [21].

LIMITATIONS

Firstly, our cohort of medical trainees had a mean age of 23.8 years, which may not represent a broader population of smokers. At younger ages, people tend to have a more robust autonomic regulatory response, and this may mask any potential smoking-related effect. Also, only 1.8% of the people with symptoms of VVS were previously diagnosed by a physician, and even though we used standardized syncope parameter scores, like the Calgary Syncope Score, the lack of head-up tilt testing and real-time hemodynamic monitoring may lead to a degree of diagnostic uncertainty and possibly missing some borderline cases. And lastly, due to the cross-sectional design of the study, the temporal relationship between initiation of smoking and the episodes of syncope is unclear, as we believe that some patients may have begun smoking following the start of VVS episodes in an attempt to reduce anxiety and stress, known to be provocative factors of episodes of VVS.

Clinical Implications and Future Directions

The pronounced 8.7-fold increased VVS risk associated with family history should encourage physicians to act with increased medical vigilance when encountering a patient with presyncope symptoms and a positive family history of VVS, particularly with females who have demonstrated a significantly higher susceptibility (p=0.008), and provide medical guidance on possible high-risk activities. We believe the recommendation of moderate exercise for patients with VVS may prove to be beneficial in cases where low orthostatic tolerance is a trigger of syncopal episodes. Future studies should focus on investigating the effect of vaping on larger populations of varying ages, as the borderline results in our study and the supporting literature mentioned previously indicate that different forms of nicotine delivery seem to have different effects on the autonomic nervous system. To resolve the issue of temporal ambiguity, we believe a retrospective study on a cohort of VVS patients who smoke will provide a clearer timeline of the events and help us identify the motivation behind the initiation of smoking.

CONCLUSION

One in five medical trainees reported VVS episodes. Contrary to our primary hypothesis, we found no evidence that smoking is associated with a reduced prevalence of VVS. The strong association with family history and female gender aligns with existing literature. These null findings challenge the hypothesis of a strong protective sympathomimetic effect from smoking in this young population. This study found no significant association between smoking and reduced vasovagal syncope incidence, though a borderline trend with vaping suggests

potential autonomic effects requiring further investigation. Female sex, family history, and moderate physical activity emerged as key predictors of vasovagal syncope, with family history conferring an 8.7-fold increased risk. Limitations include the cohort's young age, diagnostic reliance on self-reported symptoms, and the cross-sectional design, which precludes causal inferences. Clinically, heightened vigilance for patients with familial predisposition, particularly females, and promotion of moderate exercise are warranted. Future studies should prioritize longitudinal designs to clarify temporal relationships between smoking initiation and syncope onset, as well as mechanistic comparisons of nicotine delivery methods across broader age groups.

COMPETING INTERESTS

None

CONTRIBUTORSHIP

None

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DISCLAIMER

The views expressed in the submitted article are the author's own and not an official opinion of the institution or funder.

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PATIENT AND PUBLIC INVOLVEMENT

No closed involvement

DATA SHARING STATEMENT

Available from the population upon request

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