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Clinical Reviews

The Role of Troponin Testing in Patients with Supraventricular Tachycardia, Systematic Review and Meta-Analysis

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Background: Supraventricular tachycardia (SVT) is commonly evaluated in the emergency department (ED). While troponin has been shown to be elevated in SVT, its usefulness for predicting coronary artery disease and future adverse cardiovascular outcomes has not been shown. Objectives: We aimed to evaluate the prognostic utility of troponin measurement as part of SVT management in the ED. Methods: We performed a literature search in the PubMed and Scopus databases from inception to August 30, 2023, including all studies reporting troponin measurements in adult patients (age > 18 years) presenting to the ED with supraventricular tachycardia. The primary outcome of interest for this study was the prevalence of elevated troponin in patients with SVT. Secondary outcomes included the prevalence of major adverse cardiac events (MACE) and additional cardiac testing with significant findings. Results: We included 7 studies (500 patients) in our analysis. Six studies reported the number of patients with SVT and elevated troponin, with a pooled prevalence of 46% (95% CI 27–66%, I^2 93%). The pooled prevalence of all MACE in our study was 6% (95% CI 1–25%), while the prevalence for MACE among patients with elevated serum troponin levels was 11% (95% CI 4–27%). Conclusions: Troponin levels are frequently ordered for ED patients with SVT and are often elevated. However, this review suggests that they have low prognostic value in predicting MACE. © 2024 Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

□ **Keywords—supraventricular tachycardia; troponin; SVT; cardiac biomarker**

Introduction

Supraventricular tachycardia (SVT) is a type of cardiac dysrhythmia commonly evaluated in the emergency department (ED). SVT refers to any dysrhythmia with an atrial rate of more than 100 bpm and is thought to be due to disruption of the electrical impulses at or above the AV node.[1] Presenting symptoms commonly include chest discomfort or pressure, palpitations, and dyspnea. Less common symptoms include chest pain, diaphoresis, nausea, and presyncope or syncope.[2] According to one dated study, SVT accounts for approximately 50,000 ED visits every year and has an estimated annual incidence rate of 35 per 100,000 persons.[2,3] While SVT has a favorable prognosis and is often managed with cardioversion (medical or electrical) and discharge, 1 out of 4 ED visits for SVT result in a hospital admission.[4] Hospital admission for SVT increases use of limited healthcare resources, with an average cost of $3800 per admission and $190 million per year in one study.[3,5]

Patients with SVT may present with symptoms often seen in acute coronary syndrome (ACS). For this rea-

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son, routine management of SVT often includes ECG, chest radiography, and cardiac biomarker assays such as troponin.[2] Troponin elevation has repeatedly been shown to be an indicator of myocardial injury, but has also been shown to be nonspecifically elevated in various conditions including SVT and strenuous physical exercise.[6–10] One proposed explanation for this is demand ischemia, whereby the increased heart rate in SVT results in increased oxygen demand and a shortened diastole period, possibly contributing to myocardial ischemia and troponin release.[11] Other explanations have also been suggested, including tachycardia induced stretch of the myocytes and coronary vasospasm.[11]

While troponin has been shown to be elevated in SVT, its usefulness for predicting future adverse cardiovascular related outcomes has varied and its ability to predict coronary artery disease (CAD) at the time of admission has not been shown.[12–16] However, studies have shown that up to 79% of patients presenting to the ED with SVT had a troponin level ordered, and those with an elevated result were more likely to be admitted to the hospital and incur likely unnecessary cost to the healthcare system.[1,14,17] Furthermore, ordering a troponin level, regardless of the result, has been shown to be associated with longer ED stays.[16,18]

In this study, a systematic review and meta-analysis was conducted to evaluate the utility of troponin for SVT management in the ED. More specifically, we examined the prevalence of elevated serum troponin in participants with SVT, the prevalence of major adverse cardiovascular events (MACE), and the prevalence of significant additional testing in this population.

Methods

Search and Selection Criteria

This meta-analysis was performed in accordance with the 2020 Preferred Reporting Items for Systematic reviews and Meta-Analyses (PRISMA) guidelines.[19] PubMed and Scopus databases were searched with the assistance of a medical librarian from inception to August 30, 2023, using the search term in PubMed: ((troponin*[all fields]) AND ("SVT"[all fields]) OR "tachycardia, supraventricular"[MeSH] OR "supraventricular tachycardia" in [Title/Abstract]) and in SCOPUS: TITLE-ABS-KEY ((troponin*) AND ("supraventricular tachycardia" OR svt)). All observational, experimental or quasi-experimental studies reporting troponin values in adult patients (age > 18 years) presenting to an ED with supraventricular tachycardia were eligible. We only included studies that were published in English language or were available in full-text versions. Conference publications, abstracts, other non-original studies (reviews, meta-analysis) were excluded. The study protocol has been registered in the PROSPERO database (CRD42023479864).

Outcomes of Interest

The primary outcome of interest for this study was the prevalence of elevated serum troponin levels. Other outcomes of interest were: (1) prevalence of MACE, defined as stroke, myocardial infarction (MI), percutaneous coronary intervention (PCI), or death; (2) the prevalence of additional significant cardiac testing, defined as additional testing performed in SVT patients not considered part of a routine SVT workup (i.e., angiogram, computed tomography angiography (CTA), stress test, nuclear test) AND was found to be positive or clinically significant (i.e., significant coronary artery stenosis, myocardial perfusion defects); (3) prevalence of MACE among patients with both SVT and elevated serum troponin; and (4) the prevalence of additional significant cardiac findings among patients with both SVT and elevated serum troponin.

Quality Assessment

We planned to use the Newcastle-Ottawa Scale (NOS)[20] for any observational studies. The NOS assesses a study's quality according to its selection of cohort, comparability of groups, quality of outcomes. The NOS rates a study's quality as high if the score is 7–9, medium if the score is 4–6 and low if the score is ≤3.

Studies' heterogeneity was assessed using the I^2 value and the Cochrane Q statistic. The I2 value indicates the level of heterogeneity between the studies being included in this meta-analysis. The Cochrane Q-statistic tests the null hypothesis that the meta-analysis' overall effect size would be similar to the true effect size of a hypothetical meta-analysis that involves millions of studies. Two investigators independently assessed the quality of each study. If a disagreement arose, the two investigators would adjudicate the discrepancy via discussion or by review by a third and senior investigator. The consensus of the investigators was reported as the final result.

Data Extraction

The investigators first independently extracted data from each study into a standardized spreadsheet (Microsoft Corp, Redmond, Washington, USA), which were then compared for discrepancies. The reported results were the consensus of the investigators. Collected data included demographic study information, such as first authors' names, year of publication, study design, sample size, number of patients, and age. We also collected

patient-level clinical information, such as past medical history, maximum heart rate, duration of SVT, troponin levels, additional cardiovascular testing performed and MACE. We collected the information regarding MACE and significant additional testing as defined above in the outcomes of interest.

Statistical Analysis

Descriptive analyses were used to report categorical variables as percentages, and continuous variables as median [Interquartile Range (IQR)] or mean (+/- Standard Deviation [SD]). Random effects meta-analysis was performed for any 2 studies reporting the same outcome of interest. The prevalence of the outcomes of interest was reported as percentage and 95% Confidence Interval (95% CI). For sensitivity analysis, random-effects meta-analysis with one study removed was performed. This allowed us to identify any individual outlying study that would significantly affect the overall effect size seen in the meta-analysis.

Publication bias was not performed for this meta-analysis of prevalence because publication bias is used to assess whether there are missing studies that would change the overall efficacy of any interventions. This meta-analysis did not assess the efficacy of any interventions so publication bias would not be applicable. Since heterogeneity was anticipated from the included studies, we performed moderator analyses to identify which subgroups' characteristics with be associated with high heterogeneity. The moderator analyses also compared differences in prevalence of outcome between the subgroups. The moderator analyses used categorical variables that were commonly reported by the studies, such as study design (retrospective vs. prospective), sample size ($<$100 patients vs. $\geq$100 patients), and whether the study used high sensitivity troponin or not. For sample size, we assessed the histogram of the sample size then dichotomized the cut-off value of $\geq$100, according to the frequency of the distributions. We also attempted to identify any patients' clinical information that would be correlated with the prevalence of outcomes. For this purpose, we performed exploratory univariate meta-regression, using continuous variables for the prevalence of outcomes. Each continuous variable would be expressed as a percentage to be entered in the meta-regression, except age, which was expressed as an integer. The results were reported as the coefficient correlation with 95% CI and associated *p*-values.

All random-effects meta-analysis, sensitivity and moderator analyses, and univariate meta-regression were performed with the software Comprehensive Meta-Analysis version 4 (www.meta-analysis.com, Englewood, New Jersey, USA). All statistical analyses with p-value < 0.05 were considered significant.

Results

Study Selection

A total of 576 titles and abstracts were screened before 36 full text articles were assessed. We included 7 studies in our analysis (Figure 1).[13,16,21–25] Only 1 study was prospective [21], while the other 6 studies were retrospective (Table 1).[13,16,22–25]

Study Quality

Since all studies were observational, the Newcastle-Ottawa scale was used. The majority of included studies were considered as having high quality, except Ede et al. which was ranked as moderate due to its lack of an unexposed cohort.

Summary of Studies

The analysis included a total of 500 patients, with mean age of 56 (+/- 9) years. There were 232 (46%) female patients. Only 4 studies reported further details about their patients' SVT episodes (2011 Yedder, 2017 Sayadnik, 2021 Ede, 2022 Wang).[16,21,24,25] Among the studies that reported the information, the median (Interquartile [IQR]) was 14 (4–67) minutes, and the median heart rate was 169 (160–178) beats per minute. Six studies (2011 Carlberg, 2011 Yedder, 2017 Sayadnik, 2018 Noorvash, 2020 Ghersin, 2022 Wang) reported the number of patients who had SVT and elevated serum troponin,[13,16,21–23,25] and 4 studies (2011 Yedder, 2018 Noorvash, 2020 Ghersin, 2022 Wang) reported the rate of MACE.[16,22,23,25] Two studies reported the prevalence of significant additional cardiac tests among all patients with SVT (2011 Yedder, 2022 Wang)[16,25] and 4 studies (2011 Yeder, 2017 Sayadnik, 2021 Ede, 2022 Wang) reported the prevalence of significant additional cardiac tests among all patients with SVT and elevated troponin levels.[16,21,24,25] The study by Ede et al. only included patients with SVT and elevated troponin.[24]

Primary Outcome: Prevalence of Elevated Serum Troponin Levels

Six studies (2011 Calberg, 2011 Yedder, 2017 Sayadnik, 2018 Noorvash, 2020 Ghersin, 2022 Wang) reported the number of patients with elevated serum troponin.[13,16,21–23,25] The study by Ede et al. was not included in this analysis because it only included patients

Table 1. Characteristics of Studies and Patients Included in the Meta-Analysis

Author		Wang et al.	Ede et al.**	Ghersin et al.	Noorvash et al.	Sayadnik et al.	Carlberg et al.	Yedder at al.
Year		2022	2021	2020	2018	2017	2011	2011
Country of origin		Taiwan	Qatar	Northern Israel	Texas, USA	Iran	Virginia, USA	Quebec, Canada
Study design		Retrospective observational	Retrospective observational	Retrospective observational	Retrospective chart review	Prospective observational	Retrospective chart review	Retrospective chart review
Study setting		ED	ED	ED	ED	ED	ED	ED
Sample size		57	85	131	46	70	38	73
Age	**Troponin +**	65.5 ± 14.9	44.3 ± 10.4	61 ± 18	50 (37–55.8)†	58.92 ± 13.61*	51 ± 16.2	56.25 ± 18
	Troponin -	63.6 ± 7.6	NR	58 ± 15		49.11 ± 10.91	NR	63.2 ± 18
Female	**Troponin +**	0.553	0.26	0.32	0.674†	0.615	0.364	0.583
	Troponin -	0.7	NR	0.44		0.556	NR	0.612
Heart rate	**Troponin +**	158.2 ± 30.3	185 ± 31	NR	NR	171.5 ± 24.7	203 ± 32.7	190.8 ± 27.8*
	Troponin -	150.4 ± 34.7	NR			166.11 ±17.74	NR	170.3 ± 30
Diabetes mellitus	**Troponin +**	0.404	0.235	.18*	NR	0.212	0.18	0.125
	Troponin -	0.4	NR	0.15	NR	0.056	NR	0.143
CAD	**Troponin +**	0.298	0.259	0.20*	NR	NR	0.18	0.333
	Troponin -	0.1	NR	0.07	NR	NR	NR	0.245

(*continued on next page*)

Table 1. (*continued*)

Author		Wang et al.	Ede et al.**	Ghersin et al.	Noorvash et al.	Sayadnik et al.	Carlberg et al.	Yedder at al.
Troponin assay and threshold used		UniCel DxI 800 immunoassay analyzer; minimum detectable concentration of <0.01 ng/mL; threshold for positivity 0.04 ng/mL.	Hs-cTnT value (normal range in laboratory was 0–15 ng/L)	Serum cTn1 > 0.028 ng/dL	cTn1 > 0.05 ng/mL	hs-cTnT–positive (serum hs-cTnT level, ≥14 ng/L)	cTnI levels x > .02 ng/dL were measured by immunoassay on an integrated ARCHITECT analyzer ci8200	Roche Elecsys 2010 >0.03 μg/L
Prevalence of elevated troponin		0.825	1.00**	0.435	0.152	0.743	0.289	0.329
Prevalence of significant additional testing	**Troponin +**	0.09	0.235	NR	NR	0.19	NR	0.08
	Troponin -	0	NR	NR	0	NR	NR	0.06
MACE prevalence	**Troponin +**	0.36	0.02	0.12	0	NR	0.18	0.04
	Troponin -	0.10	NR	0	0.03	NR	NR	0
Length of follow-up		3 years	NR	23 ± 7 months	3 months	NR	30 days	NR

Values reported as either mean ± SD, median (IQR), or a proportion.
CAD = coronary artery disease; ED = emergency department; MACE = major adverse cardiac events; NR = Not reported.
* p-value < 0.05 when compared to group with negative troponin.
** Inclusion criteria included having an elevated troponin, therefore all subjects had an elevated troponin.
† Demographic data was not provided that was separated based on the patient being troponin positive versus negative.

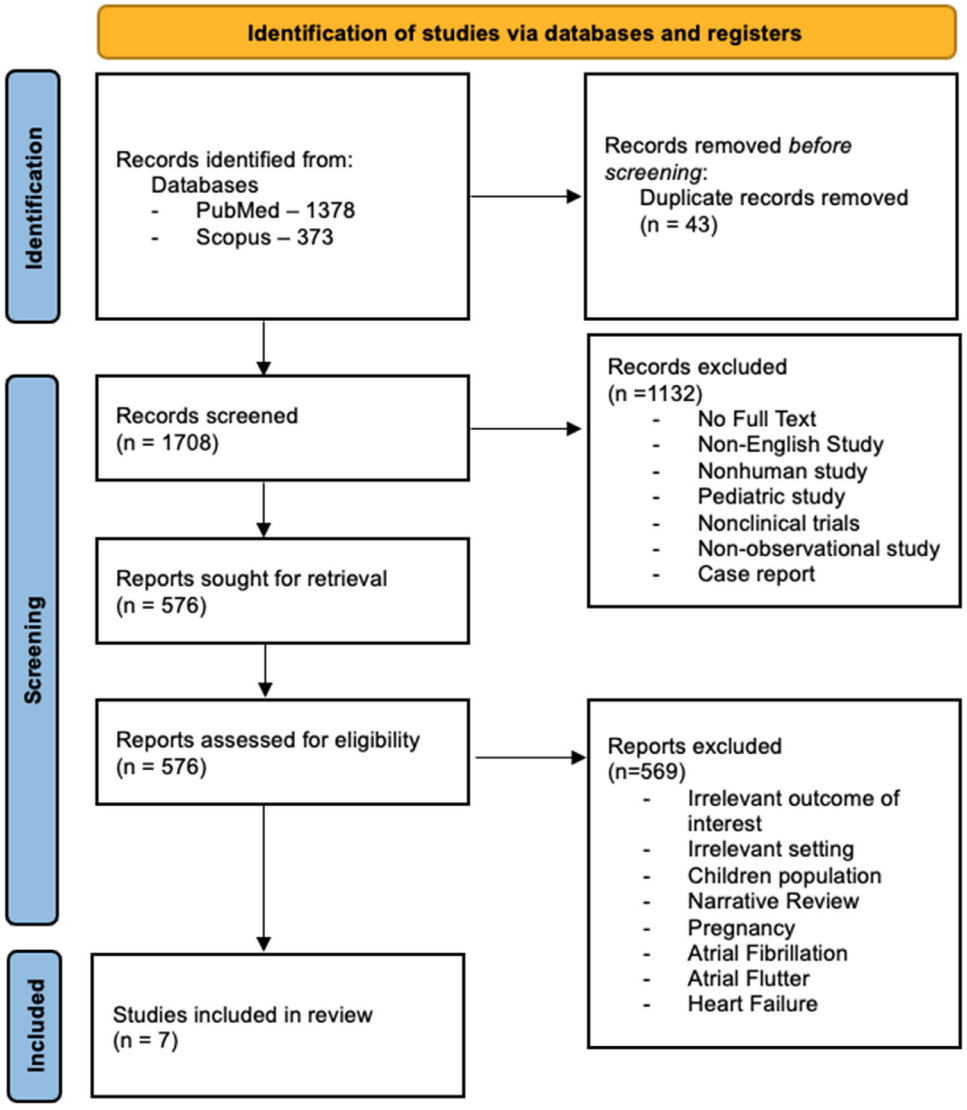

Figure 1. PRISMA diagram for selection of studies.

with SVT and positive troponin.[24] The pooled prevalence of patients who had SVT and elevated serum troponin levels was 46% (95% CI 27–66%) (Figure 2A). The I^2 value was 93%, which indicated significant heterogeneity between the included studies, and the Q-value was 68 ($p < 0.001$), which rejected the null hypothesis that our study's effect size is similar to the true effect size.

The sensitivity analysis (Figure 2B) for the prevalence of elevated serum troponin levels showed the rates of elevated serum troponin ranged from 38% to 53%, which fell within the 95% CI of the pooled prevalence. This sensitivity analysis suggested that there was no outlying study that would affect the overall effect size of this study.

The heterogeneity as represented by I^2 value was consistently high in different subgroups (Table 2A). The prevalence of elevated serum troponin levels was significantly different when we compared the subgroups according to study design (retrospective vs. prospective), sample size (<50 or ≥50 patients), and whether the authors used high-sensitivity troponin assays or not (Table 2A).

The exploratory univariate meta-regression using continuous variables showed that only age was positively correlated with the rate of elevated troponin among patients with SVT (correlation coefficient 0.16, 95% CI 0.04–0.27, $p = 0.006$) (Table 2B).

Secondary Outcome 1: Prevalence of MACE Among Patients With SVT

The pooled prevalence of all MACE in our study was 6% (95% CI 1–25%) (Figure 2C). The I^2 value was 90%, and the Q-statistic was 30 ($p = 0.001$), which in-

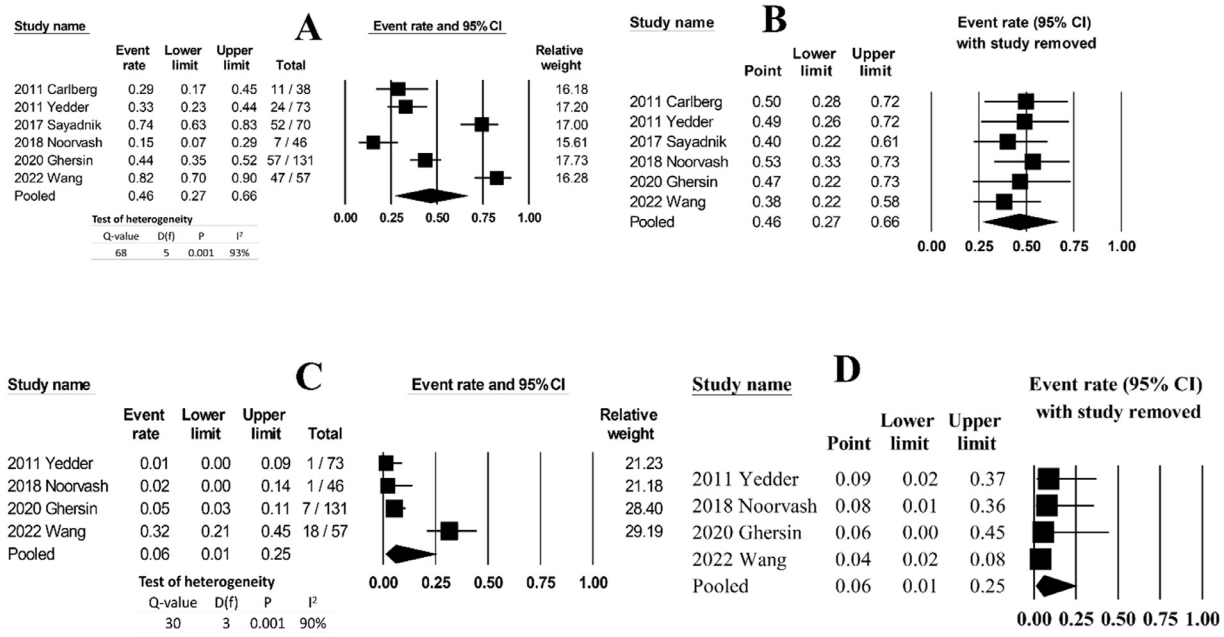

Figure 2. (A) Forest plot of prevalence of elevated troponin levels among patients with supraventricular tachycardia. (B) Sensitivity analysis of prevalence of elevated troponin levels among patients with Supraventricular tachycardia. (C) Forest plot of prevalence of major adverse cardiac events (MACE) among patients with supraventricular tachycardia. (D) Sensitivity analysis of MACE among patients with supraventricular tachycardia.

dicated high heterogeneity between the included studies. The sensitivity analysis of the studies included for this outcome (Figure 2D) did not show any study that affected the overall outcome of this meta-analysis. The I^2 values for the subgroups were still high among different subgroups (Table 2A). This suggested that heterogeneity exists among studies that reported prevalence of MACE.

Secondary Outcome 2: Prevalence of Additional Significant Cardiac Tests Among Patients With SVT

Among the 2 studies that reported significant additional cardiac tests for patients with SVT, the pooled rate was 7% (95% CI 4–13%). The I^2 value was 0%, and the Q-statistic was 0 ($p = 0.97$), which indicated low heterogeneity between the included studies. Sensitivity analysis, subgroup analysis and univariate meta-regression were not performed for this outcome because there were only 2 studies.

Secondary Outcome 3: Prevalence of MACE Among Patients With SVT and Elevated Serum Troponin Levels

The prevalence for MACE among patients with elevated serum troponin levels was 11% (95% CI 4–27%) (Figure 3A). The I^2 value was 68%, and the Q-statistic was 13 ($p = 0.013$). No individual study affected the overall outcome of this meta-analysis (Appendix 1A). No moderator analyses nor univariate meta-regression were performed for this outcome due to the small number of available studies and variables.

Secondary Outcome 4: Prevalence of Significant Additional Cardiac Tests, Among Patients With SVT and Elevated Serum Troponin Levels

The pooled prevalence of significant additional cardiac tests, among patients with SVT and elevated serum troponin levels was 16% (95% CI 10–26%) (Figure 3B). The I^2 value was 49%, and the Q-statistic was 6 ($p = 0.12$), which did not reject the null hypothesis that our study's effect size is similar to the true effect size. The sensitivity analysis for this outcome (Appendix 1B) suggested that no individual study affected the overall outcome of this meta-analysis. No moderator analyses nor univariate meta-regression were performed for this outcome due to the small number of available studies and variables.

Discussion

This meta-analysis identified that approximately 46% of patients who presented to the ED for SVT had elevated serum troponin levels. The pooled prevalence of MACE in patients with SVT was 6%, which increased to 11% for those with a positive troponin. Similarly, the pooled prevalence of significant additional cardiac testing was 7% in patients with SVT and 16% in patients with SVT and pos-

Table 2A. Moderator Analyses Using Categorical Variables for Subgroup Analyses

	Meta-Analysis			Heterogeneity			
Moderator Variables		Number of Studies	Outcome (%)	Q-Value	D(f)	*p*	I^2
Outcome 1: Prevalence of elevated serum troponin levels among patients with supraventricular tachycardia							
Study design	Prospective	1	0.74 (0.63–0.83)	NA	NA	NA	NA
	Retrospective	5	0.40 (0.22–0.61)	46	4	0.001	91%
Sample size	<50 patients	2	0.22 (0.11–0.38)	2	1	0.13	55%
	>50 patients	4	0.59 (0.36–0.79)	44	3	0.001	93%
High sensitivity troponin	No	5	0.40 (0.22–0.61)	45	4	0.001	91%
	Yes	1	0.74 (0.63–0.83)	NA	NA	NA	NA
Outcome 2: Prevalence of MACE among patients with supraventricular tachycardia							
Study design	Prospective	0	NA	NA	NA	NA	NA
	Retrospective	4	0.06 (0.01–0.25)	31	3	0.01	91%
Sample size	<50 patients	1	0.02 (0.0–0.14)	NA	NA	NA	NA
	>50 patients	3	0.08 (0.01–0.36)	26	2	0.01	92%
High sensitivity troponin	No	4	0.06 (0.01–0.25)	31	3	0.01	91%
	Yes	0	NA	NA	NA	NA	NA

MACE = major adverse cardiac event; NA, analysis was not performed due to insufficient number of variables.

Table 2B. Univariate Meta-Regressions to Measure the Association of Continuous Variables and the Prevalence of Elevated Serum Troponin Levels, MACE Among Patients With Supraventricular Tachycardia (SVT)

Variables	Number of Studies	Corr. Coeff. (95% CI)	*p*
Outcome 1: Prevalence of elevated serum troponin levels among patients with supraventricular tachycardia			
Age	4	0.16 (0.04 to 0.27)	0.006
Percentage of female	5	0.12 (-11.8 to 12.1)	0.98
Maximum heart rate	NA	NA	NA
Percentage of patients with CAD	NA	NA	NA
Percentage of patients with hypertension	4	0.74 (-4.4 to 5.93)	0.78
Percentage of patients with diabetes	4	2.3 (-7.8 to 12.3)	0.66
SVT duration, each minute	NA	NA	NA
Outcome 2: Prevalence of MACE among patients with supraventricular tachycardia			
Age	4	0.15 (-0.05 to 0.36)	0.14
Percentage of female	4	4.6 (-14.3 to 23.5)	0.63
Maximum heart rate	NA	NA	NA
Percentage of patients with CAD	NA	NA	NA
Percentage of patients with hypertension	NA	NA	NA
Percentage of patients with diabetes	NA	NA	NA
SVT duration (each minute)	NA	NA	NA

No additional analyses were performed for other outcomes as there were insufficient numbers of studies reported the same independent variables.
CAD = coronary artery disease; Corr. Coeff. = correlation coefficient; MACE = major adverse cardiac event; NA = analysis was not performed due to insufficient number of variables.

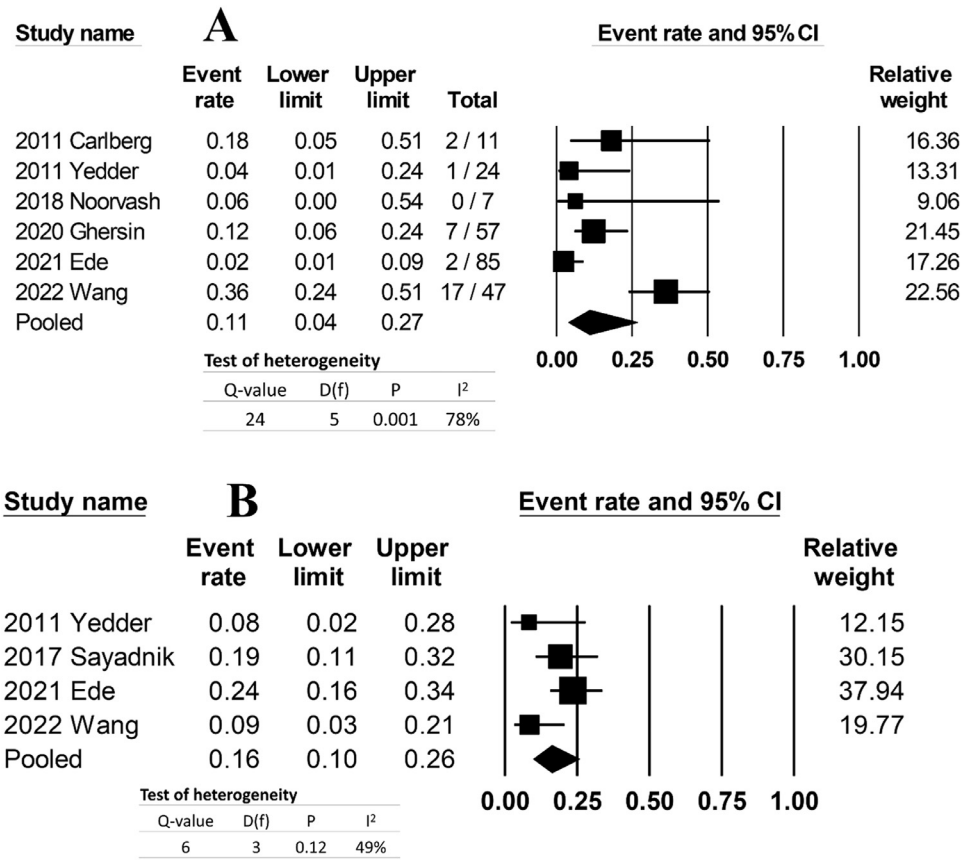

Figure 3. (A) Forest plot of prevalence of major adverse cardiac events (MACE) among patients with supraventricular tachycardia and elevated troponin. (B) Forest plot of prevalence of significant additional cardiac tests among patients with supraventricular tachycardia and elevated troponin levels.

itive troponin. The study findings suggest that given 100 hypothetical patients presenting with SVT, approximately 46 would exhibit elevated troponin levels. Of these 46 patients, only 5 (11%) would be expected to experience MACE between their index hospitalization and up to three years later—the longest follow-up period documented in any of the included studies. Assuming all MACE established by these studies were a result of SVT, there would have to be 100 patients with SVT who have a troponin level ordered to predict 5 patients with MACE. With the low number needed to treat to predict MACE, clinicians should use their clinical judgment and other established clinical decision rules to guide their further evaluation and management.

There was significant heterogeneity among the studies that reported prevalence of elevated serum troponin levels. For example, studies using high sensitivity troponins reported almost twice the prevalence of elevated serum troponin levels as conventional troponins (Table 2A). Retrospective studies identified a lower prevalence of elevated serum troponin compared to the single prospective study. There was also high heterogeneity between studies regarding the secondary outcomes of MACE and significant additional cardiac testing. Studies used different durations for follow-up and varied in their reporting of additional tests, which made it difficult to synthesize and generalize the findings. The high practice variability observed could stem from the current lack of consensus guidelines on managing ED patients presenting with SVT.

It is also important to highlight the differences across various outcomes according to patient risk factors. In the study by Ghersin et al. a multivariate model found that elevated troponin on admission to the hospital was only a predictor of MACE for patients with known CAD. Similarly, in Wang et al. CAD was the only independent predictor for 3-year MACE. In contrast, Noorvash et al. found that, despite a positive troponin, no patients with low to intermediate HEART scores had a positive cardiac catheterization, stress test, or MACE within the 3 month follow up period. This finding suggests that HEART score may be a useful tool to help clinicians make decisions about ordering troponin by risk stratifying patients.

Based on the findings of this study, troponin levels have low utility in predicting MACE and should not be routinely ordered for patients presenting to the ED with SVT without other significant cardiac risk factors. This

is especially important when considering that an elevated troponin level alone makes patients more likely to be admitted to the hospital[1,14,17] and that ordering a troponin level, regardless of the result, has been shown to be associated with longer ED stays.[16,18]

The results from this study highlight the need for more robust research evaluating the outcomes for patients presenting to the ED with SVT. Ideally, these studies should be prospective and include patients both with and without troponin measurement to adequately compare the relative incidence of short-term MACE and positive cardiovascular testing. Stratification with clinical decision rules, such as the HEART score, may also provide insight into which SVT patients may or may not benefit from troponin measurement.

Limitations

There were several limitations to our study. There was a small number of included studies and most of them were retrospective in nature, making the data more prone to bias and limited in scope. Additionally, there were only a small number of studies that reported common study demographics or patient-level clinical information. We could not perform more thorough analyses to identify other sources of heterogeneity, further limiting the generalizability of our report. One study, Ede et al. only included patients with SVT who were troponin positive, eliminating the comparison cohort entirely. Another study, Wang et al., only included patients with end stage kidney disease, making their data far less generalizable to our population of interest. Last, Noorvash et al. only included patients with low to intermediate scores on a validated scoring system (HEART score) that predicts 6-week risk of MACE, likely artificially decreasing the prevalence of MACE in their patient population.

Conclusion

This review suggests that troponin levels are often elevated in patients presenting to the ED with SVT, but they have a low prognostic value in predicting MACE or significant cardiovascular testing. Future research is needed as the current literature largely consists of small, retrospective studies with significant heterogeneity with regards to troponin testing, duration of follow up and outcomes of interest.

Declaration of competing interest

The authors do not have a financial interest or relationship to disclose regarding this research project.

CRediT authorship contribution statement

Ali Pourmand: Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Formal analysis, Data curation, Conceptualization. **Hannah Checkeye:** Writing – review & editing, Writing – original draft, Methodology, Data curation. **Bennet Varghese:** Writing – review & editing, Writing – original draft, Data curation. **Allen J Solomon:** Writing – review & editing, Writing – original draft. **Quincy K Tran:** Writing – review & editing, Writing – original draft, Methodology, Formal analysis, Conceptualization.

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Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jemermed.2024.05.010.

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Article Summary

1. Why is this topic important?

Although troponin levels may be elevated in cases of SVT, the effectiveness of troponin in predicting future adverse cardiovascular outcomes has been inconsistent. The ability of troponin levels to diagnose CAD upon admission has not been established.

2. What does this review **attempt to show?**

This study attempts to measure the prevalence of elevated serum troponin levels in patients with SVT, as well as the prevalence of major cardiac events and positive cardiovascular testing in SVT patients, both overall and in the setting of elevated troponin levels.

3. What are the key findings?

Although troponin levels are routinely ordered for patients with SVT who present to the ED, less than 50% of patients had elevated troponin levels. Elevated troponin has low prognostic value for predicting MACE or positive CAD testing among patients who present to the ED with SVT.

4. How is patient care impacted?

Providers should exercise caution when measuring serum troponin in SVT patients. Elevated troponin levels can lead to hospital admissions and further testing despite a low likelihood of adverse outcomes.