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REVIEW ARTICLE

Review article: The utility of troponin and other investigations in patients presenting to the emergency department with supraventricular tachycardia

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Abstract

Patients with supraventricular tachycardia commonly present to the ED. There is a lack of consensus regarding assessment of these patients. Our aim was to determine the utility of troponin and four other investigations (full blood examination, electrolyte levels, thyroid function tests and chest X-rays) commonly requested for these patients. MEDLINE, EMBASE and Cochrane Central Register of Controlled Trials (January 1992–March 2017) were searched for randomised controlled trials and observational studies (of sample size greater than 10). Our search strategy yielded no randomised controlled trials and seven observational studies with significant statistical heterogeneity among selected studies (I^2 87.3%, P < 0.001). Included studies investigated a total of 1155 patients. All studies reported on the utility of troponin testing in this patient population. The pooled proportion of patients investigated with troponin was 0.66 (95% confidence interval 0.44–0.88). The pooled proportion of positive troponin tests was 0.32 (95% confidence interval 0.23–0.41). Only one study reported on the utility of the remaining four investigations with abnormal results as follows: thyroid stimulating hormone 14%, haemoglobin 6%, white cell count 19% and chest X-ray 22%. Investigations are commonly requested for patients presenting with supraventricular tachycardia. Troponin testing is commonly performed with a high proportion of positive findings although these results did not appear to be associated with major adverse cardiac events. Heterogeneity among studies and low levels of evidence precluded conclusions on full blood examinations, electrolyte levels, thyroid testing and utility of chest X-rays in this patient population.

Key words: *emergency medicine, supraventricular, tachycardia, troponin.*

Key findings

- Troponin testing frequently performed after patients present to the ED with SVT.
- Troponin elevation in this population not likely to correlate with coronary artery disease.
- Insufficient evidence concerning other investigations in this patient population.

Introduction

Patients with supraventricular tachycardia (SVT) commonly present to the ED. The prevalence and incidence of paroxysmal SVT has been estimated at 2.25/1000 and 35/100 000 person years, respectively.[1]

SVT is a general term referring to any arrhythmia that relies on atrial or atrioventricular nodal tissue in order to sustain itself. Strictly speaking, this broad definition would encompass atrial fibrillation (AF) and atrial flutter. However, in this review, the term refers more specifically to atrioventricular nodal re-entrant tachycardia (AVNRT), atrioventricular re-entrant tachycardia (AVRT) and atrial tachycardia.

A study of emergency physicians treating patients with palpitations found most felt 'blood, urine and X-ray testing to be… generally low yield'.[2] Nonetheless, these investigations continue to be requested with a lack of consensus and guidelines.

The aim of this systematic review was to assess the utility of troponin and other investigations requested among patients presenting to the ED with SVT. We aimed to: (i) determine the proportion of patients presenting with SVT who undergo investigations; (ii) evaluate the frequency with which these investigations yield an abnormal result; and (iii) determine whether the results of these investigations influence clinical management.

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Methodology

Selection criteria

Randomised controlled trials of any design, non-randomised comparative studies and observational studies were considered for inclusion. Observational studies of less than 10 participants or <80% follow up were to be omitted.

Included studies were required to study a population of adult (aged above 18 years) patients who presented to the ED with SVT, defined as AVNRT, AVRT or atrial tachycardia. Studies describing investigations among patients with sinus tachycardia, Wolf–Parkinson–White Syndrome, AF, atrial flutter or multifocal atrial tachycardia were excluded.

Five investigations of interest were identified for the purposes of this review. These interventions were selected as being commonly requested in this setting. They were (i) troponin assays, (ii) electrolyte levels (including Mg, K, Na, Ca, PO_4), (iii) thyroid function tests, (iv) full blood examination/complete blood count, and (v) chest X-rays (CXRs).

Outcome measures

Eligible studies had to report on at least one of the following prespecified outcomes – proportion of abnormal findings from these investigations, coronary artery disease (CAD), mortality, thyroid disease, electrolyte imbalance, diagnosis of a new medical condition, change in medications, major adverse cardiac events.

Search methods and criteria

Electronic searches

The search strategy was conducted in March 2017. We searched for English language articles in Medline (1992 – March 2017), Embase (1992 – March 2017) and the Cochrane Central Register of Controlled Trials (CENTRAL 1992 – March 2017). Reference lists of relevant studies were scanned for further studies. The complete search strategy for each of these databases is presented in Appendix S1. Details of the protocol for this systematic review were registered on PROSPERO and can be accessed at http://www.crd.york.ac.uk/PROSPERO/display_record.asp?ID=CRD42017060029. (Registration #CRD42017060029).

Selection of studies

The primary researcher (HF) conducted a title screen followed by an abstract screen. The remaining two researchers then reviewed the inclusions. Disputes were settled by consensus. Following this, HF and BM reviewed the full texts of each article identified for inclusion.

Data extraction and management

The primary researcher extracted the data into a standard data extraction table. Extracted data included: study author, year, journal of publication, study design, setting, population, sample size, participant characteristics (type of SVT, age, sex), investigations, outcomes, duration of follow up and loss to follow up. Any discrepancies in coded information were resolved through discussion.

Assessment of risk of bias in included studies

As observational studies were expected, to evaluate risk of bias we used the Newcastle-Ottawa Scale (NOS).[3] This scale assesses quality of non-randomised studies by scoring the studies in three broad categories: selection of study groups, the comparability of the study groups and the ascertainment of either exposure or outcome of interest. Studies may score a maximum of four stars for selection, two stars for comparability and three stars for outcome.

Data synthesis and presentation of results

Studies were evaluated for heterogeneity and suitability for quantitative synthesis/meta-analysis. Only study designs of similar methodology were considered for quantitative data synthesis. When appropriate, the effect size for continuous outcomes for each study was pooled using DerSimonian-Laird random effects model.[4] Heterogeneity was assessed using the Cochrane Q test[5] and I^2 test.[6] The Q test uses the χ^2 distribution to assess whether observed differences in results are attributable to chance alone. However, this test has low power for determining overall heterogeneity when the number of studies is small.[7,8] The I^2 test is not inherently related to number of studies considered. It gives a percentage value of the variation in effect among studies that is attributable to heterogeneity as opposed to chance or random error. A high I^2 statistic is consistent with considerable heterogeneity. Statistical analysis was conducted using STATA v 13.0 (College Station, TX, USA).

Results

Search results and study exclusions

Figure 1 details the search and selection procedures. We found eight studies that fulfilled eligibility criteria. Of these, one study[9] was excluded for including AF and atrial flutter in their patient population. SVTs of interest to us were not presented as a defined sub-group.

Study and patient characteristics

A summary of study characteristics is detailed in Table 1. All seven studies were observational cohort studies (six retrospective and one prospective).[10] Only the study by Chow *et al.*[11] was a multi-centre study with the rest being conducted in a single centre.

The seven included studies contributed data from 1155 patients. Table 1 demonstrates that the definition of SVT used within the seven studies varied from exclusively AVNRT to studies reporting on AVRT and atrial tachycardia. Bukkapatnam *et al.*[12] reported sub-group specific age (mean and standard deviation) but did not report demographics for their entire cohort. Carlberg *et al.*[13] only reported demographics information for the patients who tested troponin

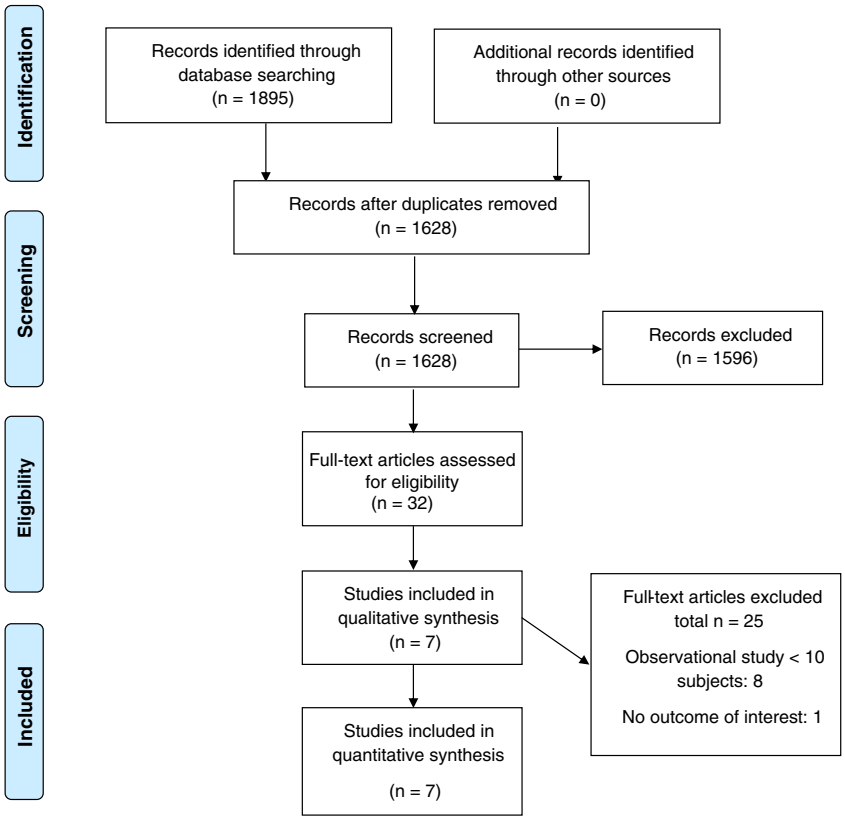

Figure 1. *PRISMA flowchart depicting study selection process.*

positive. All reported demographics figures are consistent with previously stated age and sex estimates for patients presenting with SVT.[14–16]

Study results

Table 2 details the findings of included studies by primary outcomes measured. All included studies addressed one of our pre-specified interventions of troponin levels in patients with SVT. Only one study[17] assessed the other four interventions (thyroid function tests [TSH specifically], electrolyte levels, full blood examination and CXR).

The ability of a positive troponin result to predict CAD was reported in four studies.[10,12,18,19] Future major adverse cardiac events among troponin positive patients were reported in two studies.[11,13] Ashok *et al.*[17] reported on the proportion of abnormal results among the remaining four interventions within the SVT population as well as the impact of an abnormal finding on patient management.

Among three studies that reported on the proportion of troponin testing in a population of patients presenting with SVT, the pooled proportion was 0.66; 95% CI 0.44–0.88 (Fig. 2). All seven included studies investigated the prevalence of troponin positive findings among those presenting with SVT who underwent troponin testing. A summary table and meta-analysis of these findings is detailed in Figure 3 (pooled proportion 0.32; 95% CI 0.23–0.41).

Cardiac troponin I was used in three studies,[11–13] while cardiac troponin T was used in another three.[10,18,19] Ashok *et al.*[17] used a mix of assays including a high sensitivity assay. The timing of this troponin measure also differed between studies. Chow *et al.*[11] used the first troponin level measured between 0.5 and 8 h after presentation. Schueler *et al.*[10] measured serial troponins at presentation, 3 and 6 h. Ben Yedder *et al.*[18] used the highest value from two measures at least 6 h apart. Dorenkamp *et al.*[19] used the highest measure either at admission or 4–6 h later. The timing was not stated in three studies.[12,13,17]

The association between troponin result and CAD was reported in four studies.[10,12,18,19] These studies all concluded that troponin elevation in the setting of SVT was not a predictor of CAD.

Schueler *et al.*[10] studied 45 troponin positive patients. Of these 45, six were diagnosed with new CAD and two showed progression of pre-existing CAD requiring percutaneous coronary intervention. Of the 14 troponin positive patients in Dorenkamp *et al.*'s[19] study, 13 underwent coronary angiography and none were found to have significant CAD. The studies of Schueler *et al.*[10] and Dorenkamp *et al.*[19] did not report on CAD prevalence among a troponin negative group (comparison group). The remaining two studies used their troponin negative group as comparators. Ben Yedder *et al.*'s[18] 2011 study found two of their 24 troponin positive patients to have CAD. Both these patients had known pre-existing CAD. This group is compared to the 49 patients in their troponin negative group, three of whom were found to have CAD. Pre-existing CAD was present in two of these three patients. Their study found less than 10% of patients with troponin rise in the setting of SVT to have significant myocardial ischaemia. The raw difference in CAD prevalence between the troponin positive and troponin negative groups was not stated in the study by Bukkapatnam *et al.*[12] However, they state 'neither chest pain nor troponin I increase during SVT was significantly associated with CAD'. This was determined following multivariate regression analysis. No such regression analysis was applied in the study of Ben Yedder *et al.*[18]

Two studies explicitly stated comorbidities of their patient population. Dorenkamp *et al.*[19] found those who underwent coronary angiography to be significantly more likely to have hypertension and hypercholesterolaemia. These patients were also older and more likely to present with chest

TABLE 1. *Characteristics of included studies reporting on utility of investigations in SVT patients*

Study (author, year)	Study design	Number of patients	Population	Age (years), mean (SD)	Female sex	Investigation being reported
Chow *et al.* 2010[11]	Retrospective cohort	78	AVRT, AVNRT, atrial tachycardia	62.2 (15.8)	54%	Elevated cTnI
Schueler *et al.* 2012[10]	Prospective cohort	139	AVNRT	60 (15)	66%	Elevated cardiac troponin
Ben Yedder *et al.* 2011[18]	Retrospective cohort	73	AVNRT, AVRT	60.96 (18)	60%	Elevated cTnT
Bukkapatnam *et al.* 2010[12]	Retrospective cohort	104	Adults with SVT (excluding AF, AFL, atrial tachycardia, sinus tachycardia)	NR	60%	Elevated cTnI
Dorenkamp *et al.* 2007[19]	Retrospective cohort	114	AVNRT, AVRT	50 (15)	57%	Elevated cTnT
Carlberg *et al.* 2011[13]	Retrospective cohort	51	PSVT	NR	NR	Elevated cTnI
Ashok *et al.* 2017[17]	Retrospective cohort	633	Adults with SVT	55.4 (17.7)	62%	Elevated troponin
						Full blood examination
						Electrolyte levels (including Na, K, Cl, Mg, Ca)
						Thyroid stimulating hormone
						CXR

AF, atrial fibrillation; AFL, atrial flutter; AVNRT, atrioventricular nodal re-entrant tachycardia; AVRT, atrioventricular re-entrant tachycardia; Ca, calcium; Cl, chloride; cTnI, cardiac troponin I; cTnT, cardiac troponin T; K, potassium; Mg, magnesium; Na, sodium; NR, not reported; PSVT, paroxysmal supraventricular tachycardia; SVT, supraventricular tachycardia.

pain. Bukkapatnam *et al.*[12] reported that patients who were referred for CAD evaluation were more likely to have a history of CAD compared with those who were not referred. There was no significant difference in the presence of the other cardiovascular risk factors between these two groups.

Utility of troponin rise in predicting future adverse cardiovascular outcomes

The advent of future adverse cardiovascular related outcomes in patients presenting with SVT was reported in two studies with conflicting conclusions. Chow *et al.*'s[11] primary end-points were mortality, myocardial infarction or cardiovascular re-hospitalisation. These end-points were allowed to occur for the duration of the follow-up period with duration 2.2 ± 1.7 years (mean ± SD). Their study found 17 out of 29 troponin positive patients experienced an adverse future outcome compared with six out of 49 patients in the troponin negative group. After multivariate analysis, the only significant predictor of the combined end-point in their study was an elevated cTnI (constituting a >3-fold increase in risk). In addition, event free survival by Kaplan–Meier analysis was significantly lower in those with an elevated cTnI. Carlberg *et al.*[13] found one of their 11 troponin positive patients to experience an adverse 30 day outcome. The outcomes in this study included mortality, ED return, paroxysmal SVT recurrence and exacerbation of medical co-morbidity. The single outcome achieved was an ED return for a non-cardiac purpose. The 30 day outcomes within the troponin negative group were not analysed. Neither of these studies reported whether troponin testing lead to interventions aimed at preventing future adverse outcomes.

Other investigations

Ashok *et al.*[17] found 114 of their 633 patients underwent thyroid function testing with eight findings of a high TSH result and eight of a low TSH result. Treatment was not initiated in the ED based on these results.

In the same study,[17] CXRs were requested in 190 of their 633 patients, of which 41 were found to be abnormal with all of these findings deemed coincidental and not related to the SVT presentation. None of these CXR findings altered ED management.

TABLE 2. *Effect of investigation on primary outcome measures in studies reporting on utility of investigations in patients presenting with SVT*

Investigation	Study (author, year)	Proportion of abnormal findings (%)	Primary outcome measure	Result
Troponin	Chow *et al*. 2010[11]	29/78 (37%) elevated troponin	MACE (over mean follow up period 2.2 years [SD 1.7])	cTnI elevation in setting of SVT associated with greater than triple the future risk of death, MI, or cardiovascular re-hospitalisation
	Carlberg *et al*. 2011[13]	11/38 (29%) elevated troponin	MACE (30 days)	Not significant
	Schueler *et al*. 2012[10]	45/139 (32%) elevated troponin	CAD	No comparator
	Ben Yedder *et al*. 2011[18]	24/73 (33%) elevated troponin	CAD	Not significant
	Bukkapatnam *et al*. 2010[12]	37/80 (46%) elevated troponin	CAD	Not significant
	Dorenkamp *et al*. 2007[19]	14/114 (12%) elevated troponin	CAD	No comparator
	Ashok *et al*. 2017[17]	105/302 (35%) elevated troponin	Mx in ED	NR
Full blood examination	Ashok *et al*. 2017[17]	27/514 (5%) anaemic	Mx in ED	Nil treated
		98/514 (19%) raised WCC		
Electrolyte levels	Ashok *et al*. 2017[17]	35/530 (7%) low K	Mx in ED	Eight treated with K
		11/530 (2%) high K		14 treated with Mg (all other disturbances not treated)
		11/364 (3%) low Mg		
		30/364 (8%) high Mg		
		27/530 (5%) low Na		
		8/530 (2%) high Na		
		27/530 (5%) low Cl		
		26/530 (5%) high Cl		
		2/249 (1%) low Ca		
		5/249 (2%) high Ca		
Thyroid testing	Ashok *et al*. 2017[17]	8/114 (7%) high TSH	Mx in ED	Nil treated
		8/114 (7%) low TSH		
CXR	Ashok *et al*. 2017[17]	41/190 (22%) abnormal CXRs	Mx in ED	Nil treated

Ca, calcium; Cl, chloride; CXR, chest X-rays; K, potassium; MACE, major adverse cardiac event; Mg, magnesium; MI, myocardial infarction; Mx, management; Na, sodium; NR, not reported; TSH, thyroid stimulating hormone; WCC, white cell count.

Ashok *et al*.[17] also found 514 of their 633 patients to have undergone full blood examination (FBE). Of these, 98 had a high white cell count. Twenty-nine had a low haemoglobin (Hb) count and six had a high Hb count. None of these FBE findings altered ED management.

Finally, Ashok *et al*.[17] also investigated electrolyte levels among their SVT patients. Summary of serum electrolyte testing is provided in Table 2. Of the electrolytes measures, this study found only magnesium and potassium testing to alter ED management by means of IV supplementation. Of the eight patients who received potassium supplementation,

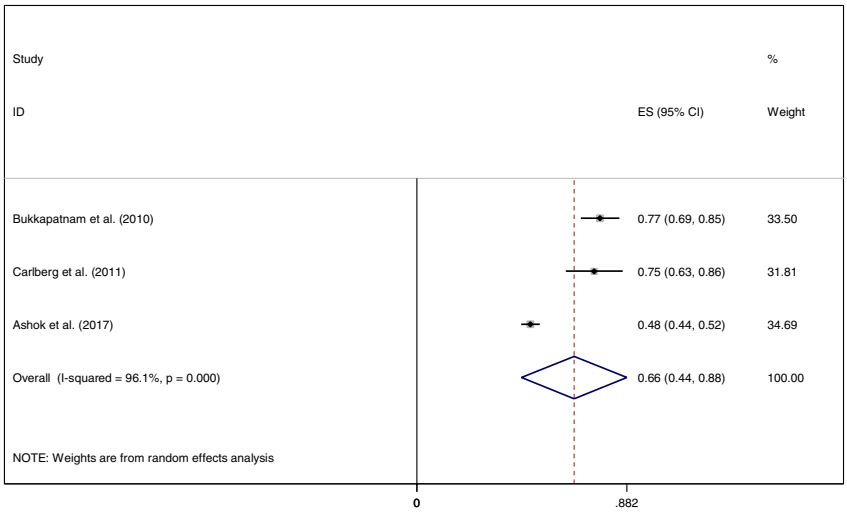

Figure 2. *Forest plot showing pooled proportion of troponin testing among patients presenting with SVT.*

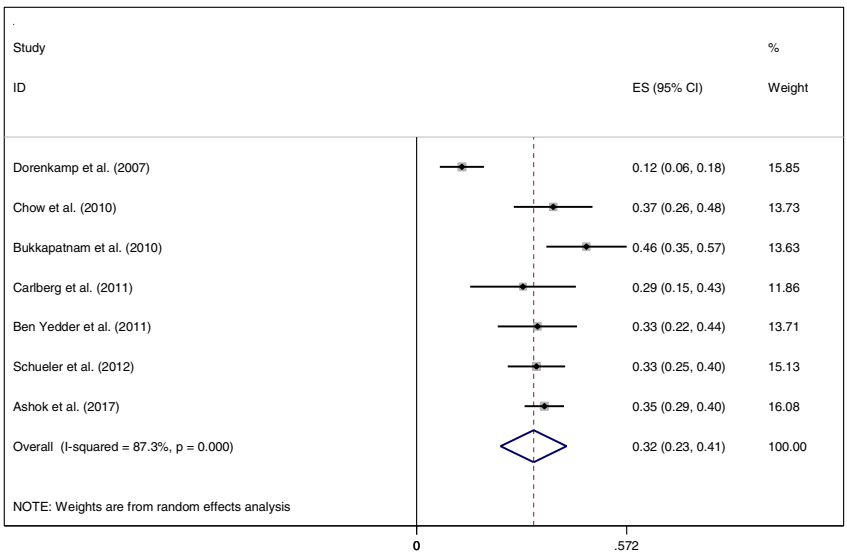

Figure 3. *Forest plot showing pooled proportion of troponin positive findings among patients presenting with supraventricular tachycardia (SVT) who underwent troponin testing.*

seven were found to have low serum potassium with one patient returning a normal result. Of the 14 patients who received IV magnesium in the ED, two had low serum magnesium, two had raised serum magnesium and a further six of these patients had serum magnesium within the normal range. Magnesium was not requested in three patients who received IV supplementation and the level was unknown in the final patient. None of the calcium, chloride or sodium findings were acted on.

Quality of included studies

Table 3 provides a summary of the quality of included studies. While four[10–12,18] of our seven studies identified important confounding variables through medical record review, only Chow *et al.*[11] and Bukkapatnam *et al.*[12] implemented regression analysis to adjust for known confounders. These two studies were scored highly in the comparability category. By the nature of our interventions of interest, most studies scored fairly high on the 'selection' category. The ascertainment of exposure was easily identified by medical record review in all studies. Schueler *et al.*[10] and Dorenkamp *et al.*[19] scored low in this category for not reporting their outcomes in a comparator group. Four studies[11–13,18] lost points in the outcome/exposure category for loss to follow up as they did not report on testing patients for their respective outcomes. This can be attributed to the retrospective nature of their studies. It was also deemed that the 30 day follow-up period in the study by Carlberg *et al.*[13] was not long enough to allow for some of their outcomes to occur.

Discussion

Seven studies with significant statistical heterogeneity assessed the utility of adjunct investigations among patients presenting with SVT to the ED. Troponin testing was commonly performed and commonly found to be positive. However, only one study suggested some benefit in its ability to predict future adverse events. All other investigations were assessed by only one study that found them to be of limited utility. The low quality of the current evidence and significant statistical heterogeneity among studies preclude from confident conclusions regarding the utility of adjunct investigations among patients presenting with SVT to ED.

This review suggests that many investigations ordered in this setting may be unnecessary and based on low levels of evidence. This potential over-investigation has implications of healthcare costs. Furthermore, over-investigation may be associated with increased ED length of stay,[18] patient anxiety and an increased likelihood of false positives that may trigger further investigations or treatment. The concern of over-investigation was echoed by Ashok *et al.*[17] who reported that some patients with elevated or unknown magnesium levels were being treated with supplementation. In addition, patients in these studies with elevated troponins were admitted for follow-up angiograms that were often normal.

TABLE 3. *Quality of included studies reporting on utility of investigations in patients with SVT*

Author	Selection (max. four)	Comparability (max. two)	Outcome (max. three)
Chow *et al.*[11]	****	**	**
Schueler *et al.*[10]	**		***
Ben Yedder *et al.*[18]	***		**
Bukkapatnam *et al.*[12]	***	**	**
Dorenkamp *et al.*[19]	**		***
Carlberg *et al.*[13]	****		*
Ashok *et al.*[17]	****		***

Quality assessed using the Newcastle-Ottawa scale for observational studies.

The mechanism behind troponin release in the setting of SVT, without CAD is still under speculation. A popular notion is that demand ischaemia is responsible, with the tachycardia causing an increase in myocardial oxygen demand while simultaneously shortening diastole and therefore reducing myocardial oxygen supply. Goette *et al.* found rapid atrial pacing in swine models to induce oxidative stress on the left ventricular myocardium and was associated with compromised microvascular blood flow and cardiac troponin elevation.[20]

Regardless of the mechanism, findings of this review suggest clinicians should be cautious in their interpretation of troponin elevation in this setting. We recommend troponin testing be considered in the setting of SVT to rule out acute coronary syndromes or CAD when patients present with acute symptoms or ST segment changes on ECG indicative of ischaemia. In the event of a residual troponin elevation following resolution of SVT, further investigations may include provocative testing with coronary angiography being reserved for those at high risk.

Investigations of electrolyte levels appear to be directed towards efforts to correct serum levels. This practice seems to be based on little evidence in the setting of AVNRT, AVRT or atrial tachycardia, instead relying on studies linking deficiencies in these two electrolytes to ventricular dysrhythmias and AF.[21–24] Although magnesium supplementation has been shown to be beneficial in post-pneumonectomy patients in preventing SVT,[25] there is limited evidence to support electrolyte supplementation in patients with SVT who present to ED.

Similarly, although hyperthyroidism has been associated with AF[26,27] and current guidelines[28] recommend thyroid testing in these patients, no evidence for association between thyroid disease and SVT (AVNRT, AVRT, atrial tachycardia) exists.

This review is limited in being able to include evidence of low quality because of retrospective study designs. However, a broad search strategy was implemented given expected paucity of evidence. We cannot exclude the possibility of publication bias when conducting this review, small studies or studies yielding negative findings may not have been published. Important studies may have also failed to report key outcomes. Additionally, our statistical analysis was limited by heterogeneity of data. When few studies for pre-specified outcomes were found, the results were presented only with descriptive statistics. Sub-group analysis was not possible due to a lack of sub-group specific data. Data extraction templates as well as methods of analysing study quality were predetermined prior to searching the databases to reduce author bias. The strength of our review lies in the comprehensive literature search strategy.

Conclusion

Limited data from observational studies suggest troponin testing is frequently performed after patients present to the ED with SVT. Elevations of troponin in this patient population were not likely to correlate with CAD at presentation. We found insufficient evidence to confidently comment on the utility of the remaining interventions (thyroid testing, full blood examination, electrolyte levels and CXRs) in this patient population.

Acknowledgements

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Author contributions

HF was the primary researcher and was responsible for searching the databases, screening the search strategy, extracting data and performing qualitative data synthesis as well as drafting the manuscript. NA and BM both made significant contributions to study conception and design and were responsible for revising and editing preliminary drafts for intellectual content. Full text review was performed by HF and BM. Quantitative data synthesis and meta-analysis was performed by

BM. All three researchers have approved of the final version and agree to be held accountable for all aspects of the work.

Competing interests

None declared.

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Supporting information

Additional supporting information may be found in the online version of this article at the publisher's web site:

Appendix S1. Complete search strategy.