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The lack of evidence-based management in electrical storm: a scoping review

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Abstract

Electrical storm (ES) is associated with a significant risk of morbidity and mortality. Despite this, there has been limited research in ES resulting in uncertainty and inconsistency in the management of this life-threatening condition. The objective of this scoping review was to define the current body of literature evaluating pharmacologic and non-pharmacologic therapies used in the management of ES. A comprehensive search of Medline, CENTRAL and Embase was completed on January 11, 2025. Primary studies on pharmacotherapies in ES were included if they reported therapy-related outcomes and included ≥ 5 adult patients. A total of 45 studies met the inclusion criteria. Four studies were randomized control trials (three trials had overlapping cohorts) and the remaining were observational studies. Amiodarone, quinidine, landiolol, isoproterenol, and mexiletine were the most studied medications. The use of sedation for ES was exclusively studied in the context of mechanical ventilation. There was an increase in the number of ES studies over time, but sample sizes remained small and unchanged. Existing evidence to guide the management of ES is predominantly based on small observational studies. High quality data to inform the management of ES is needed.

Keywords Electrical storm, Ventricular tachycardia, Pharmacologic management, Evidence-based medicine

Introduction

Electrical storm (ES), defined as three or more sustained or treated ventricular arrhythmias (VAs) within 24 h, has increased in prevalence owing to improvements in heart failure outcomes and the use of implantable cardiac defibrillators (ICDs) [1, 2]. ES occurs in 4% and 11% of patients with primary and secondary prevention ICDs during the first 4 years of device implantation, respectively, and has been reported in up to 25% certain high-risk subpopulations [3–6]. Outcomes of patients with ES are poor, characterized by significant short- and long-term morbidity and mortality. Specifically, ES is associated with a 4.8 and 2.5-fold increase in re-hospitalization

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and death compared to those with VAs in the absence of ES [7, 8].

The current tenets of therapy include anti-arrhythmic drugs (AADs), neurohormonal modulation with pharmacologic and percutaneous interventions, and catheter ablation. There has been a focus on catheter ablation and percutaneous interventions, such as stellate ganglion block (SGB) and renal artery denervation, as potential temporizing or definitive therapies, although confirmatory high-quality studies are lacking [1]. These interventions require specific resources and expertise limiting their integration and widespread use in current clinical practice [9]. Conversely, pharmacologic therapies and common critical care interventions are more commonly used and are arguably the foundation of the acute management of ES in contemporary practice. Yet, despite their widespread use, the current evidence of pharmacologic and intensive care therapies is similarly sparse.

Given the high morbidity and mortality associated with ES and the increasing frequency with which the condition is expected to be encountered in clinical practice, there is a need for contemporary and robust evidence to guide its management. The objective of this scoping review is to comprehensively summarize the current body of literature on pharmacologic management strategies for ES.

Results

Of 22,320 studies screened, 45 met the inclusion criteria (Fig. 1) [5, 10–52]. Studycharacteristics are highlighted in Table 1. In total, 1720 patients were included, with a median sample size of 14 (IQR 10.5, 29.5) patients per study. Of the included studies, 12 defined their cohort with "recurrent" or "incessant" VA but met the current definition of ES based on their inclusion criteria [18, 22–24, 26, 27, 29, 33, 34, 36, 38, 44]; 11 [18, 22, 23, 26, 27, 29, 33, 34, 36, 38, 44] were published before 1999 when ES was not formally defined; we retrospectively applied the definition. Thirty-three [12, 13, 17–20, 24–39, 41, 42, 44–52] studied a prespecified therapy [12, 13, 17–20, 24–39, 41, 42, 44–51] and the remaining 12 [5, 10, 11, 14–16, 21–23, 40, 43, 53] were exploratory studies that provided therapy-related outcomes. All studies reported arrhythmic response to therapy and 35 studies reported mortality [5, 12–25, 27, 29, 31–38, 40, 42, 44–47, 49, 51–53].

Of the 45 studies included, 37 described their studied therapy as effective or useful in their conclusion (positive study) [5, 11–14, 17–21, 23–26, 28, 30–34, 36–52], 6 did not comment on drug specific outcomes (descriptive studies) [11, 15, 16, 22, 35, 53] and 2 were negative studies [27, 29](Table 1). The two negative studies were trials published in 1995 and 1996; one trial showed no difference in arrhythmia burden or mortality between amiodarone and bretylium and the other trial showed no difference in their primary endpoint of response to therapy between doses of amiodarone [27, 29].

Randomized control trials

There were four randomized control trials (RCT; Table 2) [13, 27, 29, 44]. Three of these trials had overlapping patients in an evaluation of amiodarone and bretylium for first line therapy in ES [27, 29, 44]. Kowey et al. [27] compared low-dose and high-dose amiodarone to bretylium in 302 participants whereas Levine et al. [29] and Scheinman et al. [44] compared three different doses of amiodarone in 273 and 342 participants, respectively. In these studies, the 48-h and 90-day mortality rates were 20% and 52% respectively. The most recent RCT included 60 participants and compared propranolol to metoprolol in addition to amiodarone in initial management of ES [13]. This study found no mortality at three months.

Studied therapies

The most studied therapies were AADs (Table 3). Amiodarone was the most frequently evaluated drug (13 studies) [11, 14, 23, 26, 27, 29, 33–35, 38, 39, 44, 51]; followed by quinidine in 6 studies [17, 30, 40, 43, 47, 52]; and landiolol [24, 32, 48]isoproterenol [10, 40, 50] and mexiletine [41, 43, 46] in 3 studies each. AADs were investigated as first-line treatments in all but two studies that assessed the efficacy of esmolol [51] and flecainide [19] as second-line treatments.

Modulation of sympathetic tone in patients with ES was first described in 2000 by Nademanee et al. [37]. In their non-randomized study, they demonstrated that sympathetic suppression with SGB and/or beta-blockers decreased one-week mortality from 82 to 22% compared to advanced cardiac life support (ACLS) guidelines [37]. Excluding SGB and other percutaneous interventions, sympathetic suppression has only been studied in refractory ES with deep sedation with mechanical ventilation [12, 31]. No studies evaluated other forms of pharmacologic sympathetic suppression in either initial or refractory ES.

Studied patient subgroups

Four studies investigated ES in Brugada syndrome. Isoproterenol, as a monotherapy or in addition to quinidine or denopamine, for acute treatment was examined in three studies [10, 40, 50]. Quinidine monotherapy in the acute treatment of Brugada ES was studied once [43]. These studies had sample sizes ranging from 7 to 14 patients. Methylprednisolone was studied for the acute treatment of sarcoid ES in a case-series of five patients [25].

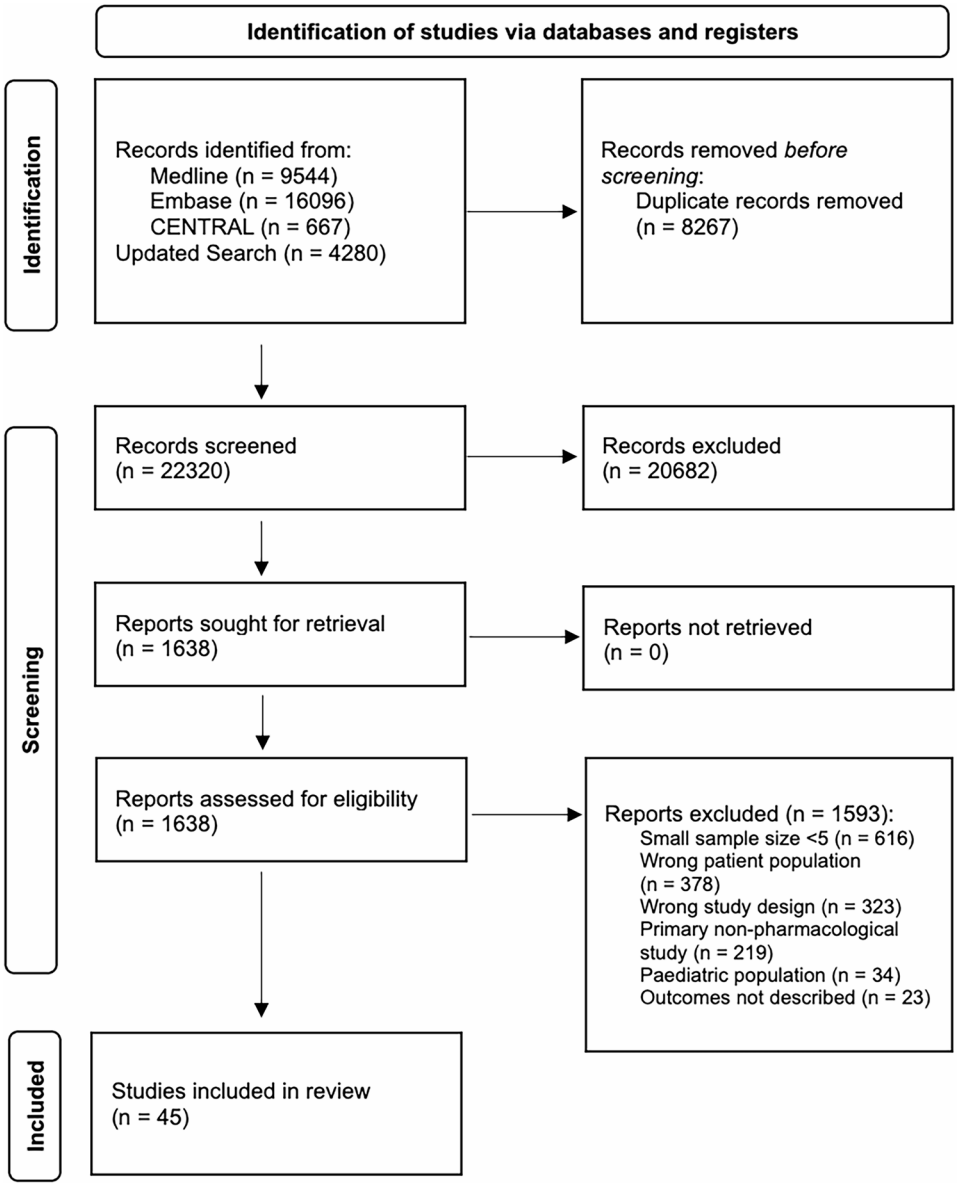

Fig. 1 Prisma Flow Diagram

Temporal trends in studies

There has been an increase in the number of relevant studies published over time ($r = 0.413$, $p = 0.03$; Fig. 2). The study sample size has remained stable over time ($r = -0.113$, $p = 0.48$; Fig. 3).

Discussion

Our scoping review identified 43 studies evaluating the pharmacologic management of ES including a total of 1695 patients. The body of literature includes 4 RCTs, 3 of which had overlapping patient cohorts, with all other studies consisting of single-arm observational studies. Pharmacologic neurohormonal modulation has only been studied in the form of deep sedation requiring mechanical ventilation and non-specific beta-blockers, whereas milder sedatives agents such as benzodiazepines and dexmedetomidine have not been studied. Amiodarone has the most data to support its use as the first-line AAD whereas the use of other AADs or the use of second-line agents are minimally supported by existing data. Lastly, while the number of published ES studies have increased over time, study sample sizes have remained small and stable.

Current European and American guideline recommendations are predominantly based on small studies, data from non-ES cohorts, animal studies, and expert opinion. This has resulted in considerable inconsistencies both in their recommendations and in clinical practice [7, 54–56]. A European Survey on ES demonstrated significant heterogeneity in prescribed therapies as well as the

Table 1 Study characteristics and outcomes

Study	Year	Study Sample Size (*n*)	ES Sample Size (*n*)	Main study Drug	Reported Outcome	ES Mortality (%)
Aizawa et al. [10]	2013	14	14	Isoproterenol	Positive	NS
Avila et al. [52]	2024	17	12	Quinidine	Positive	2 (16.7)
Awan et al. [11]	2009	25	13	Amiodarone	Descriptive	0 (0)
Bundgaard et al. [12]	2020	15	11	Deep sedation	Positive	1 (9.1)
Chatzidou et al. [13]	2018	60	60	Propranolol Metoprolol	Positive	0 (0)
Chia et al. [14]	2012	7	7	Amiodarone Lidocaine	Positive	1 (14.3)
Coromilas et al. [15]	2019	18	18	NS	Descriptive	8 (44.4)
Credner et al. [5]	1998	136	14	NS	Positive	1 (7.1)
Damonte et al. [16]	2022	61	61	NS	Descriptive	3 (4.9)
Deshmukh et al. [17]	2021	23	7	Quinidine	Positive	2 (28.6)
Dhurandhar et al. [18]	1980	18	18	Bretylium	Positive	8 (44.4)
Fuchs et al. [19]	2008	10	10	Flecainide Class III AAD	Positive	3 (30)
Giove et al. [20]	2013	48	NS	Phenytoin	Positive	11
Haissaguerre et al. [21]	2009	122	16	NS	Positive	2 (12.5)
Hariman et al. [22]	1990	17	17	NS	Descriptive	6 (35.3)
Helmy et al. [23]	1988	46	16	Amiodarone	Positive	4 (25)
Ikeda et al. [24]	2019	29	29	Landiolol	Positive	3 (10.3)
Kafil et al. [25]	2021	5	5	Methylprednisolone	Positive	1 (20)
Klein et al. [26]	1988	13	8	Amiodarone	Positive	NS
Kowey et al. [27]	1995	302	302	Bretylium Amiodarone	Negative	97 (32.1)
Kusz et al. [28]	2017	13	5	Ranolazine	Positive	NS
Levine et al. [29]	1996	273	273	Amiodarone	Negative	143 (52.4)
Li et al. [30]	2021	30	12	Quinidine	Positive	NS
Martins et al. [31]	2020	116	116	Sedation	Positive	45 (38.8)
Miwa et al. [32]	2010	42	42	Landiolol	Positive	17 (40.5)
Mooss et al. [33]	1990	35	35	Amiodarone	Positive	16 (45.7)
Morady et al. [34]	1983	15	14	Amiodarone	Positive	4 (28.6)
Morita et al. [54]	2024	44	13	NS	Descriptive	1 (7.7)
Murata et al. [35]	2015	30	30	Amiodarone	Descriptive	8 (26.7)
Nademanee et al. [36]	1985	35	20	Sotalol	Positive	2 (10)
Nademanee et al. [37]	2000	49	49	Sympathetic Modulation	Positive	30 (61.2)
Ochi et al. [38]	1989	22	NS	Amiodarone	Positive	NS
Ogiso et al. [39]	2015	14	14	Amiodarone	Positive	NS
Ohgo et al. [40]	2007	67	7	Isoproterenol Denopamine Quinidine	Positive	0 (0)
Otuki et al. [41]	2016	44	44	Nifekalant Mexiletine	Positive	NS
Rai et al. [42]	2018	6	6	Fosphenytoin	Positive	2 (33.3)
Santos et al. [43]	2011	17	17	Quinidine Nadolol Mexiletine	Positive	NS
Scheinman et al. [44]	1995	342	305	Amiodarone	Positive	71 (23.3)
Sendra-Ferrer et al. [45]	2019	6	6	Ibutilide	Positive	2 (33.3)
Sobiech et al. [46]	2017	17	12	Mexiletine	Positive	0 (0)
Viskin et al. [47]	2019	43	23	Quinidine	Positive	4 (17.4)
Wada et al. [48]	2016	51	6	Landiolol	Positive	NS
Washizuka et al. [49]	2005	11	11	Nifekalant	Positive	1 (9.1)
Watanabe et al. [50]	2006	7	7	Isoproterenol	Positive	NS
Yiping et al. [51]	2014	15	15	Amiodarone Esmolol	Positive	1 (6.7)

Table 2 Randomized control trials in electrical storm

Study	Intervention	Primary Outcome
Chatzidou et al. (2018) [13]	Group 1 ($n=30$): - Propranolol 40 mg every 6 h. - Amiodarone 300 mg over 10 min followed by 1000 mg/24 h for 48 h. Group 2 ($n=30$): - Metoprolol 50 mg every 6 h. - Amiodarone 300 mg over 10 min followed by 1000 mg/24 hours for 48 h.	- Group 1 was associated with 2.67 and 2.34 times decreased risk of VA and ICD discharges in the ICU compared to Group 2. - At 24 h, 90% and 53.3% were free of arrhythmic events with Group 1 and 2, respectively. - Time to arrhythmia termination and length of hospital stay was significantly shorter with Group 1. - There were no in-hospital deaths
Kowey et al. (1995) [27][a]	Group 1 ($n=94$): - Low dose amiodarone (125 mg/24 hours) - Supplemental dosing of amiodarone 150 mg or Bretylium 100 mg over 10 min. Group 2 ($n=105$): - High dose amiodarone (1000 mg/24 hours). - Supplemental dosing of amiodarone 150 mg or Bretylium 100 mg over 10 min. Group 3 ($n=103$): - Bretylium (2500 mg/24 hours). - Supplemental dosing of amiodarone 150 mg or Bretylium 100 mg over 10 min.	- At 6 h, more hemodynamically destabilizing VT/VF events in Group 1 compared to Group 2 and 3. - At 48 h, no significant difference in the number of destabilizing VT/VF events. - Total cohort survival at 48 h was 86% which was similar between groups, - Total cohort survival at 30 days was 68% which was similar between groups.
Levine et al. (1996) [29][a]	Group 1 ($n=88$) - Amiodarone 525 mg/24 hours - Supplementation with amiodarone 150 mg/10 minutes. Group 2 ($n=92$) - Amiodarone 1050 mg/24 hours - Supplementation with amiodarone 150 mg/10 minutes. Group 3 ($n=93$) - Amiodarone 2100 mg/24 hours - Supplementation with amiodarone 150 mg/10 minutes.	- Time to first VA was significantly longer in Group 2 and 3 in comparison to Group 1. - Number of supplemental amiodarone doses was significantly greater with Group 1 compared to Group 3. - There was no significant difference in mortality at 24 h between the groups. The 90-day mortality was 52%.
Scheinman et al. (1995) [44][a]	Group 1 ($n=117$): - Low dose amiodarone (125 mg/24 hours) - Supplemental dosing of amiodarone 150 mg Group 2 ($n=119$) - Amiodarone 500 mg/24 hours - Supplementation with amiodarone 150 mg/10 minutes. Group 3 ($n=106$) - Amiodarone 1000 mg/24 hours - Supplementation with amiodarone 150 mg/10 minutes.	- There was a non-significant decrease in median arrhythmic events with increasing doses from 0.07, 0.04, and 0.02 events per hour for Group 1, 2 and 3 respectively. - No difference in mortality at 48 h between groups; cohort survival to 48 h was 80%.

Abbreviations: ICD implantable cardioverter defibrillator, *ICU* intensive care unit, *VA* ventricular arrhythmia, *VF* ventricular fibrillation, *VT* ventricular tachycardia

[a]Trials with overlapping patients

Table 3 Most studied therapies

Study Drug	Number of Studies	References
Amiodarone	13	[11, 14, 23, 26, 27, 29, 33, 35, 35, 38, 39, 44, 51]
Quinidine	6	[17, 30, 40, 43, 47, 52]
Landiolol	3	[24, 32, 48]
Isoproterenol	3	[10, 40, 50]
Mexiletine	3	[41, 43, 46]
Bretylium	2	[18, 27]
Sedation with Mechanical Ventilation	2	[12, 31]
(Fos)Phenytoin	2	[20, 42]
Nifekalant	2	[41, 49]
Sotalol	1	[36]
Propranolol	1	[13]
Metoprolol	1	[13]
Lidocaine	1	[14]
Non-specific class III AAD	1	[19]
Flecainide	1	[19]
ACLS	1	[37]
Methylprednisolone	1	[25]
Ranolazine	1	[28]
Sympathetic Blockade	1	[37]
Ibutilide	1	[45]
Nadolol	1	[43]
Denopamine	1	[40]
Esmolol	1	[51]

threshold before advancing to more invasive therapies such deep sedation and intubation, mechanical support and percutaneous neurohormonal modulation. Agents such as sotalol and flecainide are used despite their evidence being limited to small single-cohort observational studies [19, 36]. Therapies such as procainamide, verapamil and disopyramide, are used in the acute management ES in the absence of any evidence [9]. Paradoxically, some of the most studied medications like landiolol and nifekalant are not readily available in intensive care units, or in the case of quinidine which requires special access in North America [57].

Most observational studies concluded that their studied therapy improved outcomes or were effective in the management of ES in the absence of comparator arms. In the published RCTs, three had overlapping patients and the fourth study did not include a sample size calculation, raising concerns of potential publication and ascertainment biases, and therefore overestimation of benefits and underestimation of harm [58, 59].

ES is a challenging condition to study given the heterogeneity of clinical presentation and of patients' underlying cardiac substrates or 'triggers'. Most affected patients are treated in cardiac and medical intensive care units–settings that pose unique challenges for study design and

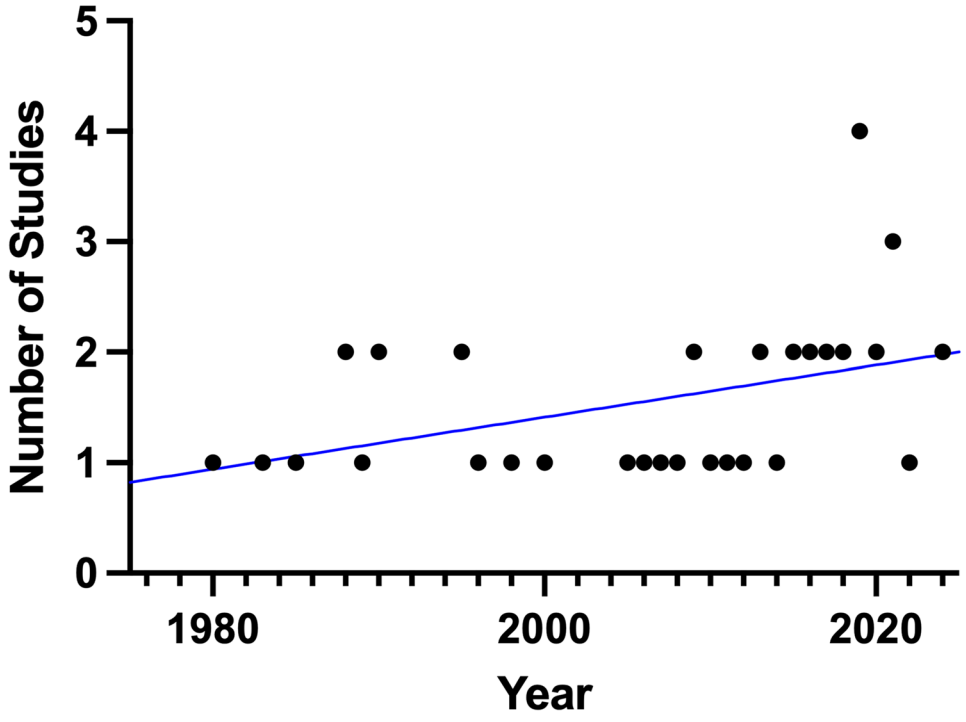

Fig. 2 Trend of the number of published studies over time. The solid line is the trend in the number of published studies over time

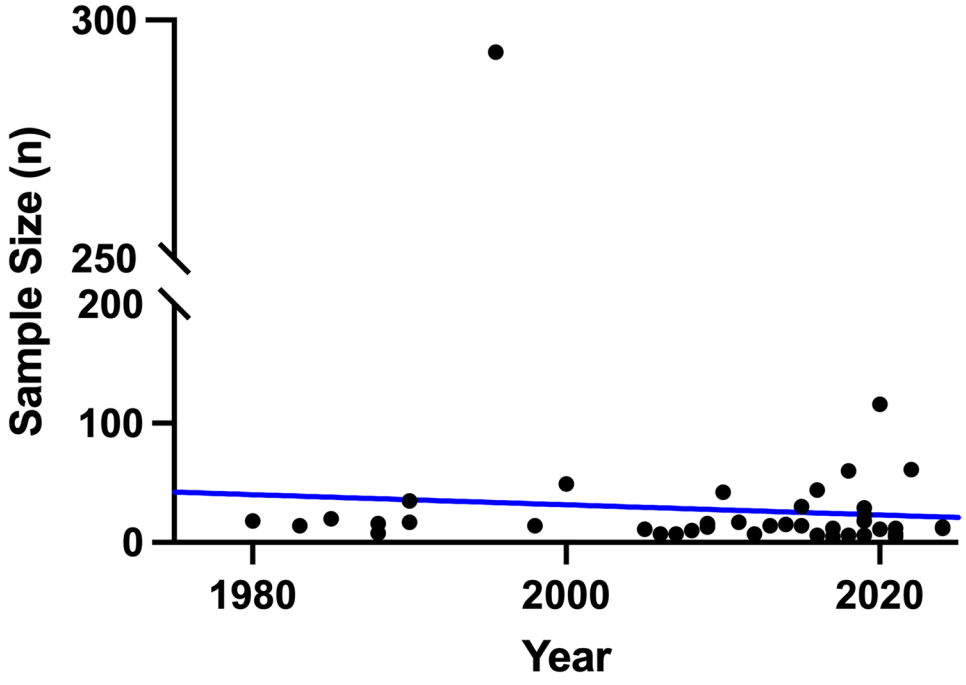

Fig. 3 Temporal trends in sample size over time. The dashed line is the trend in sample size over time

feasibility [60]. However, large studies in ES have been completed (albeit mostly in the 1990s), arguing against these challenges being prohibitive [27, 29, 45]. To advance care for patients suffering with ES, the electrophysiology and critical care communities must come together to address the current gaps in knowledge. One strategy to mitigate these challenges is the development of large, multicentre networks. The STellate ganglion block for Arrhythmic stoRm (STAR) and IntErnational eLeCTRicAl storm (ELECTRA) registries have been recent initiatives. The STAR registry is an observational before-after study and ELECTRA is prospective observational cohort

[61, 62]. Such efforts may yield higher quality observational data that could serve to guide more robust interventional studies. Currently, only one multicentre RCT is actively recruiting patients with ES. The Study Evaluating Dexmedetomidine in the Acute Treatment of Electrical storm (SEDATE), is a phase III multi-centre, double-blinded, placebo-controlled, RCT on the efficacy of dexmedetomidine in the initial treatment of ES (ClinicalTrials.gov Identifier: NCT06281977).

This review has several limitations. Title and abstract screening was completed by a single reviewer; however, a liberal approach to inclusion to full-text screening was used as evidenced by the 1627 studies that underwent full-text readings by two reviewers. Of these, only 45 studies met inclusion criteria (< 3%). It is also important to note that in the studies published before 1999, the definition of ES was retrospectively applied which can lead to confounding based on our reviewers' interpretation of the patient population. This review did not include risk of bias or quality assessment tools given the nature of scoping reviews. Given that the majority of these studies were small and observational, there is concern for bias and certainty of evidence. Furthermore, while this study aimed to assess the overall landscape of the ES literature, we cannot complete any further analysis based on the etiology of heart disease, and other important factors such as sex, as there is limited reporting for these variables. This is also limited by the heterogeneity in the clinical trials, and absence of comparator arms in the observational studies. Lastly, grey literature was not screened, which may contain additional studies in ES.

In conclusion, ES is increasing in incidence and is associated with significant morbidity and mortality. Yet, the current evidence guiding its management is limited, consisting mostly of small observational studies. High-quality studies in this underrepresented patient population are needed.

Methods

This scoping review was conducted in accordance to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) [63]. The protocol was registered prospectively on Figshare on March 8, 2023 (https://doi.org/10.6084/m9.figshare.22236532.v1).

Search strategy

A comprehensive literature search using Medline, CENTRAL and Embase was performed from inception to March 8, 2023. An updated search was completed on January 16, 2025. The search strategy was developed using a combination of free text words and Medical Subject Heading Terms (Appendix A) and included 'recurrent' and 'incessant' VA given that a number of studies published on ES used this terminology before a formal definition was created in 1999. Eligible and relevant articles were reviewed to identify additional studies. There were no restrictions on date or language of publication.

Article selection

The population, concept, context (PCC) model was used define the eligibility criteria [64]. The population include in this review was adult patients (≥ 18 years of age) with ES. The original definition of ES (≥ 2 sustained or treated VAs in 24 h) was used to prevent excluding early studies [65]. Studies with 'recurrent' or 'incessant' VAs that would have otherwise met ES criteria based on the studies' inclusion criteria or reported patient data were also included to prevent excluding earlier studies before ES was formally defined. The core concept of this review was to identify pharmacologic agents and intensive care-specific therapies (non-ablation, surgical, or percutaneous interventions) for the management of ES. Included studies needed to include primary data on treatment outcomes. The context of this study was to include all primary literature. Reviews, surveys and guideline statements were excluded as were small studies with sample sizes of < 5 patients.

Screening was completed using Covidence (Covidence systematic review software, Veritas Health information, Melbourne, Australia. Available at www.covidence.org). Covidence removed duplicates prior to manual screening; no automation tools were used during screening. Given the extensive search strategy, abstract and title screening was completed by a single reviewer. Full-text screening was completed independently by two reviewers. Disagreements were resolved by an independent third reviewer.

Data extraction and synthesis

Data extraction was completed by two independent reviewers. An extraction template was created that included variables such as study year, design, sample size, pharmacotherapies studied, number of patients per therapy (as studies included multiple therapies), ES subtype, patient demographics and studied outcomes. All study outcomes included mortality or arrhythmic response to therapy.

Descriptive statistics were used to summarize the current body of evidence for pharmacological therapy in ES. Temporal trends in the number of published studies and sample sizes were analyzed using Pearson correlation coefficients.

Abbreviations

AAD	anti–arrhythmic drugs
ACLS	advanced cardiac life support
ES	electrical storm
ICD	implantable cardiac defibrillators

SGB stellate ganglion block
VA ventricular arrhythmias

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12872-025-05024-9.

Supplementary Material 1.

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Authors' contributions
P.M., G.P.P., F.D.R. and B.H. created the study protocol. P.M., N.Q., N.B., M.M., W.K., G.P.P., W.K., S.P., P.D., O.A.R., D.N. and R.J. completed study screening and data extraction. P.M., R.J, and G.P.P. completed data analysis and figure preparation. All authors (P.M. G.P.P, M.M., N.B., W.K., N.Q., S.P., P.D.S, O.A.R., R.J., N.K.,W.B., D.N., T.S., J.C.J., R.M., G.A.W., F.D.R., B.H.) reviewed and approved the manuscript.

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Data availability
Data are available on request by contact through the corresponding author.

Declarations

Ethics approval and consent to participate
Not applicable.

Consent for publication
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Competing interests
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