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REVIEWS

The value of electrocardiography in prognosticating clinical deterioration and mortality in acute pulmonary embolism: A systematic review and meta-analysis

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The role of electrocardiography (ECG) in prognosticating pulmonary embolism (PE) is increasingly recognized. ECG is quickly interpretable, noninvasive, inexpensive, and available in remote areas. We hypothesized that ECG can provide useful information about PE prognostication. We searched MEDLINE, EMBASE, Google Scholar, Web of Science, abstracts, conference proceedings, and reference lists through February 2017. Eligible studies used ECG to prognosticate for the main outcomes of death and clinical deterioration or escalation of therapy. Two authors independently selected studies; disagreement was resolved by consensus. Ad hoc piloted forms were used to extract data and assess risk of bias. We used a random-effects model to pool relevant data in meta-analysis with odds ratios (ORs) and 95% confidence intervals (CIs); all other data were synthesized qualitatively. Statistical heterogeneity was assessed using the I^2 value. We included 39 studies (9198 patients) in the systematic review. There was agreement in study selection (κ: 0.91, 95% CI: 0.86-0.96). Most studies were retrospective; some did not appropriately control for confounders. ECG signs that were good predictors of a negative outcome included S1Q3T3 (OR: 3.38, 95% CI: 2.46-4.66, $P < 0.001$), complete right bundle branch block (OR: 3.90, 95% CI: 2.46-6.20, $P < 0.001$), T-wave inversion (OR: 1.62, 95% CI: 1.19-2.21, $P = 0.002$), right axis deviation (OR: 3.24, 95% CI: 1.86-5.64, $P < 0.001$), and atrial fibrillation (OR: 1.96, 95% CI: 1.45-2.67, $P < 0.001$) for in-hospital mortality. Several ischemic patterns also were significantly predictive. Our conclusion is that ECG is potentially valuable in prognostication of acute PE.

KEYWORDS

Clinical Deterioration, Electrocardiography, Meta-analysis, Mortality, Prognostication, Pulmonary Embolism

1 | INTRODUCTION

Acute pulmonary embolism (PE) can rapidly lead to hemodynamic collapse and death. Current guidelines endorse risk-stratifying patients, because those at high risk of clinical deterioration or death can be considered for additional treatment beyond anticoagulation, including thrombolysis or thrombectomy.[1] Patients at low risk can generally be treated as outpatients.[1] Risk-stratification approaches include hemodynamic status, clinical scores, blood biomarkers, and computed tomographic (CT) or echocardiographic findings. Although the guidelines discuss electrocardiographic (ECG) findings in PE, the use of ECG as a prognostic tool is not reviewed.[1] ECG is noninvasive, rapidly interpretable, low cost, and is one of the first tests performed in the emergency department. It is also available in remote areas with a scarcity of modern technological modalities.

Daniel et al. developed an ECG scoring system in 2001 (Daniel score) for the severity of pulmonary hypertension in patients with PE.[2] It included tachycardia, right bundle branch block (RBBB), T-wave inversion (TWI), and S1Q3T3.[2] A score was assigned from 0 to 21, with a higher score indicating a worse clinical outcome.[2–4] Since

the publication of the Daniel score, several other studies have investigated the use of ECG as a tool for PE prognostication. These studies expanded the use of ECG and included findings not included in the Daniel score, such as ST-segment depression, ST-segment elevation (STE), Qr in lead V_1, right axis deviation (RAD), and P pulmonale, among others.[5–10] A recent consensus article by the International Society of Electrocardiology, the International Society for Holter and Noninvasive Electrocardiology, and the Iberoamerican Forum of Arrhythmias in the Internet demonstrated the need for a formal and comprehensive evaluation of the evidence for the use of ECG to prognosticate PE.[11]

We aimed to comprehensively evaluate the data on ECG as a tool to prognosticate PE by performing a systematic review and meta-analysis of the available evidence. In this article, we focused on clinical deterioration and death as prognostic outcomes.

2 | METHODS

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) and Meta-Analysis of Observational Studies in Epidemiology (MOOSE) statements for reporting our systematic review and meta-analysis.

We searched MEDLINE and EMBASE through February 2017 using keywords, MeSH terms, and Emtree headings. In addition, we searched Google Scholar and the Web of Science and examined abstracts, conference proceedings, and reference lists of retrieved articles. Two authors (AQ, GD) independently screened titles and abstracts and retrieved eligible articles if they (1) reported data on the prognostication of acute PE, (2) used ECG in their prognostic model, and (3) diagnosed PE formally by CT pulmonary angiogram, ventilation-perfusion scan, or autopsy. For this article, we only included studies reporting mortality, or clinical deterioration defined as any of the following: (1) new hemodynamic collapse; (2) treatment upgrading (eg, thrombolysis, surgical thrombectomy); (3) intubation or resuscitation; or (4) systolic blood pressure consistently <100 mm Hg, refractory to volume loading and requiring vasopressors. We excluded studies not written in English.

All disagreements were resolved by consensus and consultation with a senior author (AB). We extracted data in a standardized manner using an ad hoc abstraction form containing study information and quality criteria. We systematically assessed study quality by evaluating the study population, definition of outcomes and ECG findings and their assessment, attrition bias, identification of confounders, and baseline imbalance (see Supporting Information, Table, in the online version of this article).

2.1 | Statistical analysis

We analyzed data with the R package (R Foundation for Statistical Computing, http://www.r-project.org) using the DerSimonian-Laird

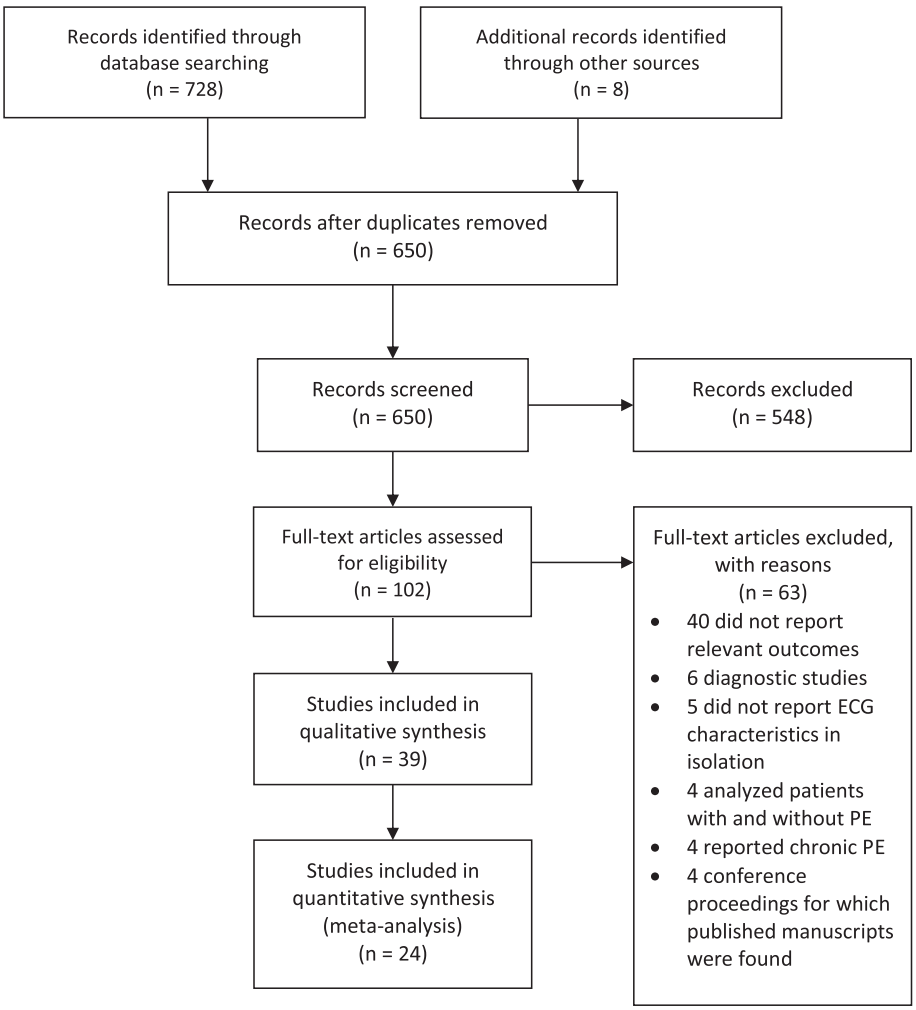

FIGURE 1 Flow chart of the selection process for inclusion of articles in this systematic review and meta-analysis. Abbreviations: ECG, electrocardiographic; PE, pulmonary embolism

TABLE 1 Characteristics of included studies

Study	Design	Outcomes	N	Male Sex, N (%)	Mean Age, y (SD)	Included in Meta-Analysis?	Comments
Agrawal 2014[5]	PC	In-hosp mortality; clin deterioration	200	123 (61.5)	43.8 (NR)	Y	—
Akgullu 2015[6]	RC	In-hosp mortality	206	97 (47.1)	61.8 (11.8)	Y	Excluded patients with missing lab values and patients with AF
Barra 2013[22]	RC	In-hosp, 1-mo, and 6-mo mortality	270	106 (39.3)	70.1 (15.8)	Y	—
Bouvier 2015[33]	PC	30-d mortality or clin deterioration	141	—	—	N (abstract)	—
Bulj 2012[39]	PC	In-hosp mortality; clin deterioration	104	38 (36.5)	68.7 (13.4)	N (abstract)	—
Buppajarntham 2014[24]	RC	In-hosp mortality; clin deterioration	300	122 (40.7)	60.3 (17.6)	N	—
Ermis 2010[25]	RC	In-hosp mortality; clin deterioration	129	69 (53.5)	58.0 (16.5)	Y	Excluded patients with AF at admission
Escobar 2007[31]	PC	1-mo mortality; 15-d mortality due to PE	644	277 (43.0)	—	Y	Only included hemodynamically stable patients at admission
Gallotta 2008[37]	PC	Clin deterioration	90	25 (27.8)	67.0 (18.0)	Y	Excluded patients with renal failure, recent ACS, and hemodynamically unstable patients at admission
Geibel 2005[30]	PC	30-d mortality	508	214 (42.1)	63.0 (15.0)	Y	—
Hariharan 2015[3]	PC	Adverse clinical event[a]	290	147 (51.0)	59.0 (17.0)	N	Performed subanalysis excluding patients with chronic lung or cardiac disease—did not change results
Huang 2011[27]	RC	30-d mortality	150	96 (64)	71.3 (14.8)	Y	Excluded patients with recent ACS
Icli 2015[29]	RC	30-d mortality	272	118 (43.4)	63.1 (16.8)	N	Excluded patients with missing lab values or echocardiograms
Janata 2012[10]	RC	In-hosp mortality; clin deterioration for STE-aVR	396	192 (48.5)	59.8 (18.5)	Y	—
Kayrak 2013[28]	RC	30-d mortality	359	168 (46.8)	63.6 (15.8)	Y	—
Koracevic 2007[26]	RC	In-hosp mortality	125	39 (31.2)	62.5 (—)	N (abstract)	—
Kostrubiec 2009[7]	RC	In-hosp mortality; clin deterioration	56	22 (39.3)	64.3 (17.9)	Y	—
Kostrubiec 2010[8]	RC	In-hosp mortality; clin deterioration	94	42 (45.0)	63.0 (19.0)	N	—
Kosuge 2006[38]	RC	In-hosp mortality; clin deterioration	40	15 (37.5)	63.0 (13.0)	N	No group with normal ECG; TWI in all groups
Kucher 2003[14]	RC	In-hosp mortality; clin deterioration	75	—	—	Y	—
Kukla 2011A[42]	RC	Clin deterioration	292	109 (37.3)	65.4 (15.5)	Y	—
Kukla 2011B[43]	RC	Clin deterioration	293	111 (38.0)	65.4 (15.5)	Y	—
Kukla 2011C[15]	RC	In-hosp mortality	225	88 (39.1)	66.0 (15.2)	Y	—
Kukla 2011D[16]	RC	In-hosp mortality; clin deterioration	292	109 (37.3)	65.4 (15.5)	Y	—
Kukla 2014A[36]	RC	Clin deterioration	500	210 (37.3)	65.4 (15.5)	Y	—
Kukla 2014B[17]	RC	In-hosp mortality; clin deterioration	245	103 (42.0)	66.3 (15.2)	Y	—
Kukla 2015A[18]	RC	In-hosp mortality; clin deterioration	437	170 (38.9)	67.4 (19.0)	Y	TWI presumed secondary to LBBB or LVH
Kukla 2015B[19]	RC	In-hosp mortality; clin deterioration	971	408 (42.0)	66.0 (15.0)	Y	Excluded 35 patients due to missing or poor-quality ECG
Kumasaka 2000[13]	RC	In-hosp mortality	139	47 (33.8)	64.0 (15.0)	Y	—
Lee 2002[35]	PC	Clin deterioration	65	25 (38.5)	59.4 (15.9)	Y	—
Ryu 2010[4]	RC	In-hosp mortality	125	56 (44.8)	62.7 (13.6)	N	Excluded uninterpretable ECGs
Stein 1997[41]	PC	Circulatory collapse[b]	123	—	—	N	Only included patients with no history of cardiac/pulmonary disease

(*Continued*)

TABLE 1 Continued

Study	Design	Outcomes	N	Male Sex, N (%)	Mean Age, y (SD)	Included in Meta-Analysis?	Comments
Subramaniam 2008[34]	PC	12-mo mortality	105	59 (56.2)	58 (median)	N	—
Tayama 2002[20]	RC	In-hosp mortality	35	7 (20.0)	62 (—)	Y	—
Toosi 2007[9]	RC	In-hosp mortality; clin deterioration	159	70 (44.0)	58.9 (17.7)	Y	Excluded those with no ECG
Vanni 2009[21]	PC	In-hosp mortality; clin deterioration	386	153 (39.6)	67.0 (16.0)	N	Excluded hemodynamically unstable and those with cardiac/pulmonary disease
Zhan 2014[40]	RC	Clin deterioration	20	8 (40.0)	58.0 (10.0)	N	Cardiac/pulmonary disease excluded
Zhan 2015[32]	RC	1-mo mortality; clin deterioration	210	92 (43.8)	57.9 (14.4)	N	Cardiac/pulmonary disease excluded
Zorlu 2012[23]	PC	In-hosp mortality	127	62 (48.8)	64.0 (13.0)	N	Patients with no lab values excluded

Abbreviations: ACS, acute coronary syndrome; AF, atrial fibrillation; clin, clinical; ECG, electrocardiogram; In-hosp, in-hospital; lab, laboratory; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; N, no; NR, not reported; PC, prospective cohort; PE, pulmonary embolism; RC, retrospective cohort; SBP, systolic blood pressure; SD, standard deviation; STE, ST-segment elevation; TWI, T-wave inversion; Y, yes.

[a] Adverse clinical event was defined as cardiac arrest, new arrhythmia, respiratory support, use of vasopressors, thrombolysis or thrombectomy, major bleeding, recurrent PE, or death from any cause within 5 days.

[b] Circulatory collapse defined as loss of consciousness or SBP <80 mm Hg.

random-effects model. We evaluated between-study heterogeneity using the I^2 index.[12] We reported associations as odds ratios (ORs) and 95% confidence intervals (CIs). We excluded instances in which studies had no events for a particular ECG finding and prognostic outcome, rather than performing a continuity correction in empty cells. We conducted a sensitivity analysis to evaluate whether performing a continuity correction would have changed the association. We used a funnel plot and the Egger test to evaluate the potential for publication bias. Whenever pooling was not possible, qualitative evaluations were made on individual studies.

3 | RESULTS

3.1 | Article selection

There was agreement between reviewers for study screening (κ: 0.91, 95% CI: 0.86-0.96). We identified 650 unique records. Seventy studies reported the prognostication of PE using ECG, but only 39 (9198 patients) reported mortality or clinical deterioration data and met inclusion criteria (Figure 1). Most included studies were retrospective cohort in design, and some studies did not appropriately control for confounders (Table 1 and Supporting Information, Table, in the online version of this article).

3.2 | In-hospital mortality

Twenty studies (4898 patients) reported data on in-hospital mortality.[4–7,9,10,13–26] Several ECG features were meta-analyzed for this outcome (Table 2). Figure 2 shows 4 sample forest plots for the association between in-hospital mortality and each of the following ECG findings: S1Q3T3, any RBBB, TWI in precordial leads, and TWI in precordial or inferior leads. Statistically significant predictors from the meta-analysis included S1Q3T3, S1Q3T3 variations, complete RBBB, any RBBB, TWI in precordial or inferior leads, ST-segment depression in leads V_4 through V_6, ST-segment depression in any lead, STE-V_1, STE-III, Qr-V_1, RAD, and atrial fibrillation (AF) at admission. Heterogeneity was generally low. Removing instances in which no events were reported for a particular ECG sign did not have a significant impact on the association compared with performing a continuity correction (data not shown). Some studies could not be pooled, and their findings are summarized in Table 2.

Some studies reported adjusted in-hospital mortality data. Only STE-V_1 could be pooled and was found to be significantly predictive (Table 2). All other adjusted in-hospital mortality data could not be pooled. Single studies identified complete RBBB and the number of leads with TWI to be significantly predictive (Table 2).

Some studies reported continuous ECG measures as predictors of in-hospital mortality. Akgullu et al[6] looked at QT-interval dispersion and P-wave dispersion and found that patients who died had a longer dispersion for both measures (median and interquartile range [IQR]: 104 [97–119] vs 78 [68–84], and 73 [54–79] vs 48 [35–55], respectively; $P < 0.001$ for both). Ermis and colleagues[25] investigated QT-interval dispersion and also found a longer mean (SD) dispersion in the group that died (89 [46] vs 65 [23]; $P = 0.001$). They also reported that a QT-interval dispersion of 71.5 ms had a sensitivity of 71%, a specificity of 73%, and an area under the curve (AUC) of 0.73 (SE: 0.54; $P = 0.001$).

Kostrubiec et al[7] reported the median (IQR) for the Daniel score and found it to be nonsignificantly higher in patients who died: 3.5 (0–15) vs 3 (0–18). The AUC (95% CI) for a score ≥ 3 in this study was 0.52 (0.37-0.64). Toosi et al[9] also found a nonsignificantly higher mean (SD) Daniel score in patients who died: 7.5 (SD not reported) vs 4.4 (5.6). The AUC (SE) with a score ≥ 3 for this study was 0.63 (0.078). Ermis et al[25] reported median (IQR) for the Daniel score and found a significantly higher Daniel score for patients who died: 6.0 (7.0) vs 4.0 (4.0), $P = 0.007$. Ryu et al[4] used a Daniel score cutoff of 12 and found that 3/38 (8%) of those in the high-ECG score group died, whereas 12/87 (14%) of those in the low-ECG group died ($P = 0.55$).

TABLE 2 ECG findings as prognosticators of mortality in acute PE

ECG Sign	No. of Studies (No. of Patients)	OR	95% CI	*P* Value	I^2, %	References
In-hospital mortality						
Sinus tachycardia	5 (737)	1.93	0.96-3.89	0.067	42.6	5,6,9,13,14
S1Q3T3	9 (2155)	3.38	2.46-4.66	<0.001	0	6,7,9,10,13,15–18
S wave in lead I	3 (250)	1.16	0.33-4.06	0.819	0	7,9,20
Q wave in lead III	3 (250)	0.47	0.13-1.71	0.251	0	7,9,20
TWI in lead III	2 (215)	1.58	0.45-5.54	0.473	38.1	7,9
S1Q3T3 variations[a]	3 (629)	1.94	1.02-3.72	0.0447	0	5,10,14
Complete RBBB	5 (1173)	3.90	2.46-6.20	<0.001	0	6,7,10,15,16
Incomplete RBBB	4 (833)	2.38	0.94-6.01	0.0672	35.9	6,9,10,14
Any RBBB	9 (1982)	3.02	2.10-4.34	<0.001	5.52	6,7,9,10,13–16,18
TWI in precordial leads	9 (1535)	1.40	0.99-1.98	0.057	1.20	5,7,9,10,13–16,20
TWI in precordial/inferior leads	11 (2197)	1.62	1.19-2.21	0.002	7.22	5,7,9,10,13–16,18,20
ST-segment depression in V_4 through V_6	2 (517)	2.50	1.43-4.36	0.0013	0	15,16
ST depression, any	5 (1258)	2.26	1.54-3.32	<0.001	0	6,10,13,15,16
STE in lead aVR	3 (913)	1.68	0.79-3.56	0.176	81.8	10,15,16
STE in lead V_1	4 (985)	4.27	2.73-6.66	<0.001	0	10,14–16
STE in lead III	2 (517)	3.08	1.63-5.81	<0.001	0	15,16
Qr sign in V_1	3 (589)	4.72	2.54-8.78	<0.001	0	14–16
RAD	4 (798)	3.24	1.86-5.64	<0.001	11.4	5,6,13,16
LAD	2 (498)	1.52	0.49-4.70	0.468	68.5	6,16
Low-voltage limb leads	2 (517)	1.59	0.63-4.03	0.323	0	15,16
Low-voltage limb/precordial	3 (723)	1.69	0.75-3.79	0.203	0	6,15,16
Clockwise rotation	3 (760)	1.44	0.83-2.51	0.190	0	10,14,16
P pulmonale	4 (1019)	1.71	0.76-3.85	0.196	35.7	6,10,15,16
AF at admission	4 (1733)	1.96	1.45-2.67	<0.001	0	15,16,19,22
AF	1 (125)	NR	NR	0.736	—	26 (abstract)
RV strain pattern[b]	1 (386)	4.13	1.22-14.0	0.023	—	21
Adjusted in-hospital mortality						
STE in V_1	2 (688)	3.23	1.71-6.11	<0.001	19.2	10,16
Complete RBBB	1 (396)	5.790	2.47-13.6	<0.001	—	10
Sum of TWI	1 (292)	0.81	0.69-0.95	0.0098	—	16
No. of leads with TWI	1 (292)	1.68	1.68-2.26	0.00068	—	16
AF at admission	1 (971)	1.4	0.8-2.3	0.2	—	19
Prolonged QTc	1 (300)	1.3	0.3-6.6		—	24
AF at admission[c]	1 (127)	0.449	0.17-1.22	0.115	—	23
30-day mortality						
Sinus tachycardia	3 (1302)	1.66	1.20-2.31	0.0025	0	27,30,31
S1Q3T3	4 (1661)	0.99	0.69-1.41	0.949	0	27,28,30,31
Any RBBB	3 (1302)	1.14	0.74-1.77	0.555	17.1	27,30,31
TWI – precordial leads	2 (658)	1.36	0.92-2.02	0.123	0	27,30
AF at admission	2 (395)	2.47	1.18-5.17	0.0167	25.6	22,27
Low-voltage peripheral leads	1 (508)	1.94	1.24-3.04	0.0039	—	30
STE in I, II, or V_4 through V_6	1 (508)	2.03	1.06-3.90	0.034	—	30
ST depression in I, II, or V_4 through V_6	1 (508)	2.52	1.30-4.90	0.0063	—	30
Q in III, aVF not II	1 (508)	1.58	1.04-2.39	0.032	—	30
Tpeak-Tend interval	1 (272)	12.9	3.05-54.7	0.001	—	29
AF[§]	1 (141)	6.3	1.05-37.7	NR	—	33 (abstract)
TWI[d]	1 (141)	6.1	1.3-29.1	NR	—	33 (abstract)
Adjusted 30-day mortality						
Sinus tachycardia	1 (644)	2.4	1.30-4.20	0.003	—	31

(*Continued*)

TABLE 2 Continued

ECG Sign	No. of Studies (No. of Patients)	OR	95% CI	*P* Value	I^2, %	References
Any abnormality[e]	1 (508)	2.56	1.49-4.57	<0.001	—	[30]
AF/flutter	1 (210)	1.36	0.39-4.80	NS	—	[32]
S1Q3T3	1 (210)	3.16	0.99-10.2	0.052	—	[32]
LAD	1 (210)	1.26	0.44-3.68	NS	—	[32]
RAD	1 (210)	1.02	0.34-3.07	NS	—	[32]
Low QRS voltage	1 (210)	1.63	0.43-6.22	NS	—	[32]
Clockwise rotation	1 (210)	0.28	0.05-1.58	NS	—	[32]
Notched S in V_1	1 (210)	1.22	0.40-3.78	NS	—	[32]
RBBB in V_1	1 (210)	0.64	0.16-2.55	NS	—	[32]
Qr sign in V_1	1 (210)	0.77	0.24-2.53	NS	—	[32]
No. of leads with TWI	1 (210)	1.18	0.96-1.45	NS	—	[32]
LV subendocardial ischemic pattern	1 (210)	3.711	0.78-17.6	NS	—	[32]
RV transmural ischemic pattern	1 (210)	4.22	1.14-15.6	0.031	—	[32]
LV subendocardial + RV transmural ischemia	1 (210)	4.02	1.13-14.3	0.032	—	[32]

Abbreviations: AF, atrial fibrillation; CI, confidence interval; ECG, electrocardiographic; HR, hazard ratio; LAD, left axis deviation; LV, left ventricular; NS, not significant; OR, odds ratio; PE, pulmonary embolism; QTc, corrected QT interval; RAD, right axis deviation; RBBB, right bundle branch block; RV, right ventricular; ST, ST segment; STE, ST-segment elevation; TWI, T-wave inversion.

[a] Includes S1Q3T3/S1Q3, S1S2S3, S1Q3/S1rSr3′/S1S2S3.

[b] Included ≥1 of: complete or incomplete RBBB, S waves in lead I combined with Q waves in lead III with or without T inversion in lead III (S1Q3T3), or inverted T waves in precordial leads V_1, V_2, and V_3.

[c] This study specifically looked at mortality related to PE and reported an HR, not an OR.

[d] Included 30-day mortality or clinical deterioration as an outcome.

[e] Any 1 of: atrial arrhythmia; complete RBBB; peripheral low voltage; Q in leads III and aVF, but not in II; STE in leads I, II, and V_4 through V_6; and ST depression in leads I, II, and V_4 through V_6.

3.3 | Thirty-day mortality

Eight studies (2354 patients) reported data on 30-day mortality.[22,27–33] Statistically significant features in the meta-analysis included sinus tachycardia and AF at admission (Table 2). The I^2 value was generally low. Individual studies also reported adjusted 30-day mortality data and found sinus tachycardia and right ventricular (RV) transmural ischemic pattern to be significantly predictive (Table 2). Numerous other unique ECG features were reported, and these are summarized in Table 2.

3.4 | Longer-term mortality

Barra et al[22] investigated the association between AF at admission and 6-month mortality and found an OR (95% CI) of 3.93 (1.95-7.94; $P < 0.001$). An adjusted model with history of AF had an OR (95% CI) of 2.49 (1.14-5.44; $P = 0.023$). Subramaniam et al[34] examined the association between the Daniel score and 12-month mortality and found no significant difference between groups: mean (SD) of 2.03 (2.34) in patients who died vs 2.40 (2.91) in those alive at 12 months ($P = 0.65$).

3.5 | Clinical deterioration

Twenty-one studies (4105 patients) had clinical deterioration as an outcome.[3,7–9,14,16,18,21,24,25,27,32,35–43] Statistically significant features from the meta-analysis included sinus tachycardia, S1Q3T3, TWI, complete RBBB, any RBBB, ST-segment depression in V_4 through V_6, STE-aVR, STE-V_1, STE-III, Qr-V_1, and AF at admission (Table 3). Findings from studies that could not be pooled are summarized in Table 3. Removing instances in which no events were reported for a particular ECG sign did not have a significant impact on the association compared with performing a continuity correction (data not shown).

Some studies reported an adjusted clinical deterioration outcome. None of these data could be pooled. Statistically significant predictors identified in individual studies included complete RBBB, RV strain, TWI, ST-segment depression in V_4 through V_6, STE-V_1, STE-aVR, Qr-V_1, fragmented QRS in V_1, low QRS voltage, prolonged QTc, left ventricular subendocardial ischemic pattern, and RV transmural ischemic pattern (Table 3).

Kostrubiec et al[7] reported the median (IQR) for the Daniel score and found the score to be significantly higher in patients who had clinical deterioration: 8 (1–17) vs 3 (0–18), $P = 0.04$. The AUC (95% CI) for a score ≥ 3 in this study was 0.73 (0.59-0.84). Toosi et al[9] also found a significantly higher mean (SD) Daniel score in patients that clinically deteriorated: 6.5 (6.1) vs 4.2 (4.3), $P = 0.036$. The AUC (SE) with a score ≥ 3 for his study was 0.64 (0.067).

3.6 | Other adverse clinical outcomes

Stein et al. used ECG to prognosticate which patients with PE would likely have circulatory collapse, defined as loss of consciousness or a systolic blood pressure < 80 mm Hg.[41] Of the ECG findings they investigated, complete RBBB was most predictive, being present in 2 of 5 patients with circulatory collapse and 5 of 118 patients with no circulatory collapse ($P = 0.0257$). Zhan et al. included hemodynamically stable patients at admission and assessed which patients would become hemodynamically unstable.[40] They found S1Q3, abnormal QRS morphology in

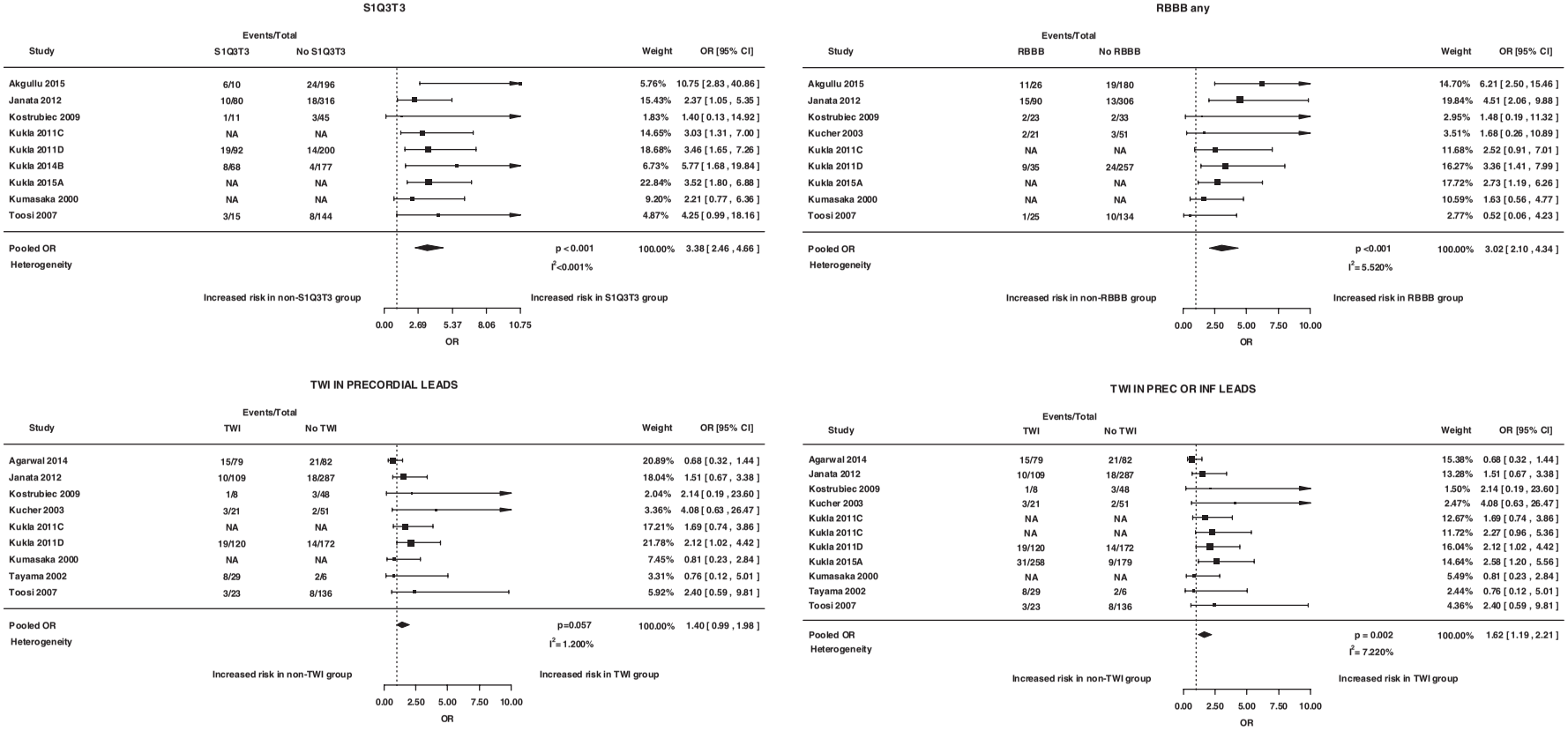

FIGURE 2 Sample forest plots. These 4 graphs show sample forest plots for the association between in-hospital mortality and each of the following ECG findings: S1Q3T3, any RBBB, TWI in precordial leads, and TWI in precordial or inferior leads. Abbreviations: CI, confidence interval; ECG, electrocardiographic; inf, inferior; OR, odds ratio; prec, precordial; RBBB, right bundle branch block; TWI, T-wave inversion

TABLE 3 ECG findings as prognosticators of clinical deterioration in acute PE

ECG Sign	No. of Studies (No. of Patients)	OR	95% CI	*P* Value	I^2, %	References
Clinical deterioration						
Sinus tachycardia	6 (631)	4.61	2.46-8.65	<0.001	40.8	7,9,14,25,27,35
S1Q3T3	8 (1822)	3.89	2.50-6.05	<0.001	59.5	9,16,18,25,27,35–37
S wave in lead I	2 (215)	1.36	0.59-3.11	0.470	0	7,9
Q wave in lead III	4 (409)	1.23	0.58-2.59	0.582	27.5	7,9,25,35
TWI in lead III	3 (280)	2.30	0.67-7.86	0.185	68.5	7,9,35
S1Q3T3 variants[a]	2 (204)	1.46	0.62-3.46	0.384	0	14,25
Complete RBBB	4 (977)	2.47	1.61-3.80	<0.001	0	7,16,25,36
Incomplete RBBB	3 (363)	2.07	0.99-4.33	0.052	0	9,14,25
Any RBBB	10 (1953)	2.04	1.51-2.75	<0.001	7.7	7,9,14,16,18,25,27,35–37
RV strain[b]	1 (386)	5.82	1.82-18.7	0.003	—	21
TWI in precordial leads	10 (1805)	2.46	1.89-3.21	<0.001	9.7	7,9,14,16,25,27,35–37,42
TWI in precordial/inferior leads	11 (2092)	2.45	1.82-3.28	<0.001	35.8	7,9,14,16,18,25,27,35–37,42
ST-segment depression V_4 through V_6	3 (1084)	2.71	2.01-3.67	<0.001	0	16,36,42
STE in lead V_1	4 (1159)	5.14	3.80-6.95	<0.001	0	14,16,36,42
STE-III	3 (1084)	3.06	2.07-4.53	<0.001	9.0	16,36,42
STE-aVR	5 (1773)	3.29	2.14-5.07	<0.001	0	10,16,36,42,43
STE in contiguous leads	1 (94)	3.04	0.47-19.7	0.243	—	8
QR in V_1	3 (864)	4.65	2.05-10.6	<0.001	63.7	14,16,36
Low QRS voltage	1 (292)	0.86	0.40-1.84	0.706	—	16
P pulmonale	1 (292)	2.06	0.85-4.98	0.109	—	16
Clockwise rotation	2 (367)	1.42	0.55-3.69	0.469	62.9	14,16
AF at admission	5 (1978)	1.78	1.35-2.36	<0.001	13.4	16,19,27,35,36
Adjusted clinical deterioration						
S1Q3T3	1 (210)	1.79	0.79-4.08	NS	—	32
Complete RBBB	1 (292)	2.87	1.15-7.19	0.02	—	16
Complete RBBB	1 (40)	NR	NR	0.50	—	38
Complete RBBB	1 (500)	2.95	1.47-5.91	0.002	—	36
RBBB	1 (104)	111	12.7-973	<0.001	—	39 (abstract)
RBBB in V_1	1 (210)	0.91	0.33-2.55	NS	—	32
RV strain[b]	1 (386)	2.58	1.05-6.36	0.038	—	21
Sum of TWI	1 (292)	0.88	0.78-0.98	0.022	—	16
No. of leads with TWI	1 (292)	1.46	1.16-1.85	0.001	—	16
No. of leads with TWI	1 (210)	1.09	0.93-1.30	NS	—	32
7+ leads with TWI	1 (40)	16.8	1.17–213	0.037	—	38
ST-segment depression V_4 through V_6	1 (500)	2.24	1.27-3.96	0.006	—	36
STE in lead V_1	1 (292)	3.99	1.96-8.18	<0.001	—	16
STE in lead V_1	1 (500)	7.62	4.50-12.9	<0.001	—	36
STE in aVR	1 (500)	2.49	1.41-4.39	0.002	—	36
Qr sign in V_1	1 (75)	8.7	1.4–56.7	0.02	—	14
Qr sign in V_1	1 (210)	1.03	0.41-2.60	NS	—	32
Qr sign in V_1	1 (500)	2.66	1.38-5.10	0.003	—	36
Fragmented QRS V_1	1 (500)	3.00	1.48-6.05	0.002	—	36
LAD	1 (210)	0.56	0.23-1.37	NS	—	32
RAD	1 (210)	1.06	0.44-2.55	NS	—	32
Low QRS voltage	1 (500)	3.44	1.57-7.56	0.002	—	36
Low QRS voltage	1 (210)	1.00	0.36-3.09	NS	—	32
Prolonged QTc	1 (300)	4.3	1.3-14.3	<0.001	—	24
Clockwise rotation	1 (210)	0.53	0.16-1.75	NS	—	32
Notched S in V_1	1 (210)	1.53	0.60-3.94	NS	—	32

(*Continued*)

TABLE 3 Continued

ECG Sign	No. of Studies (No. of Patients)	OR	95% CI	*P* Value	I^2, %	References
AF at admission	1 (292)	0.95	0.85-1.05	0.3	—	[16]
AF/flutter	1 (210)	1.02	0.35-2.95	NS	—	[32]
LV subendocardial ischemic pattern	1 (210)	4.96	1.67-14.8	0.004	—	[32]
RV transmural ischemic pattern	1 (210)	3.12	1.19-8.23	0.021	—	[32]
LV subendocardial plus RV transmural ischemic pattern	1 (210)	3.03	1.22-7.56	0.017	—	[32]

Abbreviations: AF, atrial fibrillation; CI, confidence interval; ECG, electrocardiographic; LAD, left axis deviation; LV, left ventricular; NR, not reported; NS, not significant; OR, odds ratio; PE, pulmonary embolism; QTc, corrected QT interval; RAD, right axis deviation; RBBB, right bundle branch block; RV, right ventricular; STE, ST-segment elevation; TWI, T-wave inversion.

[a] Includes S1Q3/S1rSr3′/S1S2S3/Q3T3.

[b] Included ≥1 of: complete or incomplete RBBB, S waves in lead I combined with Q waves in lead III with or without T inversion in lead III (S1Q3T3), or inverted T waves in precordial leads V_1, V_2, and V_3.

V_1, STE-V_1, STE-V_2, STE-III, STE-aVR, ST-segment depression in V_4 through V_6, and ST-segment depression in lead I to be significantly associated with the development of hemodynamic instability.[40]

Hariharan and colleagues performed a prospective study in which they used ECG to predict patients that were more likely to have an adverse clinical course within 5 days, defined as any of the following: cardiac arrest, new arrhythmia, respiratory support, use of vasopressors, thrombolysis or thrombectomy, major bleeding, recurrent PE, or death from any cause.[3] Multiple ECG findings were significantly predictive of this outcome. They then performed a multivariate analysis and found TWI in V_1 through V_3, S wave in lead I, and sinus tachycardia to have OR (95% CI) of 4.76 (1.71-13.28), 2.04 (1.17-3.54), and 2.58 (1.37-4.85), respectively.[3] They developed the "TwiST" score, with 5 points for TWI in V_1 through V_3, 2 points for S wave in lead I, and 3 points for sinus tachycardia.[3] They found a TwiST score ≤ 2 to have a 76% sensitivity and 59% specificity, whereas a TwiST score ≥ 5 had 52% sensitivity and 87% specificity.[3] They also computed test characteristics for the utility of the Daniel score in predicting the adverse clinical outcome and found a score ≤ 2 to have a 57% sensitivity and 74% specificity, whereas a Daniel score ≥ 7 had 44% sensitivity and 87% specificity.

3.7 | Risk of publication bias

Publication bias was not detected by the funnel plot, as all studies had data points falling within the 95% CI bounds (see Supporting Information, Figure, in the online version of this article).

4 | DISCUSSION

This systematic review and meta-analysis of 39 studies (9198 patients) found that ECG features predict a negative outcome in patients with acute PE, including clinical deterioration, in-hospital mortality, and 30-day mortality. Specific features most predictive of in-hospital death included S1Q3T3, complete RBBB, TWI, ST-segment depression in V_4 through V_6, STE-V_1, STE-III, Qr-V_1, RAD, AF, and RV transmural ischemic pattern. Similar findings were predictive of clinical deterioration, although other findings included sinus tachycardia and STE-aVR. Adjusted analyses were generally consistent with these findings. The cause of clinical deterioration and death in patients with PE is usually due to RV failure, so it is expected that ECG features suggesting RV failure would predict a negative outcome.[21] As for 30-day mortality, adjusted analyses from individual studies demonstrated sinus tachycardia and RV transmural ischemic pattern to be significantly predictive. Sensitivity analyses demonstrated that excluding studies with no reported events had minimal impact on the association as compared with applying a continuity correction.

In 2001, Daniel et al. developed a 21-point ECG score for the severity of pulmonary hypertension in patients with PE.[2] Subsequent studies showed that the Daniel score was significantly higher in patients with clinical deterioration, but not significantly higher for in-hospital mortality.[4,7,9,25] Furthermore, the predictive capacity of the Daniel score as measured by the AUC was found to be only modest. Hariharan et al. developed the TwiST score in 2015 with 5 points for TWI in V1 through V_3, 2 points for S wave in lead I, and 3 points for sinus tachycardia.[3] They found the TwiST score to have a slightly higher sensitivity and specificity than the Daniel score for predicting an adverse clinical outcome.[3]

A recent meta-analysis by Shopp et al. reviewed the use of 12-lead ECG to predict circulatory shock in patients with PE.[44] However, they only used ECG findings on the Daniel score (namely, tachycardia, RBBB, TWI in V_1 through V_4, S1Q3T3), in addition to STE-aVR and AF.[44] Our meta-analysis adds updated evidence for the use of ECG and includes features on the Daniel score and newly studied ECG findings since the Daniel score's publication. We found several ECG components to be predictive of clinical deterioration for in-hospital mortality. These findings may be pragmatically useful, as ECG is one of the first tests performed in the emergency department and is noninvasive, rapidly interpretable, and low cost. Additionally, it is available in remote areas with a scarcity of modern technologies. Hence, clinicians may be able to use ECG to appropriately select higher-risk patients requiring more intensive care or monitoring, even if they are deemed low-risk patients by other clinical criteria. This may include normotensive patients with a high risk of RV failure. These patients have been shown to benefit from more intensive care services, including systemic or catheter-directed fibrinolysis and pulmonary selective vasodilation.[45–48]

4.1 | Study limitations

Despite rigorous methodology, our review had some limitations. First, the assessment of publication bias was limited, as only a handful of

ECG finding and outcome associations had ≥10 studies, the minimum number recommended for testing funnel-plot symmetry.[49] Second, most studies were retrospective in design, and some did not control for confounders. As such, higher-quality studies, such as prospective cohort studies with appropriate controlling for confounders, are needed to more definitively assess which ECG findings can offer prognostic information in addition to currently used risk-stratification tools. Finally, some studies did not independently adjudicate ECG features and outcomes, potentially leading to misclassification bias.

5 | CONCLUSION

Acute PE can rapidly lead to hemodynamic collapse and death, and risk-stratifying patients is imperative to determine those requiring more intensive treatment or monitoring. This meta-analysis suggests that ECG can be a valuable tool in the prognostication of PE, especially when modern technology is not accessible. Nonetheless, most studies were retrospective, and some studies did not appropriately control for confounders. Hence, more prospective cohort studies with appropriate controlling for confounders would more definitively evaluate which ECG findings can offer prognostic information in addition to currently used risk-stratification tools. These findings can aid in developing a new ECG scoring system to assist clinicians in risk-stratifying patients with PE.

Conflicts of interest

The authors declare no potential conflicts of interest.

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SUPPORTING INFORMATION

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