The ECG in Suspected Pulmonary Embolism

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Abstract

Objective – To establish the diagnostic value of prespecified ECG changes in suspected PE.

Methods – Retrospective case control study in a district general hospital setting. We identified 189 consecutive patients with suspected pulmonary embolism (PE) whose CT pulmonary angiogram (CTPA) was positive for a first PE and for whom an ECG taken at the time of presentation was available. We matched these for age +/- 3 years with 189 controls with suspected PE whose CTPA was negative. We considered those with large (n=76) and small (n=113) clot load separately. We scored each ECG for the presence or absence of 8 features that have been reported to occur more commonly in PE.

Results – 20-25% patients with PE, including those with large clot load, had normal ECGs. The commonest ECG abnormality in patients with PE was sinus tachycardia (28%). S1Q3T3 (3.7%), p pulmonale (0.5%) and right axis deviation (4.2%) were infrequent findings. RBBB (8.5%), atrial dysrhythmias (10.1%) and clockwise rotation (20.2%) occurred more frequently but were also common in controls. Right ventricular (RV) strain pattern occurred more commonly in patients than controls, 11.2% v 2.7% (sensitivity 17.1%, specificity 97.4%; odds ratio (OR) 4.58, 95% CI 1.63, 15.91; p=0.002), particularly in those with large clot load, 17.1% v 2.6%, (sensitivity 17.1%, specificity 97.4%; OR 7.55, 95%CI 1.62, 71.58; p=0.005).

Conclusion – An ECG showing RV strain in a breathless patient is highly suggestive of PE. Many of the other ECG changes that have been described in PE occur too infrequently to be of predictive value.

Word count 245

Main messages

- The ECG finding that best predicted PE in our study was RV strain pattern.
- S1Q3T3 was uncommon in our study.
- An ECG showing RV strain when present in a breathless patient is highly suggestive of PE.
- Many of the other ECG changes that have been described in PE occur too infrequently to be of predictive value.

Research questions

- It would be interesting to repeat the study in all patients undergoing CTPA and not simply those who had both D-dimer and CTPA.
- We used an estimate of clot load rather than right ventricular dilation as our index of PE severity. It is possible that RV dilation would have been a better marker of severity.
- The use of biochemical markers such as high sensitivity troponin and NT Pro BNP could be used to further refine the assessment of severity.

Key Words

- Electrocardiogram
- Right ventricular strain pattern
- Pulmonary embolism

Introduction

Pulmonary Embolism is one of the most common cardiovascular diseases with 47,594 cases reported in the UK in 2013[1]. The majority of pulmonary artery emboli result from the propagation of a deep vein thrombosis (70-80%)[2]. Pulmonary embolism is the most common cause of vascular death after myocardial infarction and stroke and the leading cause of preventable death in patients admitted to hospital[3]. It is a common mode of death in patients with cancer, stroke and pregnancy[4].

Pulmonary embolism may be categorised as massive, submassive or low risk based on the presence or absence of hypotension and right ventricular dysfunction[5]. Most but not all patients with PE will have risk factors for venous thromboembolism, the most frequently reported of which is immobilisation[3]. The commonest presenting symptoms are dyspnoea and pleuritic pain but both may be absent even in patients with large clot load[6]. Clinical probability scores and D-dimer are recommended in order to determine which patients should have computed tomographic pulmonary angiography (CTPA) but it is well recognised that patients with low probability scores can have PE with large clot load[6] while D-dimer has low specificity for PE and is best regarded as a rule out test[7].

The role of the ECG in PE remains controversial. It is generally accepted that the ECG is of limited value in diagnosis but may predict a poor outcome when abnormal. A number of ECG changes have been reported to occur more commonly in PE including sinus tachycardia, right bundle branch block (RBBB), right ventricular strain pattern, right axis deviation, p pulmonale, S1Q3T3 pattern, clockwise rotation and atrial dysrhythmias[8,9]. Right ventricular strain pattern is recognized by simultaneous T wave inversion in the inferior (II, III, avF) and right precordial leads (V1-4) (Fig 1). These changes are considered to reflect right ventricular dysfunction in patients with large clot load[10-12] and have been shown to predict a poorer outcome when present[8-10,13-15]. Clockwise rotation is said to occur

when the transition zone from dominant S wave to dominant R wave occurs after V4. This can occur in normal subjects, in heart failure and also in patients with acute or chronic pulmonary disease.

Figure 1 about here

It is our belief that these ECG changes might also be a clue to diagnosis and that failure to recognise the significance of these changes in a breathless patient might lead to a delayed or incorrect diagnosis. A difficulty here is that the changes described can occur in patients with other causes of right ventricular dysfunction such as chronic lung disease[16] and that similar but subtly different changes may be present in acute coronary syndrome (ACS), though in ACS with T wave inversion in leads V1–V4 it would be unusual to find T wave inversion in leads III and aVF as well [17,18].

In order to address these issues further we have investigated the diagnostic value of prespecified ECG changes for PE in a cohort of patients all of whom were suspected of having PE and all of whom underwent CTPA.

Methods

This was a retrospective study carried out in Dumfries and Galloway Royal Infirmary, a district general hospital covering a population of 147,000 in southwest Scotland. We selected patients for inclusion if they had presented as an emergency and had both D-dimer and CTPA for suspected pulmonary embolism between September 2012 and March 2016[7]. From a cohort of 1397 patients with suspected PE we identified 189 whose CTPA was positive for a first PE and for whom an ECG taken at the time of presentation was available. We considered patients with large (n=76) and small (n=113) clot load separately[12] (Figure 2). We then matched patients for gender and age ± 3 years with 189 controls whose CTPA were negative. Patients were either admitted to hospital or discharged following CTPA either because this was negative or because we felt their PE could be managed safely in an ambulatory setting.

Figure 2 about here

We screened for the presence of cardiorespiratory disease by retrospective analysis of the electronic case note in order to identify conditions that might contribute to altered signal or 'noise' on the ECG. We defined pre-existing cardiorespiratory disease as myocardial infarction (MI), chronic obstructive pulmonary disease (COPD), pulmonary hypertension (PHTN), pulmonary fibrosis (PF) or obstructive sleep apnoea (OSA). If we were unable to find this information in the admission document we searched GP referrals and clinic letters. To be certain that we did not miss cases of OSA, we cross checked a database of patients that were known to the local sleep service. It had been our intention to match cases and controls for not only for age and sex but also for cardiorespiratory disease though there were too few controls to do this. We were able, however, to match 87 CTPA positive patients with 87 controls suspected of having PE whose CTPAs were negative, for age, sex and absence of cardiorespiratory disease

We printed CTPA reports for all case and controls and assigned each a unique reference number. We defined large clot load as PE in pulmonary trunk/ main pulmonary artery and either the lobar arteries or the remote branches; and small clot load as PE confined to lobar arteries or remote branches[12]. The radiologist (PH) who reviewed the CTPAs did so without knowledge of the ECG findings. We printed ECGs for all cases and controls from the electronic case note for analysis by a cardiologist (AM). Most patients had more than one ECG during their admission. When more than one ECG was present we selected the highest quality ECG that was most closely related in time to its respective CTPA. AM scored each ECG for the presence or absence of 8 features that have been reported to occur more commonly in PE[9] (Table 1) without knowledge of the CTPA result.

For each ECG finding and each patient group (all patients and subgroups by clot load and absence of cardiorespiratory disease), we calculated sensitivity and specificity for identifying PE, along with binomial confidence intervals, odds ratios with 95% confidence limits and p-values from exact Fisher tests comparing cases and controls[19].

ECG finding	Definition
Sinus tachycardia	HR > 100bpm
Complete or incomplete RBBB	QRS duration >120ms with rSR pattern V1-V3
Right ventricular strain pattern	Simultaneous T wave inversion in the inferior (II, III, avF) and right precordial leads (V1-4).
Right axis deviation	Dominant S wave lead I with dominant R wave leads II and III
P pulmonale	Peaked P waves >2.5mm in limb leads or >1.5mm in lead V1.
$S_1Q_3T_3$ pattern	The presence of S waves in lead 1 and Q waves in lead III, each with amplitudes >1.5mmm in association with negative T waves in lead III.
Clockwise rotation	Shift of the R/S transition point (the point at which the R wave becomes dominant) to V5 or beyond implying rotation of the heart due to ventricular dilation.
Atrial tachyarrthythmias	Atrial Fibrillation and Atrial Flutter.

Table 1. ECG changes said to occur more commonly in PE

Results

Our cohort of 189 patients with PE comprised 82 men and 107 women, average age 66 years with age range 20-93 years. Seventy six (40%) were judged to have large clot load and 87 (46%) no evidence of pre-existing cardiorespiratory disease. The distribution of pre-existing cardiorespiratory disease in patients and their age and sex matched controls is shown in figure 3 which illustrates that more control patients had COPD.

Figure 3 about here

Twenty to twenty five percent of patients with PE, including those with large clot load, had normal ECGs (Table 2). The commonest ECG finding in patients with PE was sinus tachycardia though this was not invariable: around 70% patients with large clot load had heart rates <100/min. S1Q3T3 (3.7%), p pulmonale (0.5%) and right axis deviation (4.2%) were infrequent findings. RBBB (8.5%), atrial dysrhythmias (10.1%) and clockwise rotation (20.2%) occurred more frequently but were also common in controls. Right ventricular (RV) strain pattern occurred more commonly in patients than controls (11.2% v 2.7%, p=0.002), particularly in those with large clot load (17.1% v 2.6%, p=0.005) (Table 2).

Table 2. Frequency of ECG findings for all patients and controls, and for subgroups by clot load and absence of cardiorespiratory disease.

ECG finding	**All cases**		**Large clot load**		**Small clot load**		**No cardiorespiratory disease**	
	Cases (%)	Controls (%)	Cases (%)	Controls (%)	Cases (%)	Controls (%)	Cases (%)	Controls (%)
Patients	189 (100)	189 (100)	76 (100)	76 (100)	113 (100)	113 (100)	87 (100)	87 (100)
Normal ECG	43 (22.7)	58 (30.7)	16 (21.1)	24 (31.6)	27 (23.9)	34 (30.1)	20 (23.0)	30 (34)
Any Abnormality	146 (77.2)	131 (69.3)	60 (78.9)	52 (68.4)	86 (76.1)	79 (69.9)	67 (77.0)	57 (66)
Sinus Tachycardia	52 (27.5)	38 (20.1)	22 (28.9)	14 (18.4)	30 (26.5)	24 (21.2)	27 (31.0)	16 (18)
RBBB	17 (9.0)	17 (9.0)	8 (10.5)	5 (6.6)	9 (8.0)	12 (10.6)	8 (9.2)	6 (7)
RV Strain	21 (11.1)	5 (2.6)	13 (17.1)	2 (2.6)	8 (7.1)	3 (2.7)	9 (10.3)	1 (1)

ECG finding	All cases		Large clot load		Small clot load		No cardiorespiratory disease	
	Cases (%)	Controls (%)	Cases (%)	Controls (%)	Cases (%)	Controls (%)	Cases (%)	Controls (%)
RAD	8 (4.2)	5 (2.6)	2 (2.6)	2 (2.6)	6 (5.3)	3 (2.7)	1 (1.1)	0 (0)
P pulmonale	1 (0.5)	0 (0.0)	1 (1.3)	0 (0.0)	0 (0.0)	0 (0.0)	1 (1.1)	0 (0)
S1Q3T3	7 (3.7)	1 (0.5)	5 (6.6)	1 (1.3)	2 (1.8)	0 (0.0)	4 (4.6)	0 (0)
Clockwise Rotation	38 (20.1)	29 (15.3)	15 (19.7)	12 (15.8)	23 (20.4)	17 (15.0)	15 (17.2)	16 (18)
Atrial Tachyarrhythmias	19 (10.1)	24 (12.7)	7 (9.2)	9 (11.8)	12 (10.6)	15 (13.3)	6 (6.9)	11 (13)

Tables 3 and 4 show sensitivities, specificities and odds ratios for each of the 8 ECG abnormalities for all patients, and for patients with large clot load, small clot load and no pre-existing cardiorespiratory disease separately. Sensitivity for each individual ECG finding was always lower than specificity and never more than 31% which is too low for the ECG to be considered a rule out test in patients suspected of having PE. Specificity approached 100% for RV strain, RAD, p pulmonale and S1Q3T3 but with the possible exception of RV strain the prevalence of these ECG abnormalities was too low for the ECG to be of value as a rule in test. RV strain, which was present in 11.1% of all cases, 17.1% cases with large clot load and 10.3% of those with no pre-existing cardiorespiratory disease, was associated with specificities of 97.4% (95% CI 93.9,99.1%), 97.4% (95% CI 90.8,99.7%) and 98.9% (95% CI 93.8,100.0%) respectively (Table 3). Corresponding odds ratios were 4.58, 7.55 and 9.82 though confidence intervals were wide (Table 4). Sensitivities were higher (76-79%) and specificities lower (27-35%) when we combined all ECG findings and considered any ECG abnormality. We did not calculate positive and negative predictive values as these are prevalence dependent risk factors and this was a case control study.

Table 3 Sensitivities and specificities in % for all patients and subgroups by clot load and absence of cardiorespiratory disease, with binomial 95% zconfidence intervals

ECG finding		All cases	Large clot load	Small clot load	No cardiorespiratory disease
Normal ECG	Sensitivity	22.8 (17.0, 29.4)	21.1 (12.5, 31.9)	23.9 (16.4, 32.8)	23.0 (14.6, 33.2)
	Specificity	69.3 (62.2, 75.8)	68.4 (56.7, 78.6)	69.9 (60.6, 78.2)	65.5 (54.6, 75.4)
Any Abnormality	Sensitivity	77.2 (70.6, 83.0)	78.9 (68.1, 87.5)	76.1 (67.2, 83.6)	77.0 (66.8, 85.4)
	Specificity	30.7 (24.2, 37.8)	31.6 (21.4, 43.3)	30.1 (21.8, 39.4)	34.5 (24.6, 45.4)
Sinus Tachycardia	Sensitivity	27.5 (21.3, 34.5)	28.9 (19.1, 40.5)	26.5 (18.7, 35.7)	31.0 (21.5, 41.9)
	Specificity	79.9 (73.5, 85.4)	81.6 (71.0, 89.5)	78.8 (70.1, 85.9)	81.6 (71.9, 89.1)
RBBB	Sensitivity	9.0 (5.3, 14.0)	10.5 (4.7, 19.7)	8.0 (3.7, 14.6)	9.2 (4.1, 17.3)
	Specificity	91.0 (86.0, 94.7)	93.4 (85.3, 97.8)	89.4 (82.2, 94.4)	93.1 (85.6, 97.4)
RV Strain	Sensitivity	11.1 (7.0, 16.5)	17.1 (9.4, 27.5)	7.1 (3.1, 13.5)	10.3 (4.8, 18.7)
	Specificity	97.4 (93.9, 99.1)	97.4 (90.8, 99.7)	97.3 (92.4, 99.4)	98.9 (93.8, 100.0)
RAD	Sensitivity	4.2 (1.8, 8.2)	2.6 (0.3, 9.2)	5.3 (2.0, 11.2)	1.1 (0.0, 6.2)
	Specificity	97.4 (93.9, 99.1)	97.4 (90.8, 99.7)	97.3 (92.4, 99.4)	100.0 (95.8, 100.0)

Table 3 Sensitivities and specificities in % for all patients and subgroups by clot load and absence of cardiorespiratory disease, with binomial 95% zconfidence intervals

ECG finding		All cases	Large clot load	Small clot load	No cardiorespiratory disease
P Pulmonale	Sensitivity Specificity	0.5 (0.0, 2.9) 100.0 (98.1,100.0)	1.3 (0.0, 7.1) 100.0 (95.3,100.0)	0.0 (0.0, 3.2) 100.0 (96.8, 100.0)	1.1 (0.0, 6.2) 100.0 (95.8, 100.0)
S1S2S3	Sensitivity Specificity	1.6 (0.3, 4.6) 98.4 (95.4, 99.7)	2.6 (0.3, 9.2) 98.7 (92.9, 100.0)	0.9 (0.0, 4.8) 98.2 (93.8, 99.8)	2.3 (0.3, 8.1) 100.0 (95.8, 100.0)
S1Q3T3	Sensitivity Specificity	3.7 (1.5, 7.5) 99.5 (97.1, 100.0)	6.6 (2.2, 14.7) 98.7 (92.9, 100.0)	1.8 (0.2, 6.2) 100.0 (96.8, 100.0)	4.6 (1.3, 11.4) 100.0 (95.8, 100.0)
Clockwise Rotation	Sensitivity Specificity	20.1 (14.6, 26.5) 84.7 (78.7, 89.5)	19.7 (11.5, 30.5) 84.2 (74.0, 91.6)	20.4 (13.4, 29.0) 85.0 (77.0, 91.0)	17.2 (10.0, 26.8) 81.6 (71.9, 89.1)
Atrial Tachyarrthythmias	Sensitivity Specificity	10.1 (6.2,15.3) 87.3 (81.7, 91.7)	9.2 (3.8, 18.1) 88.2 (78.7, 94.4)	10.6 (5.6, 17.8) 86.7 (79.1, 92.4)	6.9 (2.6, 14.4) 87.4 (78.5, 93.5)

Table 4 Odds ratios for ECG as predictor of pulmonary embolism for all patients and subgroups by clot load and absence of cardiorespiratory disease.

ECG finding	All		Large clot load		Small clot load		No cardiorespiratory disease	
	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p
Normal	0.67 (0.41, 1.08),	0.103	0.58 (0.26, 1.28),	0.197	0.73 (0.39, 1.37)	0.369	0.57 (0.27, 1.16),	0.131
Any Abnormality	1.50 (0.93, 2.45),	0.103	1.72 (0.78, 3.88),	0.197	1.37 (0.73, 2.59)	0.369	1.76 (0.86, 3.65),	0.131
Sinus Tachycardia	1.51 (0.91, 2.51),	0.116	1.80 (0.79, 4.20),	0.181	1.34 (0.69, 2.61)	0.436	1.99 (0.93, 4.35),	0.078
RBBB	1.00 (0.46, 2.16),	1.000	1.67 (0.45, 6.80),	0.564	0.73 (0.26, 1.98)	0.648	1.36 (0.39, 5.00),	0.782
RV Strain	4.58 (1.63, 15.91),	0.002	7.55 (1.62, 71.58),	0.005	2.78 (0.65, 16.71)	0.215	9.82 (1.31, 438.97),	0.018
RAD	1.62 (0.46, 6.44),	0.573	1.00 (0.07, 14.13),	1.000	2.05 (0.42, 12.99)	0.499		1.000
P Pulmonale		1.000		1.000		1.000		1.000
S1S2S3	1.00 (0.13, 7.56),	1.000	2.02 (0.10, 121.07),	1.000	0.50 (0.01, 9.67)	1.000		0.497
S1Q3T3	7.20 (0.91, 327.08),	0.067	5.23 (0.57, 252.76),	0.209		0.498		0.121

Table 4 Odds ratios for ECG as predictor of pulmonary embolism for all patients and subgroups by clot load and absence of cardiorespiratory disease.

ECG finding	All		Large clot load		Small clot load		No cardiorespiratory disease	
	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p	OR (95%CI)	p
Clockwise Rotation	1.39 (0.79, 2.46),	0.281	1.31 (0.52, 3.33),	0.672	1.44 (0.69, 3.08)	0.384	0.92 (0.39, 2.17),	1.000
Atrial Tachy-arrthythmias	0.77 (0.38, 1.53),	0.517	0.76 (0.23, 2.43),	0.792	0.78 (0.31, 1.88)	0.682	0.51 (0.15, 1.60),	0.307

Table shows odds ratio, 95% confidence interval and p-value from Exact Fisher Test comparing cases and controls. If the confidence interval does not have finite limits, only the p-value is displayed.

Discussion

We believe that ours is the first study of the ECG as predictor of diagnosis in PE to use age and sex matched controls and also to consider clot load. The ECG finding that best predicted PE was RV strain pattern. S1Q3T3 was uncommon in our study. More of our control patients had COPD, suggesting that PE is frequently suspected but not confirmed in patients with COPD who present with worsening respiratory symptoms.

S1Q3T3 and RV strain pattern, when present, are the two ECG changes most frequently associated with right ventricular dysfunction in PE. S1Q3T3, probably the most well known ECG finding, was first described in 1935[20]. The frequency with which it occurs varies from 3.7%[21] to 50%[10], the higher figure reflecting the high proportion of patients with massive PE (76%) in that study. The prevalence of S1Q3T3 in our patients was 4% which is at the lower end of this range. We recognise that the determination of S1Q3T3 pattern can sometimes be difficult, particularly in relation to the presence or absence of a small R wave in lead III and wonder if this might contribute at least in part to the variation in prevalence.

The literature on the role of the ECG in the diagnosis of PE includes some who believe the ECG is of limited value[22,23], and others who feel that ECG changes are a possible clue to a diagnosis that must then be confirmed by CTPA[21,24]. Rodger and colleagues examined 212 consecutive inpatients and outpatients with suspected PE. All had a VQ scan and anyone with an indeterminate scan had pulmonary angiography. PE was confirmed in 49 patients and excluded in 163. Only sinus tachycardia and incomplete RBBB predicted PE in this dataset. Patients and controls were not age or sex matched and no attempt was made to stratify by clot load or pre-existing cardiorespiratory disease. These authors concluded that the ECG was of limited value in the diagnosis of PE[22]. Chan et al, reviewing the literature, came to a similar conclusion, namely that the overall utility of the electrocardiogram is limited due to the variable presence, frequency, and transient nature of most of the ECG abnormalities associated with the disease[23].

By contrast, Marchick and colleagues were able to show positive likelihood ratios for S1Q3T3 pattern and precordial T wave inversion among 6049 emergency department patients suspected of having PE, in 354 of whom a diagnosis was subsequently confirmed. No attempt was made to age or sex match patients and controls or to stratify by clot load, though their results were not influenced one way or another by the presence of pre-existing

cardiorespiratory disease[24]. More recently Co et al used patients as their own controls by comparing ECG at diagnosis with a previous ECG in a cohort of 352 PE patients. Seventy six percent had a significant change in their ECG, most notably T wave inversions in just over one third of cases. These TWIs occurred in all leads but were most common inferiorly[21].

How may these apparently conflicting views be reconciled? The prevalence of RV strain varies from 7.3% (24) to 68% (10), reflecting the fact that most studies include a mixture of patients with large and small clot load. In our study 76/189 (40%) patients were judged to have large clot load, a similar proportion to that reported by Co et al[21]. We might expect the ECG to be less predictive for PE in studies with a higher proportion of small PEs. It is also likely that studies with smaller numbers of patients may not have the power to detect the findings of right ventricular dysfunction. Six of the 11 studies reviewed by Chan comprised 50 or fewer patients[23].

Strengths and limitations

Our study has strengths and limitations. Strengths are our comparison with age and sex matched controls, and stratification by clot load. The fact that more control patients had pre-existing cardiorespiratory disease may also be a strength as we were still able to show that RV strain predicted PE despite using controls likely to have RV strain for other reasons. We would have preferred to match cases and controls for presence or absence of cardiorespiratory disease but had too few control patients to enable us to do this. Our main limitation is that we could find no electronic record for 96 of our 313 CTPA positive patients. It is likely that this group includes more patients with massive and submassive PE who died early during the course of their admission as case sheets for such patients are often sent straight to file and may not always be scanned. We used an estimate of clot load rather than RV dilation as our index of PE severity. RV dilation and biochemical markers such as high sensitivity troponin and NT-Pro BNP could further refine the assessment of severity. We did not attempt to assess inter observer variability when analysing ECGs and CTPAs but eliminated interpretation bias by ensuring that our radiologist and cardiologist were unaware of each other's findings. As this was a case control study, we made no attempt to calculate positive and negative predictive values as these are prevalence dependent factors.

Conclusion

In conclusion, an ECG showing RV strain in a breathless patient with no previous history of cardiorespiratory disease is highly suggestive of PE with specificity 97.4% for large clot load. S1Q3T3 also has high specificity for PE but was a relatively uncommon finding in our study. Many of the other ECG changes that have been described in PE occur too infrequently to be of predictive value.

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Contributorship

CI, DT, GK, RT designed the study. PH and AM were responsible for scoring the CTPAs and ECGs respectively. C-MM undertook the statistical analyses. CI wrote the first draft and worked with DT and GK on the second draft. All authors contributed to the final draft.

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Ethical approval

Not required as no patient identifiable data, in keeping with Scottish Health Boards' policies.

Competing interests

All authors have completed the ICMJE uniform disclosure form at www.icmje.org/coi_disclosure.pdf and declare: no support from any organisation for the submitted work; no financial relationships with any organisations that might have an interest in the submitted work in the three previous years; no other relationships or activities that could appear to have influenced the submitted work

Transparency declaration

The lead author affirms that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned (and, if relevant, registered) have been explained.

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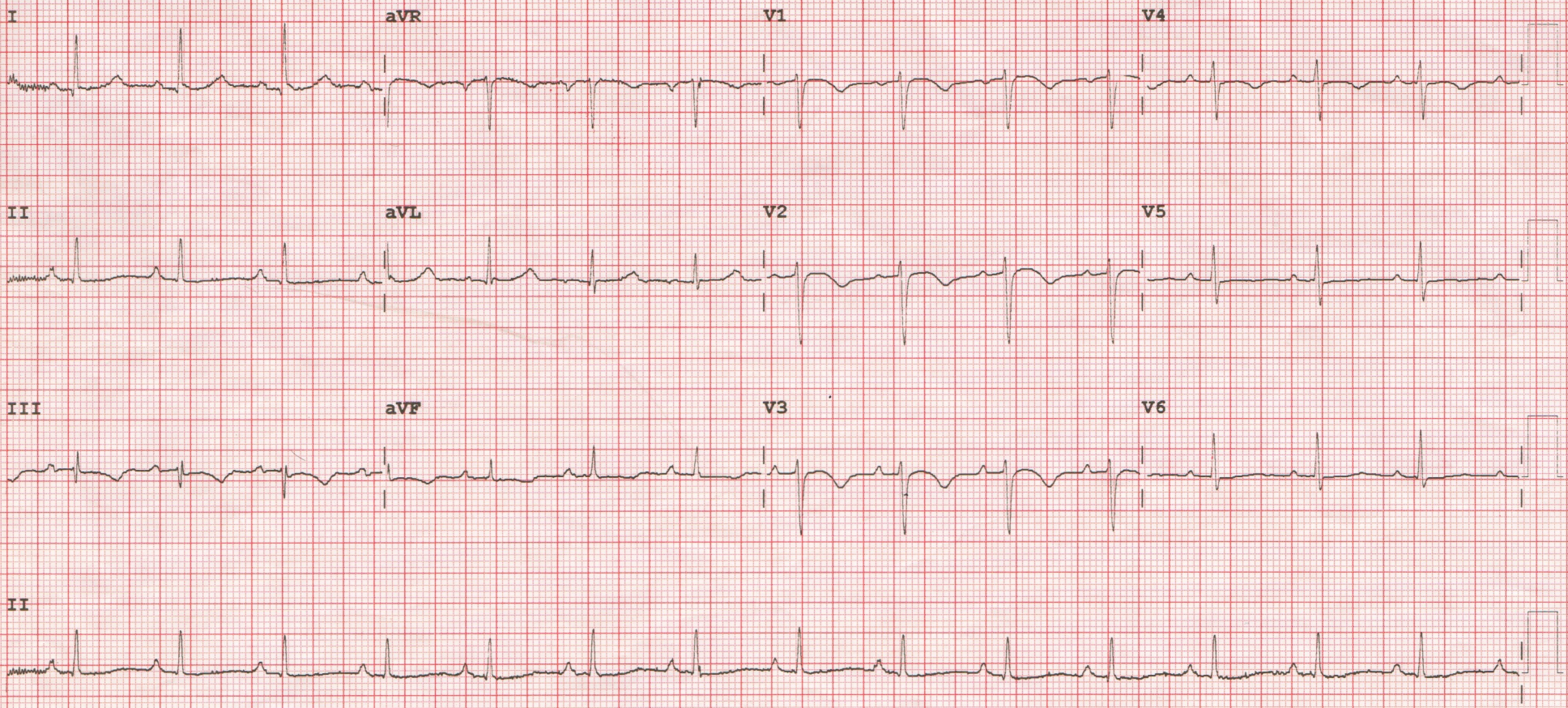
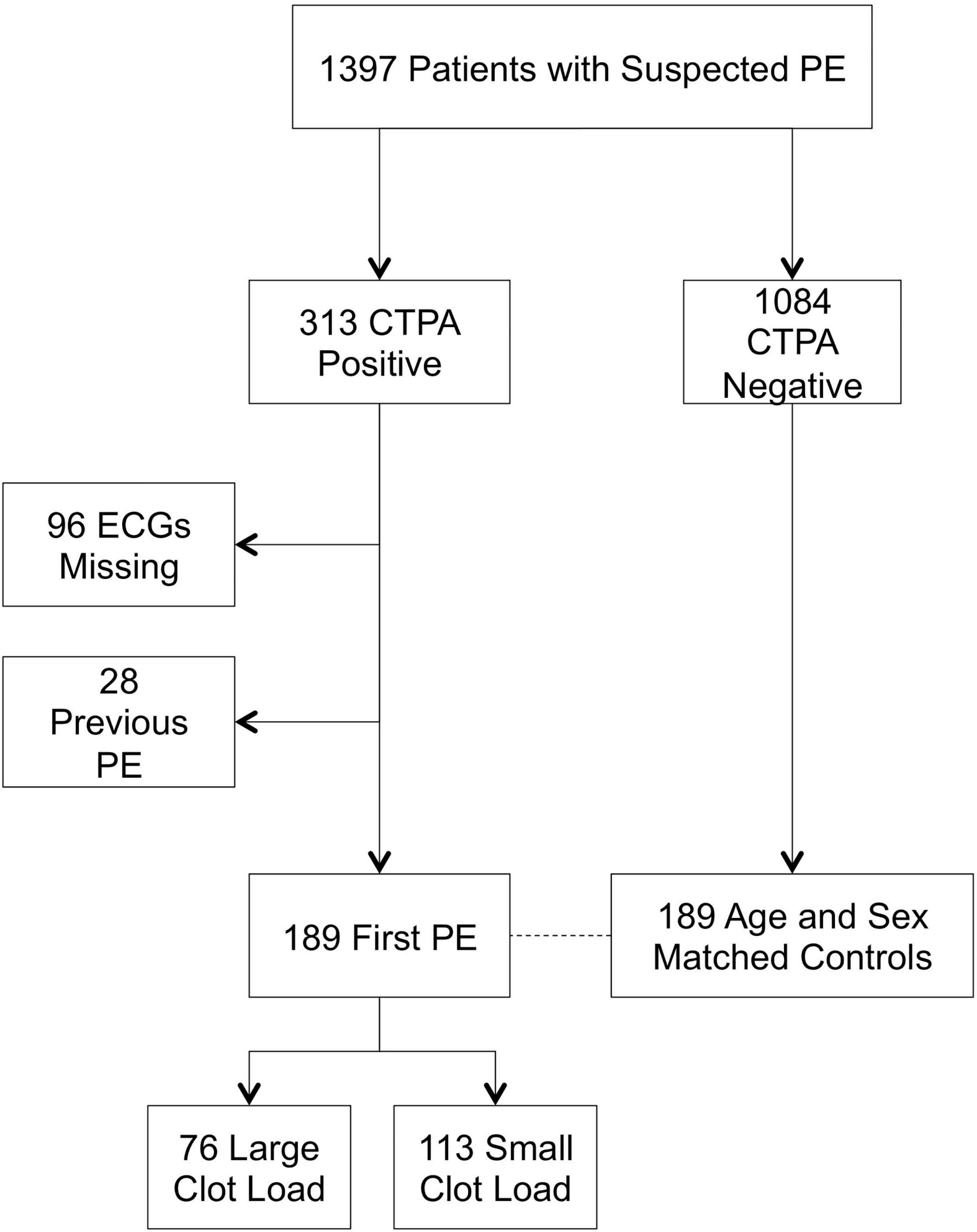
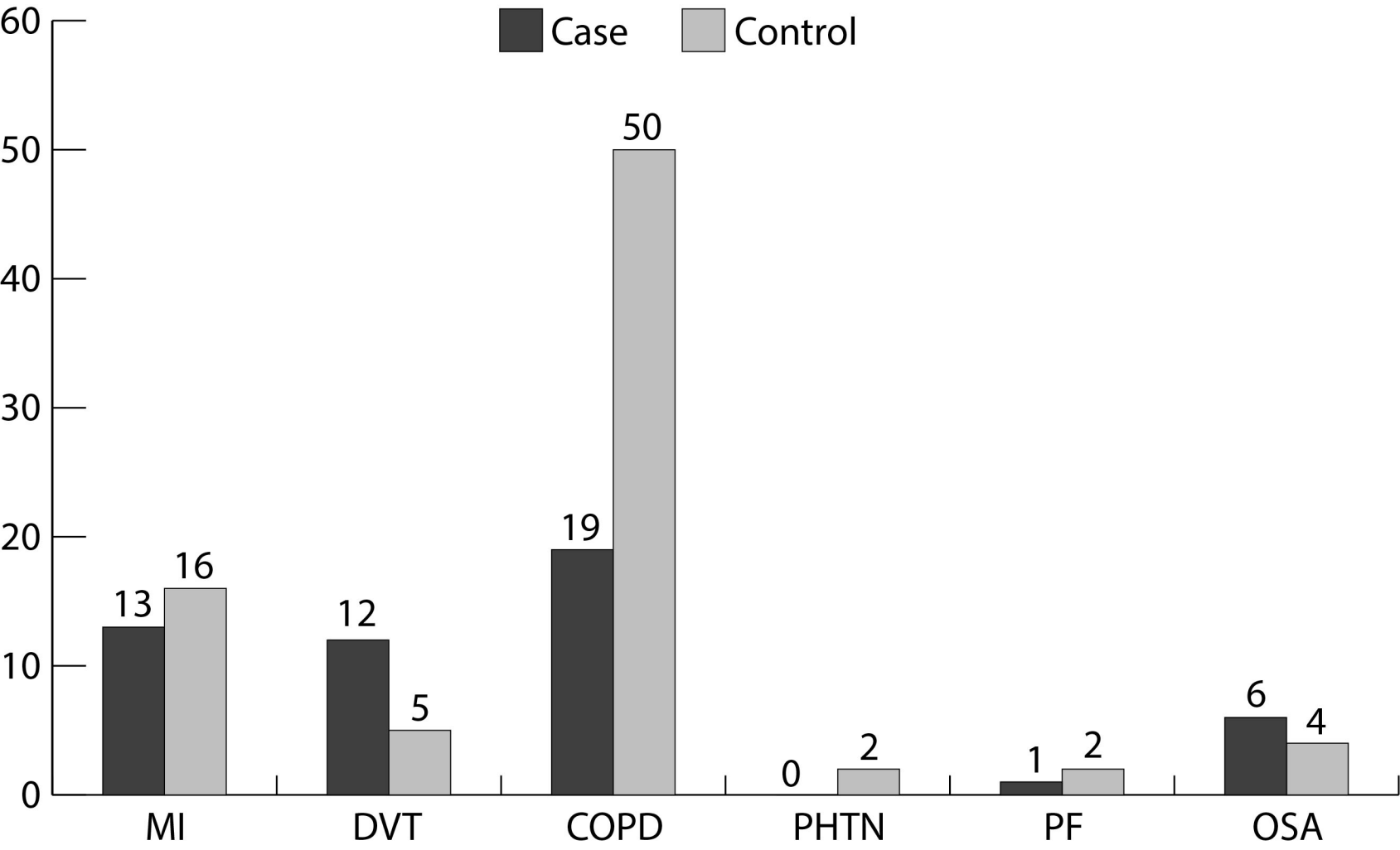

I
aVR
V1
V4
II
aVL
V2
V5
III
aVF
V3
V6
II

Figure 2.


1397 Patients with Suspected PE
313 CTPA Positive
1084 CTPA Negative
96 ECGs Missing
28 Previous PE
189 First PE
189 Age and Sex Matched Controls
76 Large Clot Load
113 Small Clot Load


Case
Control
60
50
40
30
20
10
0
13
16
12
5
19
50
0
2
1
2
6
4
MI
DVT
COPD
PHTN
PF
OSA