Research Square

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Evaluation of D-dimer Levels for the Differential Diagnosis of Acute Chest Pain in Hong Kong: A Single-Centre Retrospective Study D-dimer for Chest Pain Diagnoses

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Abstract

Background

D-dimer is elevated in acute aortic syndrome (AAS) and pulmonary embolism (PE), but local comparative data is limited. This study explores D-dimer's diagnostic value for AAS to inform acute chest pain management.

Methods

This retrospective single-centre study in Hong Kong evaluated adult emergency department (ED) patients presenting with chest pain between 2020 and 2025. Patients with D-dimer testing were grouped by diagnosis: AAS, PE, ischaemic heart disease (IHD), or non-specific chest pain (CP). Outcomes included D-dimer levels, diagnostic performance of D-dimer for AAS and PE, computed tomography (CT) utilization, 7- and 30-day mortality, hospital length of stay, and 28-day readmission rates.

Results

Among 1,019 included patients, median D-dimer levels were 329, 531, 3738, and 4693 ng/mL for CP, IHD, PE, and AAS, respectively. The AUROC for distinguishing AAS from non-AAS was 0.84 (95% CI: 0.79–0.89). D-dimer ≥ 2000 ng/mL was associated with significantly higher mortality. ED-based CT scans were performed in only 3 (27.3%) of AAS and 15 (6.3%) of PE patients.

Conclusions

This study reports the first local data characterising D-dimer levels across acute chest pain aetiologies, with the highest values observed in AAS and PE. The notably low CT rates in both the AAS and PE groups highlight an unanswered clinical need, and prospective multicentre studies integrating biomarkers with validated clinical decision tools are warranted to optimise risk stratification and CT utilisation for high-risk ED patients with chest pain.

Introduction

Chest pain is a common emergency department (ED) presentation, accounting for about 5% of ED attendances at our local hospital according to the hospital chest pain registry. Diagnoses range from benign musculoskeletal pain to life-threatening conditions such as acute aortic syndrome (AAS), pulmonary embolism (PE) or myocardial infarction (MI). Rapid differentiation between low- and high-risk patients is crucial yet challenging.

D-dimer is a fibrin degradation product found in blood following fibrinolysis, and it serves as a biomarker for thrombotic activity. Clinically, D-dimer testing is widely utilized in the evaluation of suspected deep vein thrombosis and other coagulation-related disorders[1]. However, because of the non-specific nature of this biomarker, the use of D-dimer outside of suspected deep vein thrombosis (DVT) varies considerably depending on institutional protocols. In Hong Kong, D-dimer is not routinely performed for patients with chest pain symptoms in most EDs. It is usually performed only when the treating physician suspects DVT or PE and the turnaround time is a few hours. A positive D-dimer results (i.e. >500ng/mL) does not always trigger a computed tomography (CT) scan due to resource constraints, the lack of a standardized protocol, and the local perception that DVT and PE are less common in Chinese patients compared to the Western hemisphere.

Elevated D-dimer levels are typical in AAS and PE compared to ischaemic heart disease (IHD), providing some degree of diagnostic and risk stratification value[2]. Comparative studies have quantified this difference. In the IRAD-Bio sub-analysis, median D-dimer levels in AAS were approximately 11,200 ng/mL, whereas patients with IHD had median values of only 1,110 ng/mL[3]. Similarly, Shimoney et al. reported median D-dimer levels of 3,200 ng/mL in patients with PE compared to 600 ng/mL in those with IHD[4]. Recent studies have shown that a negative D-dimer can safely exclude AAS in low-risk patients; however, these studies were conducted mainly in Western populations, and the evidence for using D-dimer in Chinese populations is limited in this scenario[5–9].

Beyond diagnosis, elevated D-dimer levels have also been linked to prognosis. In PE, higher D-dimer levels correlate with clot burden and adverse outcomes, with studies showing associations between elevated levels and increased mortality[10]. In AAS, high D-dimer values have similarly been associated with worse in-hospital outcomes[11]. A large study showed that baseline D-dimer predicts long-term cardiovascular and all-cause mortality[12]. Existing literature demonstrates heterogeneity in the assays used as well as the thresholds applied for D-dimer interpretation, which complicates the comparison of findings across different studies and populations. However, data on undifferentiated chest pain cohorts are limited, particularly from Chinese populations where the incidence of AAS and PE may differ.

Given these variations, we wanted to better understand the characteristics of ED chest pain patients who underwent D-dimer testing, especially in populations where standardized protocols are lacking. This study aims to retrospectively compare D-dimer levels across chest pain cohorts in a predominantly Asian Chinese population, aiming to provide exploratory data on the D-dimer test's diagnostic value for AAS and to inform future implementation of D-dimer testing in acute chest pain management.

Methods

Study design and setting

This was a single-centre retrospective observational study at Prince of Wales Hospital (PWH), a university teaching hospital in Hong Kong. Ethics approval was obtained from the Joint Chinese

University of Hong Kong-New Territories East Cluster Clinical Research Ethics Committee prior to data collection (CREC Ref. No. 2026.127) and this study was reported in accordance with the STROBE Statement for reporting observational studies (*checklist is in supplementary material*) [13]. Clinical trial number: not applicable. The primary outcome was the D-dimer level of different chest pain-related diagnoses. Secondary outcomes included diagnostic performance of D-dimer on AAS, hospital length of stay, computed tomography scans performed, 7- and 30-day mortality, and readmission rates within 28 days of discharge. We extracted a screening list of patients who underwent D-dimer testing and received a chest pain-related diagnosis during the study period (1 January 2020 to 31 December 2025) from Hong Kong's Clinical Data Analysis and Reporting System (CDARS).

Since the system does not allow users to filter patients by their ED presenting symptoms recorded on the ED triage note, patient identification and classification was based on the first five diagnosis codes of the index visit. The following ICD-9 codes were used: chest pain (786.5), IHD (410–414), PE (415.1), and AAS (441). Exclusion criteria were patients with a D-dimer > 500ng/mL and any of the following: (1)patients with untraceable medical records; OR (2)D-dimer measured during or after cardiopulmonary resuscitation or after primary coronary intervention or surgical intervention; OR (3)patients with known active malignancy; (4)trauma patients; OR (5)patients with active bacterial or viral infection, pneumonia or sepsis recorded in diagnosis; OR (6)patients with end stage renal failure; OR (7)patients in shock defined as systolic blood pressure < 90mmHg; OR (8)patients with aortic aneurysm but not acute aortic syndrome.

D-dimer values were recorded from the first result obtained after ED admission. D-dimer levels were measured by the hospital laboratory using an immunoturbidimetric assay.

Statistical analysis

Continuous variables were presented as median with IQR or mean ± SD, as appropriate. Categorical variables were presented as frequencies and percentages. Baseline characteristics were compared between diagnosis groups using χ^2 or Fisher's exact test for categorical variables and Kruskal-Wallis with Dunn's multiple comparisons test for continuous variables. ROC analysis was performed to evaluate D-dimer's sensitivity and specificity for distinguishing AAS from other diagnosis groups, and PE from other diagnosis groups.

Given the low AAS prevalence (n = 13) yielding an imbalanced dataset, percentile bootstrapping (1000 resamples with replacement) was implemented to derive nonparametric 95% CIs (2.5th–97.5th percentiles of the bootstrap distribution) for key diagnostic metrics. Kaplan-Meier curves estimated survival probabilities across D-dimer strata (< 500, 500–1999 and ≥ 2000 ng/mL), compared by log-rank (Mantel-Cox) test. Spearman's r and simple linear regression examined D-dimer and LOS associations.

Subgroup analyses of D-dimer levels according to the time interval from ED admission to laboratory testing (< 24 hours, 24–48 hours, > 48 hours) were performed. Post hoc pairwise comparisons were performed using Tukey's test for all pairwise group comparisons. All hypothesis testing was 2-sided and

p value < 0.05 were considered statistically significant. Participants with missing data for the variables included in each analysis were excluded. Analyses were performed using GraphPad Prism 11.0.0 for macOS and RStudio 4.5.2 for macOS.

Results

A total of 1,232 emergency admissions with D-dimer testing were included from 1 January 2020 to 31 December 2025, of which 213 patients were excluded according to the exclusion criteria, leaving 1,019 patients for final analysis (Fig. 1). 13 were AAS (6 type A aortic dissection; 5 type B aortic dissection; 2 type B intramural hematoma), 177 were IHD, 281 were PE, and 548 were non-specific chest pain (CP) (i.e. patients with ICD-9 code of 786.5). Overall, 520 patients (51%) were male with a mean age of 63(± 17). Mean D-dimer level in the dataset (n = 1,019) was 1,840.1 ± 2,836.1 ng/mL in which 532 patients (52.2%) had a 'positive' D-dimer result defined as > 500ng/mL. Patient characteristics, clinical outcomes, and D-dimer levels were presented in Table 1. The proportion of males was significantly higher in the AAS and IHD groups than in the PE and CP groups.

Table 1
Patient characteristics (n = 1019)

Male sex (n, %)	**All (n = 1019)**	**AAS (n = 13)**	**IHD (n = 177)**	**PE (n = 281)**	**CP (n = 548)**
	520 (51.0%)	**9 (69.2%)**	**112 (63.3%)**	**118 (42.0%)**	**281 (51.3%)**
Age (Mean, SD)	63 (17)	64 (11)	70 (13)	68 (16)	59 (17)
Triage category (n, %)					
1 (Critical)	32 (3.1%)	0 (0%)	13 (7.3%)	13 (4.6%)	6 (1.1%)
2 (Emergency)	102 (10.0%)	3 (23.1%)	12 (6.8%)	53 (18.9%)	34 (6.2%)
3 (Urgent)	587 (57.6%)	5 (38.5%)	111 (62.7%)	156 (55.5%)	315 (57.5%)
4 (Semi-urgent)	298 (29.2%)	5 (38.5%)	41 (23.2%)	59 (21.0%)	193 (35.2%)
5 (Non-urgent)	0 (0%)	0 (0%)	0 (0%)	0 (0%)	0 (0%)
Investigations					
D-dimer (Median with bootstrap 95% CI)	541 (484–597)	4693 (2671–5805)	531 (454–617)	3738 (3183–4967)	329 (308–356)
D-dimer > 500ng/mL (n, %)	532 (52.2%)	13 (100%)	91 (51.4%)	269 (95.7%)	159 (29.0%)
D-dimer > upper limit of normal age-adjusted cutoff (n, %)	358 (35.1%)	12 (92.3%)	64 (36.2%)	260 (92.5%)	103 (18.8%)

** 1 patient had CT done at private hospital, 1 patient did not undergo CT during the index visit as a recent scan had already been performed*

AAS, acute aortic syndrome; IHD, ischaemic heart disease; PE, pulmonary embolism; CP, non-specific chest pain; ED; emergency department, CT; computed tomography

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	520 (51.0%)	**9 (69.2%)**	**112 (63.3%)**	**118 (42.0%)**	**281 (51.3%)**
CT thorax with contrast performed (n, %)	417 (40.9%)	11* (84.6%)	44 (24.9%)	240 (85.4%)	122 (22.3%)
Performed in ED (n, %)	33 (7.9%)	3 (27.3%)	2 (4.5%)	15 (6.3%)	13 (10.7%)
Clinical outcomes					
7-day mortality (n, %)	23 (2.3%)	0 (0%)	8 (4.5%)	15 (5.4%)	0 (0%)
30-day mortality (n, %)	70 (6.9%)	0 (0%)	34 (19.2%)	36 (15.8%)	0 (0%)
Days of hospital length of stay (Median, IQR)	4 (2–8)	13 (6–19)	7 (4–11)	8 (5-13.5)	2 (1–4)
Readmission within 28 days after discharge (n, %)	107 (10.5%)	2 (15.4%)	13 (7.3%)	41 (14.6%)	51 (9.3%)

** 1 patient had CT done at private hospital, 1 patient did not undergo CT during the index visit as a recent scan had already been performed*

AAS, acute aortic syndrome; IHD, ischaemic heart disease; PE, pulmonary embolism; CP, non-specific chest pain; ED; emergency department, CT; computed tomography

The median (bootstrap 95% CI) D-dimer levels were 329 (308–356) ng/mL for CP, 531 (454–617) ng/mL for IHD, 3,738 (3,183–4,967) ng/mL for PE and 4,693 (2,671–5,805) ng/mL for AAS, respectively. The Kruskal–Wallis test showed significant differences in D-dimer levels across groups ($p < 0.01$), and Dunn's multiple comparisons test confirmed significant differences between all group pairs except PE versus AAS ($p > 0.99$) (Fig. 2, *analytical results are reported in the supplementary material, Table S1*).

The area under the ROC curve for differentiating AAS from other chest pain diagnoses (i.e. IHD, PE and CP) was 0.84 (95% CI: 0.79–0.89), with an optimal cutoff at 1,787 ng/mL, yielding a sensitivity of 92.3% (95% CI: 66.7–99.6%) and a specificity of 78.0% (95% CI: 75.4–80.5%) (Fig. 3). Other performance metrics are summarised in *supplementary material, Table S2*.

The ROC analysis for distinguishing PE from other chest pain diagnoses (i.e. AAS, IHD, and CP) yielded an AUC of 0.93 (95% CI: 0.91–0.95). At an optimal cutoff of 987 ng/mL, the model achieved a sensitivity

of 89% (95% CI: 84.8–92.1%) and a specificity of 85.2% (95% CI: 82.5–87.6%) (Fig. 3). Additional performance metrics are presented in *supplementary material, Table S3*.

Simple linear regression analysis indicated a weak association, with every 702 ng/mL increase in D-dimer level corresponding to a 1 day increase in LOS.

257 patients (25.2%) had a D-dimer test done in the ED while 762 patients (74.8%) had it done after admission to an inpatient ward. Among the 834 patients with triage time data available, the majority had a D-dimer test performed within 6 hours from triage (n = 251, 24.6%), with 198 of these tests conducted in the ED and the remaining tests on the ward (*Details in supplementary material, Figure S1*). A secondary peak was observed in patients tested at ≥ 48 hours (n = 137, 16.3%). The median time from triage to D-dimer testing was 14.1 hours (IQR: 4.2–36.2 hours).

Subgroup analysis of time from ED triage to D-dimer result time was performed. AAS and PE showed higher D-dimer levels than IHD and CP (*Details in supplementary material, Table S4*). No significant between-strata differences in D-dimer levels were observed for AAS, IHD, or CP; however, PE showed a significant difference between the < 24-hour and 24–48-hour strata ($p = 0.003$).

Survival analysis revealed significant differences in survival by D-dimer level. Kaplan-Meier curves demonstrated the highest mortality in the group with D-dimer ≥ 2,000 ng/mL. The Mantel-Cox log-rank test with Bonferroni's post-hoc test confirmed significant differences between D-dimer ≥ 2,000 ng/mL and D-dimer < 500 ng/mL and 500-1,999 ng/mL groups (Fig. 4).

Clinical outcomes varied significantly according to D-dimer stratification. Notably, patients with elevated D-dimer levels showed higher 7- and 30-day mortality rates alongside prolonged lengths of hospital stay (*supplementary material, Table S5*). Patients with D-dimer levels of 500–1,999 ng/mL had the highest 28-day readmission rate after discharge, whereas those with D-dimer levels ≥ 2,000 ng/mL had the lowest ($p = 0.01$) (*supplementary material, Figure S2*).

In terms of CT scans, 417 patients (40.9%) underwent CT thorax, with 33 (7.9%) performed in the ED. Of these 33 patients who underwent CT scanning in the ED, 3 (27.3%) had AAS, 2 (4.5%) had IHD, 15 (6.3%) had PE, and 13 (10.7%) had CP.

Discussion

Our study demonstrates that D-dimer levels vary significantly across different chest pain diagnoses, with markedly higher concentrations observed in the AAS and PE groups compared with the IHD and CP groups. Elevated D-dimer levels were also associated with prolonged hospital stay and increased mortality.

Consistent with previous studies, D-dimer levels demonstrated substantial differences between different chest pain diagnoses [14, 15]. PE and AAS both trigger massive vascular disruption with systemic fibrinolysis, yielding markedly elevated D-dimer levels due to extensive thrombus formation and

degradation. The observed overlap between PE and AAS reflects shared pathophysiology, where acute endothelial injury activates coagulation cascades, producing cross-linked fibrin that plasmin degrades into D-dimer[16]. For AAS discrimination, D-dimer alone achieved an AUC of 0.83, indicating fair diagnostic performance. A D-dimer cutoff at 1,500 ng/mL had a sensitivity of 92.3% and a specificity of 74.5%, which is comparable to Xie's 2021 study (sensitivity 92.5% and specificity 83%)[17]. Prior studies have shown that the combination of D-dimer with structured clinical risk scores, such as the Aortic Dissection Detection Risk Score, improves diagnostic performance and enhances safety in AAS rule-out algorithms[6, 7]. This diagnostic algorithm is recommended in the 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases[18]. In this context, D-dimer serves as an adjunct to clinical assessment, with robust prospective studies needed to validate the algorithm's diagnostic performance in the Chinese population. D-dimer also showed promising diagnostic ability for differentiating PE from other chest pain diagnoses, yielding an AUC of 0.93.

Beyond diagnosis, our results suggest that D-dimer may also serve a prognostic role in ED patients with chest pain. In the survival analysis, patients with D-dimer ≥ 2,000 ng/mL experienced the steepest decline in survival, indicating that markedly elevated levels identify a high-risk subgroup warranting immediate imaging and intervention. This supports D-dimer's potential utility not only for diagnostic triage but also for early risk stratification in selected chest pain cohorts.

Our study highlights an underutilisation of D-dimer in Hong Kong EDs. Although D-dimer is widely available, it is not routinely incorporated into chest pain evaluation protocols, likely due to concerns about its poor specificity, relatively long turnaround time of several hours and the absence of consensus on how to act on D-dimer results, particularly in settings where access to ED CT is limited. More local data are therefore needed to support the routine implementation of D-dimer testing and to optimize its integration into Hong Kong chest pain pathways, thereby enhancing diagnostic sensitivity for AAS and PE in EDs.

Furthermore, our findings reveal a critical gap in the timely delivery of definitive imaging for patients with AAS and PE in the ED, where only 27.3% and 6.3% of those who ultimately required CT underwent scanning during their ED stay. In AAS, where CT serves as the gold standard for confirmation, the low ED CT rate indicates potential delays due to resource limitations and the absence of standardized pathways. Similarly, the low ED CT rate for PE indicates underutilization in potentially high-risk cases. Earlier ED CT for PE patients could allow earlier diagnosis and risk stratification and potentially allow outpatient management of selected, clinically stable, low-severity patients [19]. In contrast, 10.7% of non-specific CP patients underwent urgent CT scan in the ED, underscoring the need for integrated diagnostic pathways that combine risk scores with biomarkers to more objectively stratify chest pain patients, prioritize CT access, and ultimately reduce diagnostic delays while improving outcomes.

Limitations

This single-centre retrospective study included only a small number of patients with AAS, which may limit the generalisability of the findings and the statistical power of subgroup analyses, while resulting in wide confidence intervals around the sensitivity and specificity estimates. Secondly, we only included patients with chest pain-related diagnoses rather than all patients presenting with chest pain which might have introduced selection bias toward more severe cases. Thirdly, we excluded patients with certain confounding conditions, which may have further introduced selection bias and potentially overestimated the D-dimer's performance by evaluating it in a cleaner cohort than is typical of undifferentiated chest pain presentations.

Conclusions

This study provides the first Hong Kong data on D-dimer levels across acute chest pain diagnoses, demonstrating significant differences between major diagnostic groups. Elevated D-dimer are associated with AAS and PE, and also predicted longer hospital stays and poorer survival. These findings support D-dimer's value in stratifying chest pain patients by risk level and underscore the need for prospective multicentre validation studies in undifferentiated cohorts alongside the use of clinical decision tools to safely differentiate AAS and PE from other chest pain aetiologies.

Abbreviations

AAS
acute aortic syndrome
CT
computed tomography
CP
non-specific chest pain
DVT
deep vein thrombosis
ED
emergency department
IHD
ischemic heart disease
LOS
length of stay
MI
myocardial infarction
PE
pulmonary embolism

Declarations

Consent to Participate declaration

The requirement for informed consent was waived by the local Institutional Review Board because of the retrospective nature of the study._

Funding

No authors received any funding.

Conflicts of Interest

The authors declare that they have no conflict of interest._

Data Availability

The data underlying this article will be shared on reasonable request to the corresponding author.

Author Contribution

R.L. conceived the study idea and developed the initial research concept and objectives. She led the design of data-collection instruments, coordinated and performed participant recruitment and data collection, and oversaw data management. Rebecca conducted the primary statistical analyses, generated all figures and tables, and prepared the visualisation elements for the manuscript. She drafted substantial portions of the Methods and Results sections and contributed to iterative revisions of the full manuscript.K.L. contributed to the statistical analysis plan and performed secondary and sensitivity analyses to validate the primary findings. She interpreted the analytical output in the context of existing literature and clinical relevance. Karuna drafted the first complete version of the manuscript, integrating Methods, Results, and Discussion sections, and incorporated feedback from co-authors during early revisions.G.C. played a key role in structuring and writing the first full draft of the manuscript, particularly refining the Introduction and Discussion for clarity and scholarly tone. He contributed to subsequent rounds of revision, responding to reviewer comments, updating references, and ensuring consistency across sections in later manuscript versions.L.L. assisted with data cleaning, variable derivation, and execution of predefined statistical analyses. She contributed to interpreting key findings and their clinical implications. Ling Yan Leung participated in drafting the initial manuscript, particularly the Methods and Results sections, and reviewed and approved the final version for submission.K.H. contributed to drafting the first complete manuscript, with emphasis on contextualising findings within current emergency medicine and diagnostic literature. He actively participated in later revisions, addressing peer-review comments, refining arguments in the Discussion, and ensuring alignment with journal reporting guidelines.R.C. and S.M. provided senior clinical and methodological expertise throughout the study. They reviewed the study design, interpretation of results, and clinical implications, and critically appraised the manuscript for scientific rigour and relevance. Both authors endorsed the final version of the manuscript and approved it for publication.C.G. originated and conceptualised the research question

and overall study framework. He provided continuous supervision of the research process, including oversight of data collection, analysis strategy, and manuscript development. Colin critically revised multiple drafts of the manuscript, guided responses to reviewer feedback, and gave final approval of the version to be published.

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Figures

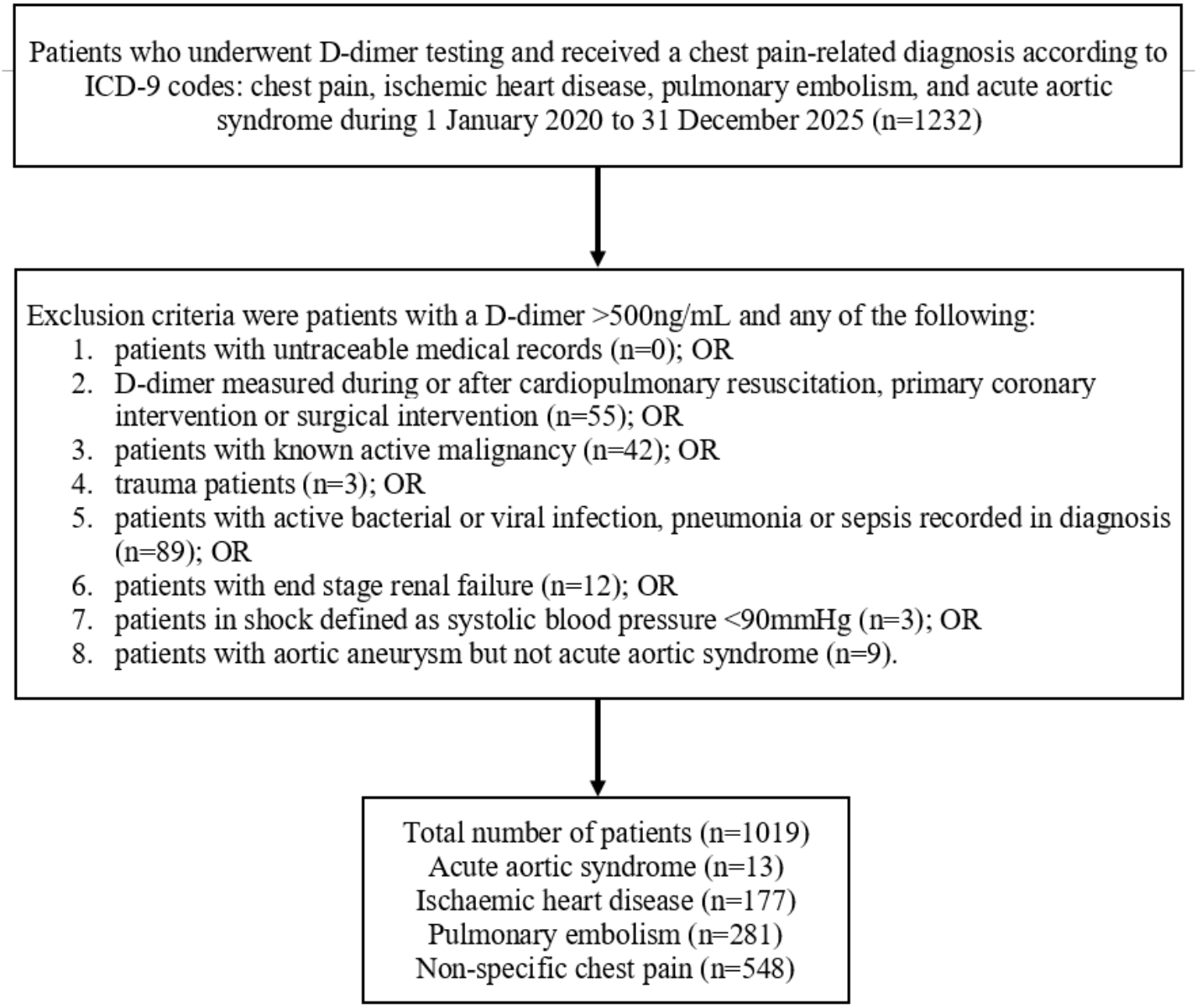


Figure 1

Study flowchart

ICD-9 codes: chest pain, 786.5; ischaemic heart disease, 410-414; pulmonary embolism, 415.1; acute aortic syndrome, 441

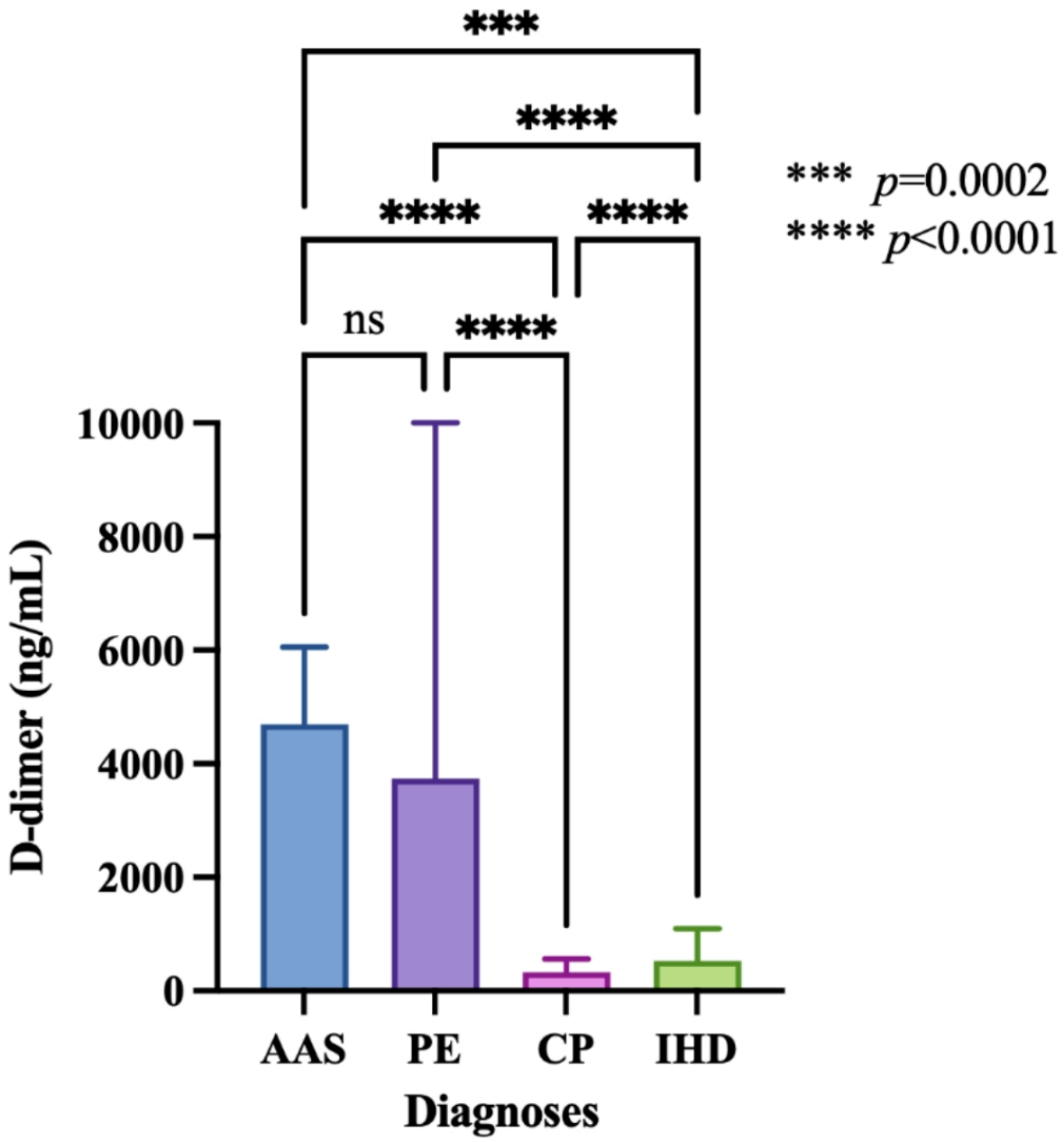


Figure 2

Median and IQR of D-dimer levels across different diagnoses. *AAS, acute aortic syndrome; PE, pulmonary embolism; CP, non-specific chest pain; IHD, ischemic heart disease; ns, non-significant*

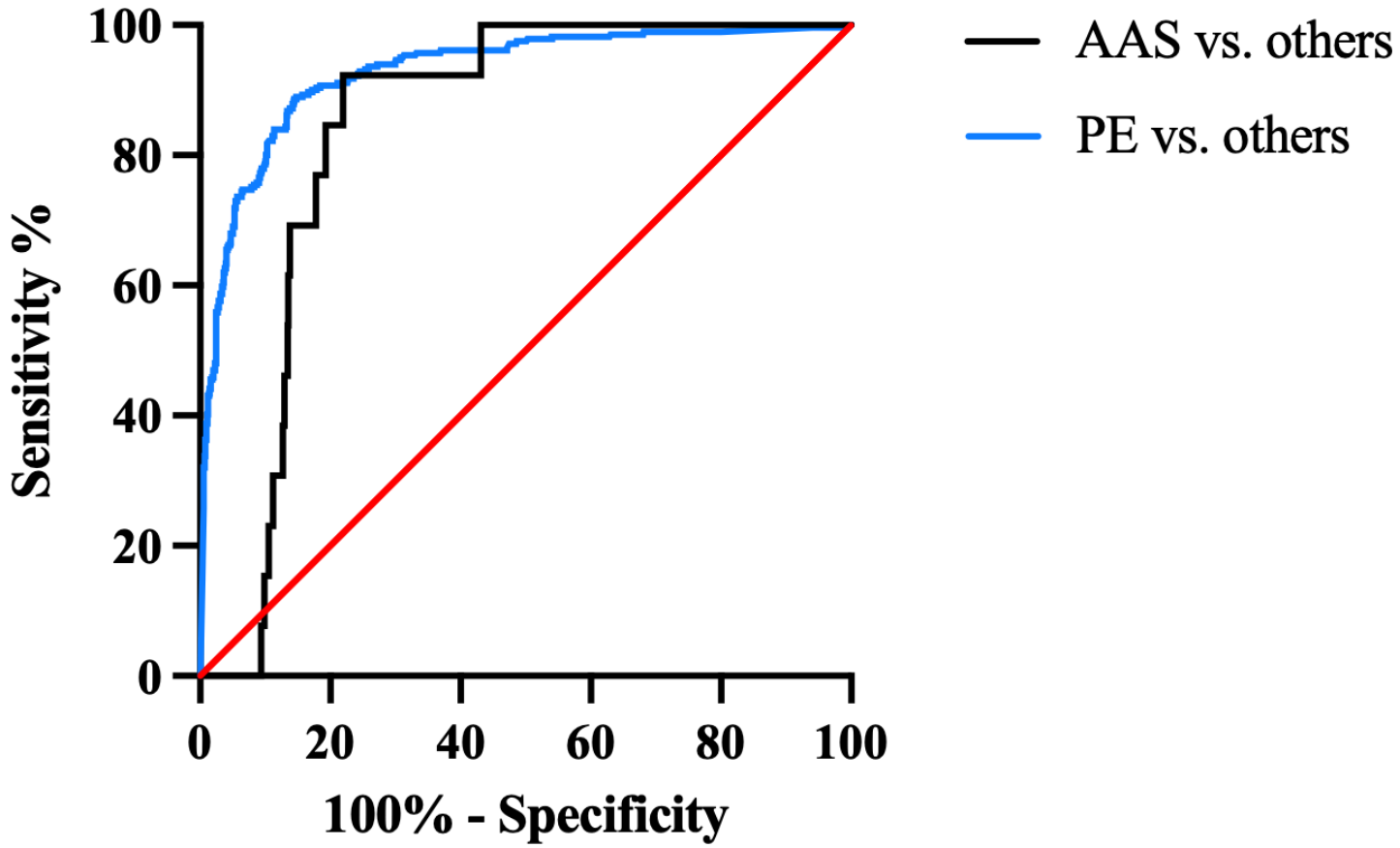


Figure 3

Receiver operating characteristic curve of D-dimer for distinguishing AAS from non-AAS, PE from non-PE. *AUROC, area under the receiver operating characteristic; AAS, acute aortic syndrome; PE, pulmonary embolism*

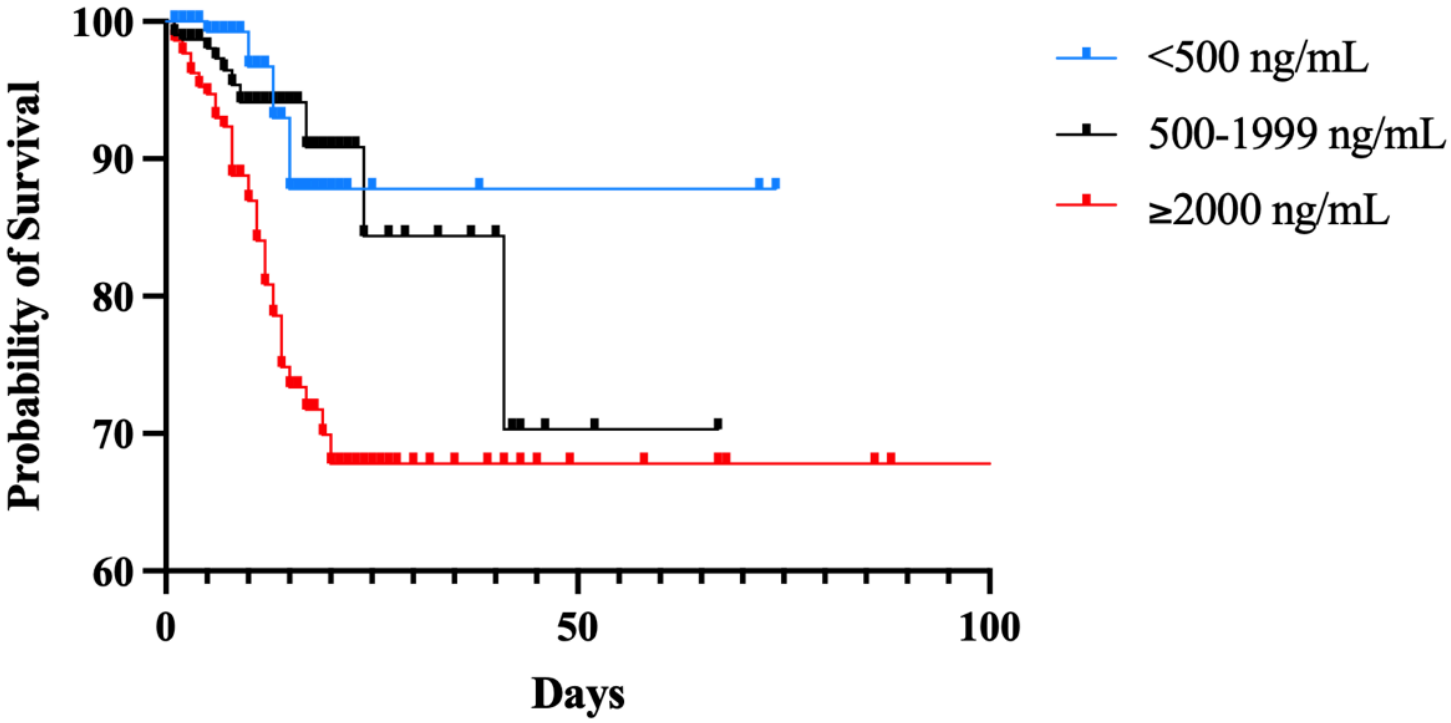


Figure 4

Survival curves by D-dimer level

Supplementary Files

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- Supplementarymaterial20260924.docx