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Original Research Article

Prediction of in-hospital atrial fibrillation after acute myocardial infarction: a cross-cohort study in Italian and Finnish patients

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ABSTRACT

Background: The development of atrial fibrillation (AF) during hospitalization for acute myocardial infarction (AMI) is common and associated with worse prognosis. We aimed to train and, for the first time, cross-nationally validate a machine learning model to predict AF developing during the cardiac intensive care unit (CICU) stay in post-AMI patients admitted in sinus rhythm, using only routine, non-electrocardiographic clinical parameters available at CICU admission.

Methods: We developed and tested classification pipelines on two independent cohorts of post-AMI patients from Italy (ITA, Sinus Rhythm = 2,200, AF = 240) and Finland (FIN, Sinus Rhythm = 3,631, AF = 452). Models were first evaluated via nested cross-validation on held out samples of the training cohort (internal testing) and then cross-cohort (external testing).

Results: An L2-regularized logistic regression model performed optimally in each case, achieving comparable performances in internal (AUROC: 0.740 ITA, 0.755 FIN) and external (AUROC: 0.735 ITA-on-FIN, 0.723 FIN-on-ITA) testing. A combined-cohort model achieved an AUROC of 0.749. *A posteriori* feature relevance analysis confirmed the predictive value of established risk factors including advanced age, lower left ventricular ejection fraction and hypertension in both cohorts. Ongoing aspirin therapy and non-ST-elevation myocardial infarction emerged as additional protective factors exclusive to cohort ITA, whereas a higher high-sensitivity C-reactive protein (risk factor) and the current smoking status (protective factor) were specific to cohort FIN.

Conclusions: Our study demonstrates that machine learning models using routinely collected clinical parameters could help predict in-hospital AF in post-AMI patients across different populations and national healthcare systems. Notably, a simple and fully interpretable logistic regression model achieved robust performance in the first cross-national validation for this prediction task, suggesting that more complex, black-box approaches may offer limited additional benefit for this specific task. Further improvements are needed to reach real-world application in clinical contexts for early risk stratification.

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1. Introduction

Acute myocardial infarction (AMI) is the leading cause of cardiovascular death worldwide [1]. Atrial fibrillation (AF) is a frequent complication during the acute phase, with incidences ranging between 2% and 22% [2]. The development of AF post-AMI has been associated with increased short- and long-term mortality [2–5]. Specifically, new-onset AF (NOAF), occurring in patients without a prior history of the arrhythmia, carries significant prognostic weight, linked to increased risks of stroke, heart failure, recurrent AF, and death, even when the episode is transient [2–9].

The pathophysiology of AF in this setting is multifactorial, involving acute triggers such as ischemia, inflammation, autonomic imbalance and hemodynamic changes [10–14]. Consistent findings have identified key clinical risk factors including advanced age, reduced left ventricular ejection fraction (LVEF), signs of heart failure, elevated inflammatory markers and hypertension [2,13,15–18].

Identifying high-risk patients at cardiac intensive care unit (CICU) would allow for tailored monitoring strategies and preventive interventions. However, predicting AF within the high-risk environment of a CICU following an AMI presents distinct challenges, and has been the focus of a limited body of research [19,20]. Moreover, the existing body of work shares several limitations relative to the present aim. Almost all prior models target strictly new-onset (first-ever) AF [4,5,13,15–18,21–28], rely solely on traditional statistical approaches, not taking advantage of the potential of modern machine learning methods; they have seldom addressed non-Asian cohorts [4,13,17]. Many also incorporate predictors that are not reliably available at the moment of CICU admission – whether ECG-derived features such as fragmented QRS or P-wave indices [22,24,27], or variables contingent on angiography and revascularization [25,28]. Finally, model evaluation has almost always been confined to a single cohort, with external validation a rare exception [28] and, to our knowledge, never spanning different countries [29]. We aimed to address these gaps by developing a machine learning pipeline for in-hospital AF after AMI that relies solely on routine, non-electrocardiographic clinical parameters available at CICU admission, and – for the first time – validating it across cohorts from two countries, Italy and Finland.

2. Methods

2.1. Study population and data collection

Two datasets of retrospective data were compiled from Italy and Finland, respectively. The Italian cohort (ITA) included 2,440 consecutive patients admitted to the CICU of Centro Cardiologico Monzino between 2010 and 2018 following an AMI, with CICU admission occurring within 24 h of the event. Among these patients, 240 (9.9%) developed AF before CICU discharge.

The Finnish cohort (FIN) comprised 4,083 consecutive patients admitted to the CICU of Tampere University Hospital Tays from 2007 to 2018, with 452 (11.1%) who developed AF during the CICU stay.

For both cohorts, patients were monitored with continuous ECG recordings during their CICU stay. AF diagnosis was based on the inspection of raw ECG signals from continuous ECG recordings and/or 12-lead resting ECG (10 s) recorded during CICU stay. All AF diagnoses were verified by an experienced cardiologist. Patients with AF already present at CICU admission, or with a history of persistent or permanent AF, were excluded from the study; a history of paroxysmal AF was instead allowed, as these patients present to the CICU in sinus rhythm and require the same monitoring and risk stratification as those without prior AF history. Throughout this work, the target outcome is consequently defined as AF developing during the CICU stay in patients admitted in sinus rhythm, without AF at admission and without a history of persistent or permanent AF, rather than strictly new-onset AF in the lifetime sense.

The prediction task was framed at a single, unambiguous timepoint – the moment of CICU admission – and the model inputs were accordingly restricted to the routine demographic, clinical-history, ongoing-treatment and first-line laboratory parameters available at that timepoint (Table 1). Variables derived from coronary angiography and revascularization, namely the treatment type (medical therapy, percutaneous coronary intervention or coronary artery bypass grafting), the number of treated vessels and the culprit vessel, were deliberately excluded from the models, as their availability at CICU admission is not guaranteed. They are nonetheless retained as descriptive baseline characteristics in Table 1.

Prior to analysis, all variables were harmonized to common measurement units across the two cohorts. The estimated glomerular filtration rate was computed with the CKD-EPI equation and the left ventricular ejection fraction was assessed by echocardiography in both centers, while high-sensitivity C-reactive protein and serum creatinine were measured with standard clinical laboratory assays; all laboratory values correspond to the first blood sample drawn at CICU admission. The proportion of missing values for each variable, stratified by cohort and outcome group, is reported in Supplementary Table 1.

2.2. Models evaluation

With two available cohorts, our model evaluation setup included different scenarios. Every model trained and optimized on cohort ITA was first tested on cohort ITA itself (internal testing), and then on cohort FIN (external testing). *Vice versa*, those trained and optimized on cohort FIN were initially tested on cohort FIN, and then on cohort ITA. Finally, any model trained on the combined cohorts (ITA + FIN) was only evaluated on that combined cohort itself. In each case, the internal testing step, i.e., a test on unseen data coming from the same cohort, was carried out through nested cross-validation (nested CV) with 10 inner and 10 outer folds, rather than by setting aside a fixed percentage of the dataset as test set, to avoid test set selection bias and yield a predicted label for each sample of the train cohort. After the internal testing via nested CV, the models trained on cohorts ITA and FIN for feature analysis and external validation were obtained by (1) selecting the triplet of preprocessing technique, feature selection algorithm and classifier that performed best in the internal testing, (2) carrying out a 10-fold CV on the entire cohort for hyperparameter optimization, and finally (3) training the model on the whole cohort with those hyperparameters.

Fig. 1 displays the general sequential structure of the learning pipelines, with the entire set of algorithms evaluated at each pipeline step available in Supplementary Table 2. Hyperparameter optimization within nested CV and when training the models for external testing was carried out via randomized search with balanced accuracy as optimization criterion, with the randomized search restricted to 100 hyperparameter combinations per fold.

The main preprocessing steps of each learning pipeline are summarized here. Features were first filtered by discarding those whose proportion of missing values exceeded a tuned threshold and, for each pair of features whose correlation exceeded a tuned threshold, the less informative one; the retained variables were then standardized via z-score and their residual missing values were imputed through k-nearest-neighbors imputation. Class imbalance was subsequently addressed at training time through resampling. Crucially, all of these preprocessing steps – missingness- and correlation-based feature filtering, scaling, imputation and resampling – were fitted strictly within the training partition of each fold, during both the inner and outer loops of the nested cross-validation, so that no information from held-out data could influence model learning. A direct consequence of this within-fold design is that the exact set of filtered-out features can vary slightly across folds.

Importantly, the pipeline configuration was not fixed a priori but selected from the data: the search space comprised several resampling strategies (SMOTE, random undersampling and Edited Nearest

Table 1
Cohorts' characteristics. For continuous variables, value distributions per subgroup are reported as *mean* ± *std* in case of normally distributed parameters and as *median [first quartile – third quartile]* for the remaining ones. For binary variables, the number of subjects fulfilling the criterion within the subgroup is presented, with the corresponding percentage value. The treatment type, the number of treated vessels and the culprit vessel are reported here as baseline characteristics but were not used as model input features (see Section 2.1).

	Sinus Rhythm		Atrial Fibrillation	
	Cohort ITA n = 2,200	Cohort FIN n = 3,631	Cohort ITA n = 240	Cohort FIN n = 452
Demographics and clinical history				
Age [years]	66 ± 12	66 ± 12	74 ± 10	75 ± 9
Male sex, *n* (%)	1644 (74.7%)	2506 (69.0%)	159 (66.3%)	283 (62.6%)
Body Mass Index [kg/m^2]	26.6 ± 4.5	28.0 ± 5.1	26.1 ± 4.6	28.0 ± 4.7
Hypertension, *n* (%)	1395 (63.4%)	1974 (54.4%)	193 (80.4%)	309 (68.4%)
Diabetes, *n* (%)	466 (21.2%)	725 (20.0%)	81 (33.8%)	137 (30.3%)
Current smoker, *n* (%)	747 (34.0%)	1140 (33.0%)	57 (23.8%)	60 (13.3%)
Former smoker, *n* (%)	442 (20.1%)	683 (19.8%)	41 (17.1%)	89 (19.7%)
Previous AMI, *n* (%)	569 (25.9%)	443 (12.2%)	77 (32.1%)	105 (23.2%)
Previous CABG, *n* (%)	254 (11.6%)	203 (5.6%)	39 (16.3%)	47 (10.4%)
Previous PCI, *n* (%)	575 (26.2%)	311 (8.6%)	68 (28.3%)	61 (13.5%)
Ongoing treatment at hospital admission				
Aspirin, *n* (%)	820 (37.3%)	444 (12.2%)	100 (41.7%)	85 (18.8%)
Anticoagulants, *n* (%)	56 (2.6%)	34 (0.9%)	29 (12.1%)	39 (8.6%)
Antidiabetics, *n* (%)	432 (19.7%)	281 (7.8%)	78 (32.5%)	63 (13.9%)
Statins, *n* (%)	744 (33.8%)	394 (10.9%)	99 (41.3%)	79 (17.5%)
Beta blockers, *n* (%)	769 (35.0%)	427 (11.8%)	104 (43.3%)	79 (17.5%)
Thienopyridines, *n* (%)	398 (18.1%)	106 (2.9%)	41 (17.1%)	31 (6.9%)
RAAS inhibitors, *n* (%)	858 (39.1%)	535 (14.8%)	120 (50.0%)	90 (19.9%)
Infarction and treatment procedure characteristics				
NSTEMI, *n* (%)	1184 (53.8%)	1698 (46.8%)	110 (45.8%)	237 (52.4%)
Medical therapy, *n* (%)	268 (12.2%)	493 (13.6%)	58 (24.2%)	70 (15.5%)
Urgent PCI (within 24 h), *n* (%)	1573 (71.5%)	2662 (73.3%)	144 (60.0%)	274 (60.6%)
Non-urgent PCI, *n* (%)	277 (12.6%)	182 (5.0%)	28 (11.7%)	20 (5.8%)
CABG, *n* (%)	82 (3.7%)	294 (8.1%)	10 (4.2%)	88 (19.5%)
Left ventricular ejection fraction [%]	51.30 ± 11.1	49.56 ± 11.0	43.16 ± 13.19	45.54 ± 12.55
N. of treated vessels				
0, *n* (%)	361 (16.4%)	743 (20.5%)	61 (25.4%)	143 (31.6%)
1, *n* (%)	1401 (63.7%)	2608 (71.8%)	133 (55.4%)	263 (58.2%)
2, *n* (%)	344 (15.6%)	256 (7.1%)	33 (13.8%)	38 (8.4%)
3+, *n* (%)	91 (4.1%)	24 (0.7%)	11 (4.6%)	8 (1.8%)
Culprit vessel				
Unidentified, *n* (%)	288 (13.1%)	750 (20.7%)	53 (22.1%)	145 (32.1%)
Left Anterior Descending, *n* (%)	848 (38.6%)	1276 (35.1%)	80 (33.3%)	127 (28.1%)
Right Coronary Artery, *n* (%)	492 (22.3%)	847 (23.3%)	53 (22.1%)	85 (18.8%)
Circumflex Artery, *n* (%)	325 (14.8%)	339 (9.3%)	32 (13.3%)	48 (10.6%)
Vein Graft, *n* (%)	80 (3.6%)	54 (4.9%)	5 (2.1%)	13 (2.9%)
Left Main Coronary Artery, *n* (%)	48 (2.2%)	41 (1.3%)	4 (1.7%)	14 (3.1%)
Other, *n* (%)	117 (5.3%)	324 (8.9%)	11 (4.6%)	20 (4.4%)
Laboratory values at CICU admission				
Hemoglobin [g/dL]	13.78 ± 1.81	13.65 ± 1.56	13.21 ± 1.92	13.24 ± 1.63
Troponin [ng/L]	400 [70–2335]	130 [45–405]	1077 [130–6703]	200 [63–653]
Glycemia [mg/dL]	147 ± 60	151 ± 60	167 ± 72	168 ± 69
Glycated hemoglobin [%]	6.1 ± 1.3	6.1 ± 1.2	6.1 ± 1.1	6.4 ± 1.2
Serum creatinine [mg/dL]	0.93 [0.79–1.11]	0.74 [0.63–0.89]	1.02 [0.83–1.35]	0.82 [0.68–1.04]
Total Cholesterol [mg/dL]	178 ± 45	175 ± 42	162 ± 41	153 ± 37
LDL Cholesterol [mg/dL]	111 ± 40	113 ± 37	98 ± 36	92 ± 33
HDL Cholesterol [mg/dL]	43 ± 13	44 ± 13	43 ± 16	43 ± 13
Triglycerides [mg/dL]	104 [76–149]	119 [89–167]	96 [69–133]	97 [75–130]
Estimated glomerular filtration rate [mL/min/1.73 m^2]	79 ± 26	83 ± 21	68 ± 26	70 ± 22
High-sensitivity C-reactive protein [mg/L]	3.4 [1.4–10.7]	3.8 [1.9–10.4]	7.0 [2.1–36.7]	5.3 [2.2–24.4]

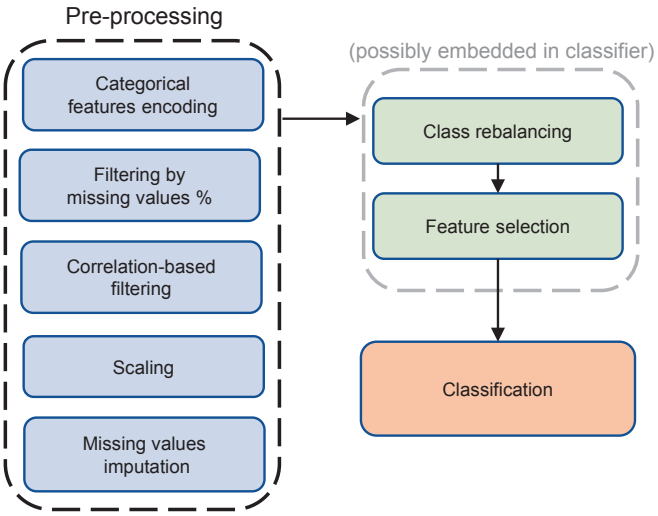

Fig. 1. General structure of the machine learning pipeline employed to train the classifier based on each cohort's data.

Neighbors) and allowed logistic regression both with and without class weighting, alongside the other candidate classifiers. Balanced accuracy was adopted as the optimization criterion because, given the marked class imbalance (approximately 10% AF prevalence), it prevents the majority class from dominating model selection and appropriately rewards correct identification of the minority – and clinically more relevant – AF class. The combination of Edited Nearest Neighbors undersampling with a balanced-class-weighted logistic regression therefore emerged as the best-performing configuration rather than being imposed by design.

2.3. Statistical analysis

In order to compare the variable distributions across and within the two datasets, we utilized the Mann-Whitney *U* test and the Chi-square test for continuous and categorical variables, respectively; the statistical significance threshold was set at $p < 10–4$ to account for the sample size and the number of variables tested. Regarding model prediction performances, all metrics presented in the paper are accompanied by confidence intervals computed through bootstrap resampling (1,000 iterations). For both the univariate and the multivariate logistic regression models, p-values were obtained via two-tailed Wald tests to test *a posteriori* feature relevance. Association between anticoagulation therapy and the potential underlying confounders evaluated were computed via Fisher's Exact test, with results considered statistically significant where $p < 0.05$.

2.4. Implementation

All code was implemented in Python 3.11.5, primarily leveraging Scikit-Learn 1.3.0 [30] and Imbalanced-Learn 0.12.0 [31] libraries for machine learning tasks and Scipy 1.15.1 [32] and statsmodels 0.14.4 [33] for statistical analyses. All experiments were run on a standard laptop (Dell XPS 13 7390; Intel Core i7-10510U, 10th generation; 16 GB LPDDR3 2133 MHz RAM; Ubuntu 24.04), with all algorithms being CPU-based and requiring no dedicated GPU.

3. Results

3.1. Dataset analysis

Table 1 displays the baseline characteristics for the Italian (ITA, N = 2,440) and Finnish (FIN, N = 4,083) cohorts. In both populations, AF patients were significantly older, had lower left ventricular ejection fraction (LVEF) and estimated glomerular filtration rate (eGFR), and higher prevalence of hypertension compared to those in sinus rhythm. Biochemical markers such as high-sensitivity C-reactive protein (HsCRP) and blood glucose were consistently higher in AF patients. Regarding inter-cohort differences, cohort ITA had a higher prevalence of previous cardiovascular events and revascularization procedures, and a higher frequency of ongoing medication use (e.g., aspirin, statins). Conversely, cohort FIN had a higher mean Body Mass Index.

The complete results of the statistical tests ran for value distribution comparisons between all relevant cohort − or sub-cohort − combinations (e.g., ITA vs FIN, ITA SR vs ITA AF, ITA SR vs FIN SR, etc.) can be found in Supplementary Table 3.

3.2. Preliminary models

Table 2 displays performance metrics on unseen data obtained in each considered scenario. For all three trained models (cohort ITA, FIN and ITA + FIN), the pipeline that performed best during the inner phases of the nested cross-validation combined undersampling via Edited Nearest Neighbors with an L2-regularized logistic regression for classification, without any additional feature selection, and with minor differences in hyperparameters among the three cases. A comparison of the best pipeline built around each candidate classifier (Supplementary Table 4) confirmed that L2-regularized logistic regression outperformed all the alternative classifiers in every scenario, with only random forest approaching its performance. In all the five considered evaluation scenarios, the classification performance appeared comparable, with only the internal testing on cohort FIN slightly outperforming the others. Internal and external testing for cohort ITA scored highly similarly, and the external test of the FIN model still achieved a reasonably good performance, despite suffering a setback compared to the internal one. The combined cohorts model achieved results comparable to those of the individual models, likely benefitting from the higher amount of data and considering the limited differences between the two cohorts' characteristics.

3.3. Feature importance

Given that logistic regression was the best classifier in all considered scenarios, the relevance of the individual predictors was assessed in each cohort by computing odds ratios with 95% confidence intervals from the logistic-regression coefficients, deriving p-values via two-tailed Wald tests. Fig. 2 displays the forest plots of these multivariate analyses for the preliminary models, with the respective significant markers. Cohort ITA showed six independent predictors, with hypertension and diabetes barely achieving statistical significance, and age as the undisputed most relevant individual marker. Cohort FIN, instead, displayed eight separate predictors − although several with limited statistical significance − with age being the strongest risk factor, once more. Differently from cohort ITA, high-sensitivity C-reactive protein, glycemia and triglycerides, along with the current smoking status, emerged as additional predictors in cohort FIN. An ongoing anticoagulation therapy was the only other marker whose significance was confirmed in both cohorts: odds ratios' 95% confidence intervals (OR 95% CI) for this variable spanned from 1.16 to 1.41 and 1.19 to 1.37 for cohorts ITA and FIN, respectively.

3.4. Anticoagulation therapy analysis

To investigate potential confounding by indication regarding anticoagulation therapy, we analyzed a subset of 831 patients from cohort ITA with detailed clinical notes. We identified three comorbidity categories: prosthetic heart valves, history of paroxysmal AF, and prior thromboembolism. While Fisher's Exact tests confirmed significant associations between anticoagulation therapy and these conditions (Table 3), a multivariate regression including these specific comorbidities did not eliminate the predictive significance of anticoagulation therapy (OR 1.32, 95% CI: 1.08–1.65). This suggested the presence of residual, unmeasured confounding, prompting the exclusion of this variable from the final clinical models.

3.5. Final models

We ultimately trained and evaluated models where the anticoagulation treatment variable was excluded. Table 4 displays the classification performances for these revised models, and Supplementary Table 5 reports all confusion matrices. Across all internal

Table 2
Classification performances on unseen data in all considered scenarios, with 95% confidence intervals.

	Cohort ITA		Cohort FIN		ITA + FIN
	Internal test	External test	Internal test	External test	Internal test
Balanced accuracy	67.2 [64.0–70.3]	68.2 [66.0–70.5]	70.3 [68.1–72.5]	66.0 [63.2–68.6]	69.3 [67.5–71.1]
AUROC	74.8 [71.4–78.0]	74.3 [72.0–76.7]	76.9 [74.7–79.0]	73.4 [70.1–76.7]	76.2 [74.4–77.9]
Sensitivity	67.1 [60.9–73.2]	73.7 [69.4–77.8]	76.3 [72.5–80.6]	80.8 [75.7–85.7]	75.1 [71.9–78.3]
Specificity	67.3 [65.3–69.3]	62.8 [61.2–64.4]	64.2 [62.7–65.8]	51.2 [49.1–53.3]	63.6 [62.3–64.9]
Precision	18.3 [15.7–21.0]	19.8 [17.8–21.7]	21.0 [19.0–22.9]	15.3 [13.4–17.2]	19.6 [18.1–21.2]
F1-score	28.7 [25.2–32.3]	31.2 [28.6–33.7]	32.9 [30.2–35.5]	25.7 [22.9–28.5]	31.1 [29.1–33.2]

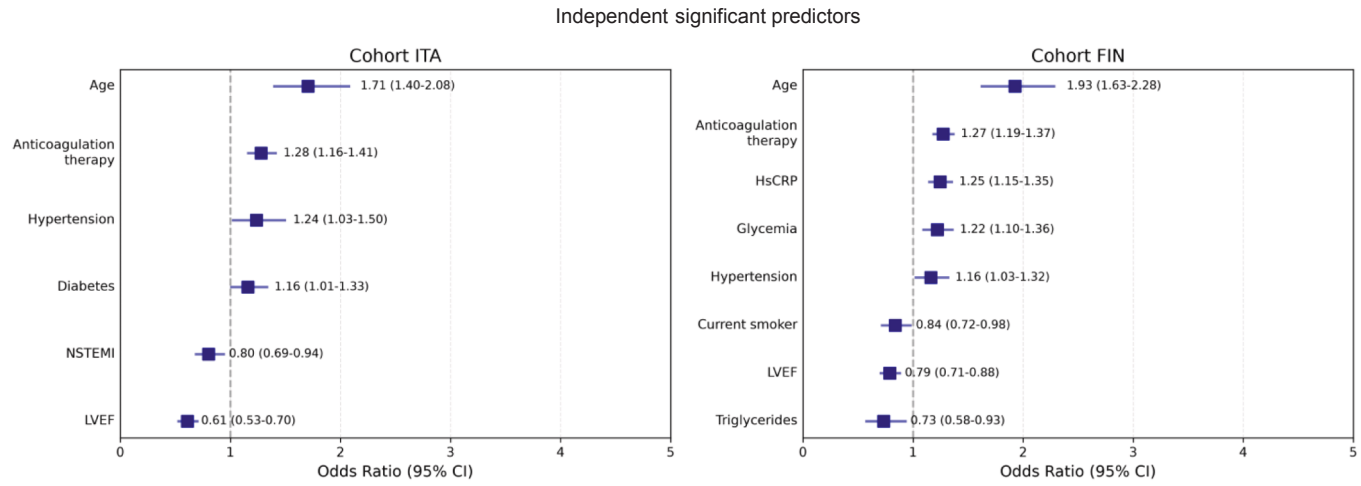

Fig. 2. Forest plots of independent significant predictors of in-hospital AF for cohorts ITA and FIN (odds ratios with 95% confidence intervals) in the preliminary models, i.e., including the anticoagulation therapy variable.

Table 3
Contingency tables and Fisher's Exact test results regarding the association between ongoing anticoagulation therapy and the investigated underlying comorbidities.

		Has history of paroxysmal AF		Carries a prosthetic valve		Has history of DVT/PE	
		No	Yes	No	Yes	No	Yes
Is undergoing anticoagulation therapy	No	776	17	782	11	783	10
	Yes	34	4	34	4	35	3
Fisher's test result		$p = 0.013$		$p = 0.004$		$p = 0.018$	

and external testing scenarios, the performance metrics remained highly comparable to those of the anticoagulation-reliant models. The optimal pipeline configurations, detailed in Table 5, also remained consistent, with an L2-regularized logistic regression combined with Edited Nearest Neighbors for class rebalancing emerging as the best-performing approach in all cases. As for the preliminary models, a comparison against the best pipeline built around each alternative classifier (Supplementary Table 6) confirmed the superiority of L2-regularized logistic regression, with only random forest approaching its performance.

Fig. 3 shows the relative feature importance (L2-normalized logistic-regression coefficients) of the most relevant variables in each cohort's final model. Fig. 4 shows the forest plots for the *a posteriori* feature relevance analysis of the final models. The core predictors – advanced age, lower LVEF and hypertension – maintained their strong, independent predictive value in both cohorts. Beyond these, in cohort ITA, NSTEMI and ongoing aspirin therapy were independently associated with a lower AF risk, whereas in cohort FIN higher high-sensitivity C-reactive protein emerged as an additional risk factor and the current smoking status as a protective one. The persistence of the core predictors across both the preliminary and the final models confirms that our models captured genuine risk factors, independent of the confounding signal from anticoagulation therapy.

Reflecting the within-fold preprocessing described in Section 2.2, the feature-filtering steps of the final models removed a small number of variables. In cohort ITA, only glycated hemoglobin was discarded, by the missingness threshold; in cohort FIN, LDL-cholesterol was removed through correlation filtering, being correlated with total cholesterol, while glycated hemoglobin, diabetes, HDL-cholesterol, total cholesterol, triglycerides, body mass index and glycemia were discarded by the missingness threshold; in the combined ITA + FIN model, again only glycated hemoglobin was removed. The exclusion of glycemia and triglycerides from the Finnish model, in particular, explains their absence among its predictors despite their relevance in the preliminary analysis.

Beyond discrimination, we evaluated the calibration of the final models (Supplementary Table 7 and Supplementary Fig. 1). Across all five scenarios the models systematically over-estimated the absolute AF risk: calibration slopes were below 1, calibration-in-the-large (intercept) was negative, and the Brier skill scores were negative relative to a prevalence-based reference, with the miscalibration most pronounced under external testing.

4. Discussion

In this study, we trained machine learning models for predicting in-hospital AF post AMI, relying exclusively on routine parameters available at CICU admission, and evaluated them on two patient cohorts from Italy and Finland. Our models' performances appear aligned with others reported in literature devoted to the same prediction task, emphasizing the difficulty of predicting AF in hospital and without ECG data. Specifically, two other studies predicting in-hospital new-onset AF after AMI reported an AUROC of 79.1% (95% CI: 69.2–89.1%) [18] and

Table 4
Classification performances on unseen data in all considered scenarios, with 95% confidence intervals, for the models without the anticoagulation therapy variable.

	Cohort ITA		Cohort FIN		ITA + FIN
	Internal test	External test	Internal test	External test	Internal test
Balanced accuracy	66.5 [63.1–69.6]	66.5 [64.1–68.9]	69.5 [67.4–71.6]	66.0 [63.5–68.4]	68.5 [66.8–70.3]
AUROC	74.0 [70.6–77.2]	73.5 [71.1–75.9]	75.5 [73.3–77.6]	72.3 [69.4–75.6]	74.9 [73.2–76.6]
Sensitivity	66.8 [60.6–73.1]	66.2 [61.7–70.6]	76.1 [72.3–80.0]	85.8 [81.2–90.1]	76.0 [72.6–79.2]
Specificity	66.2 [64.2–68.2]	66.9 [65.3–68.4]	62.9 [61.3–64.4]	46.2 [43.9–48.3]	61.1 [59.8–62.4]
Precision	17.7 [15.3–20.2]	19.9 [18.0–21.9]	20.4 [18.5–22.2]	14.8 [13.1–16.7]	18.8 [17.4–20.3]
F1-score	28.0 [24.5–31.5]	30.6 [28.0–33.4]	32.1 [29.5–34.5]	25.3 [22.6–28.1]	30.1 [28.2–32.3]

Table 5
Characteristics of the best-performing classification pipeline without the anticoagulation therapy variable for each cohort, with the corresponding optimal hyperparameter values.

Pipeline step	Algorithm employed + hyperparameter values Cohort ITA	Cohort FIN	ITA + FIN
NaN%-based filtering	Features with more than 20% NaN values discarded	Features with more than 10% NaN values discarded	Features with more than 30% NaN values discarded
Correlation-based filtering	Not applied	Keep one for each pair of features with correlation >= 80%	Not applied
Feature scaling	Z-score	Z-score	Z-score
Missing values imputation	1-Nearest-Neighbors (Euclidean distance)	3-Nearest-Neighbors (Euclidean distance)	3-Nearest-Neighbors (Euclidean distance)
Class rebalancing	Edited Nearest Neighbors (2 neighbors, Euclidean distance)	Edited Nearest Neighbors (5 neighbors, Euclidean distance)	Edited Nearest Neighbors (5 neighbors, Euclidean distance)
Feature selection	None	None	None
Classification	Logistic Regression (C = 0.7, class weight = balanced, tol = 10–5, penalty = L2)	Logistic Regression (C = 0.5, class weight = balanced, tol = 0.0005, penalty = L2)	Logistic Regression (C = 0.5, class weight = balanced, tol = 10–5, penalty = L2)

81.8% (no CI reported) [15], respectively, slightly higher than that obtained by our models. However, both studies were carried out on much smaller datasets – 333 and 311 patients – one of which encompassed exclusively patients older than 65 years, and with both datasets also relying on clinical variables not available for our cohorts. Hence, our study arguably provides a more robust performance estimate for ML-based in-hospital prediction of AF post AMI. Moreover, it represents, to our knowledge, the first cross-cohort, cross-nation evaluation of this kind, additionally allowing us to observe any baseline population differences between the two considered populations, and to evaluate their impact on the prediction task. Comparing the value distributions of the analyzed clinical variables between the two cohorts, however, there appeared to be only a few, predictable and overall minor differences, such as a higher BMI and prevalence of diabetes in the Finnish patients. One notable exception was the significantly higher troponin levels observed in the Italian cohort. However, several possible explanations for this discrepancy exist: systematic variations in clinical pathways between the two hospitals, such as the timing of blood sampling relative to symptom onset; the use of troponin assays from different manufacturers, or inconsistencies in the application of the recommended guidelines for defining the upper reference limits; other cohort characteristics acting as confounders, given that age, renal dysfunction (as estimated by eGFR) and sex are known to influence absolute troponin values by up to 300%, and both eGFR and sex distribution are different between the ITA and FIN cohorts [34,35]. Additionally, the study's long inclusion period (2007–2018) spanned the widespread clinical transition to high-sensitivity troponin assays, which might have happened at different time points in the two hospitals [34]. Regardless, the variables showing the most pronounced distribution differences between the two cohorts – such as troponin – were not retained as independent predictors, and the core predictors of advanced age, lower LVEF and hypertension proved common to both populations, underscoring how the principal risk factors and their impact on the outcome appear largely shared, with only a few exceptions. Indeed, this observation is also supported by the closely aligned performances obtained in the internal and external testing of each cohort's model, as well as by the performance of the multi-cohort one.

Analyzing the individual risk factors via relative feature importance in the ML models and multivariate regression, we initially observed an alleged high prognostic significance for ongoing anticoagulation therapy, in both cohorts. This apparent influence was hard to account for causally, and certainly represented an example of confounding by indication, with anticoagulation treatment identifying untracked underlying comorbidities. Our additional analyses on the matter did indeed show a link between anticoagulation therapy and three specific categories of underlying comorbidities. However, we were unable to fully discount the predictive power of the variable, likely due to other untracked independent risk factors, such as a general greater burden of vascular disease, frailty or higher CHA_2DS_2-VASc scores (not fully captured by individual variables). This unresolved confounder represented a limitation of the preliminary models, and highlighted how routinely tracked clinical parameters were unable to fully capture the spectrum of underlying frailty of the patients, at least in the context of the present study. To overcome this limitation and develop more clinically interpretable models, we trained models excluding the anticoagulation therapy variable, a step that confirmed the robustness of our approach as it resulted in a negligible impact on overall predictive performance.

Analyzing the significant predictors in these final models, we confirmed for both cohorts ITA and FIN the well-known prognostic significance of advanced age, lower LVEF and hypertension. Beyond this shared core, each cohort retained a few additional, cohort-specific predictors. In cohort ITA, NSTEMI and ongoing aspirin therapy were associated with a lower risk, albeit with odds ratios' confidence intervals

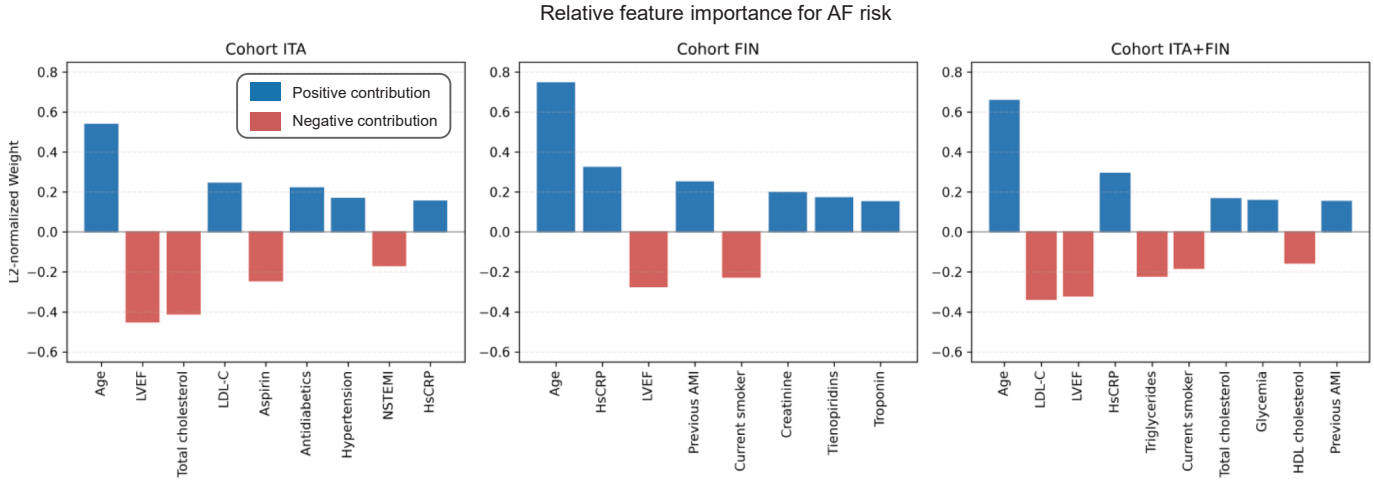

Fig. 3. Relative feature importance (L2-normalized logistic regression coefficients) of all features with relevance above the median value for the ITA, FIN and ITA + FIN final-model classifiers (i.e., without the anticoagulation therapy variable).

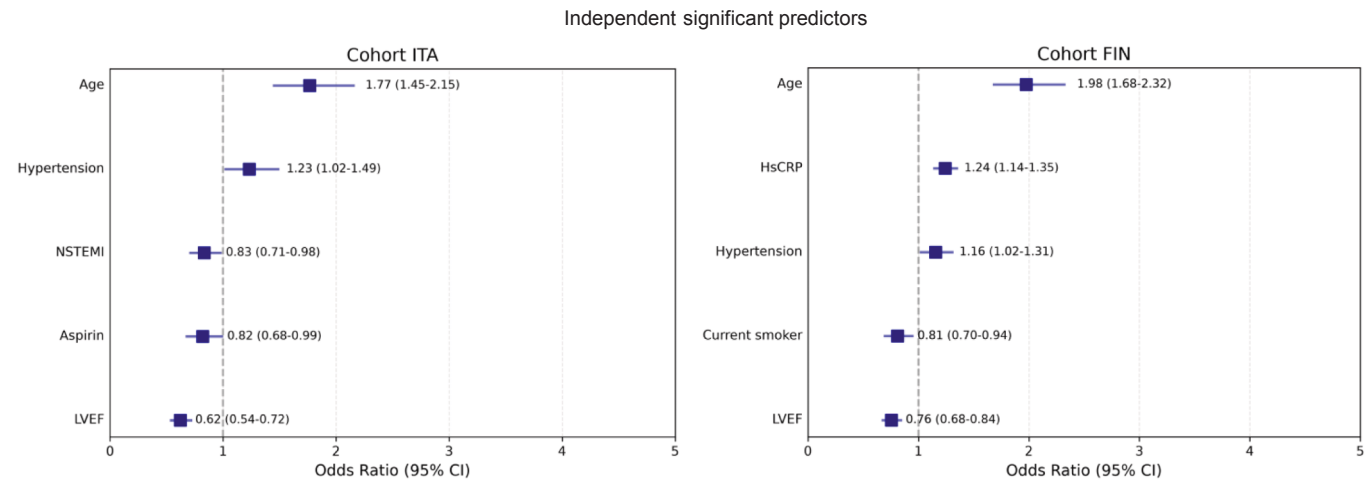

Fig. 4. Forest plots of independent significant predictors of in-hospital AF for cohorts ITA and FIN (odds ratios with 95% confidence intervals) in the models without the anticoagulation therapy variable.

almost reaching 1; plausible explanations are the lower severity of NSTEMI relative to STEMI and the anti-inflammatory effect of aspirin in the acute setting. In cohort FIN, a higher high-sensitivity C-reactive protein (HsCRP) at CICU admission was associated with a higher risk, while the current smoking status was protective: higher HsCRP, signaling systemic inflammation, is a known independent risk predictor for AF [18,36,37], whereas the apparent protective effect of current smoking is consistent with the well-documented "smoker's paradox," often attributed to residual confounding, as smokers tend to present with their first ischemic event at a younger age [38]. Notably, some established metabolic risk factors that had emerged in the preliminary models did not persist among the final-model predictors: diabetes in cohort ITA – a condition with a well-established link to higher AF risk [36] – lost statistical significance once the anticoagulation confounder was removed, whereas glycemia and triglycerides in cohort FIN, recognized AF-associated metabolic markers [37,39–41], were excluded from the final model by the within-fold missingness filtering of the restricted feature set. The cohort-specific appearance of the remaining predictors likely reflects both the difference in sample size, which affects the statistical significance reached in each cohort, and baseline population differences.

Overall, this study was able to provide the most robust estimate so far of the performance of in-hospital AF prediction after AMI via machine learning applied to routinely tracked clinical variables. Additionally, we displayed how the models performed similarly when evaluated on a completely independent cohort from a different country, emphasizing the validity and generalizability of the identified risk factors. Notably, however, despite all the advanced feature selection procedures and classifiers evaluated, a simple L2-regularized logistic regression turned out to be the best performing model in each considered case. Although aided by undersampling, missing values imputation and other feature filtering steps, this result suggests that, in the context of the present study and for this specific prediction task and feature set, more complex, black-box machine learning techniques offered limited additional utility over a simpler, traditional statistical approach; this finding should not, however, be generalized to other clinical prediction tasks without further dedicated evidence.

Beyond discrimination, the calibration analysis (Section 3.5, Supplementary Table 7 and Supplementary Fig. 1) showed that the models, while ranking patients by risk well, systematically over-estimate the absolute probability of AF. This behavior is an expected consequence of our training strategy: both the Edited Nearest Neighbors undersampling and the balanced class weighting deliberately alter the class distribution seen by the classifier to improve minority-class sensitivity, which inflates the predicted scores relative to the true population prevalence. The model outputs should therefore be interpreted as relative risk scores for stratification rather than as calibrated absolute probabilities; should calibrated probabilities be required for a specific deployment, standard post-hoc recalibration (e.g., Platt scaling or isotonic regression) could be applied as a deployment-stage step beyond the scope of the present work.

To make the practical implications of the current performance concrete, we further quantified the expected alert burden at the models' operating point (Supplementary Table 8). In the two cross-cohort (external) scenarios, the model would flag approximately 37 of every 100 admitted patients when trained on cohort ITA and tested on cohort FIN, and approximately 57 of every 100 when trained on cohort FIN and tested on cohort ITA, of whom only about 7 and 8 per 100, respectively, would actually develop AF. In other words, at this operating point roughly 80–85% of the flagged patients would be false alarms (positive predictive value approximately 15–20%), which concretely illustrates the limitation imposed by the modest precision and reinforces that the models, in their current form, are not yet suitable for direct clinical deployment.

The present study has some limitations. First, its retrospective nature and the partially paper-based records spanning the enrolment periods prevented the reconstruction of a complete patient-flow diagram with per-step exclusion counts, as well as the reliable identification of all patients with a history of paroxysmal AF, which could be ascertained only for the subset of the Italian cohort with detailed clinical notes. Including patients with prior paroxysmal AF is nonetheless clinically justified, since AF arising in the acute post-AMI setting is predominantly driven by transient, ischemia-related mechanisms – acute ischemia, inflammation, autonomic imbalance and hemodynamic stress – that are largely distinct from the atrial remodeling underlying typical paroxysmal AF, and these patients present to the CICU in sinus rhythm and require the same risk stratification as those without a prior AF history. Second, the enrolment periods (ITA 2010–2018, FIN 2007–2018) predate some recent developments in clinical practice, such as the broader adoption of high-sensitivity troponin assays and updated revascularization guidelines; although the core risk factors for post-AMI AF are well established and biologically grounded, and thus unlikely to have substantially changed in their predictive value, validation on more recent cohorts would be valuable. Finally, it is important to acknowledge that while our models demonstrate predictive capabilities to some extent, their direct implementation into clinical practice still appears out of reach. The modest precision values achieved, implying a high rate of false positives, appear not good enough to allow for the implementation

of additional surveillance and preventative measures – such as extended telemetry into the general ward stay and optimized therapies for controlling left ventricular dysfunction and other risk factors – for a subset of patients identified at higher risk that likely remains too large.

5. Conclusion

In conclusion, we developed and, for the first time, cross-nationally validated machine learning models for predicting in-hospital AF after AMI from routine, non-electrocardiographic clinical parameters available at CICU admission. Across two independent cohorts from different countries and healthcare systems, a simple and fully interpretable L2-regularized logistic regression matched the performance of more complex classifiers, and the identified risk factors – led by advanced age, lower LVEF and hypertension – proved largely consistent across populations. Nonetheless, the modest precision, the associated false-positive burden and the imperfect calibration indicate that the models are not yet ready for direct clinical deployment. Future work should focus on prospective validation in more recent cohorts, on improving precision, and on probability calibration for absolute-risk estimation, as steps toward integrating such tools into CICU risk-stratification workflows.

CRediT authorship contribution statement

Matteo Bulloni: Writing – review & editing, Writing – original draft, Software, Methodology. **Miriana Torquati:** Writing – review & editing, Visualization. **Roni Ahola:** Writing – review & editing. **Alice Bonesi:** Writing – review & editing, Data curation. **Marion Taconné:** Writing – review & editing. **Antti Vehkaoja:** Writing – review & editing, Supervision. **Felix Tirschmann:** Writing – review & editing. **Antti Kallonen:** Writing – review & editing. **Pedro Moreno-Sánchez:** Writing – review & editing. **Leo-Pekka Lyytikäinen:** Writing – review & editing, Data curation. **Jussi Hernesniemi:** Writing – review & editing, Supervision, Resources. **Erica Rurali:** Writing – review & editing. **Valentina Corino:** Writing – review & editing, Supervision. **Kirsten Brukamp:** Writing – review & editing, Supervision. **Giancarlo Marenzi:** Writing – review & editing, Supervision, Resources. **Linda Pattini:** Writing – review & editing, Supervision. **José Pablo Werba:** Writing – review & editing, Supervision, Data curation. **Claudio Tondo:** Writing – review & editing, Supervision. **Mark van Gils:** Writing – review & editing, Supervision. **Luca Mainardi:** Writing – review & editing, Supervision, Project administration, Funding acquisition, Conceptualization.

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Data availability

The data underlying this article cannot be shared publicly due to privacy reasons.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ijmedinf.2026.106595.

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