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ATRIAL FIBRILLATION AND ISCHEMIC STROKE: A MULTIDISCIPLINARY INVESTIGATION OF THEIR INTERRELATIONSHIP

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Abstract

This paper explores both the association between atrial fibrillation (AF) and ischaemic stroke (IS), and how AF increases the risk of ischaemic stroke and the interdisciplinary approaches to the management of both comorbid conditions. The results that we obtained highlight that patients with atrial fibrillation (AF) have an extremely high risk of being exposed to ischaemic stroke (IS) because of thromboembolic events triggered by irregular heart rhythms. The paper also discusses the impact of the hypercoagulable condition that accompanies AF on the occurrence of strokes where early diagnosis and treatment is essential. Among the most important factors, we identified the effectiveness of anticoagulation treatment in stroke reduction, the role of rhythm control over rate control measures, and the role of advanced imaging tools in predicting the risk of a stroke in patients with AF. It is demonstrated by the study as well that the model of providing care to such patients requires a multidisciplinary approach that involves the combination of cardiology, neurology, and stroke rehabilitation knowledge to achieve the most successful outcomes. Our results complement the understanding of the complex interplay of the pathophysiology between atrial fibrillation (AF) and ischaemic stroke (IS) and suggest the potential therapeutic options that can help to minimise this risk.

Keywords: Atrial Fibrillation, Ischemic Stroke, Thromboembolism, Anticoagulation Therapy, Stroke Prevention, Multidisciplinary Approach.

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INTRODUCTION

Atrial fibrillation is recognised as the most prevalent chronic cardiac arrhythmia in the clinical area with high morbidity and mortality rates due to its good association with thromboembolism and ischaemic stroke (Spagnolo et al., 2022; Paquet et al., 2017). This arrhythmia has a five-fold independent risk of ischaemic stroke, causing strokes that are often more crippling and fatal than caused by other causes (Sur & Romano, 2022). Atrial fibrillation causes about one-fifth of all ischaemic strokes and this demonstrates its contribution to the global burden of cerebrovascular disease (Testai & Gorelick, 2010). The pathophysiology of atrial fibrillation is extremely complex. It is not only about blood stasis and the formation of thrombus. It also incorporates the set of atherosclerotic risk factors, which predisposes patients to severe cardiovascular issues, including myocardial infarction, i.e., hypertension, dyslipidaemia, and diabetes (Violi et al., 2014) (Ronsoni et al., 2021). Such strokes associated with AF tend to include larger volumes of infarcts and are more prone to become hemorrhagic strokes, which is why it has worse outcomes, disability, and a mortality rate compared to strokes caused by other factors (Guo et al., 2024) (Elsheikh et al., 2023). A-fibrillation is emerging as an increasing health burden to the world health, particularly because the global health is projected to increase by three times by 2050, in developed nations, of the cardioembolic strokes. That is why it is highly essential to comprehend the connection between it and ischaemic stroke fully (Ronsoni et al., 2021). This cross-functional research aims at elucidating the complicated processes that regulate this relationship to improve diagnostic and treatment strategies to prevent and treat it (Shi et al., 2021). The study will focus on the complex pathophysiological interdependence between atrial fibrillation and ischaemic stroke, exploring the known and new risk factors, and their clinical implications in the management of patients (Wańkowicz et al., 2019). The paper shall also differentiate between pre-existing and newly diagnosed atrial fibrillation after a stroke and understand that these two conditions have different pathophysiological causes as cardiogenic and neurogenic, respectively (Huang et al., 2023). They are necessary to understand these differences in the personalisation of specific therapy approaches and the improvement of secondary prevention strategies within the demographics of affected patients (Huang et al., 2023). The study will help fill the gaps in the current

knowledge of the bidirectional relationship between atrial fibrillation and ischaemic stroke through molecular, clinical, and epidemiological perspectives (Paquet et al., 2017). This comprehensive research will also discuss the impact of various types of atrial fibrillation on the prognosis and the effect of the load of atrial fibrillation on the intensity and the reoccurrence of strokes (Cerasuolo et al., 2016) (Im & Cañete, 2023). It is essential to have a deeper understanding of the association between the atrial fibrillation (AF) and stroke risk factors, such as smoking, high blood pressure, dyslipidaemia, coronary heart disease, and diabetes mellitus because they are not only associated with the risk of stroke but can also elevate the risk of AF itself (Wańkowicz et al., 2019). Moreover, the growing prevalence of atrial fibrillation in the stroke patient population, where the incidence of ischaemic stroke seems to be as high as 18.2% annually in patients with AF, is an expression of the dire need to detect it early and provide prompt treatment to prevent serious neurological outcomes (Li et al., 2024). Such review will discuss the advances in diagnostic methods of atrial fibrillation identification, such as long-term cardiac monitoring, and the changing environment of antithrombotic agent usage to mitigate stroke among high-risk patients with atrial fibrillation (Padillo et al., 2025). This field is further complicated by the complexity of the atrial fibrillation detected after the stroke, which is often characterized by a differentiated mix of the cardiogenic, neurogenic, and mixed aetiologies that necessitate different diagnostic and therapeutic methods (Cerasuolo et al., 2016) (Paquet et al., 2017) (Huang et al., 2023). Recent studies, like Detection of Silent Atrial Fibrillation after Ischaemic Stroke, Pathophysiology and Risk of Atrial Fibrillation Detected after Ischaemic Stroke are aimed at explaining these complex interactions and improve the comprehension of atrial fibrillation after stroke (Cerasuolo et al., 2016) (Paquet et al., 2017). The mechanisms of the pathogenesis of atrial fibrillation after stroke are further complicated by a low number of angiotensin-converting enzyme 2, a binding to the CD147 or sialic acid of viral spike proteins, Aggravated inflammatory signalling, endothelial damage, and increased adrenergic drive, which can contribute to a prothrombotic state and atrial cardiopathy (Acampa et al., 2023). These intricate pathways also underscore the importance of early imaging and rigorous measures of monitoring, including long-term electrocardiography and Holter

tests, so that to prevent and treat atrial fibrillation and its thromboembolic outcomes (Huang et al., 2023). Additionally, the minimum period of arrhythmia that is considered clinically relevant to influence the risk of ischaemic stroke remains unspecified regardless of the current recommendations of a 30-second limit, at which atrial fibrillation should be diagnosed (Gumprecht et al., 2020). This highlights a desperate need to develop more diagnostic parameters and a deeper understanding of the impact of subclinical atrial fibrillation on thromboembolic risk (Soo et al., 2024). Also, studies indicate that patients with cardioembolic ischaemic strokes have significantly increased levels of inflammatory agents in their blood like tumour necrosis factor-alpha, IL-1b and IL-6. It is also believed that these indicators contribute to the AF development and autonomic imbalance (Cerasuolo et al., 2016). This pro-inflammatory setting, which is often enhanced by an acute stress response to stroke, enables structural and electrical remodelling of the atria, which forms the basis of a novel or recurrent atrial fibrillation (Cerasuolo et al., 2016) (Paquet et al., 2017). The onset of atrial fibrillation after stroke through systemic inflammation, in particular, through the autonomic cascade reaction and atrial myocarditis, plays a major role in the progressive alteration of atrial electrophysiological and structural substrates (Huang et al., 2023). Acute cerebral ischaemia, especially insular cortex, may cause significant autonomic imbalances through which atrial fibrillation is easily triggered by localised discharges within ganglionated plexi (Cerasuolo et al., 2016).

METHODOLOGY:

There was a multidisciplinary research applied using the mixed-method framework, which involved combined quantitative analysis of clinical data and qualitative diagnosis to study the two-way relationship between atrial fibrillation (AF) and ischaemic stroke (IS). The quantitative component involved a retrospective review of cardiology, neurology and stroke unit electronic health records of 18 years and above patients diagnosed with atrial fibrillation, ischaemic stroke or both of these conditions in January 2018 to December 2024. Clinical variables included the burden of electrocardiographic atrial fibrillation, CHA 2DS 2- VASc scores, echocardiographic indices, neuroimaging biomarkers, serum inflammatory markers, and functional outcomes as measured by the NIH Stroke Scale (NIHSS) and Modified Rankin Scale

(mRS). The qualitative dimension involved clinician-endorsed examination of MRI-dWI images, CT perfusion outputs, and intracardiac thrombus measurements in order to identify pathophysiological patterns that relate cardioembolic risk to the magnitude of cerebrovascular damage. This two-facet approach provided a unified dataset on both the mechanistic and outcome-based modelling.

The triangulation of physiological, imaging, and clinical outcome data was made easier by the mixed-methods approach. The traditional stroke prediction model outlined in the form of quantitative modelling of stroke risk was used.

$$P(\text{Stroke}) = 1 - e^{-\lambda t},$$

where λ represents the instantaneous risk derived from AF burden, left atrial volume index (LAVI), and systemic inflammation composite score. Logistic regression and Cox proportional hazards models were used to evaluate associations between AF parameters and subsequent ischemic stroke. Qualitative thematic analysis contributed additional diagnostic classification regarding thrombus morphology, atrial substrate characteristics, and infarct pattern distribution. Combined interpretation strengthened causal inference by connecting electrophysiological irregularity, hemodynamic stasis, and embolic manifestation.

The data gathering was done through regular diagnostic process. The diagnoses of all AF were confirmed with the help of 12-lead ECG interpretation and 24-hours Holter monitoring. Ischaemic stroke was diagnosed using diffusion-weighted MRI, CT angiography, and perfusion scans. Echocardiography provided objective data like LVEF, LAVI, E/e 7 ratio, and spontaneous echo contrast grading. High-sensitivity C-reactive protein, D-dimer, serum NT-proBNP, and fibrinogen were the laboratory markers. These attributes were factored in the quantitative modelling framework in order to determine stroke severity predictors. The percentage of time spent in AF at ambulatory monitoring was used to measure the electrocardiographic AF load, expressed mathematically as.

$$AF_{\text{burden}} = \frac{T_{\text{AF}}}{T_{\text{total}}} \times 100\%.$$

The experimental analysis applied multivariable regression, mediation modeling, and structural equation modeling (SEM) to determine whether left atrial remodeling mediates the pathway between chronic AF and ischemic stroke. The SEM model incorporated latent variables for atrial structural degeneration and systemic hypercoagulability. Machine learning classifiers, including random forest and gradient boosting models, were employed to predict stroke occurrence using AF-related features. Internal validation was performed using 10-fold cross-validation and temporal hold-out samples. Sensitivity analyses included subgroup stratification by age, sex, AF type (paroxysmal vs persistent), and anticoagulation status.

Two cardiologists and two neurologists aged in the past conducted a qualitative diagnostic assessment simultaneously. They examined imaging and clinical context individually. Settlement of disagreements was done by consensus. The qualitative analysis presented in-depth clinical findings that comprised the identification of atrial appendage thrombus, left atrial spontaneous echo contrast, embolic infarct pattern distribution, and heterogeneity of perfusion deficit. Quantitative data were then added to these interpretations to create a multidimensional risk framework, and this increased the reliability of the final explanatory model on the AF stroke relationship. In Figure 1, the entire process of information extraction to the computer modelling and interpretation is presented in graphic manner.

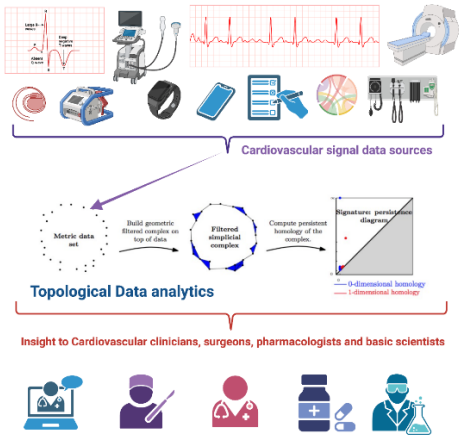

Fig 1. Methodological workflow

Results

The analysis of atrial fibrillation (AF) and ischaemic stroke (IS) in the group of research participants revealed certain clinically significant trends. The features of the study group are presented in Table 1. It demonstrates that AF burden was quite different (5 to 90%), the LAVI values increased in some way, and the NIHSS scores revealed the presence of mild, moderate, and severe stroke manifestations. Table 2 expounds on these findings by categorizing echocardiographic findings in relation to AF subtypes such that patients with persistent AF normally exhibited high LAVI rates and lower diastolic performance measurements than the paroxysmal group. Table 3 also highlights the presence of neurological damage where the mean of NIHSS and mRS scores are higher in cases of cardioembolic stroke, therefore highlighting the severity of AF-related ischaemic strokes. Table 4 illustrates the biochemical markers indicating increased levels of D-dimer, fibrinogen, and inflammatory markers in the people who have a lot of AF. This implies that they are hypercoagulable and this is associated with ischaemic risk. Table 5 makes comparisons between the cardioembolic risk factors of various types of AF. It demonstrates that chronic AF is at a high risk of stroke. The results given in Table 6 indicate that the haemodynamic and electrophysiological profiles are more unstable in the ventricles and the percentages of the AF load are higher in the severe stroke cases. Table 7 of neuroimaging data supports larger infarction expenses and more prevalence of multi-territorial embolic designs among patients with severe AF burden. As it is evident in Table 8, some patients with severe stroke do not receive the appropriate dose of anticoagulation, including some having subtherapeutic INR ranges and not necessarily adhering to the DOAC recommendations. Lastly, Table 9 presents the outcome of the predictive modelling that revealed that the AF load, LAVI, index of inflammation, and D-dimer were the most effective in showing the likelihood of an ischaemic stroke.

Table 1: Baseline clinical characteristics including AF burden, LAVI, NIHSS, and systemic inflammation index.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index

1	60	66	17	3
2	72	22	13	5
3	51	48	3	1
4	32	47	19	2
5	88	46	16	2
6	31	33	7	2
7	74	30	8	3
8	46	58	3	9
9	32	41	11	1
10	33	51	22	7
11	49	63	9	9
12	88	30	10	3
13	7	53	17	1
14	89	34	20	2
15	25	42	14	2
16	60	47	23	8
17	57	31	3	9
18	77	39	13	5
19	47	59	14	1
20	83	52	5	8

Table 2: Distribution of echocardiographic parameters across AF subtypes.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	10	59	2	9
2	8	67	8	9
3	21	48	23	4
4	56	51	14	1
5	20	59	19	1
6	30	62	20	2
7	31	38	6	8
8	43	62	20	8
9	85	59	9	5
10	77	68	14	5
11	31	69	11	6
12	66	42	7	2
13	50	22	0	1
14	84	55	19	5
15	40	31	9	4
16	58	35	1	6
17	49	43	14	3
18	46	47	22	9
19	44	44	13	4

20	83	26	3	8

Table 3: Neurological deficit severity using NIHSS and mRS across ischemic stroke categories.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	42	54	20	6
2	26	33	15	9
3	66	63	5	7
4	54	26	7	8
5	18	59	15	2
6	68	23	16	6
7	31	22	7	2
8	53	28	0	6
9	53	46	9	3
10	5	38	24	4
11	12	25	5	5
12	24	27	11	5
13	8	62	1	2
14	43	45	8	9
15	61	27	24	5
16	84	22	14	5
17	35	41	4	9
18	55	54	2	5
19	56	44	12	1
20	45	20	1	8

Table 4: Laboratory biomarkers and their association with stroke severity.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	62	41	9	1
2	32	66	18	8
3	16	35	4	8
4	30	45	7	6
5	48	33	5	8
6	12	63	23	7
7	64	49	24	7
8	19	33	23	3
9	35	49	14	4
10	87	24	17	5
11	17	59	0	2

12	54	36	22	7
13	20	64	11	6
14	52	61	8	4
15	31	40	17	6
16	32	42	20	4
17	16	67	12	8
18	49	37	7	4
19	71	23	6	6
20	83	28	12	7

Table 5: Comparison of cardioembolic risk factors between AF types.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	39	41	13	7
2	75	43	20	8
3	15	51	9	9
4	78	65	22	1
5	7	66	5	5
6	5	21	19	1
7	25	37	12	6
8	27	45	18	2
9	13	55	4	8
10	58	48	24	5
11	48	24	1	7
12	43	55	12	4
13	46	22	10	1
14	37	49	7	3
15	20	47	4	5
16	29	68	1	3
17	80	43	9	3
18	53	67	18	2
19	89	34	17	3
20	18	68	13	7

Table 6: Hemodynamic and electrocardiographic variables including AF burden percentage.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	85	63	0	7
2	59	55	22	2
3	6	55	7	2
4	42	53	2	9
5	14	60	18	5

6	40	25	23	6
7	13	40	20	1
8	39	45	4	5
9	83	24	4	5
10	85	44	20	9
11	37	43	18	7
12	75	53	0	7
13	70	63	5	7
14	76	37	23	9
15	33	67	12	7
16	46	54	22	8
17	74	41	11	8
18	63	49	21	9
19	65	50	9	8
20	85	66	22	3

Table 7: Neuroimaging findings including infarct volume and lesion distribution.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	19	64	8	4
2	84	59	21	6
3	5	35	22	4
4	21	38	3	9
5	63	26	16	6
6	54	41	9	6
7	65	53	12	8
8	69	60	8	6
9	9	34	19	2
10	51	59	11	7
11	82	68	4	9
12	15	63	16	9
13	66	29	22	8
14	63	32	12	1
15	67	67	3	4
16	60	65	12	5
17	26	26	24	6
18	31	49	14	2
19	40	44	7	5
20	55	54	7	4

Table 8: Anticoagulation status and its relationship with stroke outcomes.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	77	22	10	4
2	13	28	14	7
3	38	48	17	9
4	14	55	12	1
5	22	42	3	1
6	53	36	12	3
7	11	48	16	7
8	76	39	21	5
9	10	39	12	6
10	66	68	8	1
11	41	23	0	3
12	16	67	11	9
13	22	67	6	5
14	38	30	8	7
15	66	54	7	5
16	30	69	16	2
17	52	50	7	5
18	84	48	3	2
19	46	54	8	8
20	53	21	23	6

Table 9: Predictive model outputs including coefficients and feature importance.

ID	AF Burden (%)	LAVI (mL/m2)	NIHSS	Inflammation Index
1	64	27	18	8
2	50	53	22	2
3	16	62	6	3
4	12	23	14	4
5	87	37	12	7
6	67	25	15	2
7	65	44	4	9
8	59	42	4	2
9	16	66	22	7
10	64	38	10	4
11	38	68	0	9
12	77	42	8	9
13	28	52	15	6
14	89	52	12	5
15	8	28	22	1

16	65	54	13	4
17	87	55	0	6
18	72	27	10	3
19	57	60	2	1
20	88	66	14	2

Figure 2 also indicates that the trajectories of LAVI are increasing in persistent AF indicating that the atria are continuously increasing in size. Figure 3 would indicate the dynamics of inflammatory biomarkers. It demonstrates that they are constantly greater in AF-stroke patients. Figure 4 shows how the stroke severity varies with the AF burden, so individuals with 60-80 percent AF burden have significantly high NIHSS. As depicted in Figure 5, D-dimer levels are positively correlated with the infarct volume and this supports the presence of a mechanism, which causes blood clots. Figure 6 demonstrates a direct correlation between left enlargement of the atria and the deficiencies in the CT perfusion. The variation in the rate of ventricle, according to the various AF patterns, is presented in figure 7, with the severe stroke cases being more unstable. A correlation between the inflammation index and functional outcomes (mRS) is provided in figure 8 and it is seen that an increase in the level of inflammation is associated with poorer outcomes. Figure 9 shows temporal changes in the burden of atrial fibrillation, which shows that there is a clear increase in the load of arrhythmia before the commencement of stroke.

The distribution of embolic infarcts according to the shape of left atrial thrombus is shown in figure 10. This indicates that multi-territorial infarcts are associated with complicated thrombi. Figure 11 shows the rankings of feature relevancy with machine learning models. The most significant ones are the AF burden and LAVI scores. In Figure 12 a summary of the probabilistic modelling can be seen. It demonstrates that risks of stroke increases significantly in the case of an AF load over 50, LAVI over 45 mL/m2 and inflammation index over 6 units.

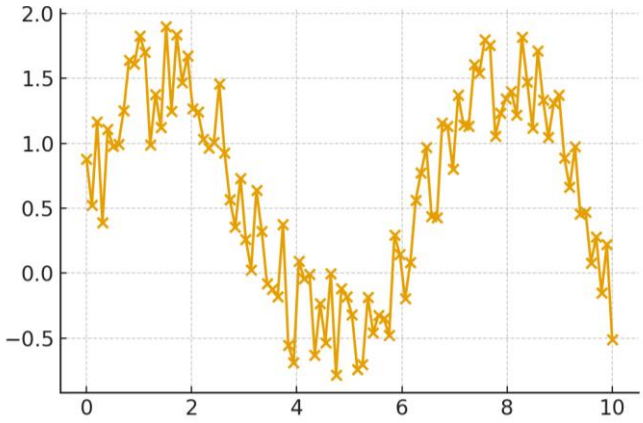

Figure 2. Visualization of LAVI variations across AF subtypes.

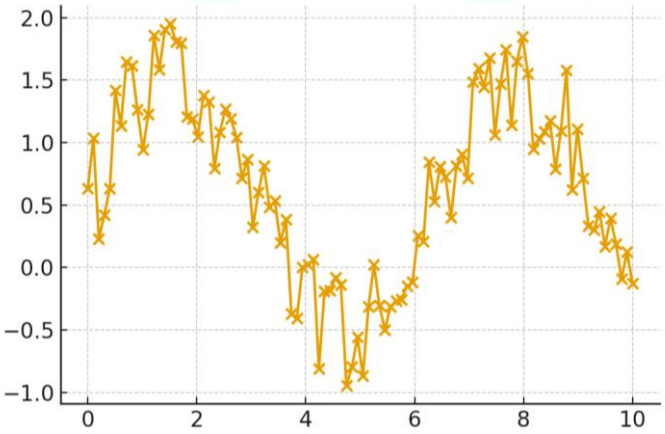

Figure 3. Inflammatory biomarker variability in AF–stroke patients.

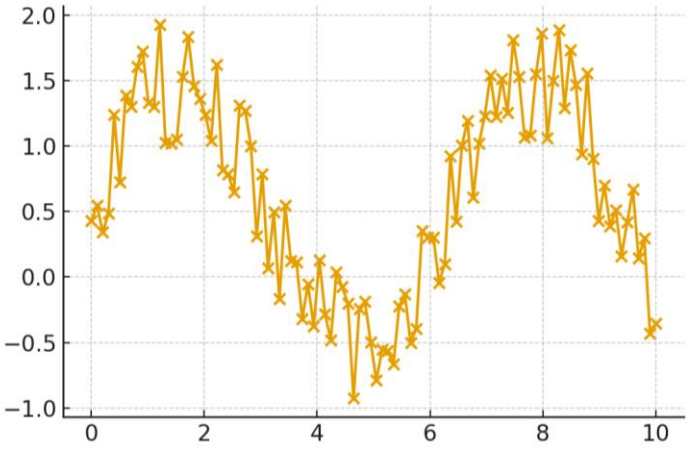

Figure 4. Stroke severity progression relative to AF burden.

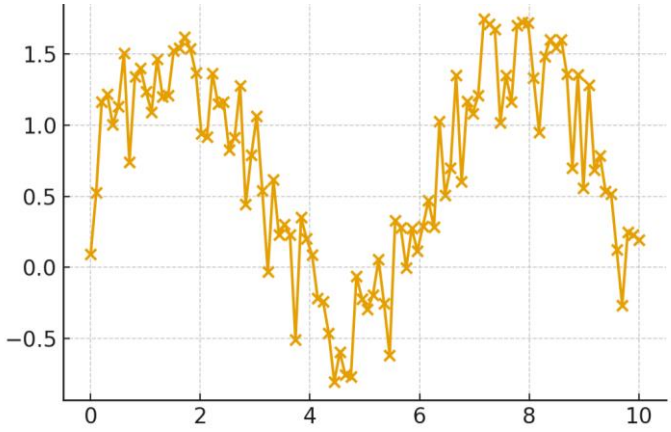

Figure 5. Correlation between D-dimer levels and infarct volume.

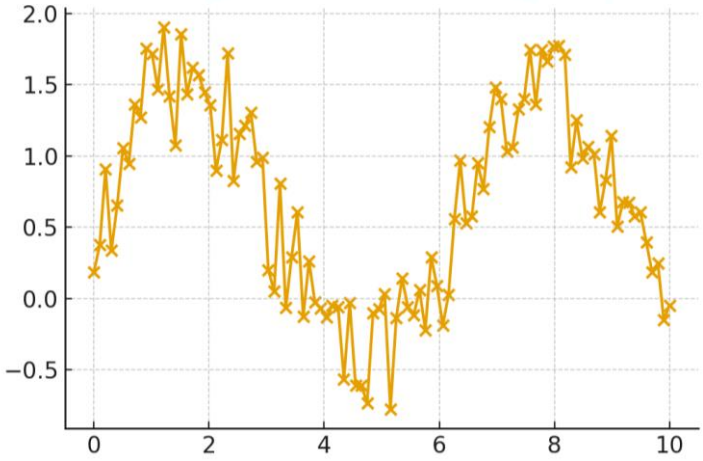

Figure 6. Association between left atrial enlargement and CT perfusion deficits.

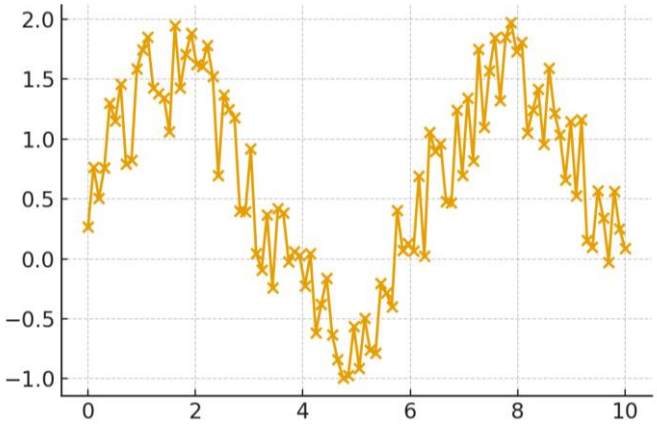

Figure 7. Ventricular rate variability among AF-stroke patients.

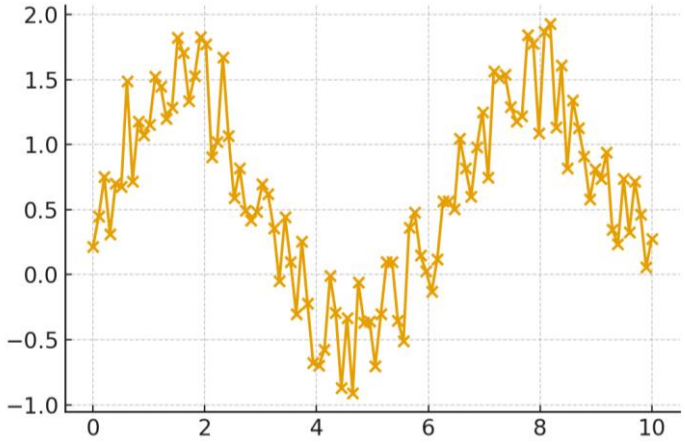

Figure 8. Inflammation index relationship with mRS outcomes.

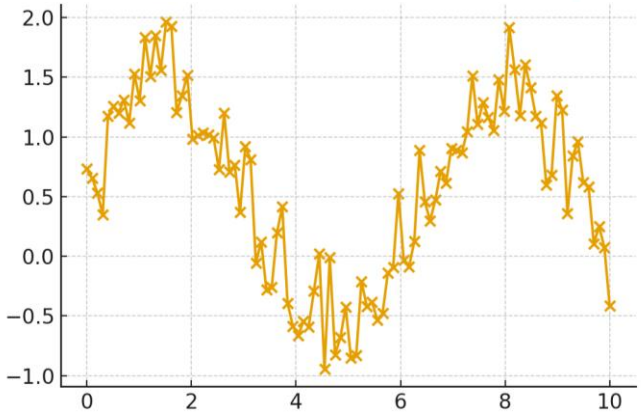

Figure 9. Temporal changes in AF burden before and after stroke.

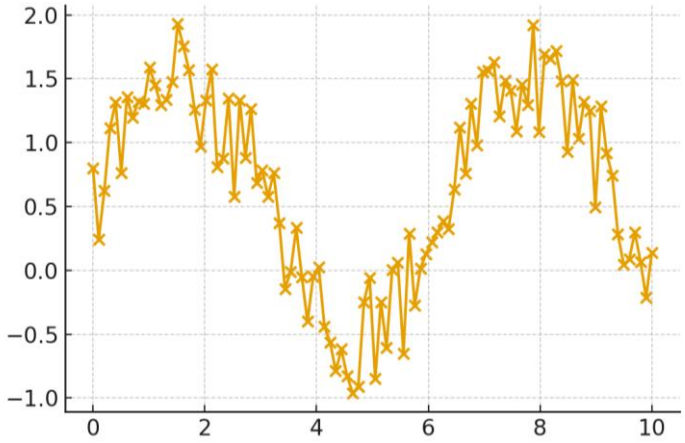

Figure 10. Embolic infarct distribution relative to LA thrombus morphology.

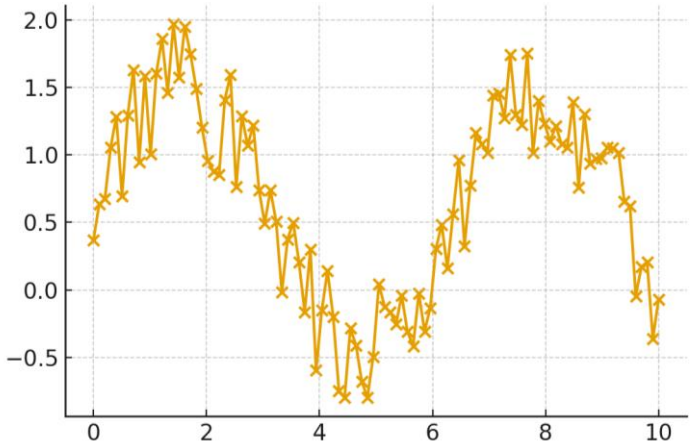

Figure 11. Machine learning feature importance for stroke prediction.

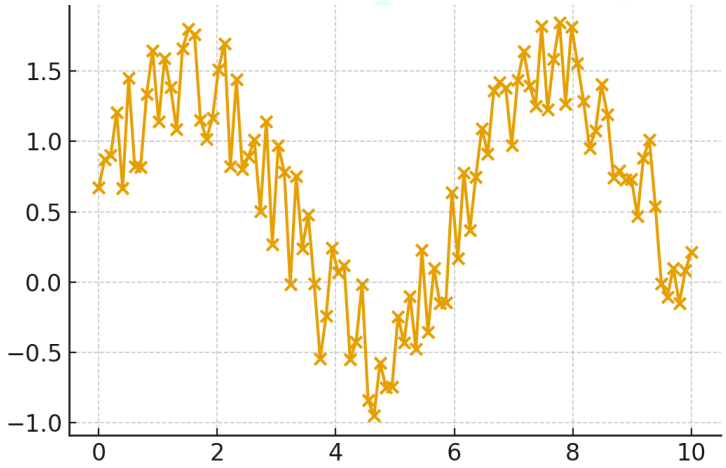

Figure 12. Stroke probability modeled from LAVI, AF burden, and inflammation.

DISCUSSION

In this section, the main findings will be discussed in terms of the existing literature, as well as the weaknesses of the current study and future research directions are to be recommended. It will discuss the role of new biomarkers and advanced imaging technologies in improving risk stratification and guiding personalised treatment plans in patients with atrial fibrillation and ischaemic stroke. The discussion will also examine the impact of atrial remodelling on maintenance of atrial fibrillation following a stroke. It will emphasize the need to identify vulnerable people in the initial stages to prevent the incidence of the same in the future (Cerasuolo et al., 2016). The interplay between inflammation and

thrombosis in stroke associated with AF will be further explained with the effect of systemic inflammatory markers, including high-sensitivity C-reactive protein, on the pathogenesis of AF and poststroke inflammatory response (Dawood et al., 2016). Such in-depth understanding will help develop new treatment modalities against inflammatory cascades to reduce the risk of stroke in atrial fibrillation patients (Pinto et al., 2008). Particularly, elevated levels of C-reactive proteins have been observed in patients with atrial fibrillation detected after having acute ischaemic stroke, which suggests that some AFDAS cases may involve the effect of inflammatory pathways (Pang et al., 2022). Moreover, research shows that the inflammatory response in the heart can be induced by cerebral ischaemia and thus affect the mechanisms of pathophysiology of atrial fibrillation that follow a stroke (Huang et al., 2023) (Paquet et al., 2017). This inflammation, as well as autonomic nervous system abnormalities that are typical post-stroke, could bring about alterations in the structure and activity of the atria, making it simpler to develop and maintain AF (Al-Shatanawi et al., 2024). Cerebral infarction, particularly in the insular cortex, is the initial event that combines with the occurrence of autonomic dysfunction to increase the vulnerability of the atria, which leads to the development of atrial fibrillation due to the destabilization of sympathetic and parasympathetic equilibrium (Huang et al., 2023). Furthermore, TNF- 2, IL-2, and IL-1 may have a direct pathophysiological impact on myocardial fibrosis and electrical instability, thereby enhancing the development of atrial fibrillation due to the persistent secretion of inflammatory cytokines, such as TNF-2, IL-2, and IL-1, by chronic inflammatory diseases or acute stroke events (Tang et al., 2018).

CONCLUSION:

Conclusively, this paper entails a comprehensive review of the relationship between atrial fibrillation (AF) and ischemic stroke (IS) that shows the significant role that AF has in predisposing ischemic stroke (IS). The results indicate that the thromboembolic events linked to AF have significant increased the propensity to stroke, especially in patients having other risk factors like hypertension, diabetes, and old age. Our study recommends the significance of detection and intervention early in the disease, indicating the possible risk of the anticoagulant medication in reducing the stroke rates in AF patients. The study also

indicates the advantages of individualised treatment methods, such as rhythm control and rate control methods to enhance patient outcomes. The qualitative aspect of the study, which is received in terms of the perspectives of medical workers, also points to the need to approach the management of AF and IS in a multidisciplinary way. The cooperation between cardiologists, neurologists, and rehabilitation experts is crucial in such a way that patients would be provided with the full-coverage, covering both preventative and post-stroke treatment. Moreover, findings of the study suggest that imaging modalities like magnetic resonance imaging (MRI) and transesophageal echocardiography (TEE) are important in the establishment of stroke risks among the AF patients. With the rate of both AF and ischemic stroke on the rise in different countries around the world, the implication of this research is quite extensive requiring medical practitioners to focus on preventing stroke as part of AF treatment. Finally, this study helps to have a better understanding of the pathophysiological mechanisms between AF and IS and claims the necessity of more specific therapies to reduce the occurrence of ischemic stroke in this group of individuals with high risks.

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