

BASEL Impaired heart rate variability triangular index to identify clinically silent strokes in patients with atrial fibrillation

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Background

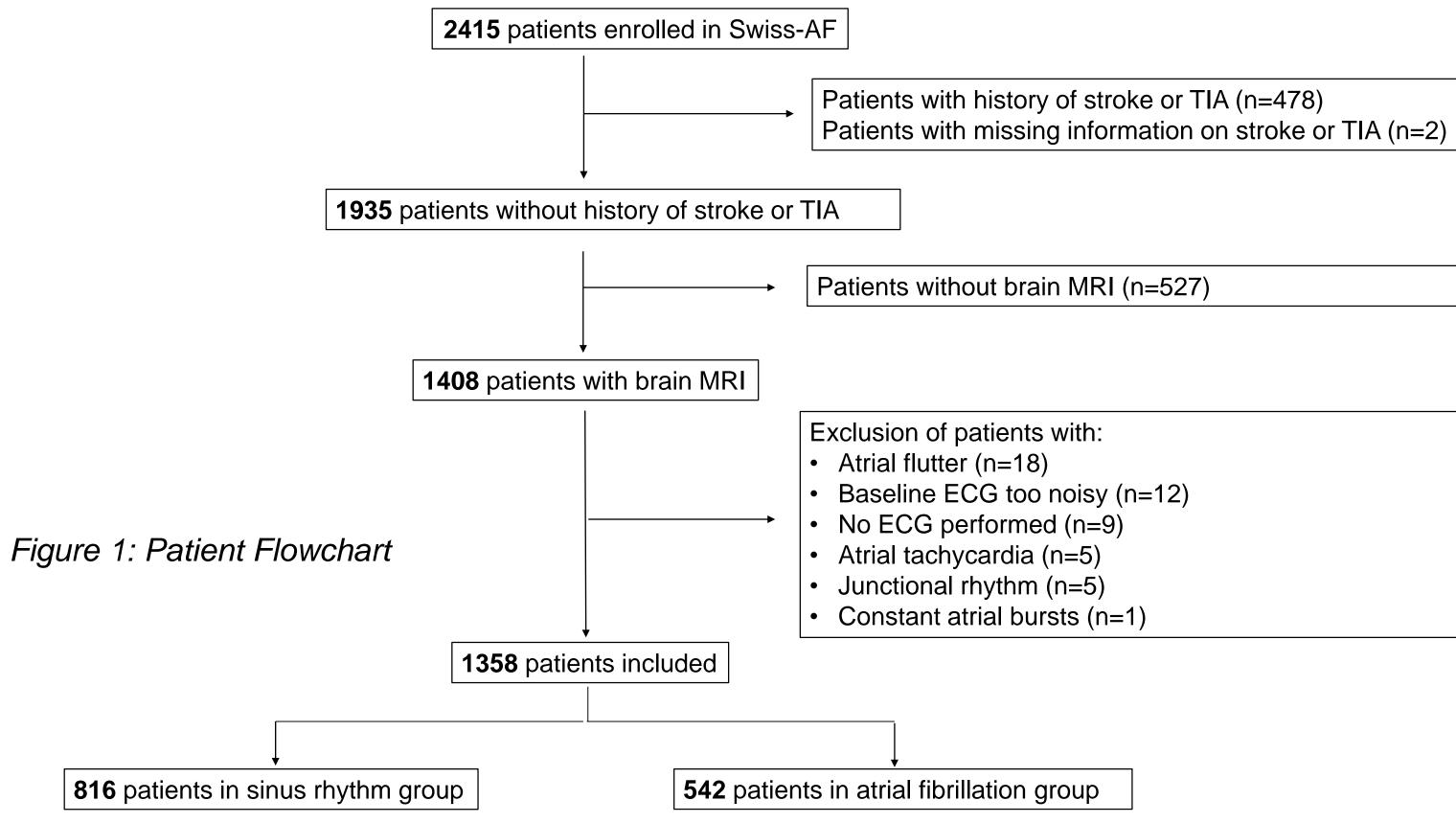
The identification of clinically silent brain infarcts in patients with atrial fibrillation (AF) is of high clinical relevance as they have been linked to cognitive impairment. Overt strokes have been associated with disturbances of the autonomic nervous system.

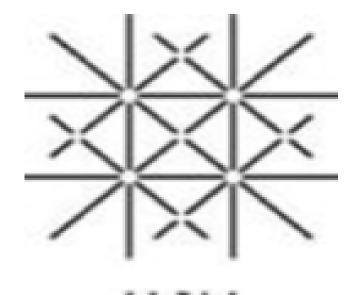
Aim

We hypothesize that impaired heart rate variability (HRV) can identify AF patients with clinically silent brain infarcts.

Methods

We enrolled 1358 patients (Figure 1) with AF without a history of stroke or transient ischemic attack from the multicenter Swiss-AF cohort study who were in sinus rhythm (SR-group, n=816) or AF (AF-group, n=542) on a 5 minute resting ECG recording. HRV triangular index (HRVI), the standard deviation of normal-to-normal intervals (SDNN) and the mean heart rate (MHR) were calculated¹. Brain MRI was performed at baseline to assess the presence of large non-cortical or cortical infarcts, which were considered to be silent brain infarcts. We constructed multivariable adjusted (age, sex, systolic blood pressure, history of hypertension, history of diabetes, history of heart failure, prior myocardial infarction, prior major bleeding, intake of oral anticoagulation, antiarrhythmics or betablockers) binary and linear logistic regression models to analyze the association between HRV parameters and silent brain infarcts.





Results

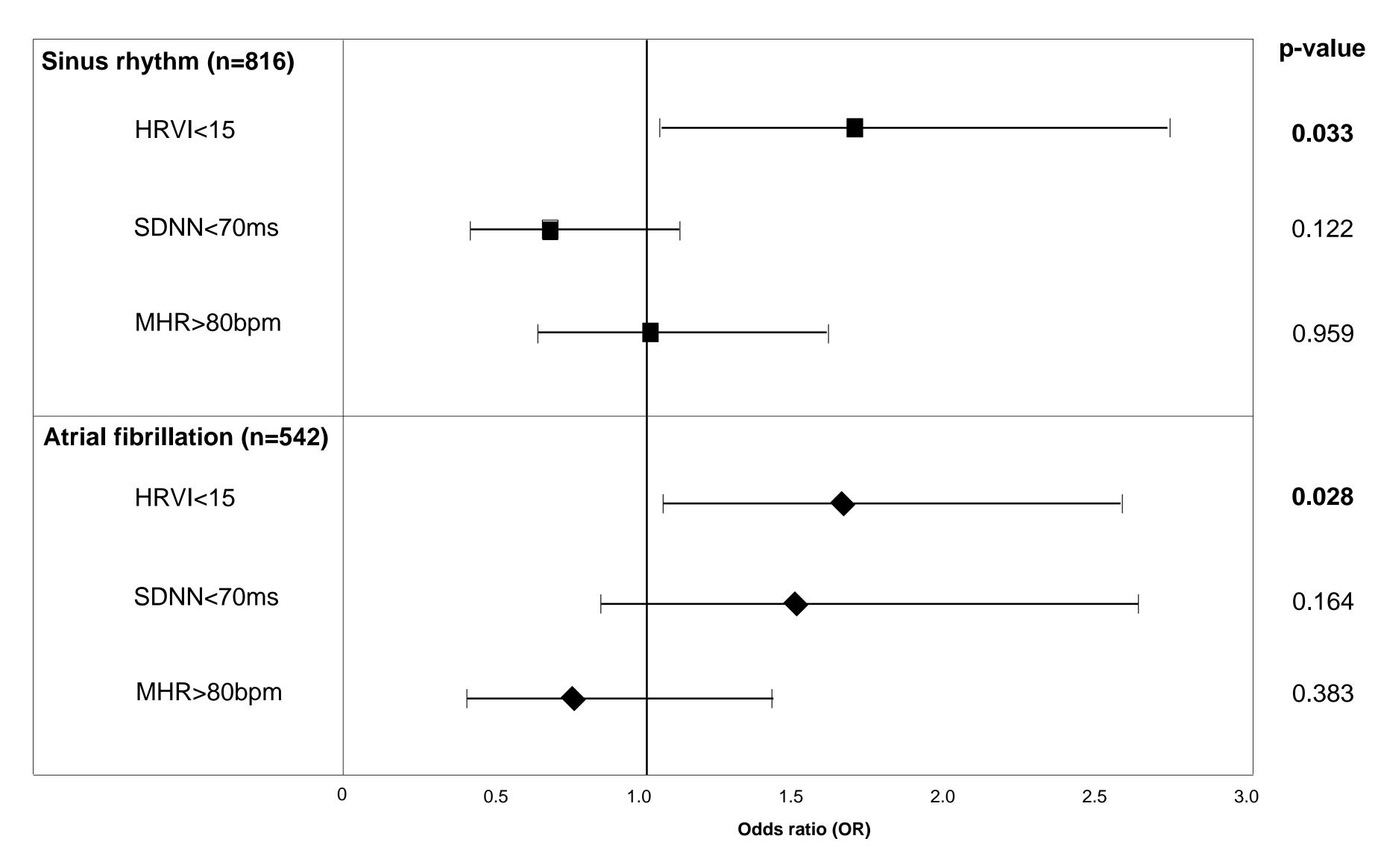
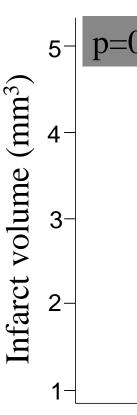


Figure 2: Association of parameters of heart rate variability with clinically silent brain infarcts when assessed in sinus rhythm and in atrial fibrillation. Data display multivariable adjusted binary logistic regression models. HRVI: heart rate variability triangular index; MHR: mean heart rate; SDNN: standard deviation of normal-to-normal intervals.

Furthermore, HRVI was associated with silent brain infarct volumes in the SR group (β (95%) CI) -0.30 (-0.87; -0.14), p=0.007, Figure 3 left panel), but not in the AF group (Figure 3 right panel). SDNN and MHR did not correlate with infarct volumes. Finally, right-hemispheric infarcts were associated with more pronounced impairment of HRVI in comparison to lefthemispheric infarcts (p=0.017).

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Mean age was 72 ± 9 years, 27% were female. Silent brain infarcts were detected in 10.5% in the SR group and 19.9% in the AF group (p<0.001). In the SR-group, HRVI <15 was the only parameter independently associated with the presence of silent brain infarcts (odds ratio (OR) 1.69; 95% confidence interval (CI): 1.04-2.72; p=0.033) in the multivariable model. Similarly, in the AF-group, HRVI<15 was independently associated with the presence of silent brain infarcts (OR 1.65, 95% CI: 1.05-2.57; p=0.028). SDNN<70ms and MHR>80bpm were not associated with silent brain infarcts, neither in the SR group, nor in the AF group (Figure 2).



HRVI was lower in right-hemispheric than left-hemispheric silent brain infarct (median 13.6 (IQR 11.3-16.2) vs 14.9 (12.1-20.0), p=0.022, Figure 4). SDNN and MHR did not differ between right- and left-hemispheric infarcts.

Conclusion

In conclusion, impairment of HRVI was independently associated with the presence and volume of clinically silent brain infarcts in a large cohort of patients with AF. Particularly, right-hemispheric brain infarcts caused disturbances of HRVI. Our data suggest that a short-term HRV analysis from routine ECG recordings provides valuable non-invasive information regarding the risk of silent brain infarcts in AF patients.

Funding

Lead University Hospital Basel

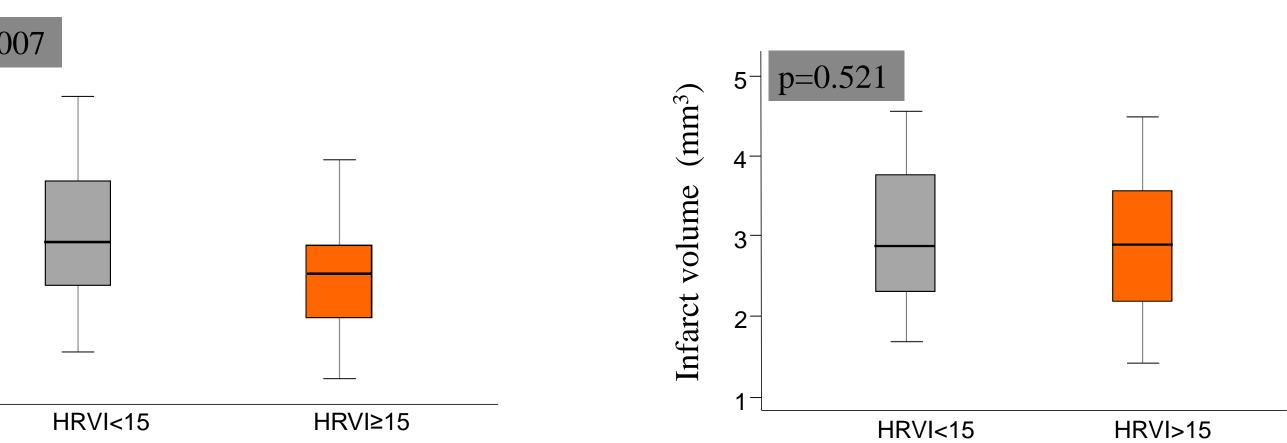


Figure 3: Heart rate variability triangular index (HRVI) and volumes of silent brain infarcts in the sinus rhythm group (left panel) and in the atrial fibrillation group (right panel). Volumes of infarcts were log-transformed.

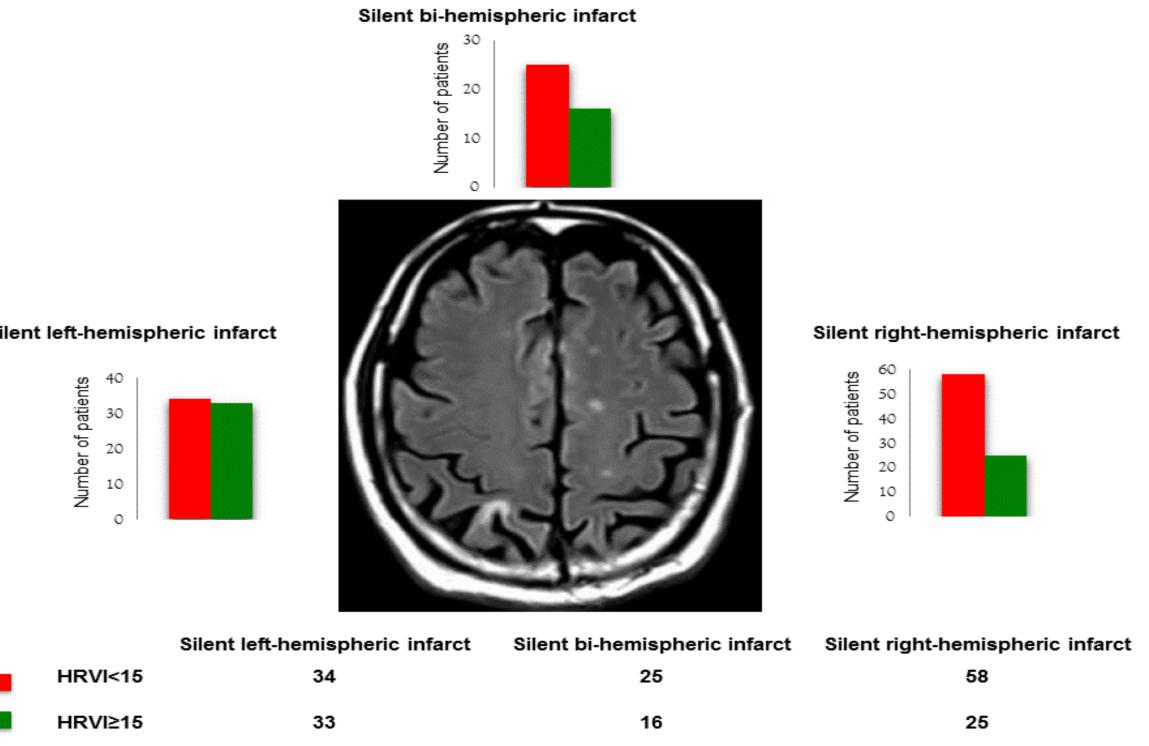


Figure 4: Performance of HRVI and topography of silent brain infarcts

