ORIGINAL RESEARCH

Incorporating Latent Variables Using Nonnegative Matrix Factorization Improves Risk Stratification in Brugada Syndrome

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BACKGROUND: A combination of clinical and electrocardiographic risk factors is used for risk stratification in Brugada syndrome. In this study, we tested the hypothesis that the incorporation of latent variables between variables using nonnegative matrix factorization can improve risk stratification compared with logistic regression.

METHODS AND RESULTS: This was a retrospective cohort study of patients presented with Brugada electrocardiographic patterns between 2000 and 2016 from Hong Kong, China. The primary outcome was spontaneous ventricular tachycardia/ventricular fibrillation. The external validation cohort included patients from 3 countries. A total of 149 patients with Brugada syndrome (84% males, median age of presentation 50 [38–61] years) were included. Compared with the nonarrhythmic group (n=117, 79%), the spontaneous ventricular tachycardia/ventricular fibrillation group (n=32, 21%) were more likely to suffer from syncope (69% versus 37%, \( P = 0.001 \)) and atrial fibrillation (16% versus 4%, \( P = 0.023 \)) as well as displayed longer QTc intervals (424 [399–449] versus 408 [386–425]; \( P = 0.020 \)). No difference in QRS interval was observed (108 [98–114] versus 102 [95–110], \( P = 0.104 \)). Logistic regression found that syncope (odds ratio, 3.79; 95% CI, 1.64–8.74; \( P = 0.002 \)), atrial fibrillation (odds ratio, 4.15; 95% CI, 1.12–15.36; \( P = 0.033 \)), QRS duration (odds ratio, 1.03; 95% CI, 1.002–1.06; \( P = 0.037 \)) and QTc interval (odds ratio, 1.02; 95% CI, 1.01–1.03; \( P = 0.009 \)) were significant predictors of spontaneous ventricular tachycardia/ventricular fibrillation. Increasing the number of latent variables of these electrocardiographic indices incorporated from n=0 (logistic regression) to n=6 by nonnegative matrix factorization improved the area under the curve of the receiving operating characteristics curve from 0.71 to 0.80. The model improves area under the curve of external validation cohort (n=227) from 0.64 to 0.71.

CONCLUSIONS: Nonnegative matrix factorization improves the predictive performance of arrhythmic outcomes by extracting latent features between different variables.

Key Words: Brugada syndrome ▪ depolarization ▪ ECG ▪ latent variable ▪ nonnegative matrix factorization ▪ repolarization ▪ risk stratification

Brugada syndrome (BrS) is an arrhythmogenic entity characterized by the high propensity of ventricular tachycardia/ventricular fibrillation (VT/VF) or sudden cardiac death (SCD).\(^1\)\(^-\)\(^3\) Subjects with spontaneous type 1 electrocardiographic (ECG) pattern and aborted SCD or syncope of arrhythmic origin are at the highest risk for future arrhythmic events and are advised to receive an implantable cardioverter-defibrillator.\(^4\) However, emerging evidence clearly underscores our inability to stratify patients with BrS,
Different risk stratification models incorporating clinical risk factors and ECG variables have been developed. However, they do not assess the interrelations between risk variables. In this study, we used nonnegative matrix factorization (NMF) to extract latent variables capturing the inherent interrelations among risk variables. NMF is a group of algorithms in multivariate analysis and linear algebra where a matrix $V$ is factorized usually into 2 matrices $W$ and $H$, with the property that all 3 matrices have no negative elements. This nonnegativity makes the resulting matrices easier to inspect. This enabled us to test the hypothesis that incorporation of latent variables can improve outcome prediction compared with logistic regression alone.

**METHODS**

Inclusion of Study Subjects

This retrospective study received ethics approval from The Joint Chinese University of Hong Kong – New Territories East Cluster Research Ethics Committee (NTEC-CUHK) and the Medical Ethical Review Committee of the Evangelismos General Hospital of Athens. Data on patients with BrS with spontaneous or drug-induced (ajmaline 1 mg/kg or flecainide 2 mg/kg) type 1 BrS ECG pattern from Hong Kong, China, were retrospectively analyzed. The analyses for our cohort (the training set) were validated against an external cohort (the validation set) of data on patients with BrS from Athens, Greece; Dalian, China; Guangzhou, China; and Osaka, Japan. The anonymized databases have been made available by the investigators at Zenodo: https://zenodo.org/record/3266179; https://zenodo.org/record/3465811.11–13

The ECG diagnosis of BrS was strictly based on the recommendations of the 2015 European Society of Cardiology guidelines for the management of patients with ventricular arrhythmias and the prevention of SCD.4 The presence of tructural heart disease was excluded in all subjects. The following demographic and clinical details were extracted from medical case records: age, sex, aborted SCD, syncopal symptoms, spontaneous VT/VF, and inducible VT/VF during programmed ventricular stimulation. Programmed right ventricular apex stimulation was performed at 3 running cycle lengths (600, 500, and 430 milliseconds) with up to triple extrastimuli (minimum coupled extrastimuli of 200 milliseconds). Inducible ventricular arrhythmia was defined as any ventricular arrhythmia (VT/VF) causing syncope/circulatory collapse or requiring intervention for its termination.

**Electrocardiographic Variables**

The 12-lead ECGs were recorded at a paper speed of 25 mm/s with an amplification of 10 mm/mV.
(sampling rate: 10 seconds of ECG at 500 Hz, filters: 0.5–100 Hz). The following automated measurements were extracted from the baseline ECG records: heart rate, PR interval, QRS duration reflecting total depolarization time (beginning of Q to the end of S), and QTc interval reflecting total repolarization time with and without correction for heart rate using Bazett’s formula.

**Statistical Analysis**

Data were expressed as median [interquartile range]. Differences between groups were tested using Kruskal-Wallis analysis of 1-way variance (ANOVA). The optimal cutoff values of the ECG variables, defined as the value maximizing the sum of sensitivity and specificity, for spontaneous VT/VF, were determined using the Youden index from receiver operating characteristic curve analysis. A \( P \) value <0.05 was considered statistically significant. Their predictive values were represented by the area under the curve (AUC) values from receiver operating characteristic analyses. Logistic regression was performed to determine the predictive value of different variables for spontaneous VT/VF. The results were presented as odds ratio (OR) with 95% CIs, with \( P \) values from receiver operating characteristic analysis. A \( P \) value <0.05 was considered statistically significant.

**Nonnegative Matrix Factorization**

An NMF approach was used to capture inherent inter-relations among risk variables. We then used the latent variables to develop a machine learning model to enhance the performance in predicting spontaneous ventricular tachycardia/ventricular fibrillation. First, we constructed matrix \( V \) representing the inter-relations between clinical variables (initial type 1 pattern, syncope, atrial fibrillation [AF]) and ECG variables (QRS duration, QTc interval). Second, nonnegative matrix factorization was used to decompose matrix \( V \) into a core matrix \( W \) multiplied by a matrix \( H \) with different component cases (ie, number of latent variables generated). The generated latent variables were then combined with the risk variables as the input for logistic regression.

**RESULTS**

**Clinical Characteristics**

This study cohort consisted of 149 patients with BrS from Hong Kong, China. The baseline demographic and clinical characteristics are presented in Table 1. The mean age was 50 (38–61) years old and 84% of the subjects were male. Syncope occurred in 65 (44%) and spontaneous VT/VF occurred in 32 (21%) patients. Atrial fibrillation was found in 10 (7%) patients. An initial spontaneous type 1 ECG pattern was recorded in 79 patients (53%). An implantable cardioverter-defibrillator was inserted in 47 (32%) subjects. In the cohort, 44 (30%) underwent electrophysiological studies and 28 (64%) were positive tests.

**Significant Predictors of Spontaneous VT/VF Using Logistic Regression**

Compared with the nonarrhythmic group (n=117, 79%), the spontaneous VT/VF group (n=32, 21%) were more likely to suffer from syncope (69% versus 37%, \( P=0.001 \)) and AF (16% versus 4%, \( P=0.023 \)), as well as displayed longer QTc intervals (424 [399–449] versus 408 [386–425]; \( P=0.020 \)). No difference in QRS interval was observed (108 [98–114] versus 102 [95–110], \( P=0.104 \)). The results of logistic regression are shown in in Table 2. Age, sex, or initial spontaneous type 1 Brugada pattern did not significantly predict spontaneous VT/VF. By contrast, syncope (OR, 3.79; 95% CI, 1.64–8.74; \( P=0.002 \)) and AF (OR, 4.15; 95% CI, 1.12–15.36; \( P=0.033 \)) were significant predictors of spontaneous VT/VF. The receiver operating characteristic curves for the different variables described previously with the AUC values are shown in Figure S1A to D and Table 3.

**Table 1. Demographic and Clinical Characteristics of Patients with Brugada Syndrome From Hong Kong Included in this Study (n=149)**

| Characteristics | Spontaneous VT/VF Group (n=32) | No Spontaneous VT/VF (n=117) | \( P \) Value |
|-----------------|-------------------------------|-------------------------------|----------------|
| Male            | 30 (94)                       | 95 (81)                       | 0.087          |
| Age at presentation (y)* | 49 (35–68)                  | 50 (39–59)                   | 0.857          |
| Implantable cardioverter-defibrillator insertion | 24 (75)           | 23 (20)                       | <0.0001*       |
| Syncope         | 22 (69)                       | 43 (37)                       | 0.001*         |
| Initial spontaneous type 1 pattern | 21 (66)           | 58 (50)                       | 0.107          |
| Atrial fibrillation | 5 (16)                       | 5 (4)                         | 0.023*         |
| PVS performed   | 13 (41)                       | 31 (26)                       | 0.121          |
| Positive PVS test‡ | 10 (77)                       | 18 (58)                       | 0.235          |
| QTc (ms)        | 422 (399–449)                 | 408 (386–425)                | 0.020*         |

‡For PVS: this was carried out in 5 subjects in the high-risk group and 104 subjects in the low-risk group.
Next, we constructed a variable matrix and then generated latent variables by performing NMF on the variable matrix to predict spontaneous VT/VF. The total number of components is denoted $d$, which indicates the number of extracted latent variables. We then combined the $d$ additional latent variables and the 4 ECG variables in the 4-point score system and then use them to fit a logistic regression. The regression performance under different combination cases of $d$ latent variables are given in Table 4 and were compared with the performance with the baseline model (ie, logistic regression without latent variables). The AUC and 3 common metrics, precision, recall and F1 score, were reported to evaluate the performance. A 2-fold cross-validation was adopted to avoid overfitting concerns.

Using logistic regression as a baseline model, the AUC was 0.7101 (Table 4, left side). However, incorporating $d=2, 3, 4, 5, \text{ and } 6$ additional latent variables by NMF led to improvement in the AUC values to 0.72, 0.73, 0.80, 0.79, and 0.73, respectively. Therefore, NMF improved the prediction performance over the baseline model using simple logistic regression with latent variables. The model achieved the best performance with $d=4$ latent variables. Our models were also validated using an external cohort from Athens, Greece; Dalian, China; Guangzhou, China; and Osaka, Japan (Table 4, right side). For the external cohort, the AUC was 0.64 using logistic regression, which was improved by NMF to 0.68, 0.68, 0.70, 0.71, and 0.69, through incorporating $d=2, 3, 4, 5, \text{ and } 6$ additional latent variables, respectively. The model achieved the best performance with $d=5$ latent variables for external cohort validation.

### DISCUSSION

The main findings of this study are that:

1. Syncope, AF, QRS duration, and QTc interval were significant predictors of spontaneous VT/VF;
2. Incorporation of information on inherent higher order interrelations among variables improved prediction of VT/VF.

The genesis of BrS remains controversial. Both depolarization (conduction delay within the right ventricular outflow tract, prolonged and fractionated potentials) and repolarization (imbalance of epicardial/endocardial repolarizing currents) abnormalities have been suggested to play crucial roles in the arrhythmogenesis of BrS. Previous studies have addressed the prognostic significance of specific depolarization and repolarization ECG markers. A prolonged QRS duration has been associated with an increased risk for arrhythmic events. For depolarization markers, QRS fragmentation has been associated with a 3.9-fold increase in the risk of future arrhythmic events and a worse prognosis in BrS. The presence of a wide and/or large S-wave in lead I has been suggested as a powerful predictor of ventricular arrhythmias. The presence of a wide S-wave in lead I is possibly related to the delayed activation in the right ventricular outflow tract. Among the repolarization ECG markers, a prolonged QTc interval, which reflects delayed cellular repolarization, has been associated with an increased risk for VT/VF and SCD in BrS. Other significant ECG predictors include the presence

### Table 2. Univariate Logistic Regression for Spontaneous Ventricular Tachycardia/Ventricular Fibrillation

| Parameter                | Odds Ratio (T) (95% CI) | P Value | Odds Ratio (V) (95% CI) | P Value |
|--------------------------|------------------------|---------|------------------------|---------|
| Age at presentation (y)  | 1.00 (0.98–1.03)       | 0.918   | 1.00 (0.96–1.03)       | 0.822   |
| Sex                      | 0.29 (0.06–1.30)       | 0.105   | 1.71 (0.53–5.54)       | 0.388   |
| Syncope                  | 3.79 (1.64–8.74)       | 0.002*  | 9.09 (3.33–24.80)      | <0.0001*|
| Initial spontaneous type 1 pattern | 1.94 (0.86–4.39)   | 0.110   | 1.20 (0.47–3.07)       | 0.704   |
| Atrial fibrillation      | 4.15 (1.12–15.36)      | 0.033*  | 2.50 (0.83–7.52)       | 0.103   |
| Positive programmed ventricular stimulation test | 2.41 (0.65–10.52) | 0.243   | 0.27 (0.13–5.77)       | 0.404   |
| QRS                      | 1.03 (1.002–1.06)      | 0.037*  | 1.03 (1.00–1.08)       | 0.051   |
| QTc                      | 1.02 (1.004–1.03)      | 0.009*  | 1.02 (0.99–1.04)       | 0.150   |

*P<0.05.

### Table 3. AUC of Continuous Variables for Predicting Spontaneous Ventricular Tachycardia/Ventricular Fibrillation

| Parameter                | Cutoff (95% CI) | Cutoff (95% CI) |
|--------------------------|----------------|----------------|
| Age at presentation (y)  | 63.5 (51.9–78.9) | 43.5 (28.3–58.7) |
| QRS                      | 107.5 (106.7–108.3) | 110.5 (109.7–111.2) |
| QTc                      | 433.5 (432.8–434.2) | 409.5 (408.7–410.3) |

left: training cohort (T), right: validation cohort (V). AUC indicates area under the curve.
of an early repolarization pattern, a low-voltage type 1 ECG, AF and syncope are observed in patients with BrS and are predictive of spontaneous VT/VF, as also seen in our study.

In this study, we used a method called NMF to extract latent variables and utilize this information for risk stratification. This led to significant improvements in the regression performance over the benchmark logistic regression-based model. Only several groups have applied the NMF technique to ECG signal analysis. First, Yao et al reported that sparse constrained NMF improved classification of ECGs between the following diseases of right bundle branch block, left bundle branch block, premature ventricular contractions, paced rhythms, and patients without these abnormalities, using data from the Massachusetts Institute of Technology-Beth Israel Hospital database. Second, Guyot et al used NMF for the preprocessing of long-term ECGs, demonstrating that this simultaneously performed 3 tasks of denoising, baseline wander removal, and peak R detection. Here, we extend NMF analyses for the first time in patients with BrS, demonstrating that it can be used to improve classification accuracy of novel electrocardiographic depolarization-repolarization indices between spontaneous VT/VF and nonarrhythmic groups.

Limitations

Several limitations of this study should be recognized. First, this is a retrospective study and is therefore susceptible to bias inherent in this type of studies. Second, we fully recognize that the proposed electrocardiographic indices were measured at baseline and do not reflect the full dynamic nature of the electrophysiological changes in BrS. Indeed, improvement in risk stratification can be achieved with markers such as restitution indices or markers measured during stress or by assessing temporal variability of different ECG markers. Finally, the sample size was moderate with 376 patients, but our findings need to be further validated in larger prospective studies.

Table 4. Incorporation of Latent Variables and Its Effects on the AUC for Spontaneous Ventricular Tachycardia/Ventricular Fibrillation

| # of Latent Variables (T) | Precision | Recall | F1   | AUC    | # of Latent Variables (V) | Precision | Recall | F1   | AUC    |
|--------------------------|-----------|--------|------|-------|--------------------------|-----------|--------|------|-------|
| Benchmark using logistic regression (d=0) | 0.6901 | 0.7050 | 0.6975 | 0.7101 | Benchmark using logistic regression (d=0) | 0.5983 | 0.6131 | 0.6056 | 0.6383 |
| d=2 | 0.7117 | 0.7265 | 0.7190 | 0.7187 | d=2 | 0.6567 | 0.6552 | 0.6559 | 0.6759 |
| d=3 | 0.7278 | 0.7341 | 0.7309 | 0.7269 | d=3 | 0.6984 | 0.6567 | 0.6679 | 0.6809 |
| d=4* | 0.7749* | 0.7828* | 0.7788* | 0.7065* | d=4 | 0.7048 | 0.6899 | 0.6973 | 0.6993 |
| d=5 | 0.7719 | 0.7746 | 0.7732 | 0.7892 | d=5 | 0.7139* | 0.6960* | 0.7048* | 0.7092* |
| d=6 | 0.7806 | 0.7181 | 0.7480 | 0.7319 | d=6 | 0.7123 | 0.6738 | 0.6925 | 0.6856 |

left: training cohort (T), right: validation cohort (V). AUC indicates area under the curve.

*P<0.05.

CONCLUSIONS

The present findings suggest that nonnegative matrix factorization improves the predictive performance by extracting latent features between different variables.

ARTICLE INFORMATION

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Disclosures

None.

Supplementary Material

Figure S1

REFERENCES

1. Antzelevitch C, Yan GX, Ackerman MJ, Borggreve M, Corrado D, Guo J, Gussak I, Hasdemir C, Horie M, Huijiri H, et al. J-wave syndromes expert consensus conference report: emerging concepts and gaps in knowledge. Europace. 2017;19:666–694.
2. Antzelevitch C, Brugada P, Borggreve M, Brugada J, Brugada R, Corrado D, Gussak I, LeMarec H, Nademaneke K, Perez Riera AR, et al. Brugada syndrome: report of the second consensus conference: endorsed by the heart rhythm society and the European Heart Rhythm Association. Circulation. 2005;111:659–670.
3. Brugada P, Brugada J. Right bundle branch block, persistent ST segment elevation and sudden cardiac death: a distinct clinical and electrophysiologic syndrome. A multicenter report. J Am Coll Cardiol. 1992;20:1391–1396.
4. Priori SG, Blomstrom-Lundqvist C, Mazzanti A, Blom N, Borggreve M, Camm J, Elliott PM, Fitzsimons D, Hatala R, Hindricks G, et al. 2015
ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death: the task force for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death of the European Society of Cardiology (ESC). Endorsed by: Association for European Paediatric and Congenital Cardiology (AEPCC). Eur Heart J. 2015;36:2793–2867.

5. Raju H, Papadakis M, Govindan M, Bastiaenen R, Chandra N, O’Sullivan A, Baines G, Sharma S, Behr ER. Low prevalence of risk markers in cases of sudden death due to Brugada syndrome relevance to risk stratification in Brugada syndrome. J Am Coll Cardiol. 2011;57:2340–2345.

6. Milman A, Andorin A, Gourraud JB, Postema PG, Sacher F, Mabo P, Kim SH, Juang JMJ, Maeda S, Takahashi Y, et al. Profile of patients with Brugada syndrome presenting with their first documented arrhythmic event: data from the survey on arrhythmic events in Brugada syndrome (SABRUS). Heart Rhythm. 2018;15:716–724.

7. Kelly A, Salerno S, Connolly A, Bishop M, Charpentier F, Stolen T, Smith GL. Normal interventricular differences in tissue architecture underlie right ventricular susceptibility to conduction abnormalities in a mouse model of Brugada syndrome. Cardiovasc Res. 2018;114:724–736.

8. Nademanee K, Raju H, de Noronha SV, Papadakis M, Robinson L, Rotthier S, Makita N, Kowase S, Boonmee N, Vitayakrutskirul V, et al. Fibrosis, Connexin-43, and conduction abnormalities in the Brugada syndrome. J Am Coll Cardiol. 2015;66:1976–1986.

9. Antzelevitch C. The Brugada syndrome: ionic basis and arrhythmia mechanisms. J Cardiovasc Electrophysiol. 2001;12:268–272.

10. Meregalli PG, Wilde AAM, Tan HL. Pathophysiological mechanisms of brugada syndrome: depolarization disorder, repolarization disorder, or more? Cardiovasc Res. 2005;67:367–378.

11. Tse G, Li CKH, Lee S, Leung K, Yin C. Brugada clinical database. 2019.

12. Tse G, Li CKH, Lee S, Leung K, Yin C, Li A. Brugada ECG analysis. 2019.

13. Tse G, Li CKH, Lee S, Yin C, Li A. Brugada ECG database. 2019.

14. Tokioka K, Kusano KF, Morita H, Miura D, Nishii N, Nagase S, Nakamura K, Kusano KF, Fujimoto Y, Hisamatsu K, Fuji H, Harakoa K, Kobayashi M, Morita ST, Nakamura K, et al. Atrial fibrillation and atrial vulnerability in patients with Brugada syndrome. J Am Coll Cardiol. 2002;40:1437–1444.

15. Junttila MJ, Brugada P, Hong K, Lizotte E, De Zutter M, Sarkozy A, Fujio H, Haraoka K, Kobayashi M, Saitoh Y, Julia J, Mguial G, et al. A score model to predict risk of events in patients with Brugada syndrome. Eur Heart J. 2017;38:1756–1763.

16. Mok NS, Zhang Q, et al. Outcomes in Brugada syndrome patients with implantable cardioverter-defibrillators: insights from the SGLT2 Registry. Front Physiol. 2020;11:204.

17. Leung J, Carlino G, de Ruvo E, Guerra F, et al. A new electrocardiographic cardiac death risk in ischaemic cardiomyopathy. J Am Heart Assoc. 2012;1:e001552. DOI: 10.1161/JAHA.112.001552.

18. Nicolson WB, McCann GP, Brown PD, Sandilands AJ, Stafford PJ, Schlindwein FS, Samani NJ, Ng GA. A novel surface electrocardiogram-based marker of ventricular arrhythmia risk in patients with ischemic cardiomyopathy. J Am Heart Assoc. 2013;2:e001552. DOI: 10.1161/JAHA.112.001552.

19. Leung KMW, Ng FS, Roney C, Cantwell C, Shun-Shin MJ, Linton NWF, Whinnett ZL, Leefroy DC, Davies DW, Harding SE, et al. Repolarization abnormalities unmasked with exercise in sudden cardiac death survivors with structurally normal hearts. J Cardiovasc Electrophysiol. 2018;29:115–126.

20. Lee S, Zhou J, Liu T, Letsas KP, Hothers SS, Vassiliou VS, Li G, Baranchuk A, Chang D, Zhang Q, et al. Temporal variability in electrocardiographic indices in subjects with Brugada patterns. Frontiers in Physiology. 2020;11. https://doi.org/10.3389/fphys.2020.00953.
SUPPLEMENTAL MATERIAL
Figure S1. Receiver operating characteristic curves for syncope (A), atrial fibrillation (B), QTc interval (C) and QRS duration (D).