REVIEW

Idiopathic short-coupled ventricular tachyarrhythmias: Systematic review and validation of electrocardiographic indices

Mohammed Almehairi, Alawi A. Alshiekh-Ali, Ahmed Alfagih

Cardiac Centre, Institute of Cardiac Sciences, Sheikh Khalifa Medical City, Abu Dhabi, United Arab Emirates
Prince Sultan Cardiac Centre, Military hospital, Riyadh, Saudi Arabia

Article info

Article history:
Received 4 October 2017
Accepted 6 June 2018
Available online 22 June 2018

Keywords:
Ventricular tachyarrhythmia
Purkinje fibres
Reentry
Delayed afterdepolarization
Electrocardiography
Sudden cardiac death

Abstract

Introduction: Idiopathic short-coupled ventricular tachyarrhythmias make up a considerable proportion of ventricular tachyarrhythmias in structurally normal hearts and are the cause of 5–10% of unexpected sudden cardiac deaths. There is disparity in the literature regarding their description and a lack of formal diagnostic criteria to define them.

Objective: To validate ECG indices for the diagnosis of these ventricular tachyarrhythmias and to subsequently unify their differing descriptions in the literature under a new terminology: Idiopathic Short-Coupled Ventricular Tachyarrhythmias.

Methods: We conducted a systematic review of all published studies describing short-coupled torsades de pointes, idiopathic ventricular fibrillation and polymorphic ventricular tachycardia. Published tracings were analysed using a standard set of criteria to define the different ECG intervals. Previously proposed diagnostic indices were validated using a control group of previously published long-coupled torsades de pointes cases.

Results: Validation of the ECG indices revealed that a coupling interval < 400 ms was the most reliable measurement (sensitivity 100%, specificity 97%), followed by a coupling interval/QT < 1 (sensitivity 96%, specificity 100%).

Conclusion: Idiopathic short-coupled ventricular tachyarrhythmias encompass all previous descriptions of this tachyarrhythmia including idiopathic ventricular fibrillation, short-coupled torsades de pointes, Purkinje-related torsades de pointes and idiopathic polymorphic ventricular tachycardia. This arrhythmia can be diagnosed by newly proposed criteria with high sensitivity and specificity.

© 2018 Egyptian Society of Cardiology. Production and hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

Abbreviations: ECG, electrocardiography; CI, coupling interval; CA, cardiac arrest; ISCVT, idiopathic short-coupled ventricular tachyarrhythmia; IVF, idiopathic ventricular fibrillation; SCTDP, short-coupled torsades de pointes; SCD, sudden cardiac death; LCTDP, long-coupled torsades de pointes; MESH, medical subject headings; PMT, polymorphous ventricular tachycardia; Pr-TDP, Purkinje related torsades de pointes; Pal/Syn, palpitations/syncope; PVC, Premature Ventricular Contraction; RVOT, right ventricular outflow tachycardia; TDP, torsades de pointes; Tasc, ascending limb of the T wave; Tdesc, descending limb of the T wave; VF, ventricular fibrillation.

Peer review under responsibility of Egyptian Society of Cardiology.

* Corresponding author at: Medicine and Cardiology, Cardiac Electrophysiology and Pacing, Sheikh Khalifa Medical City, UAE University, United Arab Emirates.

E-mail address: Malmehairi@seha.ae (M. Almehairi).

https://doi.org/10.1016/j.ehj.2018.06.003
1110-2608/© 2018 Egyptian Society of Cardiology. Production and hosting by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).
1. Introduction

Idiopathic ventricular tachyarrhythmias represent a significant proportion (5–10%) of all ventricular arrhythmias. A growing body of evidence suggests that Purkinje fibres play a pivotal role in these idiopathic forms, but there is no agreement on the mechanism of this rare type. In the last two decades, different eponyms of these arrhythmias have been used which may or may not include suggestions as to the underlying mechanisms involved in their generation. These include idiopathic ventricular fibrillation (IVF), Purkinje-related torsades de pointes (Pr-TDP), short-coupled torsades de pointes (SCTDP) and polymorphous ventricular tachycardia (PMT) in structurally normal heart. These different terms may indeed be referring to the same entity.

We systematically reviewed the literature to identify reports of these arrhythmias, validate ECG algorithms for their diagnosis, and propose a unifying terminology.

2. Material and methods

We searched Medline and Pre-Medline (PubMed) and Embase (Ovid) versions from 1992 to present (last update December 2015) to identify all observational studies, review articles and case reports describing idiopathic forms of ventricular arrhythmia. Mesh terms used in the search were: “ventricular arrhythmia”, “short coupled”, “torsades de pointes”. After our initial search identified several reports describing short-coupled torsades de pointes but labelling them as PMT or IVF, we expanded our search to include the term IVF. We further reviewed reference lists within articles to complement our electronic searches.

Our main objective was to identify ECG features that may be common to the different cases of IVF described in the literature. To serve as a control to the ECG features of IVF, we also performed a limited search using PubMed and included 12 case reports each of acquired and congenital LQT syndrome with TDP episodes. The purpose of the latter was to validate previously published ECG indices used to discern “what we described as ISCVT in this study” from torsade de points that shares similar polymorphic pattern. We restricted the search to papers published in English. The search strategies are available on request.

2.1. Study selection

We selected articles using two reviewers (MA, AA). The full text manuscripts and the extracted data were reviewed carefully to ensure that all discussing the same arrhythmia. The quality of the published ECGs and/or rhythm strips was also reviewed to ensure accurate measurements of the intervals. Disagreement was solved by consensus. Published 12 lead ECGs or rhythm strips were reviewed and relevant intervals were measured by one reviewer using an electronic caliper, blinded to the study’s published measurements (Figs. 1 and 2). ECGs or rhythm strips without clear scale standards or a well-defined QT interval of a normally conducted ventricular beat were excluded from the analysis.

2.2. Data extraction

Data were extracted in duplicate, including the number and characteristics of patients (age, gender, presence of structural heart disease or other co-morbidities). History of associated syncope or resuscitated cardiac arrest was also extracted. The calculation of the electrocardiographic indices was performed on the published rhythm strips/ECGs in each article. All extracted and calculated indices were plotted in a constructed two by two contingency table. Indices from the acquired and congenital Long QT tachyarrhythmia were used as a control group to validate ECG diagnostic indices of idiopathic forms (Table 2).

2.3. ECG parameters and definitions

The following parameters were measured:

1. Coupling interval (CI): CI was defined as the interval measured from the initial deflection of the preceding QRS to the initial deflection of the coupled premature ventricular complex (PVC), Fig. 1.

2. CI/QT ratio was defined as the ratio of the PVC CI and the QT interval of the preceding normally conducted ventricular beat, Fig. 1.

Tangent angle technique was utilised to determine the exact terminal point of the T wave. No standard ECG lead was

Fig. 1. ISCVT episode with calculation method of electrocardiographic indices. Shiga et al.8, ISCVT = idiopathic short coupled ventricular tachyarrhythmia.
specifically used to perform the measurements due to the variation in the published ECGs/tracings. If more than one lead was available for measurement, we used the one with clearest tracing. A CI/QT ratio of <1 was used to define "short-coupling" PVC.7

### 2.4. Statistical analysis

Data were entered into an Excel spreadsheet and imported into IBM SPSS (Version 21.0 for Windows, Armonk, New York) for statistical analysis. Standard descriptive statistics were used to summarize continuous data with means/standard deviation and categorical data with frequencies and percentages. Optimal cut-points for CI/QT and CI, for both the studied cohort and the control group of LQT, were identified using ROC Curves. Sensitivity and specificity for the CI/QT ratio and CI thresholds were calculated. Chi-Square test was used to calculate the P value of 2

### 3. Results

Our search strategy identified 42 articles that were further reviewed for reported cases of IVF, SCTDP, PMT and Pr-TDP. A total of 32 articles described single cases with the inclusion of relevant clinical details and measurable intervals on the surface ECG. The other 10 articles were excluded due to poor quality of the published ECGs or rhythm strips. A detailed listing of the clinical and electrocardiographic characteristics of the studied cohort is shown in Table 1. Most patients were male (n = 17), and approximately half of them were 40 years of age or younger (n = 17). All patients except two8,9 had documented normal cardiac structure by echocardiography. In the two papers8,9 where the authors did not discuss work up details, they settled for describing the arrhythmia as being "idiopathic". In accordance to that we assumed that there was no evident structural heart disease in those subjects and therefore were considered in the study. Family history of SCD was documented in only one patient. No single case was described

### Data were entered into an Excel spreadsheet and imported into IBM SPSS (Version 21.0 for Windows, Armonk, New York) for statistical analysis. Standard descriptive statistics were used to summarize continuous data with means/standard deviation and categorical data with frequencies and percentages. Optimal cut-points for CI/QT and CI, for both the studied cohort and the control group of LQT, were identified using ROC Curves. Sensitivity and specificity for the CI/QT ratio and CI thresholds were calculated. Chi-Square test was used to calculate the P value of 2

### Table 1

| Article                  | Sex | Age | Presentation | BBB | PVC morph | Episode | Pause | CI/QT | CI/RR | CI | R on T |
|--------------------------|-----|-----|--------------|-----|-----------|---------|-------|-------|-------|----|--------|
| Leenhardt et al.10        | F   | 31  | Pre-syncope/Syncope | LBBB | POLYM     | PMT/VF  | NO    | 0.67  | 0.25  | 255| Asc limb |
| Leenhardt et al.10        | M   | 15  | Pre-syncope/Syncope | RBBB | UNI       | PMT/VF  | NO    | 0.7   | 0.35  | 280| Asc limb |
| Eisenberg et al.7         | –   | 36  | Pre-syncope/Syncope | –   | UNI       | PMT/VF  | YES   | 1     | 0.33  | 408| Desc limb |
| Eisenberg et al.7         | F   | 58  | Cardiac Arrest | –   | UNI       | PMT/VF  | NO    | 0.85  | 0.5   | 355| Desc limb |
| Anter E. et al.11         | M   | 59  | Cardiac Arrest | LBBB | UNI       | PMT/VF  | NO    | 0.65  | 0.2   | 290| Asc limb |
| Takeuchi T et al.12       | M   | 51  | Cardiac Arrest | RBBB | UNI       | PMT/VF  | YES   | 0.7   | 0.27  | 260| Desc limb |
| Viskin et al.13           | F   | 50  | Cardiac Arrest | LBBB | POLYM     | PMT/VF  | NO    | 0.75  | 0.4   | 300| Asc limb |
| Viskin et al.13           | F   | 55  | Pre-syncope/Syncope | LBBB | UNI       | PMT    | NO    | 0.89  | 0.3   | 330| Desc limb |
| Suh et al.17              | F   | 40  | Cardiac Arrest | LBBB | UNI       | PMT    | NO    | 0.75  | 0.47  | 270| Asc limb |
| Betts et al.18            | M   | 32  | Cardiac Arrest | LBBB | UNI       | PMT    | NO    | 0.7   | 0.3   | 300| Asc limb |
| Allrocia et al.19         | F   | 47  | Cardiac Arrest | LBBB | –         | PMT    | NO    | 0.87  | 0.58  | 365| Desc limb |
| Mechleb et al.13          | F   | 55  | Cardiac Arrest | LBBB | –         | PMT    | NO    | 0.86  | 0.42  | 355| Desc limb |
| Burrows et al.14          | F   | 24  | Cardiac Arrest | LBBB | UNI       | PMT    | NO    | 0.85  | 0.41  | 330| Desc limb |
| Mouz et al.21             | –   | –   | –             | –   | –         | PMT/VF  | NO    | 0.54  | 0.27  | 200| Asc limb |
| Nogami et al.22           | M   | 54  | Cardiac Arrest | RBBB | UNI       | PMT    | NO    | 0.81  | 0.48  | 260| Asc limb |
| Brendan et al.23          | M   | 51  | Cardiac Arrest | LBBB | –         | PMT/VF  | NO    | 0.71  | 0.39  | 270| Asc limb |
| Chiladakis et al.24       | F   | 50  | Cardiac Arrest | –   | –         | PMT/VF  | NO    | 0.73  | 0.3   | 345| Asc limb |
| Mourad et al.15           | M   | 38  | Cardiac Arrest | LBBB | POLYM     | PMT/VF  | NO    | 0.85  | 0.36  | 325| Desc limb |
| Almehairi et al.          | M   | 25  | Cardiac Arrest | RBBB | UNI       | PMT    | NO    | 0.83  | 0.52  | 275| Desc limb |
| Landen et al.20           | M   | 53  | Cardiac Arrest | LBBB | UNI       | PMT/VF  | NO    | 0.79  | 0.59  | 346| Asc limb |
| Rak Cho et al.27          | M   | 17  | Cardiac Arrest | LBBB | UNI       | PMT/VF  | NO    | 0.92  | 0.26  | 370| Desc limb |
| Aizawa Y et al.27         | M   | 13  | Cardiac arrest | RBBB | UNI       | PMT/VF  | YES   | 0.9   | 0.45  | 370| Desc limb |
| Takatsuki et al.27        | M   | 62  | Pre-syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.8   | 0.39  | 300| Desc limb |
| Naik N et al.28           | M   | 24  | Pre-syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.7   | 0.26  | 280| Asc limb |
| Kohsaka S et al.29        | F   | 21  | Pre- syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.84  | 0.28  | 350| Desc limb |
| Yamazaki M et al.30       | M   | 21  | Pre-syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.84  | 0.3   | 240| Asc limb |
| Shiga et al.21            | M   | 41  | Pre-syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.66  | 0.3   | 240| Asc limb |
| Wafa et al.21             | F   | 30  | Pre-syncope/Syncope | –   | UNI       | PMT/VF  | NO    | 0.78  | 0.27  | 300| Deschlimb |
| Yanfei et al.22           | M   | 30–40| Pre-syncope/Syncope | –   | UNI       | PMT/VF  | NO    | 0.7   | 0.26  | 300| Deschlimb |
| Yanfei et al.22           | F   | 30–40| Pre-syncope/Syncope | –   | UNI       | PMT/VF  | NO    | 0.71  | 0.26  | 300| Deschlimb |
| Bogaard et al.30          | M   | 36  | Pre-syncope/Syncope | LBBB | UNI       | PMT/VF  | NO    | 0.78  | 0.34  | 300| Asc limb |

Fig. 2. Example of LCTDP episode demonstrating coinciding R on terminal part of the TW. LCTDP = long coupled torsade de pointes, TW = T wave.
in association with exercise. All cases were reported to have normal electrolyte and metabolic indices. Only one article was published as a pure ECG case of SCTDP, with no further clinical data.

Work up for evidence of coronary artery disease (CAD) was described in 31 cases. No obstructive coronary artery disease was reported in the 27 cases in whom coronary angiograms were performed. In the other three cases CAD was ruled out by non-invasive methods. Cardiac MRI was only performed in three patients and showed normal cardiac structure. RV biopsy was performed in 3 patients and showed no evidence of arrhythmogenic right ventricular dysplasia.

Successful ablation for PVCs foci was achieved in 7 subjects. One subject underwent electrophysiology study and was negative for inducible arrhythmia following verapamil therapy.

Genetic testing was only done in one subject and was negative for inherited channelopathies.

Fourteen subjects received ICD implantation whereas 13 subjects were maintained on verapamil and 4 on B blockers (atenolol and propranolol).

### 3.1. Clinical features

The age was reported in 32 subjects only and the mean was calculated as \( \text{age} = 39.9 \pm 13.7 \) and the median as 40 years. Gender was reported in 26 subjects of which 14 were males and 12 were females.

Reported symptoms were aborted cardiac arrest (CA) in 17/32 and palpitations or syncope in 14/32. One case was reported without description of symptoms and only included ECG findings (Table 1).

### 3.2. ECG characteristics

There were 27 cases with identifiable ectopic beats that underwent further analysis for the purposes of consistency of which 24 presented unifocal morphology.

Pauses preceding the short-coupled arrhythmia was identified in 5/28 subjects. Four patients in this category presented clinically with palpitation/syncope whereas only one presented with CA.

Pure polymorphic pattern was noticed in 24 patients of the total cohort and 7 patients degenerated into VF.

### 3.3. Validation of the ECG indices

The CI/QT index was applied with a score of < 1 used to specify the short-coupled arrhythmia. CI/QT index of < 1 was 96% sensitive and 100% specific for it. The mean CI for the short coupled arrhythmia and LCTDP were 304 ± 47 ms and 638.08 ± 47.177 ms; respectively (95% CI, 294.6–379.7). Two cut-off points for CIs were analysed for their accuracy in identifying the short-coupled arrhythmia. A CI < 400 ms was 100% sensitive 97% specific, whereas CI ≤ 300 ms is 100% sensitive and 50% specific (Table 3).  

### 3.4. Timing of coinciding PVCs with the T waves

Comparative analysis of the coinciding PVCs on the ascending (\( T_{\text{asc}} \)) and descending (\( T_{\text{desc}} \)) limbs of the T wave were performed to assess the predictability of the clinical presentation. R on \( T_{\text{asc}} \) was observed in a total of 14 patients, 64% in the CA group and 36% in the Pal/Syn group. R on \( T_{\text{desc}} \) was comparatively seen in a total of 18 subjects, 44% in CA group and 55% in Pal/Syn group. The correlation between the type of clinical presentation and the timing of R on T was not statistically significant (\( P \) value 0.47). Interestingly, 6 patients from 7 who presented with VF degeneration had R on \( T_{\text{asc}} \). Despite that, CA occurred in some of them.

### 3.5. Relation of the coupled PVCs to the Purkinje system

Of the 9 patients that underwent further ectopic mapping and ablation, 5 were identified to be Purkinje related arrhythmia whereas the remaining 4 were found to be myocardial in origin.

### 4. Discussion

This type of short-coupled arrhythmias is rare and accounts for 5–10% of out-of hospital aborted cardiac arrests.1 Variable eponyms used to describe this arrhythmia created confusion surrounding its underlying mechanism. We reviewed the literature for case reports/series describing IVF, SCTDP, Pr-TDP and idiopathic PMT and the similarities in their features indicated that all presenting same type of arrhythmia.

Leenhardt et al.6 first described an arrhythmia they termed SCTDP in 14 patients with structurally normal hearts without associated channelopathies. Yet the term TDP historically relates to long QT arrhythmias that conceptually conflicts with the proposed mechanism for a TDP specified as short-coupled. In contrast, Eisenberg et al.7 utilized the term polymorphous VT in structurally normal heart, a description that is not sensitive enough to exclude inherited channelopathies that could complicate similar pattern of short-coupled arrhythmias. Haïssaguerre et al.3 and Knecht et al.10 preferred the descriptive term IVF, whereas Nomami refined Leenhardt’s description to be Pr-TDP.

One may argue that using IVF as a unifying diagnostic term potentially creates conceptual conflicts for an arrhythmia that is always PMT/TDP in pattern and combined with degeneration to VF in some cases11,12. VF degeneration, duration of the arrhythmia, frequency of recurrences/shocks and clinical presentation, could have a close relation to the coinciding PVCs on the T-wave. Viskin et al.8 indicated that the propensity for poor outcomes associates with presentations of coinciding R on \( T_{\text{asc}} \). Alshekh-Ali et al.14 demonstrated that VF induction in an experimental commotio cordis model has close relationship with critical timing of chest impact during the repolarization phase. As indicated above, we deemed CA type of initial clinical presentation a poor outcome. An inference from the coinciding PVC on the T wave (ascending

### Table 2

| Congenital QT/RR Index CI CI/QT index | Acquired QT/RR index CI CI/QT index |
|--------------------------------------|-------------------------------------|
| Sami et al.15 0.41 ms 640 ms 1.04 ms | Gregorio et al.42 0.52 ms 590 ms 1.00 ms |
| Monahan et al.17 0.56 ms 707 ms 1.06 ms | Gibson et al.41 0.3 ms 635 ms 1.35 ms |
| Sami et al.38 0.57 ms 600 ms 1.03 ms | Hiede et al.34 0.4 ms 495 ms 1.21 ms |
| Shimizu29 0.35 ms 630 ms 1.30 ms | Arce et al.40 0.4 ms 588 ms 1.06 ms |
| Basmaad et al.40 0.36 ms 765 ms 1.25 ms | Colombo et al.20 0.58 ms 640 ms 1.2 ms |
| Pijperoa et al.41 0.44 ms 640 ms 1.25 ms | Bass et al.23 0.3 ms 400 ms 1.05 ms |

### Table 3

| Sensitivity and specificity analysis of the electrocardiographic indices. |
|----------------------------------|------------------|
| ECG indices | Se & Sp |
| CI < 300 ms | 100–50% |
| CI < 400 ms | 100–97% |
| CI/QT < 1 ms | 96–100% |

Calculated indices for the control group of acquired and congenital long QT syndrome.
or descending) was attempted to assess the propensity for the type of the clinical presentation. The comparative analysis suggested that R on Tasc or Tdesc does not predict the likelihood of CA over Pal/Syn type of presentation. (P = 0.47). However, 7 patients in from the total cohort presented with true VF degeneration and 6 had a significant correlation with R on Tdesc, which may indicate that the R on T timing plays a significant role in VF degeneration but does not necessarily lead to CA. Notably, fifteen patients (45%) presented with syncope of which 3 had occasional VF degeneration during the episode. Leenhardt’s group has described SCTDP purely consisting of PMT/TDP pattern, while in some cases has been in association with VF degeneration. In contrast, the majority of IVF cases presented as pure PMT/TDP pattern, although this observation was limited by the available ECGs displayed in the studied articles. Nogami et al.6 specified SCTDP as Purkinje-related arrhythmia and used this terminology interchangeably between the two entities. In the studied cohort, however, 4 of 9 patients underwent formal Electrophysiological Study and were identified to have non-Purkinje related PVCs. Similarly, Both Haïssaguerre et al.7 and Knecht et al.8 reported non-Purkinje related PVCs in some cases.

We propose the term “idiopathic short-coupled ventricular tachyrhythmias” to be a unifying eponym for this group of ventricular tachycarrhythmias. “Idiopathic” is proposed to avoid an overlap with aetiologies known to precipitate similar short-coupled ventricular arrhythmias such as catecholaminergic polymorphic ventricular tachycardia, Brugada syndrome, short QT syndrome or ischemia. “Short-coupled” characterises those critically coupled PVCs which are required to occur in the vulnerable phase, in the context of normal cardiac structure. Ventricular Tachyarrhythmias was used as a general term to accommodate any possible form of this arrhythmia, whether it is polymorphic VT, VF or in both combinations. As known, pauses almost always are the trigger in LCTDP,15,16; however, our search revealed 5 patients who demonstrated pauses preceding the short-coupled arrhythmia and yet had no association with long QT syndrome. Validation of the CI that was depicted by Leenhardt et al.6 as <300 ms was performed. The purpose was to set a discriminating threshold to help rule out LCTDP. In that study, 14 patients were studied and the longest CI was 300 ms. We applied this criterion to our cohort and found a sensitivity of 100%, but with a specificity that did not exceed 50%. Of the several CIs analysed, the most effective threshold was found to be <400 ms, which demonstrated 100% sensitivity and 97 % specificity. Eizenberg et al.9 has proposed the CI/QT index as a useful discriminator of idiopathic subtypes of PMT in structurally normal hearts. Our validation of this index is in agreement, with sensitivity and specificity of 96% and 100%, respectively. Several case reports and two original studies6,10 showed that ablation strategy in these cases is of great clinical benefit. Our analysis revealed that 9 patients underwent successful suppression of PVCs with radiofrequency ablation. Knecht et al.10 reported successful ablation of 38 patients in a multi-centre trial with 89% of patients remaining free of recurrences at 5-year follow up. Additionally, Haïssaguerre et al.7 achieved successful ectopic ablation in 27 patients. It is important to note that attempting ablation is a purely palliative strategy and that defibrillator implantation is mandatory to prevent SC.10 Conservative medical therapy using verapamil has been shown in a majority of patients to be a valuable strategy in suppressing PVCs, reducing the frequency of the short-coupled arrhythmia and delaying the CI of the PVC.17,18

5. Limitations

We present a systematic review of case reports published under different and unrelated titles. Many ECGs and tracings met only moderate quality in terms of their clarity that necessitated computer-based editing. Many case reports used Leenhardt’s term SCTDP or PMT, which precluded them from further invasive investigation to determine if they were related to Purkinje fibres. Unfortunately, that limited our assessment to define the prevalence of non-Purkinje related ISCVT. The comparative analysis between CA and Pal/Syn groups were based on available ECGs to assess the outcome of the coinciding PVC on the T-wave. A few patients in the cohort had had cardiac MRI, RV biopsy and genetic testing.

6. Conclusion

Substantial inconsistency in literature exists over regarding the description of ventricular tachyarrhythmias in structurally normal hearts. Therefore, ISCVT as a new diagnostic term may help to unify these descriptions. ECG indices, in particular CI and CI/QT demonstrated good discriminatory performance to distinguish ISCVT from LCTDP.

Conflict of interest

We would also like to state that we have no conflicts of interest to declare.

References

1. Survivors of out-of-hospital cardiac arrest with apparently normal heart: need for definition and standardized clinical evaluation: consensus Statement of the Joint Steering Committees of the Unexplained Cardiac Arrest Registry of Europe and of the Idiopathic Ventricular Fibrillation Registry of the United States. Circulation J 1997;95:265–72.
2. Scheinman MM. Role of the His-Purkinje system in the genesis of cardiac Arrhythmia. Heart Rhythm J 2009;6:1050–1058.
3. Haïssaguerre M, Shoda M, Jais P, et al., Mapping and ablation of idiopathic ventricular fibrillation. Circulation J 2002;106:962–967.
4. Bernfeld O, Jalife J. Purkinje-muscle reentry as a mechanism of polymorphic ventricular arrhythmias in a 3-D dimensional model of the ventricles. Circulation Res. 1998;82:1063–1077.
5. Nogami A, Purkinje-related Arrhythmias Part II: Polymorphic ventricular tachycardia and ventricular fibrillation.PACE J 2011;34:1034–1049.
6. Leenhardt A, Glaser E, Burguerua M. Short coupled variant of torsade de pointes: spectrum of idiopathic ventricular tachyarrhythmia’s. Circulation J 1994;89:206–215.
7. Eisenberg SJ, Scheinman MM, Dullet NK. Sudden Cardiac Death and Polymorphous Ventricular Tachycardia in patients with normal QT intervals and normal systolic cardiac function. Am J Cardiol 1995;75:687–692.
8. Viskin S, Belhassen B. Polymorphic ventricular tachyarrhythmia’s in the absence of organic heart diseases: classification, differential diagnosis and implications for therapy. Prog Cardio Dis. 1998;41:17–34.
9. Mochielie BK, Haddadin TZ, Said B, et al., Idiopathic polymorphic ventricular tachycardia with normal QT interval in a structurally normal heart. PACE. 2006;29:791–796.
10. Leenhardt A, Glaser E, Burguerua M. Short coupled variant of torsade de pointes: spectrum of idiopathic ventricular tachyarrhythmia’s. Circulation J 1994;89:206–215.
11. Eisenberg SJ, Scheinman MM, Dullet NK. Sudden Cardiac Death and Polymorphous Ventricular Tachycardia in patients with normal QT intervals and normal systolic cardiac function. Am J Cardiol 1995;75:687–692.
12. Viskin S, Belhassen B. Polymorphic ventricular tachyarrhythmia’s in the absence of organic heart diseases: classification, differential diagnosis and implications for therapy. Prog Cardio Dis. 1998;41:17–34.
13. Alsheik-Ali AA, Madias C, Supran S, Link MS, et al., Marked variability in susceptibility to ventricular fibrillation in an experimental commotio cordis model. Circulation. 2010;122:2489–2504.
14. Viskin S, Fish R, Roth A, et al., Prevention of torsade de pointes in the congenital long QT syndrome: use of a pause prevention pacing algorithm. Heart J. 1998;79:417–419.
15. El-Sherif N, Mechanism of ventricular arrhythmias in the long QT syndrome: on hermeneutics. J Cardiovasc Electrophysiol. 2001;12:973–976.
16. Suh WM, Fowler SJ, Yeh T, Krishnan SC, et al., Successful catheter ablation of focal ventricular fibrillation originating from the right ventricle. J Interv Card Electrophysiol. 2009;26:139–142.
17. Betts TK, Yue A, Roberts PR, et al., Radiofrequency ablation of idiopathic ventricular fibrillation guided by noncontact mapping. J Cardiovasc Electrophysiol. 2004;15:957–959.
19. Allocca G, Vallone C, Nucifora G, et al., A dramatic storm of idiopathic ventricular fibrillation. Cardiol. 2009;98:62–65.
20. Burrows KDO, Fox JNP, Lee A Biblo. Pregnancy and short coupled Totsade De Pointes. PACE J. 2010;1–3.
21. Munos JJS, Garcia-Alberto A, Martinez-Sanchez J, et al., Premature ventricular complexes as a trigger for ventricular fibrillation. Rev Esp Cardio. 2010;6:798–801.
22. Nogami A, Sugiyasu A, Kubota S, et al. Mapping and ablation of idiopathic ventricular fibrillation from the Purkinje system. HR 2005;2:646–649.
23. Branden BVD, Wever E, Boersma L. Totsade De Pointes with short coupling interval. Acta Cardiol. 2010;65:345–346.
24. Chiladakis JA, Spiroulias G, Koutsogiannis N. Short coupled variant of torsade de pointes as a cause of electrical storm and aborted sudden cardiac death: insights into mechanism and treatment. Hellenic J Cardiol. 2008;49:360–364.
25. Mourad H, Selma K, Zahra K, et al. Torsade de pointes A couplage court compliqué D’unue Mort Subite. 2004;82:542–545.
26. Landen K, Schmidt K, Poponick J. ECG changes in polymorphic ventricular tachycardia. Consultant. 2007;47:7.
27. Rak Cho YR, Park JS. Radiofrequency catheter ablation for unifocal premature ventricular complexes triggering recurrent ventricular fibrillations in a young man without structural heart disease. Korean Circulation J. 2012;42:575–579.
28. Aizawa Y, Tamura M, Chinushi M, et al., An attempt at electrical catheter ablation of the arrhythmogenic area in idiopathic Ventricular fibrillation. Am Heart J. 1992;1:257–260.
29. Takatsuki S, Mitamura H, Ogawa S. Catheter ablation of a monofocal premature ventricular complexes triggering recurrent ventricular fibrillations in a young man without structural heart disease. Korean Circulation J. 2012;42:575–579.
30. Naik N, Juneja R, Sharma G, Yadav R, et al., Malignant idiopathic ventricular fibrillation “cured” by radiofrequency ablation. J Interv Card Electrophysiol. 2008;23:143–148.
31. Kohsaka S, Razavi M, Massumi, et al. Idiopathic ventricular fibrillation successfully terminated by radiofrequency ablation of the DistalPurkinje fibres. PACE J. 2007;30:701–704.
32. Yamazaki M, Osaka T, Yokoyama E, et al., A case of Short Coupled Variant of Totsade De Pointes characterized by spatial heterogeneity of action potential duration and its restitution kinetics. J Interv Card electrophysiol. 2006;17:35–40.
33. Shiga T, Shoda M, Matsuda N, et al., Electrophysiological Characteristics of a Patient exhibiting the Short Coupled Variant of Totsade De Pointes. J Electro. 2001;34:271–275.
34. Wafa F, Salem A, Zahreddine S, et al., La torsade de pointes A couplage court: Notre experience et revue de la literature. La tunisie Medicale. 2004;82:867–874.
35. Yanfui R, Lin W. Short coupled variant of torsade de pointes. J Tongji Med Univ. 2001;21:30–31.
36. Bogaard K, Steen VA, Tan HL, et al. Short coupled variant of Totsade De Pointes. Netherl Herar J. 2008;16:7/8.
37. Monahan BP, Ferguson CL, Cleave ES, Lloyd BK, et al., Torsade de pointes occurring in association with terfenadineuse. JAMA. 1990;264:2788–2790.
38. Viskin S, Justo D, Falkin A, Zeltser D. Long QT syndrome caused by non-cardiac drugs. Prog Cardiovasc Dis. 2003;45:415–427.
39. Shimizu W, Aiba T, Antzelevitch C. Specific therapy based on the genotype and cellular mechanism in inherited cardiac arrhythmias. Long QT syndrome and Brugada syndrome. Curr Pharm Des. 2005;11:156–172.
40. Basamad Z, QT Interval: the proper measurement techniques. Shiraz E-Med J. 2010;11:No. 2.
41. Figueroa JA, Clavero MH, Serra JL, et al., Long QT syndrome with amiodarone. Rev Fed Arg Cardiol. 2012;41:1.
42. Gregorio CD, Morabito G, Cerrotti M, Dattilo G, et al., Citalopram-induced long QT syndrome and torsade de pointes: role for concomitant therapy and disease. Int J Cardio. 2011;14:226–228.
43. Gibson CM. Torsade de pointes electrocardiogram. http://www.wikidoc.org/index.php/Torsades_de_pointes_electrocardiogram.
44. Der Heide KV, De Haes A, Wietasch GJK, et al. Torsades de pointes during laparoscopic adrenalectomy of a pheochromocytoma: a case report. J Med Case Rep. 2011;5:368. Accessed date 2012.
45. Arce JS, Romero R, Solozano PA. Case of prolonged QT interval and Torsades de Pointes due to Ciprofloxacin. Rev Esp Cardiol. 2010;63:111–112.
46. Colombo N, Civelii M, Cardinale D, et al. Long QT syndrome and torsade de pointes after antirachycine chemotherapy e cancer. 2009;3:147.
47. Bass AS, Tomasselli ASC, Bullingham R, Kinter LB. Drugs effects on ventricular repolarization: a critical evaluation of the strengths and weaknesses of current methodologies and regulatory practices. J Pharma Toxicol Meth. 2005;52:12–21. Accessed date 2012 <http://www.sciencedirect.com/science/article/pii/S1056871905000596>.