Clinical Characteristic and Clinical Course of Aborted Sudden Cardiac Death Patients with Structurally Normal Heart in King Chulalongkorn Memorial Hospital

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Background: *PED (Primary electrical disease) is an arrhythmogenic disease group that causes serious ventricular tachyarrhythmia in the absence of recognized structural heart disease. Although Thailand, which is a part of Southeast Asia, is an endemic area of PED, especially Brugada syndrome, there is little known about demographic data and clinical outcome of PED among survivors of sudden cardiac death (SCD).*
Objective: *To study demographic data and clinical outcome of PED among survivors of SCD in Thailand.*
Material and Method: *The present study was approved by the ethics committee of the Faculty of Medicine, Chulalongkorn University, Bangkok. The authors reviewed patient medical records for clinical characteristics, etiology, and clinical outcome of survivors of SCD between January 2002 and December 2008. The patients with PED who had normal structural heart and no obvious non-cardiac causes of sudden death were enrolled.*
Results: *Fifty-two survivors of SCD with PED (mean age 39±12, 49 males), mainly from the northeast and middle regions of Thailand, were recruited for this study. SCD mainly occurred during times when patients were asleep or resting (50% and 15.4%) respectively. Of the 52 survivors, 21 patients (40%) had a documented family history of SCD. The etiology of survivors of SCD is composed of Brugada syndrome (63.5%), Early repolarization syndrome (ERS) (9.6%), Congenital long QT syndrome (LQTS) (7.7%), and idiopathic VF (3.8%). Remaining 15.4% had abnormal ECG but not compatible with any etiology. A large number of patients (78.8%) completely recovered without neurological sequelae. The recurrence of severe ventricular arrhythmia occurred in 27 patients (51.9%) during mean follow-up period 56.5±35.4 months (4 to137 months). The recurrence was highest during the first year (32%) of follow-up.*
Conclusion: *The Brugada syndrome is the most common etiology of survivors of SCD with normal structural heart in Thailand. Although the prognosis after resuscitation was good, recurrence was especially high during the first year and as a result, an ICD implantation is needed for prevented recurrence of SCD.*

Keywords: *Sudden cardiac arrest survivors, Aborted sudden cardiac arrest, Primary electrical disease*

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Sudden cardiac death (SCD) is an unexpected death due to cardiac causes occurring in a short time period (generally within one hour of symptom onset) in a person with known or unknown structural heart disease. The majority of sudden cardiac deaths occurs outside the hospital and before any medical intervention can take place. Previous studies have shown that the structural heart disease is the most etiology of SCD. However, most survivors of SCD who have a normal structural heart, the SCD is caused by primary electrical disease (PED).

The abnormal electrophysiological mechanism of PED is a cause of polymorphic ventricular tachycardia (PMVT) or ventricular fibrillation (VF) which is detected during CPR[1]. PED is composed of Brugada syndrome (BS), long QT syndrome (LQTS), short QT syndrome (SQTS), catecholaminergic polymorphic VT (CPVT), early repolarization syndrome (ERS), ventricular pre-excitation, and idiopathic VF[2-8]. The diagnosis of PED is predominantly based on findings in ECG including clinical history and provocation test that could induce arrhythmia. Although preliminary data originating from Western countries suggests that PED has less than 10% etiology of SCD and associated

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with 30% recurrence rate of SCD[9,10], no previous study has described clinical characteristics and clinical outcomes in Asian and Thai populations. The main objective of the authors is to describe the clinical characteristic and clinical outcome of the survivors of SCD who have a normal structural heart.

Material and Method

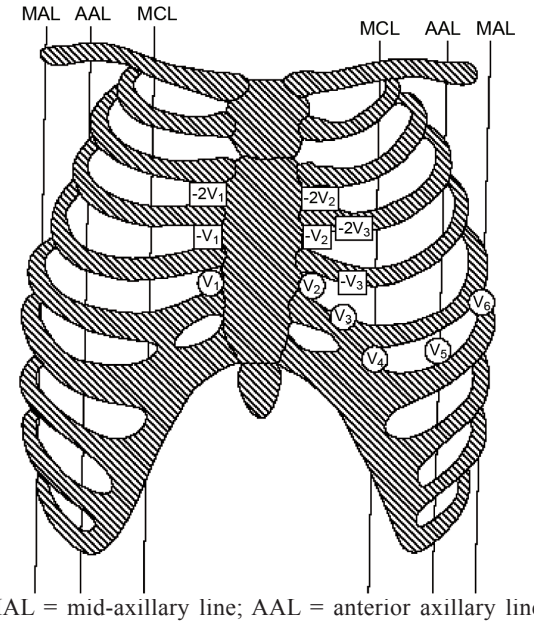
The present study utilizes descriptive retrospective design. The data was gathered by reviewing medical records from King Chulalongkorn Memorial Hospital (KCMH) in Bangkok, Thailand between January 2002 and December 2008. The present study was approved by the ethics committee of the Faculty of Medicine, Chulalongkorn University, Bangkok. The inclusion criteria included survivors of SCD including PMVT or VF which were identified during resuscitation or inducible PMVT or VF in Electrophysiological (EP) study laboratory by programmed ventricular stimulation up to 3 extrastimuli and 3 cycle length driving trains at right ventricular apex or right ventricular outflow tract.

The standard 12 leads ECG (SECG) and/or higher intercostal space leads ECG (HECG) (Fig. 1) with or without provocation by class I antiarrhythmic drugs were performed and mainly used to define the etiology in every patients. The transthoracic echocardiography (TTE) was performed on all participants for screening of structural heart disease associated with SCD. Additionally, biochemical analysis, cardiac markers (creatine phosphokinase: CPK, creatine kinase-MB: CK-MB, and Troponin T: Trop-T) were analyzed. Coronary angiogram (CAG), exercise stress test (EST) or cardiac magnetic resonance imaging (MRI) was needed in patients who had rising of cardiac markers to exclude coronary artery disease. The enrolled patients would be free from structural heart disease and no obvious non-cardiac cause of SCD.

Definition

The diagnosis of PED in the present study is a group of BS, LQTS, SQTS, CPVT, ERS, ventricular pre-excitation, and idiopathic VF. The diagnosis of BS was established only if patients had BS type 1 ECG at baseline or in higher intercostal space leads ECG (Fig. 1)[11] with or without provocation with class I antiarrhythmic drugs (flecainide 400 mg PO or procainamide 10 mg/kg given over a 10 minute period). The BS type 1 ECG defined as a prominent coved ST elevation ≥2 mm (0.2 mV) at its peak were followed by a negative T wave >1 right precordial lead V_1 to V_3[3]. LQTS was defined as QTC ≥0.44 in men and ≥0.46 in women[5]. SQTS was defined as QTC <0.32 with tenting T wave[6]. CPVT was defined as non-sustained PMVT that could be induced by isoproterenal intravenous infusion[7]. ERS was defined as J point elevation at least 1 mm (0.1 mV) with QRS slurring or notching in the inferior leads (II, III, aVF), lateral leads (I, aVL and V_4 to V_6) or both[8]. Ventricular pre-excitation was defined as delta wave[4]. Idiopathic VF was defined as normal ECG. Uncategorized ECG group was meant abnormal ECG that non-compatible with other description.

The degree of disability after resuscitation and during hospital discharge was described by Modified Rankin scale[12], 0-6 grade were classified, 0 was no neurological sequelae, 1 was non-significant neurological symptom, 2 was mild disability, 3 was



MAL = mid-axillary line; AAL = anterior axillary line; MCL = mid-clavicular line

Fig. 1 The standard precordial positions of conventional chest leads V_1 to V_6 (open circles) and the higher intercostal space ECG leads: $-V_1$ to $-V_3$ and $-2V_1$ to $-2V_3$ (open squares) are shown. The lead $-V_1$ is just to the right of the sternum in the third intercostal space; the lead $-V_2$ is to the left of the sternum in the third intercostal space; the lead $-V_3$ is one intercostal space higher than standard lead V_3; the lead $-2V_1$ and $-2V_2$ are just to the right and left respectively of the sternum in the second intercostal space. The lead $-2V_3$ is two intercostal spaces higher than standard lead V_3 [11].

moderate disability, walk without assistance, 4 was moderate disability, walk with assistance, 5 was bedridden, and 6 was dead.

Statistical analysis

The data were presented as a mean ± standard deviation, range, and percentage. Categorical variables were analyzed by using the Cox proportional hazard model, 95% confidence interval with statistical significant $p<0.05$.

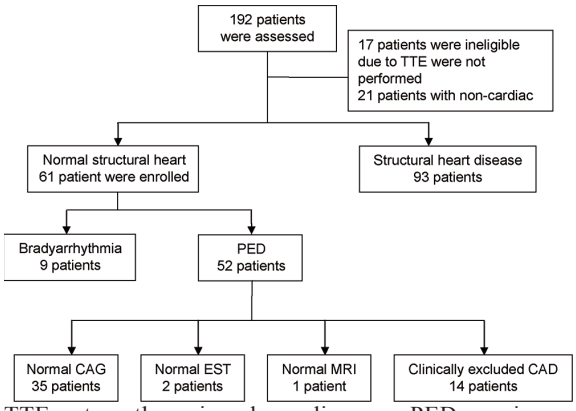
Results

The authors reviewed the patient medical records from 192 survivors of Sudden death, regardless of causation (Fig. 2). From this review, it was determined that 154 patients were defined as survivor of SCD. The mortality rate after resuscitation was 40% and acute myocardial infarction was the most common cause of SCD (Fig. 3).

One hundred two patients who did not achieve eligible criteria were excluded. There were 52 patients enrolled as survivors of SCD from PED. In this group, TTE of all patients showed normal structural heart. Subsequently, 38 patients who had abnormal cardiac markers, significant coronary artery disease were excluded by coronary angiography (35 patients), EST (2 patients), and cardiac MRI (1 patient). The vast majority were male (94%) and mean age was 39±12.5 years (Table 1). Fifty percent of the patient's events occurred during sleep, 17 patients (33%) had previously at least one episode of syncope, two patients (4%) had agonal respiration, two patients (4%) had a previous history of sudden cardiac arrest, and 21 patients (40%) had a family history of SCD.

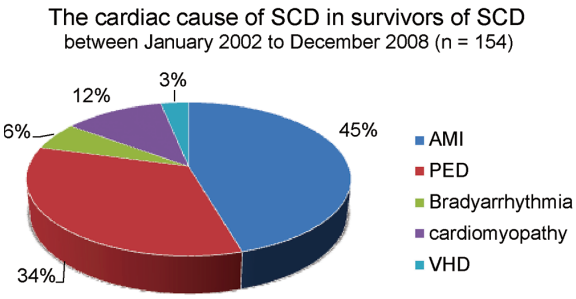
Etiology of SCD

The SECG could detect BS in 25 patients (48%). When HECG was applied, the authors could unmask BS in six patients (Fig. 4). Two patients with clinically suspected BS could induce ECG manifestation of BS by class I antiarrhythmic drugs (1 with normal in SECG provoked by procainamide and 1 with ST elevation in right precordial leads in SECG provoked by flecainide). Finally, 33 patients (63.5%) were defined as BS with the mean age was 41±13 years old. They were predominately male (97%). Fourteen patients (42%) had a family history of SCD. In the 33 patients, all family members victim were male. ERS was diagnosed in five patients (9.6%) of PED. Their mean age was 36±11 years old, all were male, and three patients (60%) had a history of SCD in family



TTE = transthoracic echocardiogram; PED = primary electrical disease; CAG = coronary angiogram; EST = exercise stress test; MRI = magnetic resonance imaging; CAD = coronary artery disease

Fig. 2 The survivors of sudden cardiac death diagram.



AMI = acute myocardial infarction; PED = primary electrical disease; VHD = valvular heart disease

Fig. 3 The cardiac causes of sudden cardiac death who survived up to KJMH from January 2002 to December 2008.

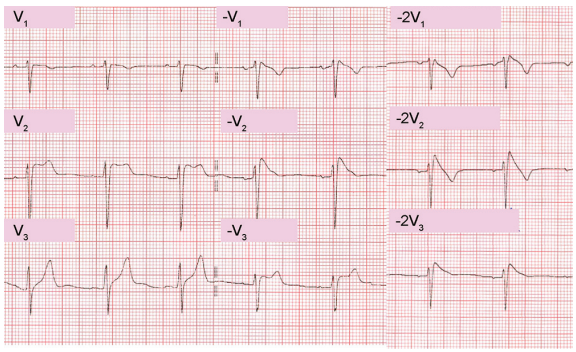


Fig. 4 A 34-year-old male from the northeastern part of Thailand who had sudden cardiac arrest on July 2004 while he was sleeping at home; he completely recovered after successful resuscitation and SECG (first row) showed ST elevation (saddle back) in right precordial leads, after HECG was applied with one intercostal space (second row) and two intercostal space (third row) moved up of the V_1-V_3 the BS type 1 was obviously appeared.

Table 1. Clinical characteristic of survivor of sudden cardiac death in primary electrical disease group

Clinical characteristic	Total (n = 52)
Age (year)	39±12.5
Sex (male,%)	49 (94.2%)
Activity during SCD	
Sleep	26 (50.0%)
Rest	8 (15.4%)
Minimal activity	11 (21.2%)
Exercise	3 (5.8%)
Unknown	4 (7.6%)
Region of Thailand	
Middle	20 (38.5%)
North	2 (3.8%)
Northeast	27 (51.9%)
East	2 (3.8%)
South	1 (1.9%)
Previous symptom	
No	29 (55.8%)
Syncope	17 (32.7%)
Agonal respiration	2 (3.8%)
Cardiac arrest	2 (3.8%)
Palpitation	1 (1.9%)
Chest discomfort	1 (1.9%)
SCD in family	21 (40.4%)
First arrhythmia detected	
PMVT/VF	47 (90.4%)
Asystole turn to VF	3 (5.8%)
Inducible PMVT/VF	2 (3.8%)
Etiology	
Brugada syndrome	33 (63.5%)
LQTS	4 (7.7%)
Early repolarization	5 (9.6%)
Idiopathic VF	2 (3.8%)
Uncategorized	
ST elevation in right precordial leads	6 (11.5%)
Complete RBBB	1 (1.9%)
Slur ST segment in II, III, aVF	1 (1.9%)
Electrophysiologic study	
Performed	31 (59.6%)
Inducible PMVT/VF	25 (48.0%)
Non-inducible PMVT/VF	6 (11.5%)
Not performed	21 (40.0%)
Discharge status (modified Rankin Scale)	
Normal (0)	41 (78.8%)
Mild disability (1-2)	0 (0%)
Moderate disability (3-4)	3 (5.8%)
Severe disability (5)	6 (11.5%)
Dead (6)	2 (3.8%)

SCD = sudden cardiac death; PMVT/VF = polymorphic ventricular tachycardia or ventricular fibrillation; LQTS = long QT syndrome; RBBB = right bundle branch block

members (1 with father SCD, 1 with mother SCD, 1 with older sister SCD). LQTS was diagnosis in four patients (7.7%) of PED with the mean age was 31±12 years old, 50% were female, and one patient (25%) had a history of SCD of father. Idiopathic VF was diagnosis in two male patients (age 51 and 39 years old) and one patient (50%) had a history of SCD of an older brother.

The remaining 8 patients were defined as uncategorized ECG abnormalities, 1 patient (1.9%) with complete right bundle branch block, 1 patient (1.9%) with slurring S wave in II, III, aVF, and 6 patients (11.5%) with ST elevation in right precordial leads but not compatible with BS type 1.

Clinical outcome

There were 50 patients (96.2%) alive at discharge, 41 patients (78.8%) had no neurological sequelae, three patients (5.8%) had moderate disabilities but could do actual activities, and six patients (11.5%) stayed in bedridden status (Table 1). In the patients who survived until discharge, 20 patients received a transvenous implantable cardioverter defibrillator (ICD), seven patients received a transvenous ICD with medication (6 patients with Beta-blocker and 1 patient with amiodarone) to attempt to control the recurrence of PMVT or VF, eight patients received medication without ICD (6 with Beta-blocker and 2 with amiodarone) because of inappropriate status and patient's preference, and 17 patients refused management and never followed-up. The recurrence of PMVT or VF during mean follow-up period 56.5±35.4 months (4 to137 months) occurred in 19 BS,

Table 2. Long term outcome and survival rate of sudden cardiac death in primary electrical disease group

	Total (n = 52)
Recurrent of PMVT/VF	27 (51.9%)
Brugada syndrome	19/33 (57.6%)
LQTS	2/4 (50.0%)
Early repolarization	1/5 (20.0%)
Idiopathic VF	1/2 (50.0%)
Uncategorized	4/8 (50.0%)
Dead	6 (11.5%)
ICD implantation	0/20 (0%)
ICD implantation + medication	0/7 (0%)
Medication	2/8 (25.0%)
No (against to treatment)	4/17 (23.5%)

PMVT/VF = polymorphic ventricular tachycardia or ventricular fibrillation; LQTS = long QT syndrome; ICD = implantable cardioverter defibrillator

two LQTS, one ERS, one idiopathic VF, and four uncategorized. The recurrent rate was 30% during the first year after the first episode of SCD. Subsequently, six patients in the non-ICD group died from recurrence of PMVT or VF (2 patients in the medication group, 4 patients in the refused management group) (Table 2).

Discussion

The survival rate of SCD in tertiary care center on average was 25 cases per year. Although successful resuscitation was performed, the hospital mortality rate was up to 40%. PED was considered as SCD etiology in 34% of the patients who survived at the hospital. The present study showed a higher incidence of PED in survivors of SCD than in western countries[2]. Possible reason for this was selection bias in the tertiary care center that had specialists of cardiac arrhythmia. Therefore, some patients who were referred from a rural province. The other reason was the difference incidence of arrhythmogenic disease in each ethnic group especially Brugada syndrome that was commonly found in East and Southeast Asia[13,14].

Clinical characteristic and etiology

The SCD from PED mainly occurred in young to middle aged healthy men. BS was the most common etiology of SCD in the Thai population followed by ERS. Healthy men could have ERS, normally discovered during patient checkup via ECG. It is usually a benign abnormality. Most patients will never have any cardiac symptoms, but the data supported the existence of the ERS, which causes SCD in the Thai population. The incidence of ERS in this study was lower than in western countries (9.6% in this study vs. 31% from Western countries)[8,15]. The clinical characteristics of ERS in the Thai population were similar to white ethnic populations, who were predominately male and had occurrences of this during sleep or rest, with a mean age 36±11 years (35±13 years in white), and history of previous unexplained syncope 40% (38% in white ethnic populations). The higher rate of families with a history of SCD in ERS in the Thai population (60% vs. 16%) could possibly be related to higher genetic penetration in the Thai population and further study might be needed. The mechanism of arrhythmogenic in ERS could be related to decrease in inward current e.g. I_{Na}, I_{Ca} and/or increase in outward repolarization current e.g. I_{to}, I_{k}, $I_{Cl(Ca)}$, $I_{K\text{-}Ach}$, $I_{k\text{-}ATP}$[15] that occurred predominantly in the apical region of left ventricular epicardium. There was a considerable amount of evidence suggesting that ERS might be the variant of BS because of both patients' clinical characteristics were similar, the event usually occurred in young to middle aged males, during sleeping or rest, and response to pharmacologic agents and neuromodulation[16]. There was an overlap of mechanism of inward and outward current that created arrhyhmogenic. A previous study showed simultaneous occurrence of ECG manifestation of BS with ST elevation in inferior or lateral leads (II, III, aVF or I, aVL) that were found in ERS (11%, 32 from 280 patients of BS)[17-21]. The authors observed two of the 33 patients (6%) with BS who had coincident ST elevation in inferior or lateral leads in the present study. In the group of ST elevation in right precordial leads, two patients had coincidence of early repolarization in inferior leads (Fig. 5). In the LQTS group, the youngest patient was 15 years old and the oldest was 43 years old, which implied that the LQTS containing risk of SCD at any age groups as in previous study[22].

Clinical outcome

The hospital mortality rate of the survivor of SCD caused by structural heart disease was obviously higher than the PED group (39% vs. 3.8%) because the PED usually occurred in healthy populations. Although clinical outcome at hospital discharge was good, many patients with PED could die from high recurrent rate

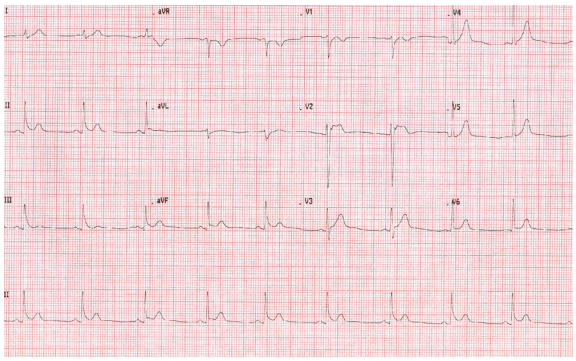


Fig. 5 A 23-year-old male from the northeastern part of Thailand survived sudden cardiac death on April 2006; he experienced SCD while walking down the street in the morning. A successful resuscitation was performed after he arrived at a nearby hospital; he completely recovered from this event. His 32-year-old father succumbed to suddenly death previously. The SECG showed the early repolarization with notching of terminal portion of QRS complex in right precordial leads and inferior leads. The HECG and provocation by class I antiarrhythmic drug was not performed in this case.

Table 3. The variable factors of recurrent of polymorphic ventricular tachycardia or ventricular fibrillation

Recurrent group (n = 27)	Cox model hazard ratios (95% CI)	p-value
Family history of SCD	1.31 (0.62, 2.76)	0.48
Previous symptom	3.04 (0.58, 15.98)	0.19
Inducible PMVT/VF in EP study	1.02 (0.23, 4.60)	0.98

CI = confidence interval; SCD = sudden cardiac death; PMVT = polymorphic ventricular tachycardia; VF = ventricular fibrillation; EP study = electrophysiologic study

of PMVT or VF (51.9%) especially within the first year (32%). As the previous study showed, beta-blocker was used to reduce cardiac arrhythmia in BS and LQTS but it could not prevent sudden cardiac death and ICD implantation was the treatment for prevent sudden cardiac death[23]. There was a trend toward association of previous symptom (hazard ratio (HR) = 3.04, 95% confidence interval (CI): 0.58, 15.98) with recurrent of PMVT/VF (Table 3).

Conclusion

SCD from PED in a Thai population was explored by the authors. BS was defined as the most common disease and commonly occurred in young to middle age healthy male with no history of cardiovascular symptom. Due to the high recurrent rate during the first year after the first symptom attacked, the authors recommended implantation of ICD within this period. HECG could unmask BS ECG especially when SECG showed obscured manifestation of BS, thus the authors recommend HECG for screening BS because it is non-invasive. The provocation by class I antiarrhythmic drugs should be applied to every survivor of sudden cardiac arrest who HECG could not unmask BS to exclude BS. ERS was interested as the second common etiology of SCD in the Thai population. Unfortunately, the authors were limited by a small population of ERS for the present study and therefore, need further study to evaluate the factors associated with high risk of SCD in ERS patients.

Potential conflicts of interest

None.

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การศึกษาลักษณะประชากรและการดำเนินโรคในผู้ป่วยที่รอดชีวิตจาก Primary electrical disease ในโรงพยาบาลจุฬาลงกรณ์

สุธาสินี ธรรมอารี, บัญชา ศันสนีย์วิทยกุล

ภูมิหลัง: Primary electrical disease เป็นกลุ่มโรคหัวใจเต้นผิดจังหวะที่รุนแรงในผู้ป่วยที่มีโครงสร้างของหัวใจปกติ ถึงแม้ประเทศในแถบเอเชียตะวันออกเฉียงใต้รวมถึงประเทศไทยเป็นประเทศที่มีความชุกของโรคชนิดนี้โดยเฉพาะ Brugada syndrome แต่ยังไม่มีข้อมูลเกี่ยวกับลักษณะประชากร สาเหตุและการดำเนินโรคของผู้ป่วยในกลุ่มโรคดังกล่าวในประเทศไทยที่เคยมีอาการหัวใจเต้นผิดจังหวะรุนแรงเกิดขึ้นมาแล้ว

วัตถุประสงค์: เพื่อศึกษาลักษณะของประชากร อาการแสดง สาเหตุ การดำเนินโรคในผู้ป่วยชาวไทยที่รอดชีวิตจาก sudden cardiac death ซึ่งเป็นผลจาก primary electrical disease

วัสดุและวิธีการ: หลังจากการศึกษาได้ผ่านการพิจารณาจากคณะกรรมการจริยธรรมการวิจัยของคณะแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัยแล้ว ผู้นิพนธ์ได้รวบรวมข้อมูลของผู้ป่วยที่รอดชีวิตจาก sudden cardiac death จากประวัติการรักษาผู้ป่วยใน และผู้ป่วยนอกที่มารับการรักษาที่โรงพยาบาลจุฬาลงกรณ์ตั้งแต่เดือนมกราคม พ.ศ. 2545 ถึง เดือนธันวาคม พ.ศ. 2551 ผู้นิพนธ์เก็บข้อมูลทางด้านลักษณะประชากร อาการแสดง สาเหตุ การรักษา และการดำเนินโรคของผู้ป่วยที่ได้รับการวินิจฉัยเป็น primary electrical disease ซึ่งมีโครงสร้างของหัวใจเป็นปกติ

ผลการศึกษา: ผู้ป่วยที่รอดชีวิตจาก sudden cardiac death ได้รับการวินิจฉัยเป็น primary electrical disease 52 ราย โดยมีอายุเฉลี่ย 39±12 ปี เป็นเพศชาย 49 ราย ส่วนใหญ่มีภูมิลำเนาอยู่ในภาคตะวันออกเฉียงเหนือ และภาคกลางของประเทศไทย ผู้ป่วยส่วนใหญ่มีอาการหัวใจเต้นผิดจังหวะอย่างรุนแรงในขณะนอนหลับ หรือ ขณะพัก (50% และ 15.4%) ผู้ป่วย 21 ราย (40%) มีประวัติสมาชิกในครอบครัวเคยเสียชีวิตกะทันหัน สาเหตุของอาการเกิดจาก Brugada syndrome (63.5%) Early repolarization (9.6%) Congenital long QT syndrome (7.7%) และ idiopathic VF (3.8%) นอกจากนี้ยังมีผู้ป่วยอีก 15.4% ที่มีคลื่นไฟฟ้าหัวใจผิดปกติแต่ไม่สามารถจัดอยู่ในกลุ่มโรคใดได้ ผู้ป่วยทั้งหมดมีอัตราการรอดชีวิตโดยไม่มีอาการผิดปกติทางระบบประสาทหลงเหลืออยู่หลังจากได้รับการช่วยเหลือฟื้นคืนชีวิต 78.8% ผู้ป่วย 27 ราย (51.9%) มีอาการหัวใจเต้นผิดจังหวะรุนแรงซ้ำระหว่างที่ติดตามอาการเป็นเวลา 56.5±35.4 เดือน (4 ถึง 137 เดือน) และโอกาสเกิดอาการซ้ำสูงสุดเกิดภายใน 1 ปี ที่ติดตามอาการ (32%)

สรุป: Brugada syndrome เป็นสาเหตุส่วนใหญ่ของภาวะหัวใจเต้นผิดจังหวะรุนแรงที่สามารถทำให้เกิดการเสียชีวิตกะทันหันในผู้ป่วยชาวไทยที่มีโครงสร้างของหัวใจปกติ ถึงแม้อัตราการรอดชีวิตสูงหลังให้การช่วยเหลือฟื้นคืนชีวิต แต่พบว่ามีโอกาสเกิดอาการซ้ำสูงโดยเฉพาะภายใน 1 ปีแรก ดังนั้นผู้นิพนธ์แนะนำให้รักษาผู้ป่วยด้วยการใส่เครื่องกระตุกไฟฟ้าหัวใจภายใน 1 ปี หลังเกิดอาการครั้งแรกเพื่อป้องกันการเสียชีวิตกะทันหันในผู้ป่วยกลุ่มนี้