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ORIGINAL ARTICLE

Significance of automated external defibrillator in identifying lethal ventricular arrhythmias

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Abstract

Automated electrical defibrillator (AED) is critical in saving children who develop unexpected cardiac arrest (CA), but its diagnostic capacity is not fully acknowledged. Retrospective cohort study of patients with aborted sudden cardiac death (SCD) was performed. Twenty-five patients (14 males) aged 1.3 to 17.5 years who presented with CA survived with prompt cardiopulmonary resuscitation. Eighteen patients had no prior cardiac diagnosis. Cardiac arrest occurred in 10 patients with more than moderate exercise, in 7 with light exercise, and in 8 at rest (including one during sleep). Twenty-two patients were resuscitated with AED, all of which were recognized as a shockable cardiac rhythm. Thorough investigations revealed 6 ion channelopathies (4 catecholaminergic polymorphic ventricular tachycardia, one long QT syndrome, and one Brugada syndrome), 5 congenital heart disease (including 2 with coronary artery obstruction), 6 cardiomyopathies, 2 myocarditis, and 2 miscellaneous. Four patients had no identifiable heart disease. In 5 patients, the downloaded AED-recorded rhythm strip delineated the underlying arrhythmias and their responses to electrical shocks. Four patients who presented with generalized seizure at rest were initially managed for seizure disorder until AED recording identified lethal ventricular arrhythmias.

Conclusions: AED reliably identifies the underlying lethal ventricular arrhythmias in addition to aborting SCD.

What is Known:

- *Although infrequent in children, sudden cardiac death (SCD) is often an unexpected and tragic event. The etiology is diverse and sometimes remains unknown despite extensive investigations.*
- *Automated external defibrillator (AED) is both therapeutic in aborting SCD and diagnostic in identifying the underlying lethal ventricular arrhythmias. However, the diagnostic aspect of AED is under-acknowledged by most medical providers.*

What is New:

- *Four of 25 patients (16%) were initially managed for possible seizure disorders until AED recording identified lethal ventricular arrhythmia.*
- *The AED recording of the lethal arrhythmia during cardiopulmonary resuscitation (CPR) should always be obtained as it plays a crucial role in the decision-making process before ICD implantation. All medical providers should become familiar with downloading cardiac rhythm strips from AED when requested.*

Keywords Automated external defibrillator (AED) · Cardiac arrest (CA) · Implantable cardioverter defibrillator (ICD) · Sudden cardiac death (SCD) · Sudden acute death syndrome (SADS) · Ventricular fibrillation (VF)

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Abbreviations

AED	Automated external defibrillator
CA	Cardiac arrest
CPR	Cardiopulmonary resuscitation
CT	Computed tomography
ECG	Electrocardiogram
ED	Emergency department
EMT	Emergency medical technician
EST	Exercise stress test
HCM	Hypertrophic cardiomyopathy
ICD	Implantable cardioverter defibrillator
LQTS	Long QT syndrome
LVOT	LV outflow tract
MRI	Magnetic resonance imaging
SADS	Sudden arrhythmic death syndrome
SCD	Sudden cardiac death
VF	Ventricular fibrillation
VT	Ventricular tachycardia

Introduction

Ventricular fibrillation (VF) and polymorphic ventricular tachycardia (VT) consist of a rare lethal cardiac arrhythmia often responsible for sudden cardiac death (SCD) in children [1, 2]. The etiology of these lethal rhythms includes congenital ion channelopathies, myocarditis, myocardial ischemia/infarction, cardiomyopathies, and advanced heart failure, and they can occur after surgical repair of certain congenital heart diseases [3–8]. In some cases, the cause remains unknown [5]. Although rare, SCD can have an immense impact on society because of its unexpected and catastrophic nature. Prompt recognition and treatment of VF with cardiopulmonary resuscitation (CPR) and automated external defibrillator (AED) are essential [9]. However, risk stratification or screening for SCD in children and adolescents has not been well established [3, 10, 11]. Moreover, the underlying molecular mechanism of VF is not fully understood [12].

The incidence of VF was previously thought to be very rare among children compared with adults, but recent reports suggest that the numbers may be underestimated in a pediatric population [2, 9, 13]. Sudden arrhythmic death syndrome (SADS) is defined as a lethal cardiac rhythm, VF or polymorphic VT, without identifiable cardiac abnormalities on the autopsy [14]. Clinical presentation of SADS is highly variable; only 20 to 30% of cases occur in association with exercise, whereas 30 to 50% are reported to occur at rest or during sleep [14]. Unlike cardiomyopathies and congenital heart disease, identification of a high-risk population for SADS is difficult because patients are usually asymptomatic until their first clinical encounter including SCD.

Here, we present aborted SCD cases experienced in our center. The presence of a lethal arrhythmia was identified by AED as having a shockable cardiac rhythm. The importance of AED as an identifying device for the underlying lethal arrhythmias is discussed.

Patients and methods

We retrospectively reviewed the medical records of patients less than 18 years of age who underwent implantable cardioverter defibrillator (ICD) implantation at the ICD/Arrhythmia Clinic, Nemours Cardiac Center, Nemours/Alfred I. duPont Hospital for Children, Wilmington, DE, from 2000 to 2017. This study was approved by the Institutional Review Board of the hospital. We analyzed patient age, sex, race, medical history, family history, underlying cardiac problems, previous symptoms, and activity at sudden cardiac arrest (CA). All patients had electrocardiogram (ECG) and echocardiogram. Selected patients also underwent cardiac magnetic resonance imaging (cMRI), exercise stress test (EST), cardiac catheterization, and genetic testing. Downloaded AED rhythm recordings were examined by the cardiologists (T.T. and J.T.). The data were shown as mean ± standard deviation (SD).

Results

Medical records of 52 patients (male 29 and female 23) who underwent ICD implantation under class I or class IIa indication according to the American Heart Association, American College of Cardiology, and European Society of Cardiology guidelines [15–17] were reviewed. We focused on 25 patients with aborted SCD who underwent ICD implantation for secondary prevention of SCD (14 males and 11 females) (Table 1). The age at CA or syncope ranged from 1.3 to 17.5 years (13.5 ± 3.6 years). Twenty-two patients were resuscitated with AED either by bystanders on site or by emergency medical technician (EMT), in which 10 were reported as VF, 5 as polymorphic VT/VF, 6 as polymorphic VT, and 1 as sustained VT (rate 250). Twenty patients presented with syncope or CA, 4 patients with generalized seizure, and 1 with palpitation followed by syncope. In relation to physical activity at the time of CA, 10 patients (40%) were engaged in more than moderate exercise and 7 (28%) were engaged in light exercise, but 8 (32%) were at rest (including 1 during sleep). After thorough clinical investigations, 6 patients were found to have congenital ion channelopathies, 5 with congenital heart disease (CHD), 6 with cardiomyopathies, 2 with myocarditis, 1 with complete heart block (CHB), and 1 with Marfan syndrome with mitral valve prolapse, but 4 patients (16%) had no identifiable underlying heart disease. Eighteen patients (72%) had no prior cardiac diagnosis at the time of CA. Although 7 patients were known to have cardiac abnormalities—5 with CHD, 1 with CHB, and 1 with Marfan syndrome with mitral valve prolapse—the occurrence of lethal arrhythmia or CA was

Table 1 Classification of 25 patients who underwent ICD implantation for secondary prevention of SCD

Age (Y)	Sex	Diagnosis	Presentation	Activities	AED	FH of SCD
I. Congenital Ion Channelopathies (n = 6)						
11.4	M	CPVT	Syncope	> Mod exercise	PMVT/VF	N
17.3	F	CPVT	Syncope	> Mod exercise	PMVT	N
9	F	CPVT	Syncope	> Mod exercise		**Y**
16	F	CPVT	Syncope	> Mod exercise	PMVT	**Y**
11.1	F	LQTS type 3 (Case 5)	Cardiac arrest	Light exercise	PMVT/VF	N
1.3	M	Brugada syndrome	Seizure	At rest (home)	VF	**Y**
II. Congenital Heart Disease (n = 5)						
14.9	M	Unrepaired PAPVC	Cardiac arrest	> Mod exercise	VF	N
14	M	VSD s/p repair LCA obstruction	Syncope	> Mod exercise	VT (250*)	N
8.6	F	d-TGA s/p ASO LCA obstruction	Cardiac arrest	> Mod exercise	VF	N
14	M	Truncus Arteriosus s/p repair s/p multiple RVOT revisions	Syncope	Light exercise	PMVT	N
14	F	Severe Ebstein's anomaly s/p BDG, TV replacement s/p RV and RA replication	Syncope	At rest (classroom)	VF	N
III. Cardiomyopathies (n = 6)						
12	F	HCM s/p myomectomy	Syncope	Light exercise		N
16.1	M	HCM (apical) (Case 2)	Cardiac arrest	> Mod exercise	VF	N
11.2	M	DCM (Barth syndrome)	Cardiac arrest	At rest (home)	VF	N
10.7	M	RCM	Syncope	> Mod exercise		N
13.5	M	LVNC	Cardiac arrest	At rest (classroom)	VF	N
17	F	ARVC	Syncope	> Mod exercise	PMVT	N
iV. Myocarditis (n = 2)						
15.3	M	Coxsackie myocarditis	Cardiac arrest	At rest (hospital)	VF	N
16.6	M	Lyme carditis	Cardiac arrest	Light exercise (school)	VF	N
V. Miscellaneous (n = 2)						
15.5	F	CHB with DDD pacer	Cardiac arrest	Light exercise (school)	PMVT	N
14	F	Marfan syndrome Mitral valve prolapse	Cardiac arrest	Light exercise (park)	PMVT/VF	N
VI. Unknown (n = 4)						
17.5	M	Unknown (Case 4)	Cardiac arrest	Light exercise	PMVT	N
15.3	M	Unknown (Case 1)	Seizure	At rest (classroom)	VF	N
15.8	M	Unknown	Seizure	During sleep§	PMVT/VF	N
16	F	Unknown (Case 3)	Seizure	At rest (classroom)	PMVT/VF	N

ICD: implantable cardioverter device, SCD: sudden cardiac death, AED: automated external defibrillator, FH: family history, CPVT: catecholaminergic polymorphic ventricular tachycardia, CA: cardiac arrest, PMVT: polymorphic ventricular tachycardia, VF: ventricular fibrillation, N: no, Y: yes, LQTS: long QT syndrome, PAPVC: partial anomalous pulmonary venous connection, VSD: ventricular septal defect, s/p: status-post, VT: ventricular tachycardia, LCA: left coronary artery, d-TGA: d-transposition of the great arteries, ASO: arterial switch operation, SVT: supraventricular tachycardia, RVOT: right ventricular outflow tract, BDG: bidirectional Glenn procedure, TV: tricuspid valve, RA: right atrium, RV: right ventricle, HCM: hypertrophic cardiomyopathy, DCM: dilated cardiomyopathy, RCM: restrictive cardiomyopathy, LVNC: left ventricular non-compaction, CHB: complete heart block.

*Heart rate of VT. †Detected by loop recorder. Electrical shock was not applied. §Resulted in severe neurological deficit.

not anticipated. A positive family history of SCD was present only in 3 patients (12%). Three patients developed severe long-term neurological deficit after resuscitation.

Although we obtained reports of AED findings or clinical responses to defibrillation from the referring institutions, we were able to obtain actual rhythm strips only from 5 patients. Clinical presentations of these 5 patients are reviewed in conjunction with diagnostic information generated by AED.

Case 1

Patient 1 is a 15-year-old, previously healthy male who suddenly collapsed in the classroom with a generalized seizure.

Because of his unresponsiveness, CPR was initiated, followed by electrical defibrillation by AED on site. He recovered hemodynamic stability after electrical shock, but he remained unconscious. En route to our emergency department (ED), he again developed generalized seizure. He was initially investigated for possible seizure disorders. Head computed tomography (CT) and magnetic resonance imaging (MRI) results were negative. Electroencephalography results were not specific. On the following day, a recorded AED rhythm strip demonstrated VF before electrical shock (Fig. 1). He was transferred to the cardiology service for further management. He was alert, oriented, and hemodynamically stable. His physical examination was normal with no family history of SCD or significant past medical history. Echocardiogram, ECG, and cardiac MRI (cMRI) results were normal. Exercise stress test showed normal exercise performance with no abnormal ECG findings. He was discharged home after ICD implantation with nadolol for unknown cause of VF.

Case 2

Patient 2 is a 16-year-old, previously healthy male who collapsed after completing intense training drills in a school basketball team practice. Immediately, CPR was started as he was unresponsive and pulseless, and electrical shock was delivered by AED. He was alert and hemodynamically stable by the time he arrived at the ED. Initial ECG results were abnormal, showing left ventricular hypertrophy pattern with negative T waves in the lateral leads (Fig. 2), which persisted on subsequent ECGs. Echocardiogram and cMRI revealed LV apical hypertrophy but no evidence of myocardial abnormality. Exercise stress test showed normal exercise performance with normal blood pressure response to maximum exercise. Five days after admission, the cardiac rhythm strip from AED at the initial CPR became available, which demonstrated the presence of VF. He underwent ICD implantation under the diagnosis of apical hypertrophic cardiomyopathy (HCM). He was discharged home with daily nadolol.

Case 3

A 17-year-old female suddenly collapsed in the hallway of her school building while talking with her friend. She had been complaining of headache and general lethargy for about a week and had not felt well since the morning. She was found apneic and pulseless, for which bystander CPR was initiated, followed by defibrillation by AED. On arrival at the ED, she developed generalized seizure. Upon admission, ECG and echocardiogram results were within normal limits. On the fourth hospital day, as she developed nonsustained VT, she underwent cardiac catheterization with endomyocardial biopsy, which was negative for myocarditis or cardiomyopathy with normal hemodynamic status. Cardiac MRI results were negative for arrhythmogenic right ventricular cardiomyopathy (ARVC). Later, preshock AED recording at the initial CPR demonstrated VF that was successfully converted to sinus rhythm after defibrillation (Fig. 3). Epinephrine/procainamide challenge test results were negative for pathological QT prolongation or Brugada pattern of ST elevation. She underwent ICD implantation and was discharged home with atenolol. The diagnosis remained unclear (idiopathic VF).

Case 4

Patient 4 is a previously healthy, 17-year-old male who collapsed on the playground of the detention center while playing basketball by himself. According to the witness, he slowed down and fell over slowly on the court, then did not move or get up. Bystander CPR was started, and AED delivered two shocks. He was transferred to our pediatric intensive care unit for possible seizure disorder. Head CT and MRI results were negative. Downloaded AED recording revealed polymorphic VT (Torsades de Pointes). He was transferred to the cardiac center, where ECG, echocardiogram, and cMRI results were all within normal limits. The EST showed mildly decreased exercise tolerance, normal QTc response to exercise, and no exercise-induced arrhythmia. Procainamide challenge test results were negative for Brugada syndrome. He underwent ICD placement for unknown cardiac etiology of polymorphic VT and was discharged home with daily nadolol.

Case 5

A 10-year-old female collapsed on the school playground. She was running laps during gym class and felt "funny." She screamed to her friend "help me" and then collapsed on the ground. She was started on CPR and was shocked once with a school AED as she was pulseless. A second shock was given when EMT arrived 6 min later. On arrival at the ED, she was hemodynamically stable with borderline prolonged QTc. Cardiac MRI was unremarkable. The EST demonstrated normal QT adaptation to exercise under submaximal effort. During general anesthesia for implantation of ICD, however, her QTc was found to be prolonged (495 ms). Genetic testing confirmed class I mutation in SCN5A (Ala 572 Phe) consistent with congenital long QT syndrome (LQTS) type 3.

The clinical presentation of these 5 cases is summarized in Table 2. Two patients collapsed at rest, 2 during light exercise, and 1 after intense exercise. All cases were witnessed. Patients 1 and 2 were active athletes participating in competitive sports without any problems, but the other three were not trained athletes. Genetic testing was performed in 4 patients, the results of which showed only patient 5 was positive for LQTS type 3.

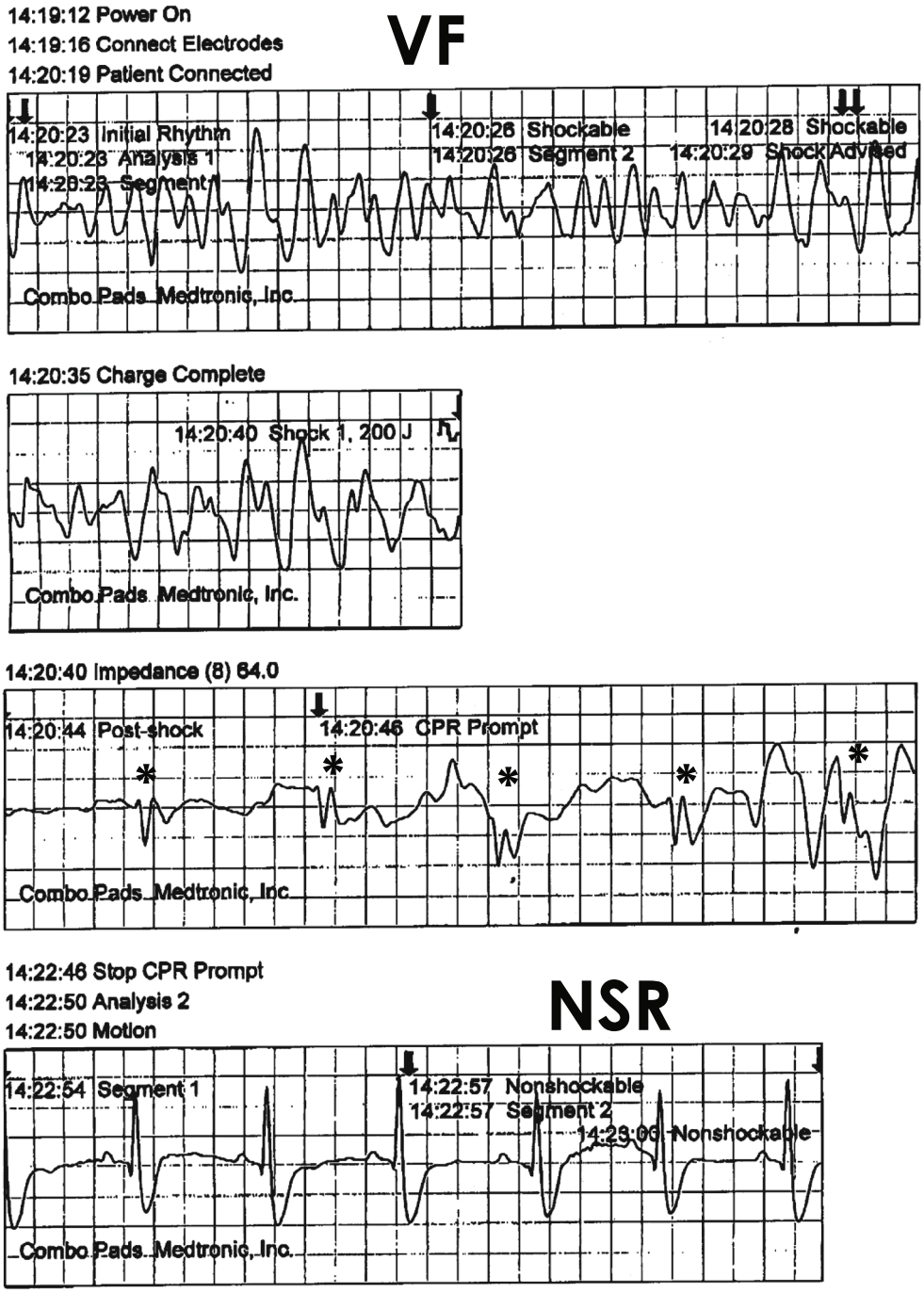

Fig. 1 Automated external defibrillation recording during CPR on patient 1. Initial rhythm was ventricular fibrillation (VF), which was converted to ventricular escape rhythm (*) after electrical shock (200 J) was delivered, followed by normal sinus rhythm (NSR)

Discussion

In this study, we found the following important clinical characteristics of aborted SCD in children: (1) 18 patients (72%) had no prior cardiac diagnosis; (2) sudden cardiac death was not always triggered by an intense exercise, as 8 cardiac arrests (32%) occurred at rest and 7 (28%) with only light physical activities; (3) 4 patients (16%) presented with a generalized seizure, which could delay the correct diagnosis; and (4) the use of an AED not only is life-saving but also can be diagnostic, providing rhythm strips that should always be saved for later interpretation by the cardiologist.

Ventricular fibrillation is a rare lethal cardiac rhythm previously thought to be responsible for approximately 10 to 20% of SCD in children and adolescents [13], but the incidence may be underestimated [18]. Recent reports reveal a higher incidence of VF in SCD, 57.4% [19] to 67.6% [20], probably due to more frequent deployment of AED in recent years [19–21]. Polymorphic VT can be a pre-VF condition that should be regarded as

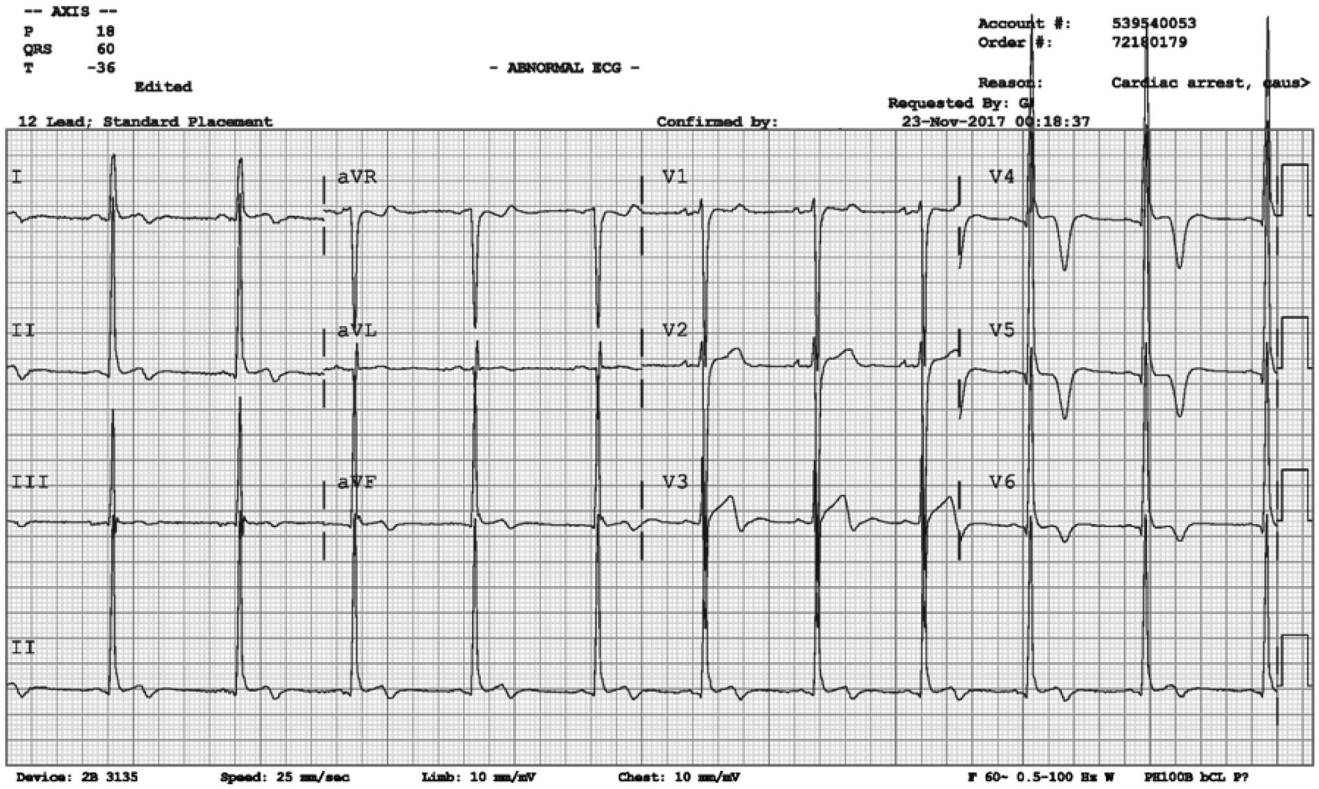

Fig. 2 Initial abnormal ECG of patient 2 at the emergency department (ED). Note T wave inversion in the lateral leads and prominent LV voltages (high R waves in V5 and V6 and deep S waves in V1 and V2)

the same clinical entity as lethal cardiac arrhythmia [22]. The etiology of VF encompasses the following five entities: (1) congenital ion channelopathies (long QT syndrome, Brugada syndrome, and catecholaminergic polymorphic VT); (2) cardiomyopathies (HCM, DCM, RCM, and LV non-compaction) and other myocardial diseases (myocarditis); (3) sudden myocardial ischemia due to congenital or acquired coronary artery abnormalities; (4) after heart surgery for certain congenital heart diseases; and (5) environmental factors (electrolyte abnormalities, hypothermia, and medications/drugs) [2–4, 7, 23, 24]. In repaired CHD, especially in tetralogy of Fallot (TOF), ventricular arrhythmia can occur because of a formation of dense fibrosis after surgical incision, because of patch materials, or because of associated hemodynamic abnormalities causing ventricular hypertrophy and dilatation [25]. Of our 5 patients with CHD, the fourth and fifth patients may have similar pathological substrate to TOF, whereas ventricular arrhythmia was most likely induced by myocardial ischemia in the second and third patients (Table 1). However, VF frequently occurs unexpectedly in previously healthy individuals as a first clinical presentation without detectable underlying cardiac pathology. In a recent study in Denmark, 43% of 87 autopsied children from 1 to 18 years of age with SCD did not show identifiable abnormalities [26]. In a similar study from Spain reporting 34 sudden unexpected non-violent deaths, 10 cases (29%) were of cardiac etiology and 11 (32%) were unknown [5]. In the United Kingdom, a structurally normal heart was reported in 56% of pediatric and adolescent (< 18 years of age) and 44% of young adult (18 to 35 years of age) SCD victims [22]. A substantial number of episodes of SCD have no identifiable cause.

Here, we presented 25 cases of aborted SCD, including 5 cases of documented VF or polymorphic VT that occurred in patients as a first presentation with no known prior cardiac abnormalities. The diagnosis of lethal cardiac arrhythmias would not have been promptly made without AED application immediately after the collapse. Generalized seizure may be an important presentation of SCD in children, but SCD should not be misinterpreted as a primary epileptic disorder [27]. Electrical shock was delivered upon the recognition of shockable cardiac rhythm by AED, but the definite identification of lethal ventricular arrhythmia could not be made until a few days later when the preshock AED rhythm strip was available. Without actively petitioning schools and/or EMT to retrieve actual rhythm strips during AED applications, the proper decision-making for ICD implantation may not have taken place. Training of medical staff to promptly download AED

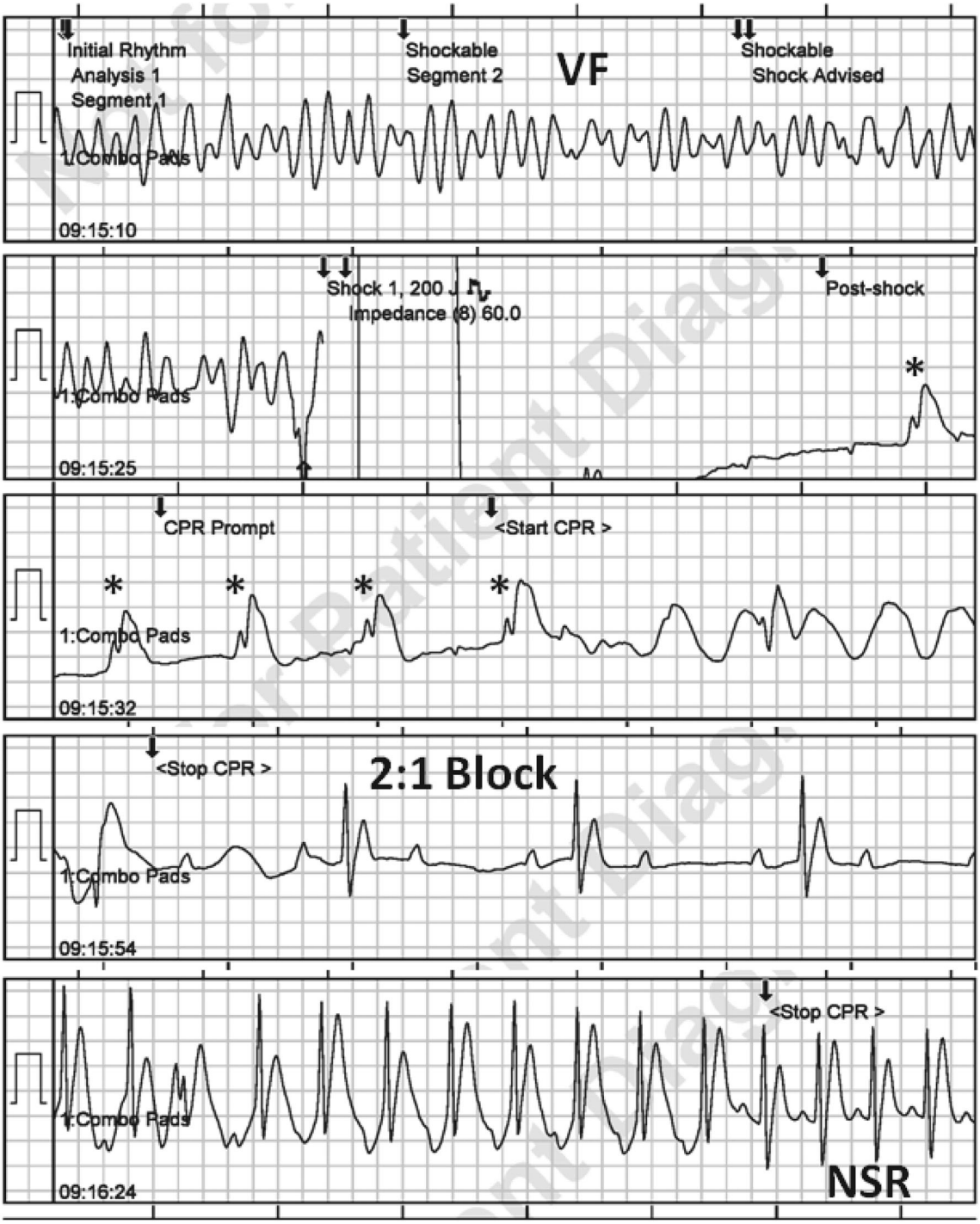

Fig. 3 Automated external defibrillation recording during CPR on patient 3. Initial ventricular fibrillation (VF) was converted to slow ventricular escape rhythm (*) by electrical defibrillation (200 J), for which cardiopulmonary resuscitation (CPR) was resumed. After CPR for 20 s, AED recording showed 2:1 block, which subsequently transitioned to normal sinus rhythm (NSR)

recordings is a crucial aspect of AED implementation, as further management strategy is highly dependent upon the mechanism of CA.

Sensitivity and specificity of AED in detecting VF and pulseless VT in children have been well studied. Atkins et al. initially reported that sensitivity and specificity for accurate rhythm analysis in 18 children with age ranging from 5 to 15 years were 88% and 100%, respectively [9]. Efficacy and safety of AED in children and adolescents have been well established, as both sensitivity and specificity were nearly 100% in recent reports [28–30]. Implementation of AED was proven effective and safe in preventing SCD in

Table 2 Clinical summary of 5 patients who were noted to have ventricular fibrillation (VF) or polymorphic ventricular tachycardia (VT) by automated external defibrillator (AED)

Patient	1	2	3	4	5
Age	15	16	16	17	10
Sex	M	M	F	M	F
Race	Caucasian	Black	Black	Black	Hispanic
Presentation	GS at rest	Syncope after intense EX	Syncope at rest	Syncope at light EX	Syncope at light EX
Place	School (classroom)	School (gym)	School (hallway)	Detention center (playground)	School (playground)
Preceding symptoms	None	None	Headache/lethargy	None	None
ECG	Normal	Abnormal Inverted T wave in V6, LVH	Normal	Normal	Normal
Echo	Normal	Borderline LV apical hypertrophy (12 mm)	Normal	Normal	Normal
EST	Normal EXP Normal BP response No VEB induced	Normal EXP Normal BP response No ectopies induced Stress echo: ↑ dynamic LVOTO	Not performed	Low EXP Normal BP response No ectopies induced	Low EXP Mildly blunted BP response No ectopies induced
cMRI	Normal	Mild mid and apical LVH with significant mid cavitary obstruction in systole	Normal	Borderline ↑ VS thickness (1.3 cm)	Normal
Genetic test[†]	HVUS	HVUS	HVUS	Not performed	SCN5A mutation
Other tests			Cath & EMB: normal EPI/PCA challenge: negative		
AED tracing	VF	VF	VF	Polymorphic VT	Polymorphic VT
Diagnosis	Unknown	Apical HCM	Unknown	Unknown	LQTS type 3

GS generalized seizure, *EX* exercise, *ECG* electrocardiogram, *LVH* left ventricular hypertrophy, *EST* exercise stress test, *EXP* exercise performance, *BP* blood pressure, *VEB* ventricular ectopic beats, *LVOTO* left ventricular outflow obstruction, *cMRI* cardiac magnetic resonance imaging, *VS* ventricular septum, *HVUS* heterozygous for variants of unknown significance, *EMB* endomyocardial biopsy, *EPI/PCA* epinephrine/procainamide, *HCM* hypertrophic cardiomyopathy, *LQTS* long QT syndrome

[†] We used "comprehensive arrhythmia panel" that covers LQTS, CPVT, Brugada syndrome, and arrhythmogenic right ventricular cardiomyopathy

elementary and middle school students in Japan [31] and even in infants and small children [32]. Installment of AED has become more common in schools in general, but low rates of CPR training and overall compliance with guidelines in the school staff remain a concern [33]. It is imperative to recognize that AED also serves as an important diagnostic tool to identify lethal cardiac rhythm that would provide clinicians with solid therapeutic guidelines for prevention of future SCD [21]. Although SCD during exercise is well highlighted among adolescents [3, 23, 34], SCD also occurs frequently at rest or during sleep [14, 35–38]. In our 25 cases of aborted SCD, 8 cases (32%) occurred at rest. According to Mellor et al. who analyzed 146 patients with SCD aged 1 to 17 years (median 14 years) with postmortem normal cardiac morphology, SCD occurred in 40 cases (27%) during exercise, whereas 75 cases (51%) were encountered at rest or during sleep. The incidence of SCD at rest or during sleep increases in older patients (71% in > 35 years of age of 335 patients) [14]. Although exercise is a common association with SCD in adolescents (around 20 to 30%), SCD is still more frequent at rest or during sleep (30 to 70%) [14, 23, 26, 36]. Because of the high incidence of SCD at rest or during sleep in young athletes, identification of life-threatening cardiac status and subsequent disqualification from competitive sports may not prevent SCD in all athletes harboring potentially fatal cardiac events [7, 39].

Our study has certain limitations. First, this is a retrospective study in a single institution with a relatively small sample size. Second, there is a selection bias as our study does not include those who did not survive resuscitation efforts. The patients who died unwitnessed or those who were refractory to CPR were not included in this study. In our cohort, all 4 patients with CPVT underwent ICD implantation because they experienced cardiac arrest despite what was considered optimal therapy at that time. These patients were diagnosed prior to the recent report indicating that ICD does not alter natural course of CPVT and that CPVT may be well controlled by β-blockers, flecainide, and left cardiac sympathetic denervation [40]. Despite these limitations, our

current 26 cases of aborted SCD and 5 cases of AED-recorded lethal arrhythmia with a detailed clinical profile underscore the importance of AED use in public places for preventing SCD and the potential for valuable diagnostic information.

Conclusion

Our current study suggests that prompt application of AED in sudden CA is essential not only in aborting SCD but also in identifying lethal ventricular arrhythmias and ultimately justifying ICD implantation as a long-term management to prevent SCD. With advances in technology in analyzing and recording cardiac rhythm, AED has become a powerful and reliable tool in identifying VF and polymorphic VT that comprise SADS. It is imperative that first-line providers and tertiary providers be familiar with downloading cardiac rhythm strips from AED during CPR, which provides critical information for making correct diagnosis and proper management.

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Authors' contributions T. T. conceptualized and designed this retrospective study, collected and organized the data, wrote the manuscript, and revised the manuscript according to the co-authors' feedback. E. G. reviewed the individual patient charts in detail and collected the data. J. T. is in charge of ICD clinic who provided critical information regarding AED recording and patients' profiles, carefully examined the provided data, and critically read the manuscript. All authors read and approved the final manuscript as submitted and agree to be accountable for all aspects of the work.

Compliance with ethical standards

Ethical approval All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standard.

Conflict of interest The authors declare that they have no conflict of interest.

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