VIA MEDICA

CASE REPORT

Cardiology Journal
2007, Vol. 14, No. 1, pp. 91–94

ISSN 1507–4145

Lightning-induced ventricular fibrillation

Deborah R. Levy[1] and Toshio Akiyama[2]

[1]HVMA Division of Medicine, Brigham & Women's and Faulkner Hospital, Boston, MA, USA
[2]Covance Cardiovascular Sciences, Reno, Nevada, USA

Abstract

We present a case of a previously healthy 17 year-old white male boy scout who collapsed after a lightning strike, and was found to be in ventricular fibrillation when emergency medical services arrived. The ventricular fibrillation was defibrillated into sinus rhythm after a single direct current (DC) countershock. However, the patient has remained in coma. Commotio cordis, sudden cardiac death from low-energy chest wall impact, is a phenomenon in which an exactly timed and located blow on the chest during the cardiac cycle results in ventricular fibrillation. Commotio cordis and electrical shock can both result in ventricular arrhythmias. We speculate that in this patient, ventricular fibrillation began immediately after the lightning, which probably struck at the peak of the T wave. (Cardiol J 2007; 14: 91–94)

Key words: lighting, commotio cordis

Introduction

Commotio cordis, sudden cardiac death from low-energy chest wall impact, is a phenomenon in which an exactly timed and located blow on the chest during the cardiac cycle results in ventricular fibrillation (VF). Impact occurring directly over the heart, especially localized over the apex or base of the left ventricle, and 10 to 30 ms before the peak of the T wave, is known to precipitate VF [1].

More commonly occurring than commotio cordis, an electrical stimulus or a premature ventricular beat can induce VF if precisely timed during the vulnerable period of repolarization during the cardiac cycle. As a result of this phenomenon, an electrical stimulus is routinely utilized to induce VF, during the implantation and testing prior to implantation of an implantable defibrillator. In a patient with myocardial ischemia or infarction, the R on T phenomenon can occur as the result of a premature ventricular beat, subsequently triggering ventricular tachycardia or fibrillation [2, 3].

Lightning strikes can result in a variety of injuries including burns and non-cardiac injuries, as well as cardiac arrest and subsequent brain death. Currently, an estimated 50–300 deaths per year in the United States are attributed to lightning causes, with an additional four to five times that number of injuries [4]. One manner in which lightning injury may occur is when a person is touching an object that is part of the pathway of lightning current, the phenomenon of side splash or flash as the current flows through the pathway of least resistance. Blunt injury can also occur, either if the person is thrown as the result of a massive muscular contraction or due to an explosive force.

Cardiovascular injuries that can result from lightning strikes include hypertension, tachycardia, non-specific electrocardiographic (ECG) changes, prolonged QT intervals, and myocardial injury [5, 6]. Lichtenberg et al. [6] followed nineteen victims of lightning strikes with several markers of myocardial damage: serial ECG, creatinine kinase MB (CK-MB) fractionation, and echocardiographic imaging. Early effects of exposure were evident on ECG, and corresponded with segmental or global ventricular dysfunction, with or without pericardial effusion,

Address for correspondence: Deborah R. Levy, MD
Hospitalist, HVMA Division of Medicine
Brigham & Women's and Faulkner Hospital
1153 Centre Street, Boston, MA 02130, USA
e-mail: drlevy@partners.org.
Received: 11.09.2006 Accepted: 2.10.2006

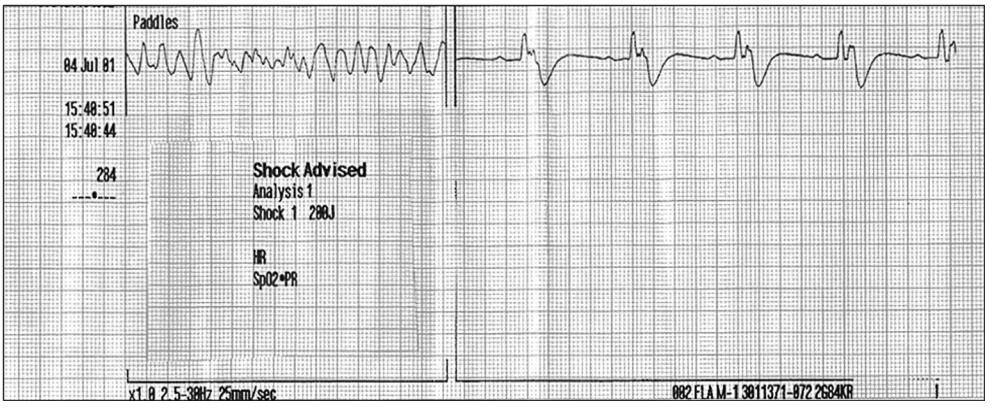

Figure 1. Initial rhythm on EMS arrival (ventricular fibrillation); rhythm after 200 Joule shock delivery (sinus rhythm at 60 bpm with first degree AV block and an intraventricular conduction delay).

on echocardiography. They concluded that only direct lightning strike hits would result in a prolonged QT interval or echocardiographic abnormalities, and that abnormal serum myocardial markers are also useful in assessing the degree of myocardial damage.

The degree of electrical bodily injury is influenced by the amount of current flowing through an object and the human body. The intensity of the current is in turn influenced by the voltage and resistance pathways in the body. The pathway, duration, amperage, and resistance of the tissues all play a role in the type and severity of the electrical injury. While 1 to 4 mA causes a tingling sensation, an electrical impulse of 60 to 120 mA could result in VF if a substantial portion of that current flows through the heart during a critical period of a cardiac cycle. Unlike electrical injuries involving AC currents, lightning-related current is not classified as direct or alternating. An extremely short period of current flows through the affected body, and can result in muscular spasms and ventricular arrhythmias [6]. Since the lightning strike is required to occur at the exact time around the peak of the T wave in order to precipitate VF, resultant asystole is considered to occur more commonly than ventricular fibrillation. Prolonged respiratory arrest resulting in severe hypoxia may also precipitate terminally refractory VF or asystole.

The following case is an example of both commotio cordis due to trauma to the chest and electrical shock from a lightning strike causing VF. Due to the lack of external evidence of obvious entry or exit sites, we suspect that the lightning traveled either through side splash or ground strike. Although the patient was resuscitated, prolonged cerebral hypoperfusion prior to the successful defibrillation of VF resulted in a permanent vegetative state. This case appears to be the first documentation of VF directly triggered by lightning.

Case report

A previously healthy 17 year-old white male boy scout collapsed after a lightning strike, and was found to be in VF when emergency medical services arrived approximately 10 min after the lightning strike. This boy scout was with his troupe, returning from target shooting, holding a rifle beneath a tree at the time of the lightning strike. The patient dropped to the ground and first aid-trained staff initiated 2-man CPR. Fifteen minutes after collapsing, he received a 200 Joule DC countershock for VF, which readily restored a sinus rhythm at 60 beats per minute with first degree AV block and an intraventricular conduction delay (Fig. 1). However, initially he remained pulseless. The patient was intubated, and epinephrine and atropine were administered via the ET tube. CPR continued until a pulse became palpable at 180 beats per minute corresponding with supraventricular tachycardia, 25 min after initial collapse. The patient's care was transferred to a community hospital. On physical examination, the patient was in deep coma and had mottling of his bilateral lower extremities to the knees, without specific evidence of surface burns.

Pertinent data: EKG on arrival to the community hospital revealed sinus tachycardia with a normal axis, and peaked T waves in the precordial leads V2–V4 (Fig. 2). Twenty-four hours later, EKG revealed normal sinus rhythm with high QRS voltage and early repolarization. Initial labs included potassium 3.5 mEq/L, Lactate 3.0, CK 327 IU/L (upper limit of normal: 230), no MB fractionation was

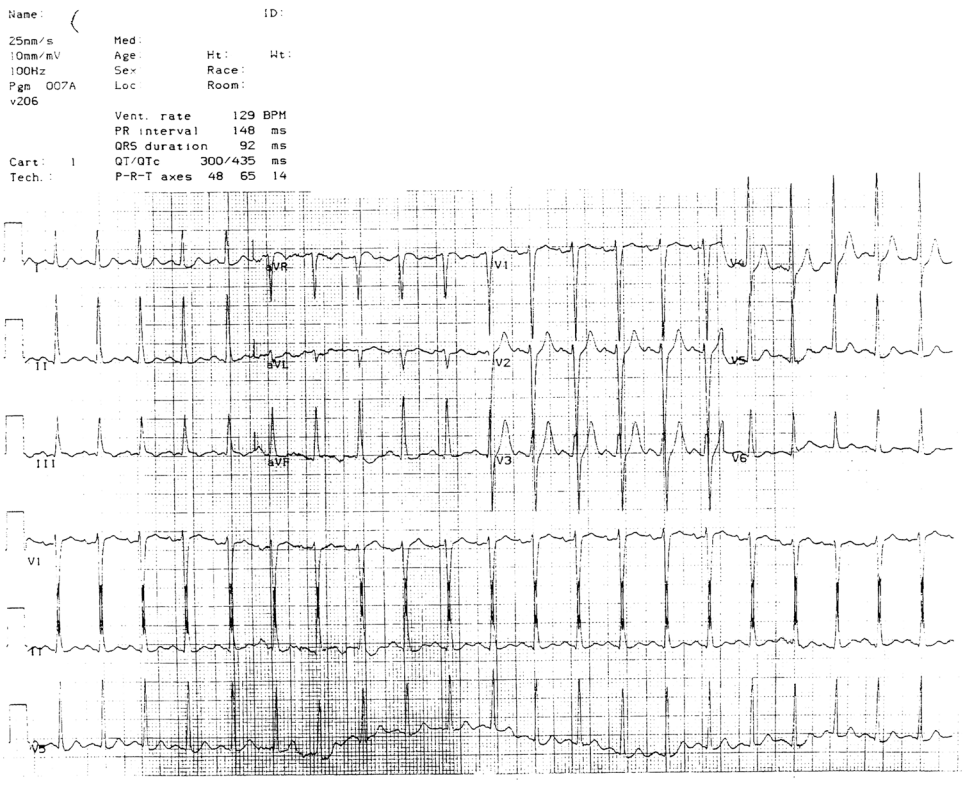

Figure 2. Twelve-lead EKG on arrival to the community hospital revealed sinus tachycardia with a normal axis, and peaked T waves in the precordial leads V2–V4.

performed, BUN 15 mg/dL and Cr of 1.4 mg/dL, and INR of 1.2. Twenty-four hours later, troponin T was 0.95 ng/mL (upper limit of normal: 0.10), CK 196 IU/L, with CK-MB 85 IU/L, the CK-MB fraction was 16%. Chest X-ray and Head CT were both within normal limits. Cardiac echo at that time showed no significant valvular or wall motion abnormalities and normal left ventricular function. The patient remained in coma, and EEG and MRI were consistent with severe hypoxic-ischemic brain injury.

Discussion

Commotio cordis and precisely timed electrical shock can both result in cardiac arrhythmias. Lightning-related injuries more commonly result in asystole and respiratory arrest than in VF, based on the flow of intense transient current and the force, which often results from the lightning strike. Our case provides a diagnostic challenge as to which abnormal cardiac rhythm he was in immediately after the lightning strike, since an electrocardiographic monitor was begun ten minutes after the lightning strike, and before any electrical therapeutic measures were taken.

The events leading up to the cardiac arrest in this patient could have occurred in several different scenarios. We focused on the two most likely, including VF induced by the exactly timed lightning strike, versus asystole initially with deterioration to ventricular fibrillation. Based upon the observation that asystole never occurred following the successful defibrillation of VF and that the VF was easily defibrillated with a single DC countershock, we speculate that VF began immediately after the lightning, which probably struck at the peak of the T wave. Lichtenberg et al. [6] noted that none of their reported cases experienced a reversible documentation of severe ventricular dysfunction. They hypothesize that cardiovascular injury can occur either by direct mechanical or electrical trauma or through systemic adrenergic stimulation. Lichtenberg et al. [6] focused more on the potential secondary adverse effects to the cardiovascular system, when systemic damage is also noted. In none of the cases that we were able to obtain, was commotio cordis entertained as the etiology of the ventricular arrhythmia following lightning strike. In most of the cases reviewed, cardiovascular damage was noted in association with electrocardiographic

changes, and systemic evidence of lightning-induced damage, which our case lacked.

If we focus on the scenario that VF was induced by an exactly-timed lightning strike, the issue of the path of the current remains. One possiblity is that the current did not actually traverse the patient's body, but instead the mechanical trauma or mblunt force of the lightning timed during the peak of the T wave led to the ventricular arrhythmia. In another secnario, while the patient had no burns or external evidence of electrocution, there is still a significant possibility that the a low voltage current resulted in the ventricular fibrillation. While current that induces ventricular arrhythmias is usually of high voltage, with timing during the peak of the T wave, it is possible that a relatively smaller voltage current could have travelled through his body, and his heart and led to the arrhythmia. The lower voltage would have made it more difficult to detect, and might not have caused the expected skin changes.

Our proposed pathophysiology for VF occurrence, in which we implicate commotio cordis, differs from other cases reported in the literature. The patient did not have the typical burns seen in other cases of electrical shock leading to ventricular arrhythmias. This seemed to be the most significant difference from other cases we found in the literature.

Aside from the anoxic brain damage sustained during the low-flow period while he was in the field, he did not sustain other organ damage. One would expect other sequelae of electrical shock including burns at entrance and exit routes of the current. If the shock had entered through the hand holding the rifle, one would expect that the entrance pathway (in that case, his hand) would show evidence of severe burns. That scenario would also imply an exit-route for the current (frequently through the feet, in the literature). He did not have burns on any skin surface of his body, which made the direct current strike theory seem less likely.

In addition, in the cases reviewed in which ventricular arrhythmias were observed and documented, most noted VF following ventricular tachycardia or VF which did not respond to DC cardioversion. In our case, the patient was shocked once and reverted to a sinus rhythm. We found no other cases where VF was documented, and was subsequently returned to any sustainable rhythm. When VF did occur, it was nearly always accompanied by significant signs of electrical and/or lightning-induced damage including burns, or muscle damage. Our patient's echocardiogram and electrocardiogram within 24 hours of the event were normal, which has been observed in some cases of lightning strike, although his CKs were not elevated to the extent often seen in the literature.

Conclusions

Lightning-induced ventricular arrhythmias can occur, but are rarely documented. Although lightning-induced arrhythmias have been reported, VF secondary to lightning with therapeutic countershock has not been captured, according to the literature. We present a case of ventricular fibrillation, following lightning strike, in a healthy 17 year-old Boy Scout. This case provides insight into the importance of the timing of a lightning strike, and on the mechanism of the resultant cardiac rhythm disturbance. Further investigation is warranted into the causation of VF through low-energy chest wall impact resulting from a lightning strike and not a traditional mechanical force.

Acknowledgement

Special thanks to Finger Lakes Ambulance Corps, Rochester, NY.

References

1. Link M, Mason BJ, VaderBrink BA et al. Impact directly over the cardiac silhouette is necessary to produce ventricular fibrillation in an experimental model of commotio cordis. J Am Coll Cardiol, 2001; 37: 649–654.
2. Link M, Wang PJ, Pandian NG et al. An experimental model of sudden death due to low-energy chest-wall impact (commotio cordis). NEJM, 1998; 338: 1805––1811.
3. Wiggers C, Wegria R. Ventricular fibrillation due to single, localized induction and condenser shocks applied during the vulnerable phase of ventricular systole. J Physiol, 1939; 128: 500–505.
4. Price TG. Electrical and lightning injuries. In: Marx J, Hockberger R, Walls R (eds.) Rosen's emergency medicine. 5th ed. Mosby, 2002: 2010–2014.
5. Smirk FH, Palmer DG. A myocardial syndrome: with particular reference to the occurrence of sudden death and of premature systoles interrupting antecedent T waves. Am J Cardiol, 1960; 6: 620–629.
6. Lichtenberg R, Dries D, Ward K et al. Cardiovascular effects of lightning strikes. J Am Coll Cardiol, 1993; 21: 531–536.