Cardiac thrombus developing after an accidental high-voltage electric shock in a child

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Electric shock is a condition that may affect various organ systems and potentially cause death. Cardiac findings vary from asymptomatic mild injury to fatal myocardial involvement. Herein we present a five-year-old boy with a cardiac thrombus developing after an accidental electrical shock. Cardiac arrhythmias and evidence of ischemia have been reported after electric shock; we were, however, unable to identify an earlier case report of intracardiac thrombosis related to electric shock. Findings such as elevated cardiac enzymes and systolic dysfunction, which indicate myocardial damage following electric shock, were present in our patient. We think that the cardiac thrombus might have resulted from the myocardial damage and the slowed intracardiac blood flow related to systolic dysfunction. As the thrombus was thought to have been formed through known mechanisms, it was treated traditionally. However, further data regarding the etiology and management of such thrombi is needed.

Key words: electric shock, cardiac thrombus, child.

Electricity can cause injury in a variety of ways, including but not limited to electrocution; that is, a severe electric shock may or may not be fatal. Electrical injuries can result from low-voltage, high-voltage, lightning strike or electrical arc exposure. Although reported electrical injuries among children are usually caused by low-voltage (<1000 V) current, the intensity of the current as expressed in milliamperes (mA) may be higher in children than in adults[1]. High-voltage (> 1000 V) electrical injuries are also reported on rare occasions, most notably in teenagers. It has previously been reported that high-voltage electric current may cause asystole through myocardial ischemia or an electrical shock similar to defibrillation[1-3]. Herein we present a five-year-old boy with a cardiac thrombus developing after an accidental electrical shock.

Case Report

A five-year-old boy had been exposed to high-voltage electric current upon direct contact with a high-tension cable; he had experienced cardiac arrest, but sinus rhythm had been reestablished after 20 minutes of resuscitation. The patient had been intubated and transferred to our intensive care unit. Mechanical ventilatory support was initiated. On admission he was unconscious and unresponsive, with dilated pupils and a Glasgow Coma Score of 3. His heart rate was 108/minute and arterial blood pressure 85/60 mmHg. Initial arterial blood gas analysis showed metabolic acidosis (pH 7.11, HCO3 11.7 mmol/l, PCO2 37 mmHg, PO2 77 mmHg, lactate 7.5 mmol/l). Other laboratory results are given in Table I. Electrocardiography (ECG) showed nonspecific ST-T changes but no dysrhythmia, including atrial or ventricular fibrillation. A left ventricular ejection fraction (LVEF) of 40% and slight mitral insufficiency were found on echocardiography. Cardiac inotropic support was provided with dopamine and doputamine. His blood pressure was normalized and remained within normal limits. Cardiac enzymes, ECG and echocardiography were followed up daily. The cardiac enzymes in his blood analysis improved gradually from the 6th day of hospitalization, and his troponin I and CK-MB returned to 0.88 ng/ml and 12.2 ng/ml, respectively. Improvement

Table I. Clinical, Laboratory and Echocardiographic Findings of the Patient over the Course of Hospitalization

	Day 0	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 12	Day 14
Troponin I (ng/ml)	21.5	>95	46.2	22.2	25.9	24.43	15.8	14.8	0.09	0.88
CK-MB (ng/ml)	>305	>305	157	61.4	42.5	100.5	41.5	30	12.1	12.2
CK (U/L)	>4267	>4267	>4267	3285	1671	776	346	288	220	
WBC (K/uL)	11.9	13.3	9.2	4.3	7.5	8.7	6.2	8.1	7.7	5.9
Hemoglobin (gr/dl)	15.5	12.2	9.8	8.1	12.4	12.8	11.3	10.6	12.1	10.2
Platelet (K/uL)	400	297	197	158	188	155	123	98	68	131
Urea (mg/dl)	36	44	33	45	31	17	15	23	46	41
Creatine (mg/dl)	0.39	0.54	0.48	0.42	0.4	0.46	0.52	0.5	0.6	0.85
ALT (U/L)	165	225	152	131	120	102	89	67	36	361
AST (U/L)	504	806	306	172	89	69	46	43	32	902
aPTT (sn)	66.9		29.5					67	34	
INR	1.27		1.18					1.28	1.1	2
LVEF (%)				40		45		55	70	
GCS	3	3	3	3	3	3	3	3	3	3

ALT: Alanine transaminase; AST: Aspartate transaminase; aPTT: Activated partial thromboplastin time; WBC: White blood cell; CK: Creatine kinase; CK-MB: Creatine kinase-myocardial band; GCS: Glasgow Coma Score, INR: International normalized ratio; LVEF: Left ventricle ejection fraction

in LVEF (to 55%) occurred on the 7th day of hospitalization, while an image suggestive of a thrombus was observed on the lateral wall of the left ventricle, close to the apex (Fig.1). He had no history of previous thrombosis, nor did his family. Neither a central venous nor an arterial catheter that would predispose him to thrombosis had been inserted. Finally, his blood and urine cultures revealed no bacterial growth, and he had no other documented infection. After detection of the thrombus, measurement of prothrombotic factor levels was planned but could not be performed for technical reasons. Given the impossibility of excluding the presence of intracranial bleeding in the unconscious patient, thrombolytic treatment could not be initiated. The patient was heparinized; on the second day of treatment, heparin was suspended due to thrombocytopenia, even though a 20% reduction of the thrombus mass had been achieved. Low-molecular-weight (LMW) heparin (Fraxiparin®) treatment was started. The mass shrank by a further 10% on the second day of the latter treatment. On the third day of LMW heparin treatment (the 12th day of hospitalization), cutis marmorata developed in the right lower extremity, in which pulses could not be palpated. Echocardiography indicated a normal LVEF of 65%. The ventricular mass could no longer be observed; it was thought that it had moved into the right lower limb. An embolectomy was performed by the cardiovascular surgery department, and the embolized thrombus was removed. ECG and cardiac functions on echocardiography were normal for two consecutive days following the intervention. On the 14th day of hospitalization, however, the patient was lost to cerebral death.

Discussion

Although the occurrence of vascular thrombosis following high-voltage electric shock has been reported previously, no case of intracardiac thrombus associated with electric shock has

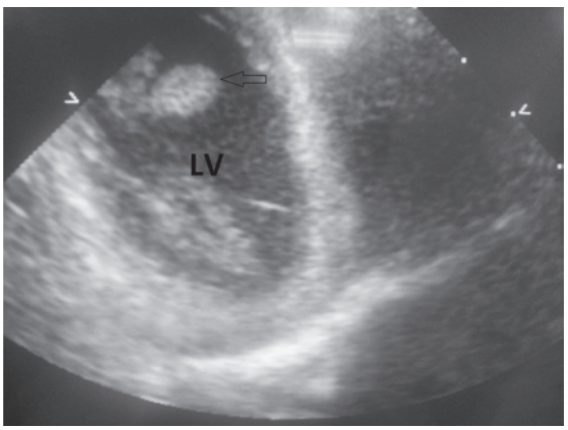

Fig. 1. Echocardiography showing a thrombus on the lateral wall of the left ventricle, close to the apex (LV: left ventricle).

been described in the English-language medical literature so far. There are various hypotheses as to the formation of such thromboses. The possibility of a substantial fall in tissue plasminogen activator (t-PA) concentration after exposure to an electrical current, especially in the early period, was noted in one report; such a disturbance of the coagulation-fibrinolysis balance was suggested as a possible explanation of progressive thrombosis[4]. Another study reported a significant increase in platelet alpha-granule membrane protein GMP-140 and endothelin-1 (ET-1) levels in the early stage following electric shock damage. Plasminogen activator (PLGA) levels were also shown to be significantly disturbed. The report assigned an important role in triggering post-electrocution thrombosis to these factors[5]. A study performed in rabbits by Chen et al[6]. found an elevation of antithrombin III (AT-III) and protein C (PC) levels; there was in addition a marked change in t-PA concentration. The authors also reported an increased risk of thrombosis, with two episodes occurring 6-12 hours and five days after the electrocution.

Systolic dysfunction is well known to be a significant risk factor for intracardiac thrombosis and embolism; reported cases consist mainly of patients with severe systolic dysfunction[7,8]. Given that the lowest value of LVEF in our patient was 45% on admission, and it then gradually recovered, we think that even though the reduced LVEF may have contributed to thrombosis, the main causative factor consisted in problems in the coagulation system due to the electric shock. While arrhythmias are another risk factor for cardiac thrombosis[8], no arrhythmia had been documented in our patient.

Various measures, including observation, systemic anticoagulation, thrombolysis and thrombectomy, have been proposed for the treatment of intracardiac thrombi[7]. Thrombolytic treatment is recommended in acute events; resolution with heparin infusion has also been reported[7,9]. John et al[7]. reported on 31 pediatric patients, 30 of whom received medical treatment, principally in the form of heparin infusion. None of these patients subsequently developed a need for thrombolysis. The authors reported that 55% of the (mostly left ventricular) thrombi in these patients diagnosed with dilated cardiomyopathy had resolved under aspirin, warfarin or heparin therapy. In our patient, the presence of the intracardiac thrombus was first observed on 7th day of hospitalization; the last unremarkable echocardiography having been made on 5th day suggests that the thrombus developed in the 48 hours preceding its diagnosis. Fibrinolysis, considered as a possible treatment, was not initiated because an intracranial hemorrhage could not be excluded given the unconscious status of the patient, who had a GCS of 3. A shrinking in the dimensions of the thrombus was observed under treatment, first with heparin, and then with LMW heparin.

In conclusion, the cardiovascular effects of exposure to an electric current may vary from nonspecific changes to fatal results. The ECG, cardiac enzymes and echocardiography of patients exposed to high-voltage electric power in particular should be followed up; it should be kept in mind that an intracardiac thrombus may develop even several days past the event, and appropriate anticipatory measures should be taken in this regard.

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