Cardiology in the Young

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Survival following cardiac arrest due to lightning strike

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Brief Report

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Abstract

Lightning strike is rare, with a reported incidence of around 1:100,000. There are few reports of survival after cardiac arrest due to lightning strike. We report the case of a 12-year-old male survivor. Though he had a prolonged out of hospital cardiac arrest and initial poor systolic function, widespread ST segment elevation on electrocardiography, and elevated cardiac troponin I, he survived with a good cardiac and neurological outcome.

Case report

A 12-year-old male was struck by lightning during a football match. Instantaneous collapse was witnessed, cardiac arrest was confirmed, and cardiopulmonary resuscitation with an automated defibrillator was initiated within 5 minutes. He received one DC shock for a "shockable rhythm" of unclear aetiology then remained in pulseless electrical activity. A prehospital emergency medical team arrived and resuscitation continued according to European Resuscitation Council guidelines.[1] He had return of spontaneous circulation 38 minutes after the initial strike. He was intubated, ventilated, and transferred by air ambulance to our hospital.

Examination revealed reactive pupils, superficial partial thickness burns over chest and abdomen (Fig 1), burns to extremities with a likely exit wound on the right heel, and total area of burns <10%.

On electrocardiography, he had frequent premature ventricular contractions with short runs of non-sustained ventricular tachycardia lasting up to 10 beats. 12-lead electrocardiogram showed inferolateral Q waves and ST segment elevation (Fig 2). Magnesium, calcium, and potassium levels were actively optimised, and no antiarrhythmic infusion was required. Initial CT brain showed normal appearances with preserved grey-white differentiation. CT chest, abdomen and pelvis showed no evidence of traumatic injury.

On the paediatric intensive care unit, he remained mechanically ventilated for five days, with a neuroprotective strategy for the initial 48 hours to maximise neurological recovery, including targeted temperature management to maintain normothermia.

Initial echocardiography demonstrated severely impaired systolic function with paradoxical septal motion. Cardiac index was evaluated to be 2.44 L/min/m.[2] Proximal coronary artery anatomy appeared normal, with no evidence of thrombus.

Clinically, he had profound peripheral vasoconstriction with cold extremities, poor peripheral pulses, and hypertension with a blood pressure of 150/110. After discussion with the regional Paediatric Cardiology Unit, milrinone was started at 0.5 mcg/kg/minute for lusitropic and inotropic effects and to reduce afterload by systemic vasodilation.[2] Nitroglycerin was considered but not used as the hypertension improved with milrinone and the index of suspicion for an anatomical coronary lesion was low. Following the introduction of milrinone, the clinical picture improved. Captopril was introduced and milrinone was discontinued after 72 hours.

Cardiac troponin I was above the limit of the assay at >25000 ng/L (reference range 0–54 ng/L) for 5 days before falling to a measurable level. Electrocardiography remained abnormal, with ST segment changes and inferolateral Q waves for several days after admission. CT angiogram one day post injury demonstrated patent coronary arteries.

He had mild acute kidney injury with elevated urea and creatinine. Maximum creatine kinase was 10,805 U/L one day post injury, indicating a degree of rhabdomyolysis. Renal support was not required.

Post extubation, neurological examination revealed normal motor and sensory function, but significant cognitive impairment with agitation, confusion and memory loss. He was transferred to the paediatric ward for ongoing neurorehabilitation 8 days after admission.

He was discharged home six weeks post cardiac arrest on oral captopril with continued outpatient rehabilitation and review with the Paediatric Cardiology and Neurology teams.

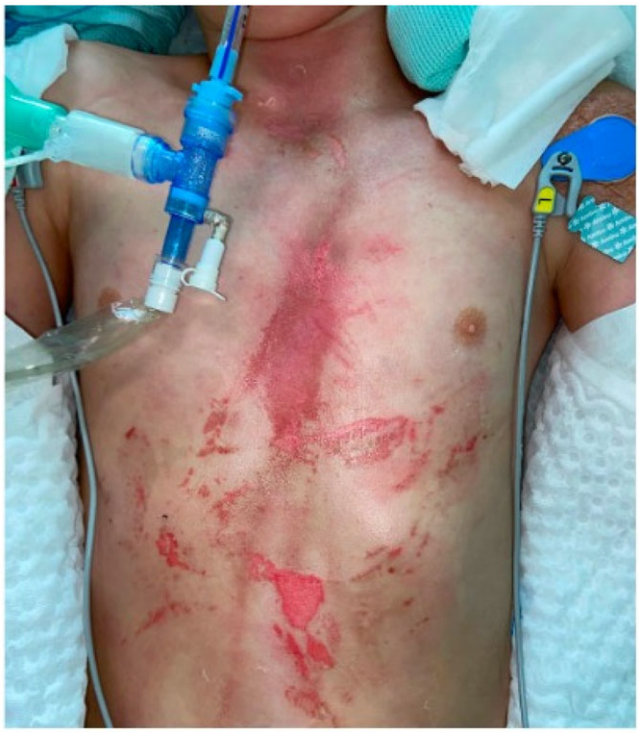

Figure 1. Burns to anterior torso and neck.

At that point, cognitive function and neurological status were on an improving trajectory. He had ongoing fatigue, memory loss, and limb pain but was able to talk and was mobilising independently.

24-hour electrocardiogram showed sinus rhythm with infrequent ventricular ectopics. Echocardiography showed some resolution of the previously noted abnormalities, with fractional shortening of 35% and normalising regional wall dyskinesia.

Cardiac MRI at 9 weeks post injury showed akinesia and thinning of the infero-lateral left ventricular wall from mid-base to apex, likely consistent with infarction, and moderately impaired global systolic function with a left Ventricular Ejection Fraction (LVEF) of 40%. There was also a large right antero-lateral pericardial collection with a small pericardial tear.

At 5 months post event, echocardiography showed significant improvement with only mild hypokinesia of the posterior mid-ventricular segment but an overall ejection fraction of 51% and global strain of −19%. A 2-week rhythm monitor showed a ventricular ectopic burden of 2.9% with ventricular ectopic couplets at <1% and episodes of bigeminy. Repeat cardiac MRI at 7 months post injury demonstrated resolving pericardial collection in the anterior mediastinum, improved systolic function (LVEF 54%), with mild regional wall motion abnormality, thinning, and transmural scar. Despite these changes, clinically, he continues to improve and is returning to mainstream education and sport.

Discussion

Worldwide around 24,000 people die per year from lightning strike,[3] though this is likely an underestimate. Immediate cardiac arrest is thought to occur in 1% of lightning strike victims, with a mortality of up to 90%.

The transferred electrical energy causes immediate complete myocardial depolarisation, most commonly leading to ventricular fibrillation or asystole.[4] Though cardiac automaticity may soon restore circulation, secondary hypoxic cardiac arrest can also occur due to paralysis of the medullary respiratory centres.[5]

Ischaemic-like changes on electrocardiogram may have multiple aetiologies. Coronary artery vasospasm, dissection, or thrombus can occur with changes to localised coronary artery territories, whereas myocarditis and pericarditis result in widespread changes. Cardiac biomarkers do not correlate with injury or prognostic outcome.[6] In this case, the ECG changes were thought

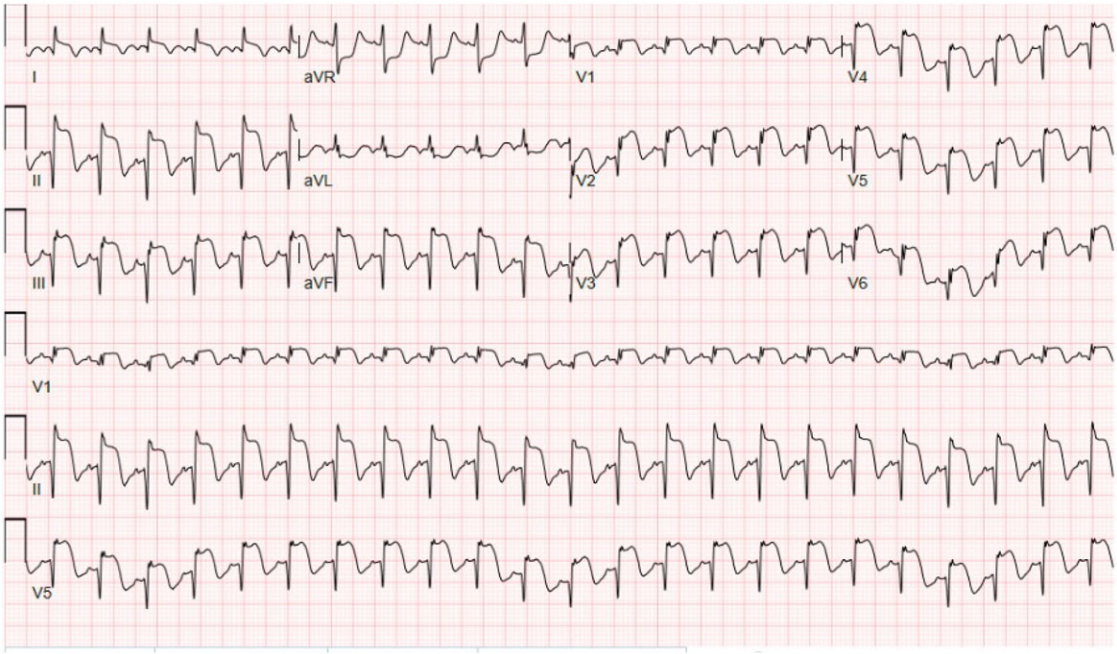

Figure 2. Electrocardiogram 8 hours post arrest.

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to be secondary to coronary vasospasm and associated ischaemia or myocardial inflammation rather than coronary dissection.[6] Hypertension was likely due to either catecholamine release or autonomic stimulation.[7]

Conclusion

This case demonstrates the potential for good myocardial recovery post lightning strike, despite initial elevated troponin, marked ischaemic changes on electrocardiography, and poor cardiac function on echocardiogram. Cases of lightning strike should receive maximum possible supportive care until the neurological prognosis is clear as the prospects for cardiac recovery may be good.

Learning points

- Although ST elevation may suggest a localising vascular lesion, the coronary arteries may be normal. Hypertension and peripheral vasoconstriction due to catecholamine release or autonomic instability may need active management.
- Elevated cardiac biomarkers are often reported, but are not typically prognostic.
- There may be good cardiac functional recovery despite ECG, biochemical and imaging findings.

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Competing interests. None.

Ethical standard. The authors declare that the procedures followed were in accordance with the regulations of the relevant clinical research ethics committee and with those of the Code of Ethics of the World Medical Association (Declaration of Helsinki).

References

1. Van de Voorde P, Turner NM, Djakow J, et al. European resuscitation council guidelines 2021: paediatric life support. Resuscitation 2021; 161: 327–387.
2. Deep A, Goonasekera CD, Wang Y, et al. Evolution of haemodynamics and outcome of fluid-refractory septic shock in children. Intensive Care Med 2013; 39 (9): 1602–1609.
3. Powell T, Charnigo A, Yee J. Lightning Strike. J Educ Teach Emerg Med 2022; 7: S78–S106.
4. Christophides T, Khan S, Ahmad M, et al. Cardiac effects of lightning strikes. Arrhythm Electrophysiol Rev 2017; 6 (3): 114–117.
5. (Vol. 112, 24 Suppl): IV154. American heart association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. Part 10.9: electric shock and lightning strikes. Circulation 2005; 112; IV154–IV155.
6. Davis C, Engeln A, Johnson EL, et al. Wilderness medical society. Wilderness medical society practice guidelines for the prevention and treatment of lightning injuries: 2014 update. Wilderness Environ Med 2014; 25 (4 Suppl): S86–S95.
7. Lichtenberg R, Dries D, Ward K, et al. Cardiovascular effects of lightning strikes. J Am Coll Cardiol 1993; 21 (2): 531–536.

https://doi.org/10.1017/S1047951125001349 Published online by Cambridge University Press