

## ST-segment elevation myocardial infarction after drowning

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Dear Editor,

A 16-year-old boy with no significant past medical history was brought to the emergency department (ED) after drowning in Lake Michigan where he was reportedly submerged in water for 15 min before being pulled out. On arrival of the paramedics, he was unresponsive with an initial rhythm of pulseless electrical activity. He was intubated at the scene, and cardiopulmonary resuscitation was performed for 8 min with restoration of spontaneous circulation. In the ED, the patient's electrocardiogram (EKG) revealed an acute infero-postero-lateral ST-segment elevation myocardial infarction (STEMI) (Fig. 1, panel a). He was started on heparin and eptifibatid (INTEGRILIN; Merck and Co., Whitehouse Station NJ) drip, and was transferred to the cardiac catheterization lab for percutaneous coronary angiogram. A repeat EKG performed immediately prior to the procedure revealed resolution of ST-segment elevation, and the procedure was therefore deferred and the patient was sent to the intensive care unit (ICU).

Upon arrival in the ICU, the patient was unconscious, hypothermic (temperature 32.5 °C), and hypotensive requiring vasopressors and inotropic support. Follow-up EKG 3 h later revealed deep Q waves in the inferior leads (Fig. 1, panel b). Laboratory investigations were significant for a troponin >50 ng/mL (*N* 0–0.08), creatinine phosphokinase (CPK) 4104 unit/L (*N* 32–294), creatinine kinase MB (CKMB) 297 ng/mL (*N* 0–5), a combined metabolic and respiratory acidosis with a pH 7.1, bicarbonate (HCO<sub>3</sub>) 16 mmol/L and partial pressure of carbon dioxide (*p*CO<sub>2</sub>) 53 mmHg, international normalized ratio (INR) 3.2 and partial thromboplastin time (PTT) >150 s. Chest X-ray study revealed bilateral patchy infiltrates consistent with pulmonary edema. Shortly, the patient developed epistaxis and hematemesis, so antiplatelet and anticoagulant medications were discontinued. The patient's level of consciousness deteriorated to a Glasgow Coma Scale of 3/15 with absence of all brainstem reflexes in addition to a positive apnea test and electroencephalogram (EEG) conformed to the diagnosis of brain death.

Drowning is a major cause of death worldwide among boys 5–14 years of age [1]. There is scant data in the literature describing the myocardial ischemic complications of drowning. In the prospective study by Checchia et al. [2], there was a trend towards a higher mortality in children with acute out-of-hospital cardiac arrest after submersion injury who had a higher troponin elevation, however, the results did not reach statistical significance. In a retrospective analysis of autopsy and laboratory findings in 197 submersion cases by Papadodima et al. [3], 23 cases died from complications of coronary artery disease. A possible mechanism by which drowning can lead to myocardial infarction includes hypoxia-either due to fluid aspiration or laryngospasm- and subsequent coronary spasm. Sheikh et al. [4] demonstrated the occurrence of transient ST

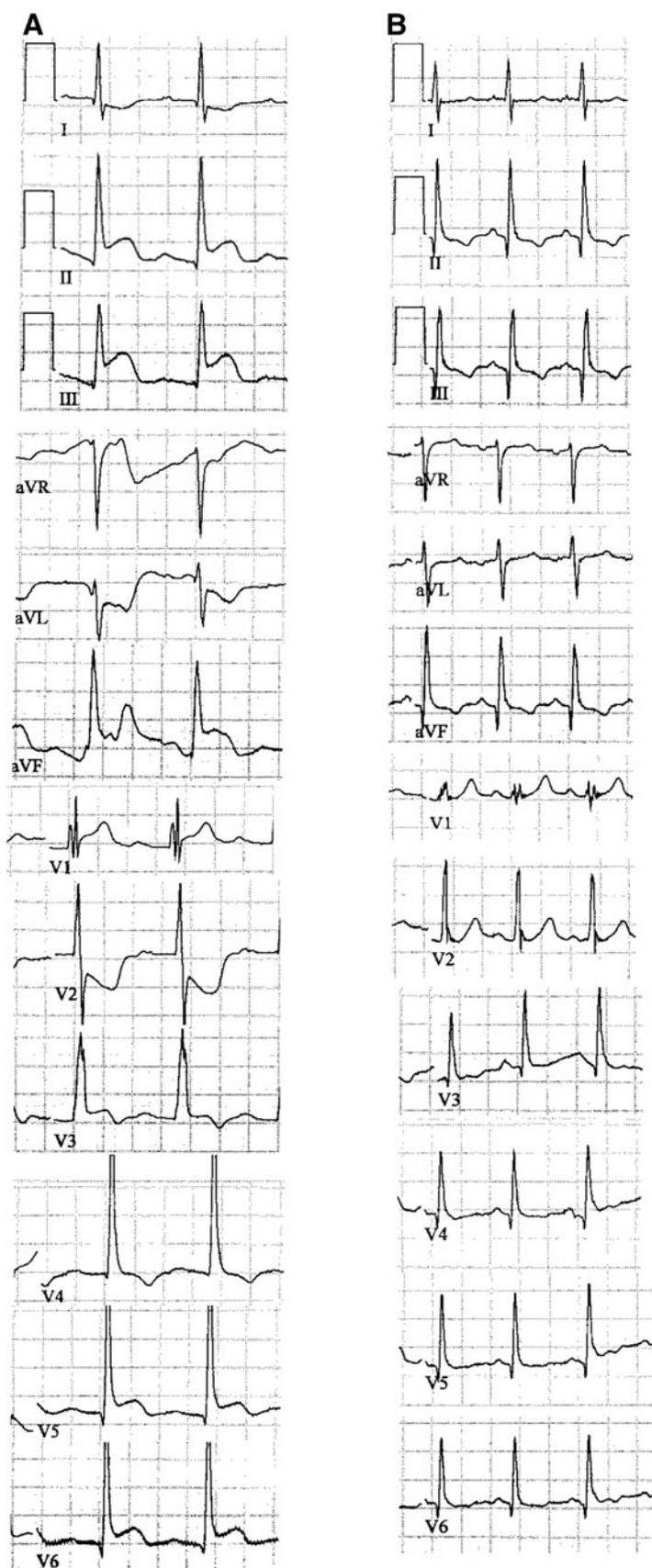
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**Fig. 1** Panel **a** (*left*) revealing ST-segment elevation in leads II, III, aVF, V5, V6, a tall R wave with ST-segment depression in V2 and reciprocal ST-segment depression in leads I and aVL conforming to acute infero-postero-lateral STEMI. Panel **b** (*right*) demonstrates the resolution of ST-segment elevation with the development of Q waves in leads II, III and aVF



elevation in a patient with hypoxic respiratory failure. STEMI after drowning can also be explained by hypothermia-induced coronary spasm. Akin et al. [5] reported the development of diffuse coronary spasm during therapeutic hypothermia that was nitrate-refractory requiring percutaneous coronary intervention (PCI). Emotionally induced coronary artery spasm due to the extreme distress during the drowning process can be another trigger for STEMI. The spectrum of sequelae that may result from coronary spasm extends from mild reversible to more severe cases complicated by myocardial infarction and death. In a study of 349 patients with vasospastic angina, myocardial infarction (diagnosed by the development of new Q waves and elevation of the cardiac enzymes) occurred in 18 (5 %) patients [6]. Other EKG findings that can be encountered after drowning accidents are mainly related to hypothermia. These include sinus bradycardia, prolongation of the PR interval, widening of the QRS complex, QT interval prolongation and the classic Osborn wave. The Osborn wave is a deflection at the R-ST junction of the EKG, is more evident in the infero-lateral leads and has an amplitude that inversely correlates with the core body temperature. It is the result of a transient, outward potassium-mediated current in the epicardium but not the endocardium creating a transmural voltage gradient during early repolarization evident as “J wave”. Other hypothermic EKG changes include various atrial and ventricular

dysrhythmias. In presenting the case described herein, we aim to emphasize that STEMI is a possible complication in submersion injuries and hence highlight the importance of monitoring serial EKGs and cardiac biomarkers for timely diagnosis and management.

**Conflict of interest** None.

## References

1. World Health Organization (2003) Injuries and violence prevention: non-communicable diseases and mental health: fact sheet on drowning. World Health Organization, Geneva. [http://www.who.int/violence\\_injury\\_prevention/other\\_injury/drowning/en/index.html](http://www.who.int/violence_injury_prevention/other_injury/drowning/en/index.html)
2. Checchia PA, Moynihan JA, Brown L (2006) Cardiac troponin I as a predictor of mortality for pediatric submersion injuries requiring out-of-hospital cardiopulmonary resuscitation. *Pediatr Emerg Care* 22(4):222–225
3. Papadodima SA, Sakelliadis EI, Kotretsos PS, Athanaselis SA, Spiliopoulou CA (2007) Cardiovascular disease and drowning: autopsy and laboratory findings. *Hellenic J Cardiol* 48(4):198–205
4. Sheikh M, Adlakha S, Bruhl S, Kanjwal S (2010) Coronary vasospasm in a patient with respiratory failure: a case report and a brief review. *Br J Med Pract* 3(3):a330
5. Akin I, Rehders TC, Kische S, Schneider H, Turan RG, Kleinfeldt T, Chatterjee TC, Ince H, Nienaber CA (2011) Diffuse coronary spasm during therapeutic hypothermia. *Int J Cardiol* 148(3):373–375
6. Nakamura M, Takeshita A, Nose Y (1987) Clinical characteristics associated with myocardial infarction, arrhythmias, and sudden death in patients with vasospastic angina. *Circulation* 75(6):1110–1116