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Case Report

ACUTE MYOCARDIAL INFARCTION TRIGGERED BY DROWNING IN AN ELDERLY PATIENT: A CASE REPORT

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ABSTRACT: Drowning is a leading cause of unintentional injury-related mortality worldwide, particularly in two vulnerable populations: children and the elderly. In addition to respiratory compromise, drowning may lead to serious complications such as hypothermia, prolonged neurological injury, and cardiovascular sequelae — including myocardial damage. Myocardial infarction following drowning is rare and frequently overlooked in the acute setting, as elevated cardiac biomarkers are often attributed to resuscitation efforts. Nevertheless, in patients with pre-existing cardiovascular risk factors, systemic hypoxia combined with acute physiological stress induced by drowning may serve as a trigger for true myocardial infarction. This report describes an incidental finding of underlying coronary artery disease following a drowning event, underscoring the critical importance of comprehensive cardiovascular assessment in drowning survivors for early recognition of life-threatening cardiac events and timely, individualized management.

Keywords: drowning; elderly patient; coronary artery disease

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1. INTRODUCTION

According to the World Health Organization, drowning is defined as a process of respiratory impairment due to submersion or immersion in liquid, resulting in acute hypoxia and subsequent multi-organ injury. Drowning remains a major global public health concern, with significant morbidity and mortality, particularly in vulnerable populations such as children and the elderly [1]. Survivors of drowning may develop a wide spectrum of complications, including acute respiratory distress syndrome (ARDS), acute pulmonary edema, aspiration pneumonia, altered consciousness, hypothermia, and electrolyte disturbances. Cardiovascular complications are largely attributed to hypoxemia and hypothermia. Cardiac rhythm disturbances reported after drowning include sinus tachycardia, sinus bradycardia, atrial fibrillation, pulseless electrical activity, and asystole. Ventricular tachycardia and ventricular fibrillation are rare and are usually observed in individuals with underlying structural heart disease or latent ischemia. Electrocardiographic ST–T segment changes resembling myocardial ischemia may arise from takotsubo cardiomyopathy, coronary vasospasm, type 2 myocardial infarction (due to oxygen supply–demand mismatch), and, in some cases, type 1 myocardial infarction resulting from plaque instability [2]. Focusing solely on respiratory management without comprehensive cardiovascular assessment following drowning may result in missed underlying coronary artery disease, thereby increasing the risk of severe cardiovascular events. We report a rare case of acute myocardial infarction following drowning in an elderly patient with significant coronary artery disease, highlighting the importance of thorough cardiovascular evaluation in atypical emergency presentations.

2. CASE PRESENTATION

A 76-year-old female was transferred to Thong Nhat Hospital after successful resuscitation from cardiac arrest secondary to drowning. Her past medical history included hypertension, type 2 diabetes mellitus, and dyslipidemia, for which she had been receiving regular and stable medical treatment. The clinical course was as follows

Day 0: The patient went swimming alone at the beach without any preceding symptoms such as chest pain or dyspnea. While swimming, she was swept away by waves and was unable to return to shore, subsequently losing consciousness. Upon regaining consciousness, she had already received initial emergency care at a local medical facility.

Day 1: Upon admission to Thong Nhat Hospital, the patient was alert, oriented, and hemodynamically stable. Physical examination revealed a regular heart rhythm without murmurs, and fine crackles were detected at the bases of both lungs. The patient reported nonspecific chest pain, which, although subtle, warranted further investigation. Electrocardiography (Figure 2) demonstrated deeply inverted, symmetrical T waves in multiple leads, suggestive of significant myocardial ischemia. Subsequent transthoracic echocardiography (Figure 3) revealed ventricular septal aneurysm with severe hypokinesia of the apical and septal regions. These findings indicated previously unrecognized ischemic heart disease, incidentally detected following the drowning event.

Day 2: After counseling, the patient and her family initially declined invasive coronary angiography (DSA) due to financial concerns. Therefore, coronary computed tomography angiography (CCTA) was performed (Figure 4). The results revealed three-vessel coronary artery disease with a total Agatston calcium score of 1773.28, indicating severe coronary calcification and reflecting a very high risk of adverse cardiovascular events. Notably, CCTA also demonstrated a thrombus causing severe stenosis in the proximal LAD-I, classified as CAD-RADS 4A, providing evidence that the patient may have developed type 1 myocardial infarction following the drowning episode. This condition necessitated timely intervention to prevent further major cardiovascular complications.

Day 3: After further explanation and counseling, the patient consented to undergo percutaneous coronary intervention (PCI) the following day. However, during coronary angiography, the lesions were found to be complex and heavily calcified, involving the left main (LM) and all three major vessels.

Therefore, stent implantation was deemed unsuitable, and coronary artery bypass grafting (CABG) was recommended as the optimal revascularization strategy, in accordance with the 2023 ESC/EACTS guidelines [3].

2.1. Laboratory tests:

Laboratory tests revealed leukocytosis (WBC 15.27 G/L, neutrophils 93.9%) and elevated procalcitonin (0.622 ng/mL). In addition, chest radiography (Figure 1) demonstrated bilateral perihilar infiltrates, suggestive of aspiration pneumonia. The patient was therefore started on antibiotic therapy.

Troponin T-hs: demonstrated a dynamic rise from 227 to 306 pg/mL within 14 hours, consistent with acute myocardial injury. Initially, we considered that the patient might have sustained secondary myocardial injury following cardiac arrest, while type 2 myocardial infarction related to coronary vasospasm or hypoxemia—direct consequences of the drowning event—could not be excluded. However, subsequent coronary MSCT revealed a thrombus causing severe stenosis of the proximal LAD-I. Although uncommon, this finding provided evidence that the elevation of cardiac biomarkers in this patient was attributable to type 1 myocardial infarction.

NT-proBNP was markedly elevated (5906 pg/mL), suggesting increased left ventricular filling pressures and indicating previously unrecognized chronic heart failure secondary to underlying coronary artery disease.

AST: 233 U/L, ALT: 164 U/L. AST was elevated to a greater extent than ALT, suggesting hepatic injury secondary to hypoxemia and hypoperfusion during cardiac arrest.

Urea: 6,8 mmol/L, Creatinine: 68 µmol/L, eGFR 77 mL/min/1,73m2, were within normal limits.

LDL-C: 2,15 mmol/L, HbA1c: 7,5%, indicating suboptimal control of diabetes mellitus and dyslipidemia.

Figure 1. Chest X-Ray: Cardiac silhouette not enlarged. Bilateral perihilar opacities. Bony thorax unremarkable.

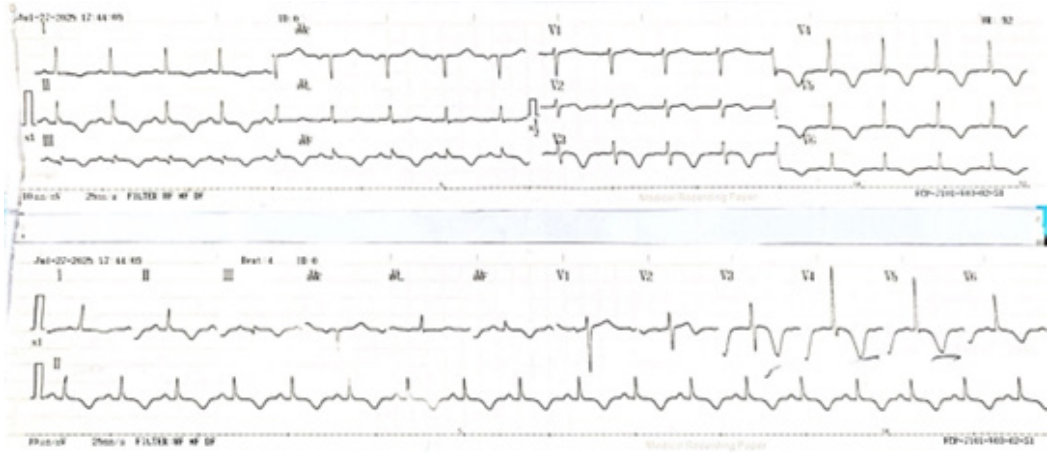

Figure 2. Electrocardiogram: Sinus rhythm, regular, 92 bpm. Intermediate axis. Deep, symmetrical T-wave inversions in leads V3–V6, II, III, and aVF.

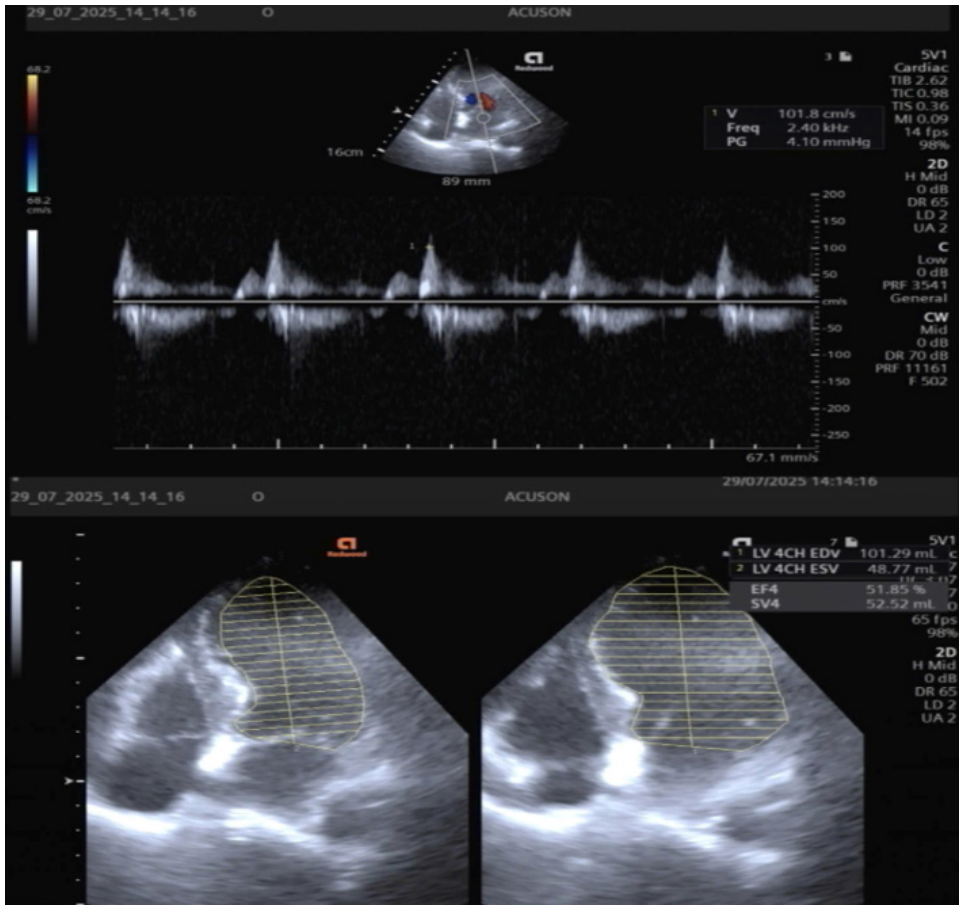

Figure 3. Echocardiogram: Left ventricular aneurysm with hypokinesia of the interventricular septum and apical segments. LVEF (Simpson) = 51%. Mitral valve: thickened and calcified, mild regurgitation (grade I), impaired relaxation pattern, E/A<1. Aortic valve: thickened, no stenosis or regurgitation. Tricuspid valve: mild regurgitation (grade I), PAPs = 25 mmHg, TAPSE =18 mm

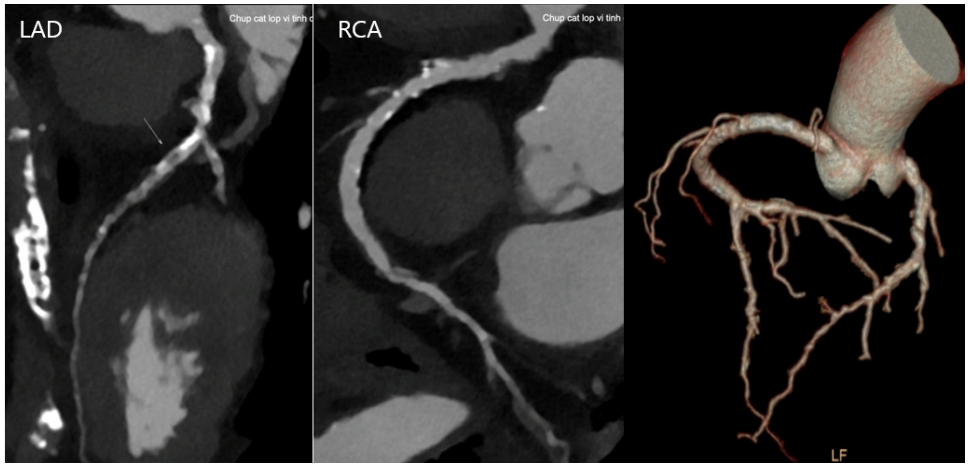

Figure 4. Coronary computed tomography angiography. LM: Calcified plaque with 30–40% stenosis at the ostium. LAD: Scattered calcified plaques causing 50–70% stenosis in the LAD-I segment, with superimposed atherosclerotic plaque and thrombus resulting in severe (#80–90%) stenosis in the proximal LAD-I. Calcified plaque with 30–40% stenosis at the ostium D1. LCX: Hypoplastic; no significant calcified atherosclerotic stenosis detected. RCA: Scattered calcified plaques causing approximately 30% stenosis in both RCA-I and RCA-II segments. LM: 11.29 LAD: 818.19 LCX: 0 RCA: 943.8. Agatston calcium score: 1773.28. Coronary dominance: Right dominant system.

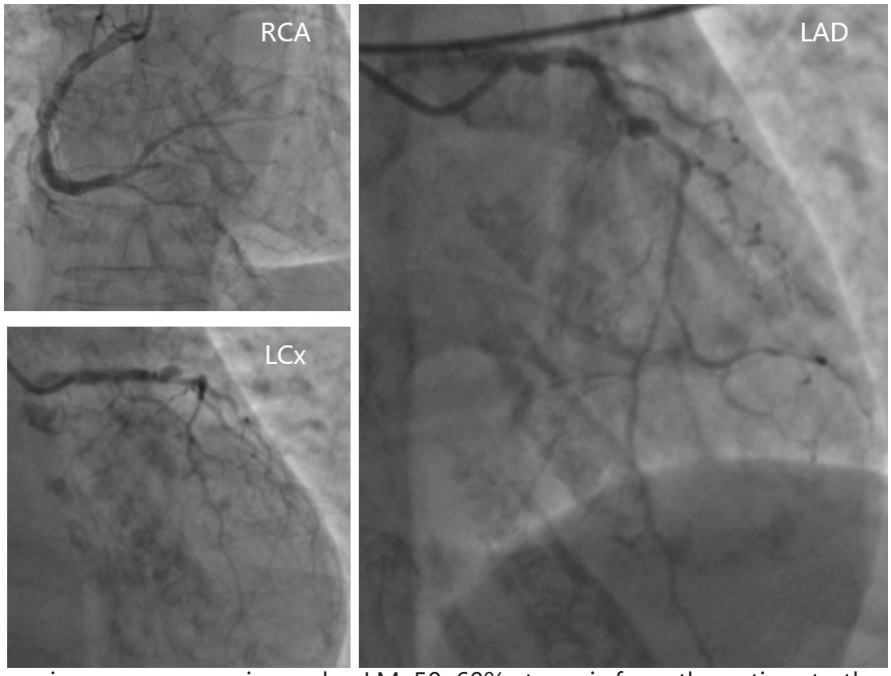

Figure 5. Invasive coronary angiography. LM: 50–60% stenosis from the ostium to the distal segment. LAD: 70–80% stenosis from the proximal to distal segments. LCx: 70–80% ostial stenosis (small-caliber vessel). RCA: dominant vessel with 50–60% stenosis in the mid segment. Severely calcified coronary artery system.

3. DISCUSSION

According to the Fourth Universal Definition of Myocardial Infarction, issued by the ESC/ACC/AHA/WHF, type 1 myocardial infarction results from atherosclerotic plaque rupture, whereas type 2 myocardial infarction is a consequence of an imbalance between myocardial oxygen supply and demand. Common underlying mechanisms include hypoxemia, coronary vasospasm, catecholamine surge, or increased oxygen demand in the setting of severe comorbid conditions [4].

In the medical literature, myocardial infarction following drowning is a rare condition, with only a few cases reported, most of which were classified as type 2 myocardial infarction. In the case described by Chen et al. (2008), an 82-year-old female who was successfully resuscitated after drowning exhibited ST-segment elevation from V2 to V6 on electrocardiography. Although initial cardiac biomarkers were normal, they rose markedly on the second day. The authors concluded that this was a case of type 2 myocardial infarction, attributed to severe respiratory failure, hypothermia, and metabolic acidosis. However, in the absence of coronary imaging, the possibility of type 1 myocardial infarction could not be completely excluded [5].

Similarly, Omar et al. (2013) reported a case of a young patient who developed transient ST-segment elevation after drowning. The authors suggested that, in addition to hypoxemia, coronary vasospasm—possibly triggered by hypothermia or emotional stress associated with the drowning event—also contributed to the underlying pathophysiological mechanism [6].

In addition, a retrospective study conducted in Greece demonstrated that among 168 cases of drowning-related deaths undergoing forensic autopsy, nearly 49% had evidence of underlying coronary artery disease, and 6% showed signs of recent myocardial infarction or coronary thrombosis [7]. This finding supports the hypothesis that underlying coronary artery disease is not only a risk factor but also a direct contributing factor to mortality in the context of drowning.

Unlike most previous reports that primarily suspected type 2 myocardial infarction, our patient demonstrated definitive imaging evidence—specifically, thrombotic occlusion of the proximal

LAD on a background of complex atherosclerosis—together with dynamic troponin elevation and echocardiographic findings, which were consistent with type 1 myocardial infarction. This suggests that drowning may also act as a triggering factor destabilizing a vulnerable plaque, thereby promoting thrombus formation and precipitating myocardial infarction.

Distinguishing between type 1 and type 2 myocardial infarction is of paramount importance, as only type 1 MI requires coronary revascularization and intensive medical therapy, including anticoagulation, dual antiplatelet therapy, and high-intensity statins. In contrast, the management of type 2 MI primarily targets the underlying trigger (e.g., hypoxemia, infection, or stress). During the diagnostic process, coronary CT angiography represents a valuable alternative when invasive angiography is declined, particularly given its ability to detect intracoronary thrombus, provide risk stratification using the CAD-RADS classification, and quantify coronary calcification by Agatston score. Finally, according to the 2020 ESC guidelines on NSTEMI, in patients with type 1 MI involving left main and triple-vessel disease, coronary artery bypass grafting (CABG) is preferred over PCI when surgical risk is acceptable and life expectancy exceeds one year; therefore, CABG was considered the most appropriate revascularization strategy in this patient [3].

4. CONCLUSION

Drowning in the elderly represents a medical emergency not only because of the immediate risk of death but also due to a wide spectrum of severe post-resuscitation complications, including acute cardiovascular events, particularly in the context of previously undiagnosed coronary artery disease. A comprehensive, multi-system approach is therefore warranted in such cases, rather than focusing solely on respiratory injury.

In all cases of drowning in the elderly, comprehensive cardiovascular evaluation is essential, including cardiac biomarkers, electrocardiography, echocardiography, and, when indicated, coronary angiography. Elevated cardiac enzymes following drowning should not be hastily attributed to "secondary" injury; instead, true myocardial infarction, including type 1 MI, must be carefully excluded using appropriate diagnostic modalities. Missing underlying coronary artery disease or myocardial infarction may result in patients failing to receive appropriate therapy, leaving them at risk of fatal cardiovascular complications despite apparent recovery from the drowning episode. Moreover, clear differentiation between type 1 and type 2 MI is critical to guide appropriate management strategies, avoiding both overtreatment and undertreatment with coronary interventions and antiplatelet therapy.

Enhancing clinical evaluation and fostering comprehensive awareness of the association between drowning and cardiovascular disease in the elderly is essential to ensure individualized and effective care for this vulnerable patient population.

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