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Letter to the Editor
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Heat and the Heart: The Unseen Microvascular Pathway to Myocardial Infarction

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To The Editor,

Surpassing floods and storms, extreme heat waves have emerged as the most lethal climate phenomenon globally, leading to thousands of deaths worldwide due to various pathologies, by altering respiratory, cardiovascular, and homeostasis mechanisms. In 2019, heat-related cardiovascular diseases were responsible for around 90,000 deaths globally (1). Although various etiologies were widely studied, the cardiovascular repercussions, especially acute myocardial infarction (MI), are often underestimated, rendering millions of lives at high risk.

Hospital admissions and deaths related to ischemic heart disease and arrhythmias are significantly increasing on hot or summer days (2), underscoring that such attributes were due to a systemic process of dehydration, hemoconcentration, autonomic stress, and electrolyte imbalances (3), neglecting vascular biology fundamental to cardiac endurance. It is worth mentioning that the human heart may be the first organ to suffer silently due to a temperature rise. One study indicates that exposure to acute heat can impact endothelial-dependent dilation and microcirculatory flow (4); however, whether the thermal endothelial dysfunction persists after a heat wave or increases the risk of myocardial ischemia is yet to be discussed (5). Identifying this overlooked association could change the healthcare professionals' perspective on evaluating cardiovascular risk in a warming world.

Heat stroke triggers several systemic and cardiac injuries that ultimately damage the heart muscle. Sasaki et al. reported markedly raised troponin I levels, ST-segment elevation, and apical ballooning on ventriculography—signs of acute myocardial injury even without blocked coronary arteries (6). This supports the hypothesis that heat stress can cause heart damage through mechanisms apart from traditional plaque rupture. Factors such as endothelial damage, oxidative stress, and metabolic disruptions in cardiac myocytes were intensified by heightened sympathetic nervous system activity and disruption of heat shock proteins, which can hinder the blood flow to the heart and weaken its contractile function (7). Such findings indicate that extreme heat can cause a type of myocardial infarction, ensuing stress on the microvasculature and direct strain on the heart muscle.

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As climate change intensifies rapidly, global warming is expected to increase in frequency and intensity throughout the 21st century, marking a new era of cardiovascular risk (8). Studies have shown that older adults, individuals with diabetes or hypertension, and those of low socioeconomic status are more vulnerable to heat-associated cardiovascular events, particularly MI (9). This rising burden calls for public health education and post-heat exposure cardiovascular risk assessment as part of healthcare policy. In low- and middle-income countries, limited access to cooling systems, greater occupational heat exposure of outdoor workers, and inadequate cardiovascular care significantly contribute to the higher number of cases of heat-treated MI in these areas (10). Addressing these disparities requires equitable access to cooling and hydration, improved heat adaptation, and stronger public health strategies.

Further validation of the relationship between heat exposure and myocardial infarction requires future research, with particular focus on microvascular dysfunction. Prospective studies integrating clinical outcomes and biomarker profiling, especially pre- and post-heat exposure changes in endothelin and nitric oxide metabolites, could clarify the role of endothelial injury in heat-related MI. Furthermore, global efforts to reduce greenhouse gas emissions, including a rapid shift away from fossil fuels and the development of green spaces, are crucial to mitigating the health impacts of global warming (11).

In summary, myocardial injury as a result of prolonged exposure to extreme heat is a serious health concern that is often overlooked. Understanding the link between heat exposure, microvascular injury, and myocardial infarction is necessary to guide preventive strategies. Public health measures such as mass education and awareness campaigns can play a vital role in reducing heat-related cardiovascular risk and promoting timely recognition of early cardiac symptoms. Screening tests should be carried out in high-climate zones, and greater emphasis should be placed on lifestyle changes to improve cardiovascular health. Worldwide efforts to reduce global warming through reforestation, reliance on renewable resources, and adoption of sustainable agriculture are crucial, particularly in low-income areas, because these areas are at high risk for heat-related cardiovascular events. Such strategies and, through coordinated and combined efforts between clinicians, policy makers, and global health organizations, cardiovascular events, especially MI due to hot climate, can be significantly reduced with good outcomes.

“Every degree saved is a heartbeat preserved.”

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