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Review Article

Adrenaline Overload as a Contributing Factor in Myocardial Infarction

Ahlam A. Alwan[1]

[1]Department of Applied Pathological Analysis,College of Science ,Al-Nahrain University,Iraq.

Abstract: A major stress hormone that the adrenal glands produce in response to intense physical or emotional stress is adrenaline, also referred to as epinephrine. Profound changes in the cardiovascular system are produced by these drugs, likely due to stimulation of - or β-adrenergic receptors that would act to increase cardiac work through increasing blood pressure, heart rate and myocardial contractility during myocardial ischemia. Physiologically, at physiological doses adrenaline predominantly leads to vasodilation and coronary blood flow increase by stimulating β_2 receptors in the coronary arteries. Conversely, higher grades of predominant α_1 receptor stimulation lead to systemic vasoconstriction, with subsequent rise in cardiac workload and peripheral resistance. High blood levels of adrenaline (epinephrine) and some aggravating pathophysiological factors, such as arrhythmia, changes in serum potassium, bleeding characteristics, and direct action on the myocardium, are linked to the first phase of myocardial infarction. High levels of adrenaline after an acute MI have been associated with infarct severity, arrhythmogenesis, symptom load and vascular injury based on a clinical biochemistry literature. Adrenaline is a very significative biomarker, it is warning and a target in therapy because appears to be the trigger for maladaptive responses by means of both latter endothelium and electrophysiologic ways. You let me know if details are required for quantitative data tables, receptor specific pathways or pharmacological modulation.

Keywords: Adrenaline (Epinephrine), Myocardial Infarction, Catecholamines, Cardiovascular Physiology, Adrenergic Receptors, Arrhythmogenesis,

1.Introduction

The body's fight-or-flight hormone, adrenaline (sometimes called "epinephrine"), is released in response to Hitting a wall during a marathon is my favorite example of when these burst hormones are generated. Other instances include short-term physical or emotional suffering. Stimulation of α and β adrenergic receptors in the cardiovascular system causes profound effects on heart rate, contractility, vascular resistance, and on the coagulation pathway (Boarescu & Boarescu, 2024). Catecholamines, especially adrenaline, often increase in the circulation following acute myocardial infarction (MI) because of ischemia and sympathetic tone. While this reaction may be helpful at first to sustain perfusion, continual augmentation becomes a maladaptive response and can result in ischemic injury, arrhythmias, endothelial disruption, and other harmful effects (Ostrowski et al., 2013).

Mediator: At low physiological levels, adrenaline's selective activation of β_2 receptors on coronary arteries leads to vasodilation and increased perfusion of the heart. Nonetheless, at high doses α_2 mediated systemic vasoconstriction mainly increases myocardial oxygen demand and afterload and paradoxically reduces microcirculatory flow in ischemic areas (Peaston & Weinkove, 2004). Of importance, a surge of adrenaline leads to platelet hyperactivity and coronary vasospasm, generating dense, fibrinolysis-resistant clot material that may cause thrombosis even in patent vessels (Boarescu & Boarescu, 2024). Clinical signs of direct cardiac tissue damage due to oxidative stress, and β receptor-induced calcium overload can be observed in endothelial dysfunction, arrhythmogenesis and cell death especially in injured or hibernating myocardium (Boarescu & Boarescu, 2024).

The likelihood of lethal ventricular tachyarrhythmias is enhanced even at levels of MI adrenaline induced hypokalemia with QTc prolongation, diminished T-wave amplitude and other repolarization disturbances. These electrophysiological effects can be severe (Struthers et al., 1983). Though adrenaline is a cornerstone of prospective step-up protocols in cardiac arrest promoting the increase of coronary perfusion pressure or achieving restoration of spontaneous circulation, evidence suggests no improvement in long

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term survival and on neurological outcome with its use and that allows us to postulate that inappropriate or uncontrolled use increases the microvascular thrombosis risk as well as there may be a proarrhythmogenic effect probably associated with multiple electrical deaths (Papastylianou & Mentzelopoulos, 2012).

The dose- and time-dependent relationship between a hormone's effects on cardiovascular pathophysiology is shown by the evidence that high doses of adrenaline chronically elevate to worse ischemia, arrhythmic events, endothelial injury and clinical outcome whereas an acute increase in adrenaline can only temporarily sustain hemodynamics in AMI. Increased adrenaline leads to increased oxygen demand of the heart in two ways - more heartbeats and stronger heartbeat. Ischemia, plaque rupture, or vasospasm may result to this mismatch in oxygen supply and demand in individuals with coronary artery disease. Adrenaline also promotes the risk of coronary artery occlusion by enhancing platelet activity, thrombus propagation and more stable clot formation through suppression of fibrinolysis (Papastylianou & Mentzelopoulos, 2012).

2. Proof from Research on Humans

2.1 Arrhythmias with Catecholamine Levels

Elevated plasma noradrenaline and adrenaline levels were also reported in 41 CCU patients, with higher levels noted during AMI, particularly in those who had VF [9]. Higher levels of noradrenaline and adrenaline were detected in a separate group of 48 liter-matched patients who were recruited less than six hours from onset of symptoms. The most severe infarct patients presented higher levels of catecholamines and some developed VF (Bertel et al., 1982). According to these investigations, the likelihood of potentially fatal ventricular arrhythmias is strongly correlated with elevated circulating adrenaline in early AMI.

2.2 Adrenaline and Hypokalemia

As you all know, adrenaline can causes hypokalemia. In healthy volunteers, an intravenous infusion of adrenaline (which gives levels comparable to those in infarction) caused a pronounced fall is blood potassium with QT prolongation and abnormalities in T wave configuration characteristic of the changes which can induce arrhythmia (Struthers et al., 1983). Plasma potassium was inversely correlated with the greater concentration of adrenaline in rabbits and those AMI patients, whose plasma adrenaline levels were higher than medians, respectively, hypokalemia and high blood concentration of adrenaline had been found to be independent predictors in ventricular arrhythmias (Zhao & Pan, 1989).

2.3 Platelet Function and Prothrombotic State

Plasma adrenaline concentrations were higher and the bleeding time was shorter in the AMI patients than in those with unstable angina and non-cardiac chest pain. The shortening of bleeding time after aspirin was present but associated with lower systemic adrenaline levels, which suggest that the balance is toward prothrombogenic state when the level of adrenalin increases (Kristensen et al., 1990).

2.4 Pain Correlation

Indeed, arterial adrenaline was higher in patients with uncomplicated MI paining than in non-paining ones suggesting that the associated adrenaline levels might reflect rather sympathoadrenal activity provoked by pain than the infarct's size itself (Husebye et al., 1990).

3. Mechanistic Insights from Biochemistry

3.1 Myocardial Catecholamine Distribution

Within 1-2 days after the infarction, postmortem analyses showed increased myocardial adrenaline predominantly in periinfarct areas. Based on this, the speculation was raised whether regional adrenaline accumulation could directly elicit arrhythmias and aggravate myocardial injury (Popov et al., 1975).

3.2 Pathophysiologic Cascade

In essence, adrenaline accomplishes this through intracellular signaling pathways (such as cAMP/PKA), which control ion channels and cardiac myocyte excitability, as well as through β_1-adrenergic receptors, which increase heart rate and contractility when stimulated by epinephrine, or α_1-receptors, which induce vasoconstriction (Moro et al., 2013). These changes can disrupt peri-infarct tissue, increase myocardial oxygen requirement, and result in dynamic electrophysiologic disturbances.

4. Rare Paradoxical Effects: Exogenous Adrenaline and MI

Even in young patients without significant CAD, acute MI secondary to therapeutic adrenaline is likely caused by coronary vasospasm and platelet activation (although it is acutely life-saving for emergencies like anaphylaxis), according to anecdotal reports in rare case reports (Moro et al., 2013). This highlights the fact that in some conditions adrenaline can induce ischemia.

5.Results

5.1 Plasma Adrenaline Elevations in AMI

- The mean (SD) plasma adrenaline (E) in 48 patients tested <6 h after onset of symptoms

was 73 pg/mL and maximal values were obtained: 1,098 pmg/mL; vegetative levels are approximately between rest at ~34pg/ml) (Karlsberg et al., (1981.

- In a different trial including 41 CCU patients, even AMI patients without VF had lower plasma adrenaline levels at admission than the VF group (Bertel et al., 1982).
- Norepinephrine was significantly elevated in anterior AMI (~60% higher than controls) and epinephrine was elevated in both types of AMI with no difference between anterior and posterior infarctions in a larger study of 84 patients (chronic ischemic heart disease and anterior and posterior AMI) (Slavíková et al., 2007).

5.2 Adrenaline Correlation with Mortality and Infarct Size

- Over the course of the 18-month follow-up period, individuals with greater peak adrenaline (>1,000 pg/mL) had a substantially higher mortality rate; all deaths occurred in the subset of patients with peak E > 1,000 pg/mL ($p < 0.01$); none of the patients with peak E < 1,000 died (Karlsberg et al., 1981).
- Plasma NE and E levels were associated with infarct size (e.g., LDH level, prognostic score), and those who later experienced VF also displayed higher catecholamine concentrations early after beginning medication (Little et al., 1986).

5.3 Adrenaline and Electrical Instability

- Patients with confirmed infarction and subsequent VF had the greatest plasma adrenaline levels, while those with congestive heart failure also showed markedly higher adrenaline but only mildly elevated norepinephrine (Bertel et al., 1982).
- Adrenaline and noradrenaline were significantly increased in ischemic regions with electric instability versus controls (Slavíková et al., 2007).

5.4 Adrenaline and Pain Correlation

In 22 patients with uncomplicated MI, the mean arterial adrenaline was 0.83 nmol/l ($P < 0.025$), compared to 0.44 nmol/1 in controls. Plasma adrenaline levels were significantly greater in pain patients (1.15±0.23 versus 0.60±0.10 nmol/L), and in this instance, there was a larger association between increased plasma adrenaline and pain severity than there was with infarct size (Husebye et al., 1990).

5.5 Endothelial Injury and Shock

In a STEMI group following the first PCI, elevated levels of noradrenaline and adrenalin were linked to increased endothelial/glycocalyx damage indicators (syndecan-1 and thrombomodulin). Elevated adrenaline was found to be an independent predictor of heart failure and both short-term and long-term mortality in a study; the group with cardiogenic shock exhibited the strongest connections (Ostrowski et al., 2013).

6.Discussion

6.1 Sympathoadrenal Activation in Acute MI

Within the first few hours of onset, it has been reported that acute MI induces marked sympathoadrenal activation with, e.g., plasma adrenaline levels often reaching 20–30× those observed at baseline [22]. The degree of infarction, early complications, and late mortality correlate well with this increase which has a relatively homogeneous pattern in all infarct sites.

6.2 The Marker and Mediator of Arrhythmia Risk: Adrenaline

Electrical storm and ventricular fibrillation: correlations to increased adrenaline levels. This gradient of catecholamine levels appears steepest in arrhythmic subjects Adrenaline could act either as a marker or a mechanism for arrhythmogenesis. The finding that individuals with heart failure had significant levels of adrenaline even when norepinephrine was only slightly elevated highlights the function of adrenaline in acutely pathological circumstances.

6.3 Pain vs. Infarct Size: Drivers of Adrenaline

Sympathetic stimulation may be pain, rather than ischemia, driven on the basis of a study that links increased adrenaline to stress (pain) related symptoms versus infarct size. From a clinical point of view, this means that even in uncomplicated MI, subjective pain severity may be an indirect measure of adrenergic stress.

6.4 Damage to Endothelium: A Catecholamine-Mediated Process

As seen in recent STEMI populations, elevated plasma adrenaline is linked to indicators of endothelial glycocalyx injury, especially during shock. Both of these interactions independently predict death and subsequent heart failure, thus suggesting that catecholamines may induce myocardial as well as vascular injury. This further broadens the deleterious

sequelae of adrenaline, beyond electrophysiologic lability.

6.5 Clinical Implications

- Measurement of adrenaline early in the course of AMI (i.e., within 4-6h) might stratify risk for adverse events such as death and VF.
- The link between endothelial dysfunction and adrenaline highlights the potential benefit of therapies like beta-blockers that target early sympathoadrenal hyperactivity to reduce both arrhythmias and vascular damage.
- The importance of assessing clinical presentation as well as objective criteria in categorizing it by severity is emphasized with the understanding that adrenaline release due to pain may not directly parallel infarct size.

7. Synthesis & Conclusions

- Plasma and myocardial (local) adrenaline levels are elevated considerably in acute MI. Levels are associated with infarct size and outcomes such a heart failure and arrhythmias.
- Adrenaline induced hypokalemia also contributes to the risk of arrhythmia by adding to direct receptor mediated repolarization abnormalities in the development of ventricular electrical instability.
- Platelet hyperreactivity and reduced bleeding times are prothrombotic effects especially after aspirin75avdrewarylAdrenaline increases early risk of thrombosis in AMI.
- Whether the infarct is large or small, pain-related stress may enhance adrenaline release, providing support for a relationship between sympathetic activation and clinical presentation.

Clinical implications: while abrupt stressor injection may temporarily raise measured catecholamine for research purposes without affecting overall mortality, adrenergic inhibition (i.e., beta blockers, such as metoprolol) may reduce adrenaline-mediated detrimental hemodynamic and arrhythmic effects.

8. Limitations and Future Prospects

- The majority of human research are observational and have large sample sizes, which restricts their capacity to draw conclusions on causality;
- Time points for adrenal reanalysis are examined and utilize different techniques between studies and may impact comparison.
- There is limited regional information suggesting that local release of adrenaline (especially in peri-infarct regions) probably precipitates arrhythmias but there remains scarce differentiation between systemic and myocardial origin for catecholamines .
- Therapeutic windows of adrenergic regulation and the possible crosstalk between long-term vascular remodeling and endothelial damage induced by catecholamines warrant further investigation.

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