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The reperfusion ST-peak in acute myocardial infarction: a topic where the current knowledge is insufficient

To the editor,

Reperfusion therapy is the primary treatment for ST-elevation myocardial infarction (STEMI). It is an attempt to minimize the myocardial damage and improve clinical outcomes by restoring blood flow. Resolution of ST-elevation on electrocardiogram is considered a positive variable assessing the efficacy of coronary flow restoration. In the last two decades several studies have demonstrated a new phenomenon named "reperfusion peak" (RP).[1,2] It is a transient ST-elevation in patients undergoing either primary percutaneous coronary intervention or thrombolytic therapy followed by complete ST-resolution. It has been observed in 68–75% of patients treated with thrombolysis and in 23–63% undergoing primary coronary intervention.[3] Early on it was interpreted as a positive sign of reperfusion. However this interpretation has now changed and most consider RP a manifestation of reperfusion injury. The RP has been associated with an increase in myocardial infarct size, lower left ventricular ejection fraction, higher level of troponin-T release and NT-pro-brain natriuretic peptide.[4] Furthermore a recent work demonstrated adverse cardiac clinical outcomes in patients who developed ST-peak during reperfusion compared with those who did not develop a ST-peak. Rates of cardiac mortality (8% vs. 3%; $p = 0.047$) and cardiac events and admission for heart failure (19% vs. 10%, $p = 0.018$) were the main findings.[5]

As for the pathophysiology the most common opinion is that RP totally or partially may be due to the rise of heart rate. This leads to a reduction of the left ventricular diastolic period and subsequently coronary blood flow.[6] Distal embolization has also been suggested as a cause of RP. However this hypothesis was not supported in the previously mentioned investigation. It is possible that RP is an electrophysiological phenomenon caused by potassium washout during reperfusion, although this needs further study. Some studies have found an association between transient ST-elevation and patients who have a large ischemic risk prior to vessel recanalization. Other investigators argue that RP is certainly caused by reperfusion injury.[4]

This is a clinically important question as it may have implications for the prognosis of patients who experience it. As discussed there is no uniform agreement as to the pathophysiologic mechanisms involved in the production of RP. Because of that lack of understanding, pharmacological or mechanical therapies have not been developed to modify it. However, the prevalence of STEMI and reperfusion therapy is fairly high, making this an important topic to discuss and understand. Future investigations should focus on the pathophysiology of this phenomenon. An adequate understanding of the pathophysiology will help us comprehend the significance of RP and design new strategies to achieve better medical care of these patients.

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