CRITICAL CARE

COMMENTARY

Clinical correlates of arterial lactate levels in STEMI patients

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Abstract

Increases in blood lactate reflect decreases in systemic blood flows associated with low blood flow states characteristic of circulatory shock. Accordingly, the report by Vermeulen and colleagues documents the use of the blood lactate measurement as a prognostic indicator in settings of ST elevation myocardial infarction. That lactate value therefore identified high-risk patients as a complication, often with clinical signs of cardiogenic shock of corresponding severities.

The contribution by Vermeulen and associates documents measurements of blood lactate in an impressively large number of patients with ST elevation myocardial infarction [1]. On admission to the cardiac catheterization laboratory, increases in arterial blood lactate in patients corresponded to the magnitude of the infarct. Some of the patients had clinical signs of circulatory shock, including increases in heart rate and decreases in arterial pressure. If there was less successful restoration of coronary blood flow after catheter interventions, disproportionately higher blood lactate concentrations were observed.

Increases in arterial blood lactate in coronary settings were therefore most probably accompanied by decreases in cardiac output and thus by decreases in systemic blood flows consistent with cardiogenic shock of corresponding severities. To the extent that lactate measurements were obtained early after admission, they were indeed likely to be predictive of both short-term and long-term outcomes. In accordance with the historically useful Killip classification, the close relationship among the outcomes in patients undergoing primary percutaneous coronary interventions was confirmed by DeGeare and colleagues

[2]. Specifically, patients without physical signs characteristic of reduced systemic blood flows due to heart failure after acute myocardial infarction had a favorable outcome, and patients with reduction in cardiac output and heart failure had a poor prognosis.

Increases in lactate in settings of cardiogenic shock were first reported by our group in 1974 [3] and secured the earlier findings [4,5] that lactate measurement is highly predictive for outcomes of circulatory shock states. In the present report, myocardial infarction led to reduced cardiac work capacity and therefore reduced systemic blood flows. The increases in lactate were then best explained by critical reduction in cardiac output, which is typically accompanied by physical signs, including tachycardia hypotension, cyanosis and pallor, third heart sounds and cold extremities. The sources of the lactate increase may in part be due to ischemic myocardium, but the likelihood is that lactic excesses were primarily due to systemic circulatory failure. Unrelated causes of increased lactate are associated with struggling, convulsive seizures and hyperthermia, and must be excluded [6].

The evolution of lactate measurement followed the early studies of Huckabee [7,8] who, largely on the basis of earlier investigations in exercise physiology and based on measurements of both serum pyruvate and lactate values, clarified the value of the so-called excess lactate as a quantitator corresponding to the systemic oxygen deficit [4]. Subsequent workers confirmed that excess lactate was a valuable measurement and yet later demonstrated that the measurement of lactate alone in blood or serum was sufficient [5,8-10]. With present methods of facile point-of-care laboratory measurements, as utilized in the study by Vermeulen and colleagues [1], lactate proved a useful prognosticator for the severity of myocardial infarction. When acute myocardial infarction was accompanied by lactic acidosis, systemic blood flow was critically reduced.

The present report, therefore, again demonstrates the early prognostic value of lactate as an indicator for the severity of decreased systemic blood flows with correspondingly poor outcomes and, in this instance, in

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catheter laboratory settings of ST elevation acute myo-cardial infarction.

Abbreviations
STEMI, ST elevation myocardial infarction.

Competing interests
The authors declare that they have no competing interests.

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