COMMENTARY ARTICLE

Electrocardiographic changes in acute pancreatitis: distinguishing cardiac events from pancreatic manifestations

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DESCRIPTION

Acute Pancreatitis (AP) is an inflammatory condition of the pancreas that can induce a variable host immune response, ranging from local tissue injury to systemic inflammatory response syndrome and organ failure [1]. Cardiovascular Diseases (CVD) have long been described as the leading cause of death worldwide, with an estimated 17.9 million deaths each year [2]. The presence of abnormal ECG findings and a cardiac event in a patient with acute pancreatitis creates a significant diagnostic challenge. Several studies have well documented this phenomenon of acute pancreatitis mimicking Acute Coronary Syndrome (ACS), and the earliest case report, dating back to 1934, describes Drummond's initial observation of ECG changes related to acute pancreatitis [3-10].

The co-occurrence of AP and ACS has generally been considered an infrequent association. A retrospective cohort study found that only 0.46% of AP patients experienced concurrent ACS. Although concomitant presence of the 2 pathologies was extremely rare, it is critical to recognize this diagnostic dilemma. Awan et al. indicated that Inpatient mortality was significantly higher in the AP with concomitant ACS cohort compared with the AP without ACS cohort (8.4% vs 0.5%, adjusted OR 9.94, p <0.05) [11]. Empirically treating all cases as a true ACS may expose the patients to thrombolytic therapy and/or invasive cardiac catheterization unnecessarily; on the other hand, the consequences of could be fatal if all pertinent ECG and biomarker changes were to be misattributed to AP alone.

AP is capable of presenting with a variety of clinical manifestations similar to ACS, thus complicating accurate identification of disease. Various ECG abnormalities were associated with AP in past research, which include tachy- and bradyarrhythmia, atrial fibrillation and flutter, premature supraventricular contractions, short PR interval, QRS prolongation, bundle branch blocks, nonspecific changes of repolarization, and ST-T changes [3,5,6,10,12,13]. Rubio-Tapia et al. concluded that more than 50% of AP patients had an abnormal ECG from baseline, with the most frequent findings being nonspecific changes of repolarization, sinus tachycardia, and left anterior hemiblock [13]. In contrast, Gu et al. found that only 2.3% patients with suspected ST-Segment Elevation Myocardial Infarction (STEMI) referred for coronary angiography and primary percutaneous coronary intervention was discharged with a final diagnosis other than ACS over a 18-month period. Notably, less than half of them (7/19, 36.8%) were diagnosed with a non-cardiac pathology, and none of them was AP [14].

Elevated cardiac biomarkers are common in AP. Myocardial injury can occur in AP, irrespective of severity, with troponin elevation observed in up to 54% of patients where measured [8]. Overall, it was challenging for physicians to delineate between the 2 pathologies based on any single clinical or laboratory feature. The exact mechanism of acute pancreatitis-induced myocardial injury and its associated impact on clinical outcomes had not been clearly understood; nonetheless, multiple hypotheses had been proposed trying to explain this phenomenon. It is well studied that proteolytic enzymes released in AP is responsible of tissue destruction, secondary pancreatic necrosis, and

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pancreatic pseudocyst formation [15]. The excessive activation of the inflammatory cascade leads to a surge of pro-inflammatory mediators entering the bloodstream, further intensifying the systemic inflammatory response through a cytokine storm. In a narrative review, Luo et al. concluded that both pancreatic enzymes (e.g. pancreatin, trypsin, pancreatic lipase) and systemic inflammatory factors (e.g. TNF-α, interleukin family, NO & iNOS, ROS, HMGB1) can contribute to laboratory-defined myocardial injury and a clinical diagnosis of myocardial infarction [16]. Coagulation cascade might be affected by these enzymes, resulting in transient formation of thrombus and acute coronary occlusion [17]. Yu et al. reported 32 cases of acute pancreatitis with ECG features mimicking acute myocardial infarction. Inferior wall infarction patterns were the most common, suggesting trans-diaphragmatic spread of epicardial inflammation as the underlying pathogenesis, due to the pancreas's retroperitoneal location relative to the heart3.

Pathological stress induced by critical illness of AP may produce regional dysfunction of the left ventricular apex and subsequent compensatory hyperkinesia in the basal walls, which is a phenomenon described as "Takotsubo cardiomyopathy" or "broken heart syndrome" and a classic mimic of ACS [3,18,19]. Lab-defined myocardial injury may result from sympathetic overactivation and excessive catecholamine release due to severe abdominal pain in AP [8]. Conversely, increased autonomic vagal tone in AP, or "cardio-biliary reflex," can also lead to transient spasm of the coronary vessels and manifest in ACS-like features [3,20]. Since the heart and biliary tract share overlapping innervation at the T4-T5 level, localized inflammation in the biliary system may trigger somatic nociception that is misinterpreted by the heart. This cross-innervation could lead to a coronary spastic event, potentially resulting in a temporary decrease in cardiac output and cardiogenic shock [20].

Despite the clinical similarities, no single definitive marker differentiates these two conditions. This atypical presentation necessitates vigilant monitoring, comprehensive assessment, and serial examinations for accurate diagnosis and timely intervention. Current ACS guidelines recommend serial ECG evaluation and point-of-care echocardiography in patients with unclear diagnoses [21,22]. This review highlights the importance of cautious interpretation by cardiologists and emergency physicians to avoid premature conclusions and unnecessary invasive procedures.

CONFLICTS OF INTERESTS

None of the authors have any conflicts of interest to declare.

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