Heart in acute pancreatitis: facts and fictions

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Abstract

Pain is the hallmark of acute pancreatitis and it is localized in the epigastrium in more than 60% of patients having mild or severe disease. Acute pancreatitis may mimic other diseases such as acute coronary syndrome. In addition, the acute illness of the pancreas is associated with a number of metabolic abnormalities, such as hypocalcemia and hypophosphatemia, which may cause hemodynamic changes and variations in the concentration of ionized calcium. In turn, these have been directly correlated to changes in myocardial contractility. The aim of this paper is to review the current literature on the involvement of heart during the course of acute pancreatitis and also to evaluate experimental and clinical data on this topic.

Introduction

Pain is the hallmark of acute pancreatitis and it is localized in the epigastrium in more than 60% of patients having mild or severe disease.1 Sometimes acute pancreatitis may mimic other diseases and, in particular, acute coronary syndrome.2 In addition, the acute illness of the pancreas is associated with a number of metabolic abnormalities, such as hypocalcemia and hypophosphatemia which may cause hemodynamic changes and variations in the concentration of ionized calcium. In turn, these have been directly correlated to changes in myocardial contractility. Our aim is to review the current literature on the involvement of heart during the course of acute pancreatitis and to evaluate experimental and clinical data on this topic.

Pathogenesis of acute pancreatitis

The main etiology of acute pancreatitis, at least in Western countries remains that of bil-
collagenization of myocardial stroma. The electrocardiographic alterations in acute pancreatitis are various, say tachyarrhythmia or bradyarrhythmias, atrial flutter and atrial fibrillation, supraventricular premature contractions, short PR interval, QRS prolongation, various bundle-branch blocks (such as left and right bundle-branch block, and left anterior hemiblock), non-specific changes in depolarization, decreased T-wave voltage, T-wave changes, and ST-segment abnormalities. These alterations are often seen in approximately 50% of patients. Evaluation of left ventricular function in the early phases of acute pancreatitis has also been investigated, showing an impairment of contractility in a significant portion of patients with acute pancreatitis. Echocardiographic assessment based on clinical parameters of severity may help to select those patients who merit highly intensive treatment.

The laboratory evidence

Easy and largely available serum markers are required to rapidly identify those patients having a cardiac involvement during the course of acute pancreatitis. In a recent study, we evaluated the presence of elevated levels of high-sensitivity cardiac Troponin (hs-TnT) in patients with acute pancreatitis, and we found that more than 35% of them had high serum levels of this cardiac marker. The time course of serum troponin T in the early phases of acute pancreatitis is reported in Figure 2. However, the absence of any clinical and electrocardiographic features of acute coronary syndrome in our patients suggests that abnormally high results should be interpreted as of non-ischemic origin. In conclusion, we believe that troponin I should be used in assessing cardiac damage in acute pancreatitis patients, but additional studies exploring this possibility are needed.

The future way

Other markers of cardiac damage should be evaluated in clinical practice for a more in-depth evaluation of cardiac involvement in acute pancreatitis of different severity. For example, arginine vasopressin (AVP) also known as antidiuretic hormone (ADH) is one of the key hormones for cardiovascular homeostasis. Despite its pivotal role in cardiovascular diseases, both the measurement and the diagnostic use of AVP have never reached clinical practicability due to the technical problems related mainly to three reasons: its short plasma half-life, its interaction with platelets in the serum, and its small circulating quantity. Another interesting molecule to test in clinical practice is copeptin, a glycosylated 39-amino acid long peptide with a leucine-rich core segment. Copeptin and AVP share the same precursor peptide, the 164-amino acid long preprovasopressin and copeptin is the C-terminal part of pro-AVP (CT-pro-AVP) and it is released together with AVP during precursor processing. In contrast to AVP, copeptin is very stable in serum or plasma at room temperature, and is easy to measure. In contrast to many other biomarkers, the copeptin plasma concentration was similar in different age groups and showed no correlation with age. A particularly interesting observation was the response of circulating copeptin levels as a result of an acute myocardial infarction since these protein
levels were higher in patients who died or were readmitted with heart failure as compared to event-free survivors. Thus, the measurement of plasma copeptin should be investigated in acute pancreatitis patients in order to select those who require more intensive support.

Finally, adrenomedullin (ADM), a 52-amino-acid peptide elevated in plasma of patients having heart failure and/or post-acute coronary syndrome, has been isolated in human pheochromocytoma and it is also present in the heart, brain, lung, kidney and gastrointestinal organs; it has a potent vasodilator activity due to an increase in cyclic adenosine monophosphate levels. It should be underlined that the quantification of ADM is quite difficult due to a short half-life and the lack of the reliable laboratory techniques. The identification of mid-regional pro-adrenomedullin (MR-proADM) has overcome these problems, because it is a stable peptide possibly reflecting the concentration of ADM. MR-proADM is probably secreted in equimolar amounts to those of ADM, and it does not have any physiological effects which might explain its apparent stability. Plasma MR-proADM concentrations predict an adverse outcome in patients in the recovery phase of myocardial infarction and appear to add information beyond the strong predictor N-Terminal pro-B-Type natriuretic peptide (NT-proBNP). There are no studies on the ADM circulating concentration in acute pancreatitis patients and this topic should be explored in order to obtain more accurate information regarding cardiac involvement in acute pancreatitis. In any way, it has been recently reported that MR-proADM is a strong predictor of rehospitalization and mortality in patients with septic shock and we believe that this molecule may have a practical role in patients with infected pancreatic necrosis. Regarding NT-proBNP, we have found that this molecule is abnormally elevated in acute pancreatitis and its elevation persists for at least 72 hours (Figure 3). In addition, similar high levels of serum NT-proBNP are present in patients with mild acute pancreatitis as well as in those with severe disease. Thus, we should be aware that elevated values of NT-proBNP in patients with acute pancreatitis cannot be useful to detect hearth failure.

Conclusions

The exact mechanism of myocardial injury during the course of acute pancreatitis still remains unclear. In clinical practice, laboratory examinations are needed to distinguish in the emergency room patients with epigastric pain having acute pancreatitis from those with acute myocardial infarction because the treatment strategy of the two diseases differs markedly. In addition, further efforts should be made to identify acute pancreatitis patients at high risk of cardiovascular disease and the new above-mentioned markers need to be explored for their possible use in clinical practice.

References


