APŽVALGINIAI STRAIPSNIAI

Predicting development of infected necrosis in acute necrotizing pancreatitis

Žilvinas Dambrauskas1,2, Juozas Pundzius1, Giedrius Barauskas1
1Clinic of Surgery, 2Institute for Biomedical Research, Kaunas University of Medicine, Lithuania

Key words: acute necrotizing pancreatitis, infected necrosis of pancreas, prognostic markers.

Summary. The incidence of severe acute pancreatitis is about 30 cases per 100,000 inhabitants, and it carries an overall mortality rate of 10–15%. Infection of pancreatic necrosis occurs in 20–30% of patients with severe acute pancreatitis and triples the mortality rate. Therefore, early prediction and diagnosis of infection in necrotizing pancreatitis are extremely important.

The aim of the studies included in this review was to investigate the potential of specific prognostic factors to predict the development of secondary pancreatic infection in severe acute pancreatitis. This is seen as an important tool allowing to perform a computed tomography- or ultrasound-guided fine needle aspiration for bacteriological sampling at the right moment, to confirm the diagnosis, and, finally, to select the subgroup of patients who would benefit from the antibiotic prophylaxis. Precise patients’ selection could possibly result in more rational use of antibiotics in patients with acute necrotizing pancreatitis and reduction of multi-resistant bacteria.

Recent studies show that C-reactive protein is an important prognostic marker of pancreatic necrosis with the highest sensitivity and negative prognostic value in this respect. Procalcitonin alone or in combination with interleukin-6 best identifies patients not at risk for infection. However, a review of the clinical studies suggests that we still do not have an optimal model, thus there is a need for new more reliable biochemical and/or clinical predictive systems.

Severe acute pancreatitis

Most patients with acute pancreatitis have a mild disease that resolves spontaneously within few days. However, 20–30% of patients develop necrosis of the pancreas and multiple organ failure, which may lead to death. Its incidence is about 30 cases per 100,000 inhabitants and it carries an overall mortality rate of 10–15% (1, 2). Most patients who die from pancreatitis belong to the severe group which has a mortality rate approaching 40% (3, 4). Infection occurs in 30–40% of patients who have more than 30% necrosis of the pancreas (5, 6). Furthermore, infection accounts for approximately 80% of deaths from acute pancreatitis. Consequently, a question arises whether prophylactic antibiotics may prevent occurrence of such complications (5, 7). Therefore, early diagnosis of infection in a necrotizing pancreatitis is extremely important. Increased abdominal pain, fever, high leukocyte count, and/or organ failure are common clinical signs that might be attributed to the development of infection in necrotic tissue. However, there are no specific signs or symptoms to differentiate sterile necrosis from infected. This can only be confirmed by computed tomography (CT)- or ultrasound-guided aspiration of necrotic material or peripancreatic fluid collections (8, 9). The current recommendations advocate fine-needle aspiration (FNA) for culture to confirm infected necrosis in patients with signs of sepsis (10).

During recent years the management of acute pancreatitis has changed. This has been particularly due to the general availability of CT, improved intensive care facilities, knowledge about the central role of pancreatic infection, and refinements in surgical and other interventional techniques. Evidence-based guidelines for management of severe acute pancreatitis, developed by International Association of Pancreatolog (IAP) in 2002, have fostered these changes (11). Conservative treatment of severe acute pancreatitis is based on early evaluation of disease severity, invasive organ system monitoring, and appropriate fluid resuscitation in the initial stage of the disease. Prophylactic...
administration of antibiotics targeted to prevent translocation of bacteria from the intestine has been shown to be useful in several studies. The effectiveness of early and adequate antibiotic treatment has been confirmed in several randomized clinical studies and by systemic meta-analysis (12, 13). Treatment of patients with sterile necrosis is mostly conservative, but might be reconsidered if the patient’s status deteriorates despite appropriate conservative treatment.

Why do we need to predict development of infected pancreatic necrosis?
Infection of pancreatic necrosis worsens the prognosis of patients with severe acute pancreatitis and triples the mortality rate (14). Despite substantial necrosis, mortality remains low (0 to 10%) as long as the necrotic tissue remains sterile and increases dramatically when infection develops (>30%) (15–17). The incidence of infection correlates with the extent of necrosis and is associated with prolonged hospital stay and increased morbidity (6, 18).

Six prospective randomized trials carried out in the 1990s suggest beneficial role of prophylactic broad-spectrum antibiotics in patients with CT-proven pancreatic necrosis (19–24). CT-guided fine-needle aspiration of pancreatic necrosis or peripancreatic collections is a key procedure to diagnose infection with bacteriological identification. However, positive culture implies worse prognosis and usually requires surgical intervention. This scenario prompted researchers to investigate the potential of several inflammatory markers and scoring systems to predict the risk of secondary infection. These could potentially be helpful to select patients who may benefit from antibiotic prophylaxis (25).

CT- or ultrasound-guided FNA is the “gold-standard” for the early diagnosis of infected necrosis. During the last decade sensitivity and specificity of FNA in necrotizing pancreatitis is reported to be approximately 90%. Unfortunately, the demand for high standard technical equipment and experienced personnel as well as the potential risk of complications such as bleeding or iatrogenic infection prevent it from being readily available and cost effective procedure. Therefore, noninvasive and universally available biochemical or clinical parameters for identifying patients with infected necrosis would definitely contribute to more simple and safe diagnosis (9, 26).

Another argument driving us towards the reconsideration of preventive antimicrobial treatment is serious side effects of broad-spectrum antibiotics in a subgroup of treated patients. These include fungal infection, pseudomembranous colitis, and emergence of multi-resistant bacteria (27, 28). The World Health Organization (WHO) also alerts that bacterial resistance is becoming very common in surgical wards and intensive care units (ICU). The misuse of antibiotics is responsible for this situation, at least in the well-developed world. The WHO has described the need for new strategies to fight surgical infection, as antibiotics will become more and more obsolete in the future. Although the prophylactic use of broad-spectrum antibiotics has reduced the incidence of infected pancreatic necrosis and, consequently, the need of surgery, it is common to find that microorganisms involved in the secondary pancreatic infection have changed. Gram-negative bacteria of gastrointestinal origin are gradually replaced with methicillin-resistant Staphylococcus aureus, Candida glabrata, as well as some other highly resistant microorganisms. This observation is confirmed by numerous authors and represents a serious problem since this type of infection leads to higher mortality rates even when appropriately treated.

In the study by M. W. Büchner et al., administration of antibiotics (i.e., imipenem) against gram-negative rods and anaerobes resulted in predominant isolation of gram-positive cocci or rods and fungi (29). The same group reported a 8% rate of fungal infection and a 3% rate of multiresistant bacterial infection (two of them were lethal) in 103 patients with acute necrotizing pancreatitis receiving prophylactic broad-spectrum antibiotics (28). H. G. Beger and C. W. Imrie have also alerted of evolving antibiotic resistance and fungal infection. They have even questioned the use of antibiotic prophylaxis in the context of the latter findings (30).

Presented data generated interest to determine early markers for identifying patients who are prone to develop an infection in necrotic pancreas. Several multifactorial scoring systems such as multiple organ failure, sepsis severity, and Acute Physiology and Chronic Health Evaluation II (APACHE II) scores, as well as various blood parameters are known to be good predictors of necrosis or overall severity in acute pancreatitis (31–35). Much effort has been spent to identify the role of inflammatory markers such as tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), C-reactive protein (CRP), and phospholipase A2 that reflect the severity of pancreatitis. However, potential of these markers to identify patients at risk to develop infected necrosis remains undefined.

To better target patients in whom antibiotic prophylaxis would be beneficial, we need to precisely identify clinical and/or serological markers capable
Table 1. Biochemical markers as early predictors of necrosis infection

<table>
<thead>
<tr>
<th>Authors/study model</th>
<th>Patients (No)</th>
<th>Analysis of biochemical markers</th>
<th>Conclusions and cut-off values</th>
</tr>
</thead>
<tbody>
<tr>
<td>B. Rau <em>et al.</em> (1997) Prospective</td>
<td>50</td>
<td>– not evaluated</td>
<td>+ AUC=0.70 p&lt;0.012</td>
</tr>
<tr>
<td>M. Armengol <em>et al.</em> (1999) Prospective</td>
<td>150</td>
<td>– not evaluated</td>
<td>not evaluated</td>
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<tr>
<td>Y. Mandi <em>et al.</em> (2000) Prospective</td>
<td>30</td>
<td>– ns</td>
<td>not evaluated</td>
</tr>
<tr>
<td>S. R. Mettu <em>et al.</em> (2003) Prospective</td>
<td>40</td>
<td>– not evaluated</td>
<td>not evaluated</td>
</tr>
<tr>
<td>F. C. Riche <em>et al.</em> (2003) Prospective</td>
<td>48</td>
<td>+ AUC=0.77 p&lt;0.003</td>
<td>+ AUC=0.78 p&lt;0.04</td>
</tr>
</tbody>
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Abbreviations: “+” – valuable marker; “–” – no proven benefit; acc. – accuracy; AUC – area under curve; not evaluated – not included in specific study; npv – negative predictive value; ns – not significant difference; ppv – positive predictive value; sens. – sensitivity; spec. – specificity; IL-6 – interleukin-6; IL-8 – interleukin-8; PCT – procalcitonin; CRP – C-reactive protein; sICAM-1 – soluble intercellular adhesion molecule-1; TNF-α – tumor necrosis factor-α; RNI – reactive nitrogen intermediate.
### Table 2. Multifactorial scoring systems as early predictors of necrosis infection

<table>
<thead>
<tr>
<th>Authors/study model</th>
<th>Patients (No)</th>
<th>Analysis of multifactorial scoring systems</th>
<th>Conclusions and cut-off values</th>
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<td></td>
<td>SAPS 2</td>
<td>Ranson</td>
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<tr>
<td>B. Rau et al. (1997)</td>
<td>50</td>
<td>not evaluated</td>
<td>+ p&lt;0.003</td>
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<td>Prospective</td>
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<td>150</td>
<td>not evaluated</td>
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<tr>
<td>Prospective</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>K. I. Halonen et al. (2003)</td>
<td>234</td>
<td>not evaluated</td>
<td>AUC=0.65 ns</td>
</tr>
<tr>
<td>Retrospective</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>S. R. Mettu et al. (2003)</td>
<td>40</td>
<td>not evaluated</td>
<td>+ p&lt;0.05</td>
</tr>
<tr>
<td>Prospective</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>F. C. Riche et al. (2003)</td>
<td>48</td>
<td>+ AUC=0.75 p&lt;0.002</td>
<td>+ AUC=0.65 p&lt;0.03</td>
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<tr>
<td>Prospective</td>
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</tbody>
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Abbreviations: “+” – valuable marker; “-” – no proven benefit; acc. – accuracy; AUC – area under curve; not evaluated – not included in specific study; npv – negative predictive value; ns – not significant difference; ppv – positive predictive value; sens. – sensitivity; spec. – specificity; CTSI – Computed Tomography Severity Index; SAPS 2 – Simplified Acute Physiology Score II; APACHE II – Acute Physiology and Chronic Health Evaluation Score II; MODS – multiple organ dysfunction scores.
to discriminate between groups of patients with and without infectious complications. Compared to FNA, the current “gold standard,” accurate and readily available biochemical parameters for identifying patients at risk to develop infected necrosis would contribute to non-invasive and cost-efficient diagnosis. However, only a few biochemical parameters and scoring systems have been investigated with respect to their clinical relevance to diagnose secondary pancreatic infection.

**Review of studies on prediction of infected necrosis in acute pancreatitis**

The aim of the six studies included in this review was to investigate the potential of specific prognostic factors to predict the development of secondary pancreatic infection (SPI) in severe acute pancreatitis (SAP). This is seen as an important tool allowing to perform a CT- or ultrasound-guided FNA for bacteriological sampling at the right moment, to confirm the diagnosis, and, finally, to select the subgroup of patients who would benefit from the antibiotic prophylaxis. Precise patients’ selection could possibly result in more rational use of antibiotics.

The ability of inflammatory markers (Table 1) in the serum (CRP, IL-6, interleukin-8 (IL-8), procalcitonin (PCT), etc.) as well as classic severity indices (Table 2) (APACHE II, Ranson, Imrie, CT Severity Index (CTSI), etc.) to predict infection of necrosis has been assessed using receiver operating characteristic (ROC) methodology in majority of included studies. The aim of study by K. I. Halonen et al. (2003) was to construct a novel prediction model to predict fatal outcome in early phase of severe acute pancreatitis and to compare this model with previously reported predictive systems.

All the multifactorial scoring systems demonstrated certain ability to discriminate between the groups of patients with infected and noninfected necrosis. Nevertheless, authors conclude that Ranson, Imrie, and multiple organ dysfunction scores are inaccurate indicators of the infected necrosis and mortality in SAP. Some evidence also showed that certain serum markers might play an important role in determining the course of necrotizing pancreatitis, but further studies should be carried out to confirm these data. Overall, the results of these studies provided a possibility to predict secondary pancreatic infection in SAP, allowing bacteriological confirmation and early treatment of this severe condition, but none of the currently used scoring systems or serum markers are fully consistent.

**C-reactive protein**

Overall median concentration of CRP in edematous pancreatitis usually remains quite low. Therefore, CRP has the highest predictive value (area under the ROC curve (AUC)=0.92) for the presence of necrosis, comparing to PCT (AUC=0.79) and IL-8 (AUC=0.52) (25). The results of our own study show that the CRP values increase significantly in early stages of necrotizing pancreatitis. The highest sensitivity and negative predictive value (94.1 and 95.7%, respectively) was obtained for C-reactive protein cut-off at 110 mg/l (36).

However, there is no significant difference shown in median concentrations of CRP in patients with infected necrosis and in those with sterile necrosis during the early period of the disease. Nevertheless, CRP levels are related to the development of secondary pancreatic infection as shown by univariate and multivariate regression analysis, and it could be used as a marker for the preoperative differentiation between infected necrosis and sterile necrosis in severe acute pancreatitis. The best calculated cut-off value for CRP is 300 mg/l (sensitivity – 83%; specificity – 78%; accuracy – 84%, AUC=0.86), however, CRP level rarely exceeds this value during the first 3–4 days from the onset of the disease. Other authors also show similar accuracy (approx. 80%) of CRP (sensitivity – 80% and specificity – 70–75%) in prediction of SPI in SAP (26, 37).

**Interleukins**

Overall serum levels of IL-8 and IL-6 in edematous pancreatitis are low. Median concentration of IL-8 is significantly higher in patients with infected necrosis when compared to those with sterile necrosis even during the first days after the onset of SAP. The best cut-off value for IL-8 is 112 pg/ml (sensitivity – 72%; specificity – 75%; accuracy – 74%, AUC=0.70). After surgical debridement values of IL-8 remain significantly higher in patients with persistent pancreatic sepsis compared to those having uneventful postoperative course. As IL-8 is known to be a potent neutrophil-activating cytokine, these results further support the hypothesis that polymorphonuclear cells (PMNC) play a central role in the development of SAP and septic multiple organ failure as the result of infected pancreatic necrosis. Therefore, monitoring of IL-8 may provide valuable information on the severity of septic complications in critically ill patients (9, 26).

IL-6 is produced at rather high concentrations in both types of pancreatic necrosis (infected and sterile), and its levels remain elevated for several days even after surgical debridement and establishment of conti-
nous lavage. Nevertheless, concentrations of IL-6 are markedly higher in patients with infected necrosis (p<0.04) during the initial 3-day period after onset of acute pancreatitis. In addition, the AUC of IL-6 (0.77) is greater than the AUC of Simplified Acute Physiology Score II (SAPS II) (0.75), Ranson score (0.65), and CTSI score (0.67). Elevated serum IL-6 levels are characteristic in systemic inflammatory response syndrome of either infectious or noninfectious origin. Immunomodulatory properties of IL-6 include triggering of PMN-mediated hyperinflammation and, interestingly, delayed host immunosuppression. Elevated IL-6 may reflect the fact that a certain level of local or general immunosuppression has been reached, allowing microorganisms to proliferate (25, 38).

**Procalcitonin**

Overall median concentrations of PCT in edematous pancreatitis are low. These are significantly higher in patients with infected necrosis (8.5±4.8 ng/ml) compared to those with sterile necrosis (<1.2 ng/ml) already during the first week of the disease (p<0.003). PCT at relatively high concentrations is also found in patients with sepsis of different origin (15±5.4 ng/ml). By comparison of the areas under the ROC curve, PCT was found to have the closest correlation with the presence and severity of bacterial/fungal infection of necrosis compared with IL-8 and C-reactive protein. In addition, the AUC of PCT (0.78) and IL-6 (0.77) were greater than that of SAPS II (0.75), Ranson score (0.65), and CT severity index score (0.67). The best cut-off value for PCT is 1.8 ng/ml (sensitivity = 94%; specificity = 91%; accuracy = 92%, AUC=0.96). Diagnostic accuracy of preoperative differentiation between infected and sterile necrosis by means of ultrasound-guided FNA was 84% (sensitivity = 90%; specificity = 79%) compared to 87% for PCT (sensitivity 80%; specificity = 93%). Other authors also showed PCT level to be an accurate, readily available parameter allowing discrimination of infected pancreatic necrosis, being a helpful marker facilitating decision-making in SAP patients. Sensitivity, specificity, and positive predictive values for discriminating infected and sterile pancreatic necrosis are 90, 100, and 100%, respectively (p<0.0001). In the present study, the combination of IL-6<400 pg/l and PCT<2 ng/l has been shown to best identify patients who are not at risk for secondary pancreatic infection and in whom antibiotic prophylaxis may be useless. The negative predictive value for proposed thresholds was 91%, whereas sensitivity and specificity were 75 and 84%, respectively (25, 26, 38).

These findings lead to the conclusion that serum levels of PCT closely correlate with both the morphological features of infected necrosis and the severity of associated systemic complications. Therefore, monitoring of serum PCT is a potential new marker for the noninvasive and accurate prediction of infected necrosis as well as for the selection of patients with persisting septic complications after surgical debridement.

**Other inflammatory cytokines**

Soluble intercellular adhesion molecule-1 (sICAM-1) is overproduced in both types of pancreatic necrosis (infected and sterile). High levels of sICAM-1 remain elevated for several days even after surgical elimination of the infected focus (widespread necrosectomy and continuous lavage). Sensitivity, specificity, and positive predictive values for discriminating infected from sterile necrosis are 90, 10, and 50%, respectively (p<1.000 n.s.). Elevated serum sICAM-1 levels are characteristic in systemic inflammatory response syndrome of either infectious or noninfectious origin (38).

The serum nitric oxide levels in the form of reactive nitrogen intermediates (RNIs) were assessed on admission and on day 3 in another study. Levels of RNI were significantly higher in patients with acute pancreatitis as compared with the healthy control group (159.1 vs. 106.0 nmol/ml, p<0.05). But there was no statistical difference between groups of patients with infected and sterile necrosis on admission (168.1 vs. 148.8 nmol/ml, p>0.05) and after 72 hours (175.4 vs. 136.5 nmol/ml, p>0.05), thus, production of RNI was not associated with the development of infected pancreatic necrosis. On the other hand, RNI levels on admission were significantly higher in the subset of patients who developed bacterial sepsis (195.5 vs. 134.7 nmol/ml, p<0.05) and in the non-survivors compared to survivors (216.0 vs. 140.1 nmol/ml, p<0.05). Hence the patients with higher serum nitric oxide levels are at a significantly higher risk of sepsis and mortality, since increased iNOS (inducible nitric oxide synthase) activity leads to enhanced mucosal injury, disruption of the barrier function of gut, and increased bacterial translocation leading to sepsis. This led to the conclusion that acute necrotizing pancreatitis is associated with raised serum nitric oxide levels at its early stage, but the authors failed to demonstrate a role of nitric oxide levels in identifying the severity of acute pancreatitis (39).
No significant difference was noted between the types of pancreatic necrosis (infected and sterile) for TNF-α (tumor necrosis factor-alpha) (AUC=0.5). So the value of this marker in recognizing patients at risk for development of infected necrosis is not established yet (25).

**Acute Physiology and Chronic Health Evaluation II**

Several scoring systems, including APACHE II, are known to be good predictors of necrosis in acute pancreatitis. The disease severity according to APACHE II score is highest in the group of patients with infected necrosis even during the first 48 hours after admission to the hospital. APACHE II score is related to the development of secondary pancreatic infection as demonstrated by multivariate analysis. In the validation set, the predictive accuracy of APACHE II, determined by the area under the receiver operating characteristic curve value, was 0.817 (6, 26, 37, 40).

**Ranson and Imrie scores**

Clinical severity of the disease as assessed by Ranson score differs significantly in patients with sterile and infected pancreatic necrosis. However, predictive accuracy, determined by area under the ROC curve value, is 0.655 for Ranson, and 0.536 for Imrie scores. Consequently, these instruments are considered to be not sufficiently accurate for predicting the development of infected necrosis and mortality in SAP (25, 26, 40).

**CT Severity Index**

Significant differences in CTSI are seen between patients with infected pancreatic necrosis and those without (p<0.04). Regression analysis also confirms that this score is significantly associated with the occurrence of infected necrosis. In addition, the AUC of CTSI (0.67) is comparable to those of Ranson and Imrie scores (25, 39).

**Other clinical severity scores**

In the study of K. I. Halonen et al., the prediction model (Model-4) considered optimal was a logistic model with four variables: age, highest serum creatinine value (within 60–72 hours after admission), need for mechanical ventilation, and chronic health status. In the validation set, the predictive accuracy, determined by the area under the receiver operating characteristic curve value, was 0.862 for this model, 0.817 for APACHE II, and 0.781 for multiple organ dysfunction score. A novel predictive model based on four variables can reach at least the same predictive performance as the APACHE II system with 14 variables. This further validates the statement that there is still a need for the future research in this area (40).

The severity of illness as assessed by SAPS II is significantly higher in the patients with infected pancreas necrosis as compared with sterile necrosis. The AUC of SAPS II is 0.75 as shown by the receiver operating characteristic curve analysis, but it is not sufficiently validated for predicting the course of the disease among the patients with acute pancreatitis (25).

**Conclusions**

The severity of acute pancreatitis and development of infected necrosis are crucial in determining the prognosis and therapeutic approach. Hence, it is of particular importance to have early markers identifying patients prone to develop secondary pancreatic infection. A range of biochemical markers and several multifactorial scoring systems have been described to differentiate between the patients with severe and non-severe acute pancreatitis, but the value of these markers to recognize patients at risk for development of infected pancreatic necrosis is still undefined. To better target patients in whom antibiotic prophylaxis would be beneficial, we need to identify more accurate clinical and/or serological markers. This would contribute to an easier, less invasive, and more cost-efficient diagnosis.

Recent studies show that C-reactive protein is an important prognostic marker of pancreatic necrosis with the highest sensitivity and negative prognostic value in this respect. Based on the data from the above studies the most valuable markers for early prediction of infected pancreatic necrosis are procalcitonin and interleukin-6. Procalcitonin alone and especially in combination with interleukin-6 best identifies patients not at risk for infection.

Scoring systems in operation are not accurate enough to predict the development of infected necrosis. However, they are useful assessing severity of the disease. Review of the clinical studies suggests that we still do not have an optimal model, thus, there is a need for new more reliable clinical predictive systems.

Determination of severity of acute pancreatitis and prediction of infected pancreatic necrosis based on a few clinical parameters and single blood test would be of utmost value in triaging patients and initiating potentially critical therapy. However, it still remains a goal for future research.
Kasos nekrözės infekavimosi prognozavimas sergantiesiems ūminių nekröziniu pankreatitu

Žilvinas Dambrauskas1,2, Juozas Pundzius1, Giedrius Barauskas1
Kauno medicinos universiteto 1Chirurgijos klinikos, 2Biomedicininių tyrimų institutas
Raktažodžiai: ūminis nekrözinis pankreatitas, kasos nekrözės, progostiniai žymenys.


Šioje apžvalgoje analizuojami moksliniai tyrimai, kur buvo vertinti įvairūs progostiniai žymenys, galintys padėti nuspėti kasos nekrözės infekavimai ligoniams, sergantiesiems sunkiu ūminių pankreatitui. Ankstų įtarius kasos nekrözės infekavimai, būtų galima atliti kasos nekrözės punkciją (kontroliuojant ultragarsu ar KT) ir paimti medžiagos mikrobiologiniam paslėpti, taip pat būtų galima tiksliai atrinkti pacientus, kuriems profilaktinis antibiotikų skyrimas būtų tikrai tikslingas bei efektyvus. Tikslesnė pacientų, sergančių sunkiu ūminių pankreatitui, atranka padeda diegti racionalią antibiotikų terapiją ir mažinti antibiotikams atsparingų mikroorganizmų vystymąsi.

Naujųjų tyrimų duomenimis, C reaktyvusis baltymas yra svarbus ir paprastai nustatomas kaip ankstyvaisis kasos nekrözės žymuo, pasižymi dideliu jautrumu bei neigiamai prognostine rieškme. Serum prokalcitoninas ir jo derinis su interleukinu-6 pada ko tiksliausiai atrinkti pacientus, kuriuos kasos nekrözės infekavimai tikimybė yra minimalai. Tačiau, apibendrinant turimus duomenis, galima teigti, jog optimalaus modelio, kurį naudojant būtų galima prognozuoti kasos nekrözės infekavimai ligoniams, sergantiesiems sunkiu ūminių pankreatitu, kol kas nėra, todėl būtina tęsti naujų progostinių žymenų bei klinikinių požymių paiešką.

Adresas susirašinėti: G. Barauskas, KMU Chirurgijos klinikos, Eivenių 2, 50009 Kaunas
El. paštas: giedrius.barauskas@kmuk.lt; giedrius.barauskas@kmuk.lt

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