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# THE UNSOLVED QUESTION OF ACUTE PANCREATITIS. PROGNOSTIC CRITERIA: A CASE REPORT

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***ABSTRACT***

*Acute pancreatitis is a severe pathologic condition that requires an early identification of patients at risk of developing potentially lethal complications. To date, the use of clinical scoring systems and evaluation of biochemical parameters are, by far, the most widely used means to stratify the risk, even if they seem approximate and inadequate. The computed tomography severity index has proved to be superior in predicting acute pancreatitis outcome. Here we report the case of an adult male, admitted to our Department with the initial diagnosis of acute edematous pancreatitis, which was proved later to be a necrotizing pancreatitis, in which the clinical and laboratory prognostic scores were inadequate and discrepant with the more accurate computed tomography severity index.*

***Key words**: Acute pancreatitis, Ranson's criteria Glasgow score APACHE II score Balthazar's score.*

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## Introduction

Acute pancreatitis is an inflammatory process of pancreas characterized by sudden onset and is, to date, one of the most important gastrointestinal causes of hospitalization, with an annual worldwide incidence ranging between 13 and 45 cases per 100.000 people[1].

In Italy its annual incidence is estimated around 5-6 cases per 100.000 people, with an average age of onset ranging between 40 and 60 years[2].

Clinical presentation could be extremely various, from quite asymptomatic forms, characterized by a dyspepsia-like framework, to severe and life-threatening ones. The mild clinical variety, also called edematous or interstitial pancreatitis, is a self-limiting disease with absent or minimal organ dysfunction, and usually characterized by an uneventful recovery without complications. The severe form, also called necrotizing pancreatitis, occurs in about 20-30% of all patients with acute pancreatitis, and is characterized by protracted clinical course, high incidence of local complications and high mortality rate. Patients with pancreatic necrosis require to be closely monitored in intensive care unit, with a strict clinical and laboratory follow-up, associated with CT and MRI examination[3].

An early assessment of severity of acute pancreatitis is crucial to guide therapy and is based on the evaluation of objective parameters useful to predict clinical complications and to identify potentially lethal frameworks, which occur in 2-10% of patients suffering from acute pancreatitis. In the last decade it has been established that the increased frequency of death in acute pancreatitis is strictly related to the development and extension of pancreatic necrosis. For this, the detection of pancreatic necrosis (i.e. necrotizing pancreatitis) is used as a critical prognostic indicator for the first clinical assessment of these patients[4-6].

Prognostic assessment of acute pancreatitis severity rank is based both on clinical and laboratory evaluation (i.e. Ranson's, Glasgow, and Acute Physiology And Chronic Health Evaluation [APACHE] II scores) and on contrast-enhanced CT[7,8].

Unfortunately, the prognostic criteria based on clinical, laboratory and radiological evidences, are not always effective and consistent each other. The following case points-out the inadequacy and discrepancy of the existing parameters, conceived by scientific community and worldwide approved, confirming the superiority of radiological criteria on clinical-laboratory ones.

**Case report**

A 48-year-old male was admitted to our Department of Internal Medicine due to the onset, from approximately a month, of bloating and abdominal pain localized, mainly, to the superior quadrants, which radiated to the back, associated with nausea and anorexia, in absence of additional symptoms and/or signs of organ involvement. His medical history revealed, in the last six years, several access to Emergency Department (ED) because of "recurrent abdominal colics". For this, on 2010 he underwent colonoscopy (without biopsies) which detected 'diverticulosis of the sigmoid colon'. About 20 days before the admission to our Department, due to a fresh outbreak of the above mentioned symptoms, he went to the ED where was subjected to abdominal ultrasonography which proved negative (the investigation was hampered by meteorism and the pancreatic region was not shown); blood tests were not performed. Few days after, with the recommendation of his general practitioner, he underwent esophagogastroduodenoscopy, which showed "esophagitis grade A, according to Los Angeles classification, in a subject suffering from gastroesophageal reflux, sliding axial hiatal hernia and bulbar erosive duodenitis". Subsequently therapy with prokinetic drugs and proton pump inhibitors (PPI) was started, with little benefit. Nevertheless, due to the persistence of symptoms, he went back to the ED of our Hospital, where routine blood tests were performed, showing only neutrophilic leucocytosis, while amylase, lipase, hepatobiliary and renal function markers where all inside the range of normality; on the contrary, abdominal CT scan without contrast showed a 'volumetric expansion of head and isthmus of pancreas, with evidence of ectasic tubular formations (pancreatic ducts increased in volume); hyperdensity of peripancreatic adipose tissue; centimetric lymph nodes and fluid imbibition of anterior renal fascia" (Figure 1).

The patient was, then, admitted to our Department with the diagnosis of "acute edematous pancreatitis". Physical examination showed pain on deep palpation of epigastrium, mesogastrium and left iliac fossa. Laboratory tests showed neutrophilic leucocytosis (WBC 16.000 x mmc, reference values 4000-11000 x mmc), while amylase, lipase, hepatobiliary and renal function were still normal, as well as serum calcium and blood glucose. All these parameters persisted normal in several samples carried out throughout the hospitalization. The arterial blood gas analysis and the acid-base balance were normal too. The application of prognostic criteria (Tables 1-4) provided, on admission, the following results: Ranson's Criteria: 1% mortality rate; Glasgow score: mild acute pancreatitis; Apache II score: 4% approximate mortality risk; Balthazar's score: mortality and complications rates 3% and 8%, respectively. Based exclusively on clinical-laboratory scores, it was formulated the diagnosis of acute edematous pancreatitis with favorable prognosis. Fasting and PPI intravenous therapy were promptly started. On the second day of hospitalization an abdominal contrast-enhanced CT scan was performed, showing, unexpectedly, the framework of 'necrotic-hemorrhagic pancreatitis' (Figure 2).

| Admission | |
|---|---|
| Age older than 55 years | Lactate dehydrogenase > 350 IU/L |
| Serum white blood cell count > 16.000 mm3 | Aspartate aminotransferase > 250 IU/L |
| Serum glucose > 200 mg/dL | |
| Initial 48 hours | |
| Hematocrit decrease > 10% | Estimated fluid sequestration > 6 L |
| Blood urea nitrogen increase > 5 mg/dL | Serum Calcium < 8 mg/dL |
| Base deficit > 4 mEq/L | PaO2 < 60 mmHg |
| Ranson's prognostic scale (96% accuracy rate) | |
| < 3 signs = 1% mortality rate | 5 or 6 signs = 40% mortality rate |
| 3 or 4 signs = 15% mortality rate | > 6 signs = 100% mortality rate |

**Table 1:** Ranson's Criteria.

| PANCREAS acronym | |
|---|---|
| PaO2 < 60 mmHg | Age > 55 years |
| Neutrophils - White Cell Count > 15.000 x mm3 | Serum Calcium < 2 mmol/L (8 mg/dL) |
| Renal Function: Urea > 16 mmol/L (44 mg/dL) | Enzymes: AST/ALT > 200 IU/L<br>or LDH > 600 IU/L |
| Albumin < 3.2 g/L | Sugar: Glucose > 10 mmol/L (180 mg/dL) |
| a score ≥ 3 indicates acute severe pancreatitis | |
| a score < 3 indicates acute mild pancreatitis | |

**Table 2:** Glasgow score.

| | HIGH ABNORMAL RANGE | | | | | LOW ABNORMAL RANGE | | | |
|---|---|---|---|---|---|---|---|---|---|
| **PHYSIOLOGICAL VARIABLE** | **+4** | **+3** | **+2** | **+1** | **0** | **+1** | **+2** | **+3** | **+4** |
| TEMPERATURE - rectal (C°) | ≧ 41 | 39- 40.9 | | 38.5-38.9 | 36- 38.4 | 34-35.9 | 32- 33.9 | 30-31.9 | ≦ 29.9 |
| MEAN ARTERIAL PRESSURE (mmHg) | ≧ 160 | 130-159 | 110-129 | | 70-109 | | 50-69 | | ≦ 49 |
| HEART RATE (ventricular response) | ≧ 180 | 140-179 | 110-139 | | 70-109 | | 55-69 | 40-54 | ≦ 39 |
| RESPIRATORY RATE (non ventilated or ventilated) | ≧ 50 | 35-49 | | 25-34 | 12-24 | 10-11 | 6-9 | | ≦ 5 |
| OXYGENATION: A-aPO2 (if FiO2>50%) or PaO2 (if FiO2<50%) | ≧ 500 | 350-499 | 200-349 | | ≦ 200<br>≧ 70 | 61-70 | | 55-60 | ≦ 55 |
| ARTERIAL pH | ≧ 7.7 | 7.6-7.69 | | 7.5-7.59 | 7.33-7.49 | | 7.25-7.32 | 7.15-7.24 | ≦ 7.15 |
| SERUM HCO3- venous (mEq/L) | ≧ 52 | 41-51.9 | | 32-40.9 | 22-31.9 | | 18-21.9 | 15 - 17.9 | ≦ 15 |
| SERUM SODIUM (mEq/l) | ≧ 180 | 160-179 | 155-159 | 150-154 | 130-149 | | 120-129 | 111-119 | ≦ 110 |
| SERUM POTASSIUM (mEq/l) | ≧ 7 | 6-6.9 | | 5.5-5.9 | 3.5-5.4 | 3-3.4 | 2.5-2.9 | | ≦ 2.5 |
| SERUM CREATININE (mg/dl) Double point score for acute renal failure | ≧ 3.5 | 2-3.4 | 1.5-1.9 | | 0.6-1.4 | | ≦ 0.6 | | |
| HEMATOCRIT (%) | ≥ 60 | | 50-59.9 | 46-49.9 | 30-45.9 | | 20-29.9 | | ≤ 20 |
| WHITE BLOOD COUNT (total/mm3) | ≧ 40 | | 20-39.9 | 15-19.9 | 3-14.9 | | 1-2.9 | | ≦ 1 |
| GLASGOW COMA SCORE (GSC): score 15 minus actual GCS | | | | | | | | | |

| Apache II score: approximate mortality interpretation (%) | | |
|---|---|---|
| **Points*** | **Medical** | **Surgical** |
| 0-4 | 4 | 1 |
| 5-9 | 8 | 3 |
| 10-14 | 15 | 7 |
| 15-19 | 24 | 12 |
| 20-24 | 40 | 30 |
| 25-29 | 55 | 35 |
| 30-34 | 73 | 73 |
| 35-100 | 85 | 88 |


| Apache II score: approximate mortality interpretation (%) | | |
|---|---|---|
| **Points*** | **Medical** | **Surgical** |
| 0-4 | 4 | 1 |
| 5-9 | 8 | 3 |
| 10-14 | 15 | 7 |
| 15-19 | 24 | 12 |
| 20-24 | 40 | 30 |
| 25-29 | 55 | 35 |
| 30-34 | 73 | 73 |
| 35-100 | 85 | 88 |


** Sum of physiological variables, age and chronic health problems points.*

| Chronic Health Problems | |
|---|---|
| **Disease** | **Points** |
| Cirrhosis of the liver confirmed by biopsy | None: 0 points<br>Non-Surgical: 5 points<br>Emergent operation: 5 points<br>Elective operation: 2 points |
| New York Heart Association Class IV 3 or Severe COPD (Hypercapnia, home O2 use, or pulmonary hypertension) | |
| On regular dialysis | |
| Immunocompromised | |

*Chronic diseases includes biopsy proven cirrhosis and documented portal hypertension; past upper gastrointestinal bleeding attributed to portal hypertension; prior hepatic failure; prior hepatic encephalopathy; NYHA class IV; chronic restrictive, obstructive, or vascular lung disease resulting in severe exercise restriction; documented hypoxemia or hypercapnia; secondary polycythemia; severe pulmonary hypertension (>40 mmHg); ventilator dependence; chronic hemodialysis. Chronic diseases also includes immunosuppression from chemotherapy, radiation therapy, long-term or recent high-dose steroids, immunodeficiency (eg, leukemia, lymphoma, AIDS).*

**Table 3:** APACHE II score.

| Grade of acute pancreatitis | Points |
|---|---|
| Pancreas undamaged (grade A) | 0 |
| Altered pancreatic volume, other tissues unharmed (grade B) | 1 |
| Altered pancreas and peripancreatic adipose tissue (grade C) | 2 |
| Fluid peripancreatic collection (grade D) | 3 |
| Two or more fluid collections, or gas in the tissues around the pancreas (grade E) | 4 |
| **Degree of pancreatic necrosis** | **Points** |
| No necrosis | 0 |
| Necrosis of 30% of pancreas | 2 |
| Necrosis of 50% of pancreas | 4 |
| Necrosis of >50% of pancreas | 6 |

*CT severity index = points for grade of acute pancreatitis + points for degree of pancreatic necrosis.*

| CT severity index | Mortality | Complications |
|---|---|---|
| 0-1 | 0% | 0% |
| 2-3 | 3% | 8% |
| 4-6 | 6% | 35% |
| 7-10 | 17% | 92% |

*CT severity index = points for grade of acute pancreatitis + points for degree of pancreatic necrosis.*

**Table 4**: Balthazar's score (CT severity index).

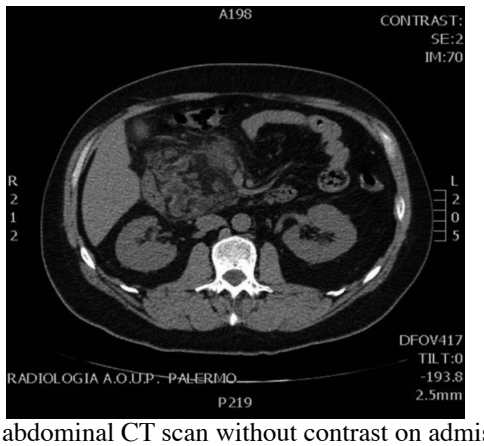


**Fig. 1**:abdominal CT scan without contrast on admission, showing a volumetric expansion of head and isthmus of pancreas associated with the presence of ectasic tubular formations (pancreatic ducts increased in volume), hyperdensity of peripancreatic adipose tissue, centimetric lymph nodes, and fluid imbibition of anterior renal fascia.

For this, a surgical consultation was required, which excluded any surgery urgency, and therapy was promptly changed with the insertion of nasogastric tube and the addition of intravenous octreotide, fluoroquinolones and carbapenems. On the second day of hospitalization, clinical-laboratory scores gave the same results, whereas Balthazar's score raised on mortality and complications rates to 6% and 35%, respectively. On the seventh day, the patient underwent to an abdominal contrast-enhanced MRI, which confirmed the diagnosis of acute pancreatitis of the head and highlighted 'presence of biliary sludge' (Figure 3), which was the reason to start therapy with ursodeoxycholic acid. In the same day, clinical-laboratory prognostic scores were still unmodified, whereas Balthazar's score fell down to mortality and complications rates of 3% and 8%, respectively. After twenty days of hospitalization, the patient was discharged in good clinical condition, completely asymptomatic, and it was recommended oral therapy with PPI + ursodeoxycholic acid.

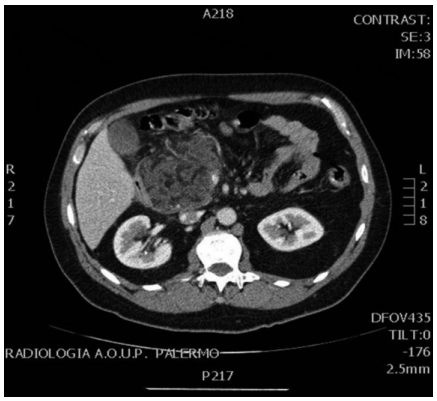


**Fig. 2**: abdominal contrast-enhanced CT scan after 48 hours of hospitalization, showing necrotic-hemorrhagic pancreatitis.

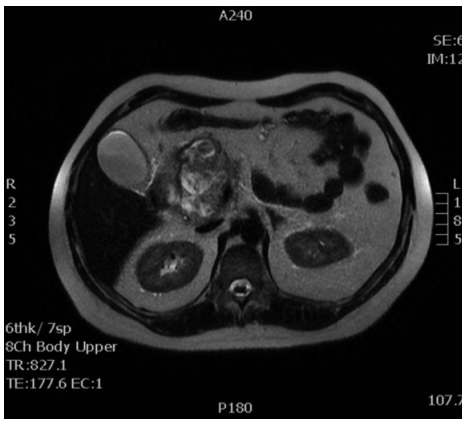


**Fig. 3**: abdominal contrast-enhanced MRI, on seventh day of hospitalization, confirming the diagnosis of acute pancreatitis (involving primary the head) and enlightening the presence of biliary sludge.

Subsequent clinical, laboratory and ultrasound follow-up after 1, 4 and 12 months showed no significant pancreatic alterations, in particular, absence of pseudocysts.

## Discussion

Several scoring systems, that combine clinical and laboratory parameters, have been formulated to identify patients with severe pancreatitis and stage the disease using the presence of specific abnormalities, called prognostic signs,. The first numeric system, proposed by Ranson et al. in 1974 (hereafter, Ranson's Criteria), is still the most widely used. It is based on 11 objective signs: five determined on admission, and six after 48 hours. A high number of risk factors correlates with increased morbidity and mortality rates. In patients with fewer than three signs, there is no mortality, while in those patients with six or more signs the mortality rate rises over 50% and usually the disease is characterized by extensive necrosis of pancreas. Unfortunately, there is a sensibility deficit between the real severity of disease (or the development of necrosis) and the group of patients with three to five severe signs. Moreover, a proper evaluation requires to complete the 11 measurements, in a time span of 48 hours of observation[9-11].

From this first approach, further systems have been formulated, such as the Glasgow criteria, focused on different parameters but, nevertheless, with a prognostic sensibility comparable to the Ranson's ones. However, the Glasgow criteria have not shown an adequate sensitivity and specificity in the initial staging of acute pancreatitis[12-16].

Currently, the APACHE II assessment and monitoring system is considered the more reliable, due to its complex structure based on the analysis of physiologic measurements, age of the patient, and co-presence of chronic health problems. Comparing the various systems each other, the APACHE II is more sensitive as early prognostic indicator, succeeding to guide the correct approach and, moreover, it can be used during the hospitalization to control the patient's response to therapy[17-19]. After 48 hours, the APACHE II score is comparable (or even better) with the Ranson's score in distinguishing mild from severe pancreatitis, with an elevated accuracy[7,20,21].

Balthazar's CT severity index is an instrumental scoring system that combines CT findings to provide an early prognostic evaluation of acute pancreatitis. Depending on CT frameworks ranging from grade A to E, respectively absence of lesions to presence of two or more fluid collections or gas in the tissues surrounding pancreas, is assigned a score from zero to four points. In addition, further points are assigned according to the presence and percentage of pancreatic necrosis, namely two points up to 30%, four points between 30% and 50%, and six points if higher than 50%. There is a strict statistic relationship between morbidity and mortality rates and the scores obtained according to the Balthazar's criteria. Patients with a CT severity index up to 1 showed negligible mortality and morbidity rates, while patients with a score of 2-3 showed a mortality rate equal to 3% but a 8% morbidity rate. In case of scores higher than 7, the mortality rate is around 17% with a 92% morbidity rate[22-24].

Unfortunately, the discrepancy between clinical-laboratory prognostic scores (i.e. Ranson's, Glasgow, and APACHE II) and Balthazar's CT severity index is often significant. Comparing the Ranson's criteria with Balthazar's ones, patients with a low CT grade (A or B) may show zero to five signs, while patients with an high CT grade (D or E) may have one to eight severe signs after 48 hours. Most of patients with more than five Ranson's prognostic signs have a high CT grade[25,26]. Differently, the APACHE II numeric system, despite its low sensitivity and specificity (56% and 72%, respectively) in distinguishing an interstitial pancreatitis from a mild necrotizing pancreatitis, showed a good capacity in identifying those patients with the need for an intensive care treatment and a strict monitoring[27]. Surprisingly, to date, a unique scoring system able to classify the severity of acute pancreatitis on admission combining together clinical, laboratory and instrumental findings has not been devised, and further clinical studies are required to identify such a system.

Our patient, although on admission showed clinical and laboratory prognostic factors (i.e. Ranson's, Glasgow and APACHE II) extremely favorable and compatible with the initial diagnosis of acute edematous pancreatitis, unfortunately was affected with a necrotic-hemorrhagic form, as evidenced by abdominal contrast-enhanced CT scan, notoriously with a worst and potentially unfavorable prognosis. This resulted in an immediate modification of the therapy previously practiced, with a both clinical and instrumental closer monitoring, adequate to the real severity of the disease.

Also notable how the prognostic criteria, excepted the Balthazar's one, remained favorable throughout the course of the hospitalization[4-6]. In other words, despite the severity of final diagnosis, the clinical and laboratory prognostic indices did not changed during the entire hospitalization, as if our patient was suffering from a mild form of acute pancreatitis, not requiring, therefore, a closer monitoring. Better was the performance of the Balthazar's criteria, as previously stated in the international literature[8,28], with the confirmation of the finding reported by the CT scan with the subsequent MRI[29].

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