# CT modern potential in acute pancreatitis visualization (part 2)

A. A. Filatau, A. A. Litvin

*Gomel Regional Clinical Hospital,*

*Gomel State Medical University, Gomel, Belarus*

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In the previous issue of the magazine we presented the first message on the CT Imaging of acute pancreatitis (AP), whose main purpose was a review of the literature on modern capabilities of computed tomography (CT) in the diagnosis and treatment of AP. We have summed up what is now the main role in diagnosis, stratification AP and differentiation in its various forms is given to CT-visualization with intravenous bolus contrast. This is due to the rather high diagnostic accuracy, availability of the method, the ability to perform in patients, fastest time scanning and a relatively simple interpretation of the obtained images of the treating physicians and radiologists working. The purpose of this communication (part 2) is a review of the current classification of CT-definitions of the various forms of AP.

The world is currently the most widely used two classification of AP: Revision of the Atlanta Classification [9] and Determinant-based Classification [16]. These classifications fundamentally differ from the number of degrees of severity AP: three and four (Table 1, 2, 3) [15] and, consequently, the main determinants.

Table 1

**Categories of AP severity according to the Revision of the Atlanta Classification**

| AP severity | Organ failure / local or systemic complications |
|---|---|
| Mild AP | - no multiple organ failure<br>- no local or systemic complications |
| Moderate AP | - intermittent (transient) organ failure (up to 48 hours)<br>- local or systemic complications without persistent organ failure |
| Severe AP | - presence of persistent organ failure (single or multiple lesions of organs and systems) |

Table 2

**Categories of AP severity according to the Determinant-based Classification**

| AP severity | Organ failure / necrosis |
|---|---|

| | |
|---|---|
| Mild AP | - no necrosis<br>- no organ failure |
| Moderate AP | - sterile necrosis<br>- and/or passing organ failure |
| Severe AP | - infected necrosis<br>- or persistent organ failure |
| Critical AP | - infected necrosis<br>- and persistent organ failure |

Table 3

**Differences between the Revision of the Atlanta Classification and Determinant-based Classification of AP**

| **Differences** | **Revision of the Atlanta Classification** | **Determinant-based Classification** |
|---|---|---|
| Number of categories | 3 | 4 |
| Form of pancreatic necrosis (sterile or infected) | not classified | classified |
| Acute fluid accumulations or cysts | classified | not classified |
| Detection of organ failure | ≥2 points according to the modified Marshall scale | >2 points according to SOFA scale |

Difference of classifications is also greater attention in the Atlanta classification on the audited morphological changes in tissues of the pancreas based on CT data; this AP has a larger CT-visualized direction compared to Determinant-based classification.

So, in the Revision of the Atlanta Classification of AP (Miami, 2012), the main two kinds of pathological process if AP is defined as edematous interstitial pancreatitis (EIP) and acute necrotizing pancreatitis (ANP). According to this classification, UNP in turn subdivided into necrosis actually pancreas parenchyma is pancreatic necrosis, necrosis, peripancreatic as well as their combination is necrosis of pancreatic parenchyma with peripancreatic necrosis [3]. The listed forms of disease can be both sterile and infected (Table 4). The division is also in the terminology of the time passed from the onset of the disease: more or less 4 weeks.

Table 4

**Terminology of changes in the pancreas and peripancreatic tissue upon AP according to the Revision of the Atlanta Classification**

| Type of pancreatitis | Type of liquid accumulation |
|---|---|
| *<4 weeks from disease onset* | |

| | |
|---|---|
| AIP | Acute peripancreatic liquid accumulation<br>— sterile<br>— infected |
| ANP | Pancreatic necrosis<br>— sterile<br>— infected |
| | Peripancreatic necrosis<br>— sterile<br>— infected |
| | Pancreatic necrosis combined with peripancreatic necrosis<br>— sterile<br>— infected |
| *≥4 weeks from disease onset* | |
| AIP | Pancreatic pseudocyst<br>— sterile<br>— infected |
| ANP | Localized necrosis<br>— sterile<br>— infected |

## Edematous interstitial pancreatitis

When conducting contrast CT in patients with EIP detected locally or diffuse increase pancreas with normal homogeneous or heterogeneous slightly increased pancreatic parenchyma associated with swelling (Fig. 1) [4]. Peripancreatic and retroperitoneal fiber may look unchanged, that often occurs in the early stages of the disease and in severe pancreatitis. Sometimes can be detected by slight inflammatory changes in the surrounding soft tissues, which manifest as "nebula" or light "pectoral" adipose tissue with a different number of peripancreatic liquid [33]. In contrast, in the first few days of the start of AP, sometimes noted increased dying of pancreatic parenchyma, heterogeneous, that cannot be attributed to either the EIP neither ANP. However, with the passage of time occurs when component fluid reabsorption EIP or melting nonviable tissue at ANP, that allows to differentiate data disease with repeated CT-study through 5-7 days.

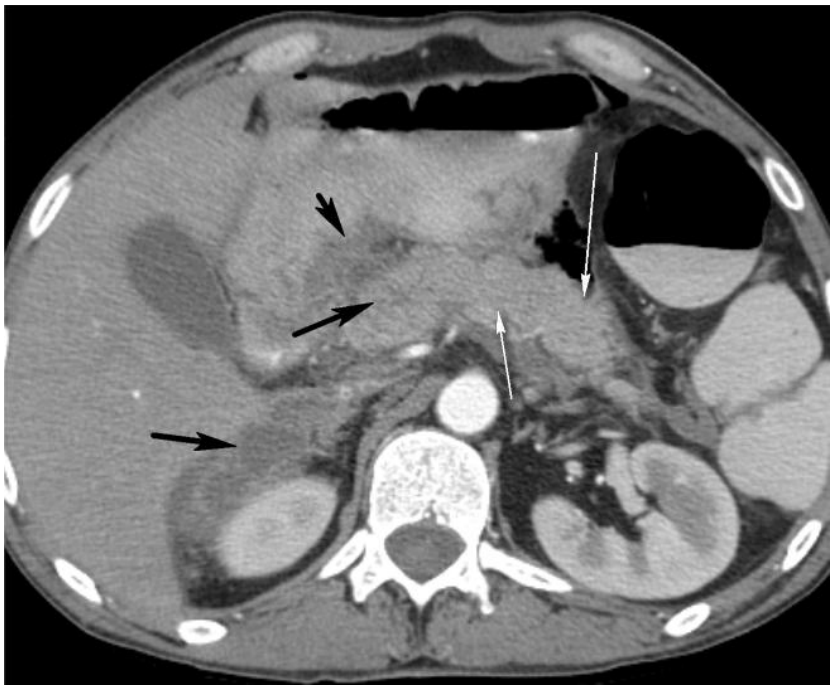

Fig. 1. CT of patient L., 45 years old, with alcoholic pancreatic necrosis. 6-th day from the onset of illness. Thin white arrows indicate an enlarged pancreas in areas of the body and tail, black — to emerging sharp peripancreatic liquid accumulation and diffuse swelling in the area of the pancreatic head.

## Necrotizing pancreatitis (pancreatic necrosis)

Pancreatic necrosis develops as a result of expressed pancreatic microcirculation. Visually this pathological process is defined as diffuse or focal sites unviable parenchyma, often combined with necrosis peripancreatic fiber. For the development of genuine pancreatic necrosis takes approximately 24-48 hours of onset, but well visible on CT, it becomes only after 48-72 hours from the onset of symptoms [6, 43]. It is for this reason that the sensitivity of CT SCAN with contrast in the diagnosis of pancreatic necrosis in the first 72 hours is only 60-70% [41].

## Necrosis of pancreatic parenchyma

Isolated necrosis of pancreatic parenchyma (without peripancreatic necrosis) is less than 5% of patients and is contrasting UNP CT images as lack of dying tissue glands in certain areas. Most often this process affects the body and the tail of the pancreas. On 1st week when conducting disease contrast CT necrotizing pancreatitis manifests itself as homogenous is not enhanced contrast area (Fig. 2), and then, during the development of pathological process, as more heterogeneous area [44]. Detected on CT changes is the result of a process in which the necrotic tissue of the pancreas gradually melted.

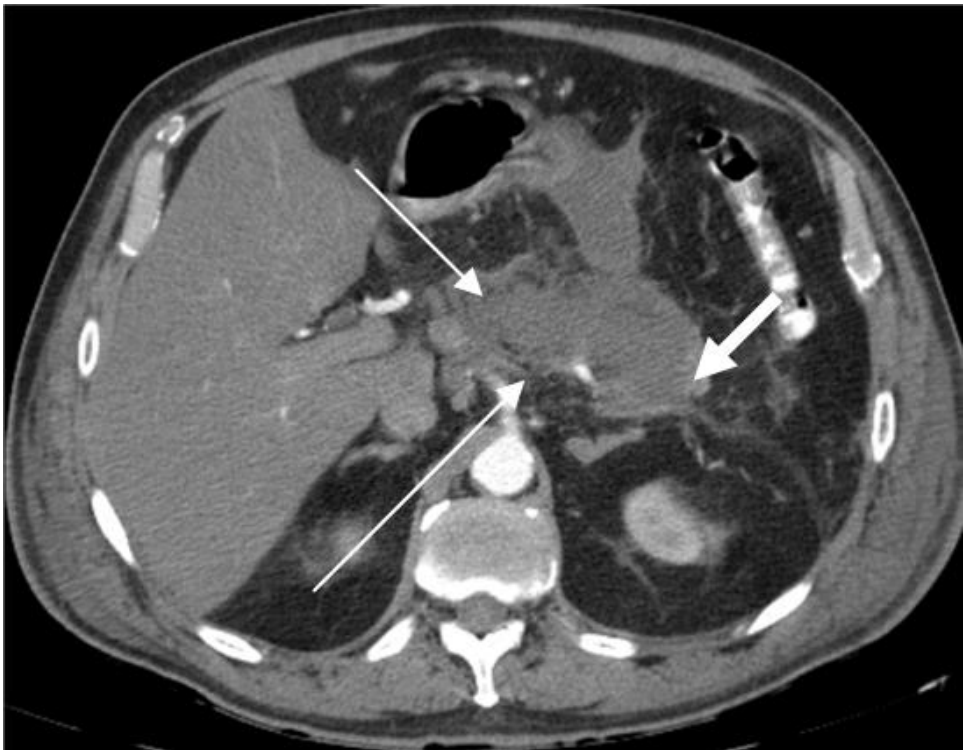

Fig. 2. CT of patient V., 61 year old, with alcoholic pancreatic necrosis. 12-th day from the onset of illness. Thin arrows indicate reduced zone and dying consumer heterogeneity, thick arrow marked plot saved tissue of the tail of the pancreas.

## Peripancreatic necrosis

Isolated peripancreatic necrosis is observed in approximately 20% of patients with UNP [44]. His presence is diagnosed in the presence of heterogeneous non-painted areas in peripancreatic tissue, which contain non-infected components (Fig. 3) [40]. Peripancreatic necrosis is usually located in the retroperitoneal space and stuffing your bag. The clinical significance of the form AP is that such patients have a better prognosis than when isolated pancreatic necrosis [32], but a greater number of complications than when EIP [35].

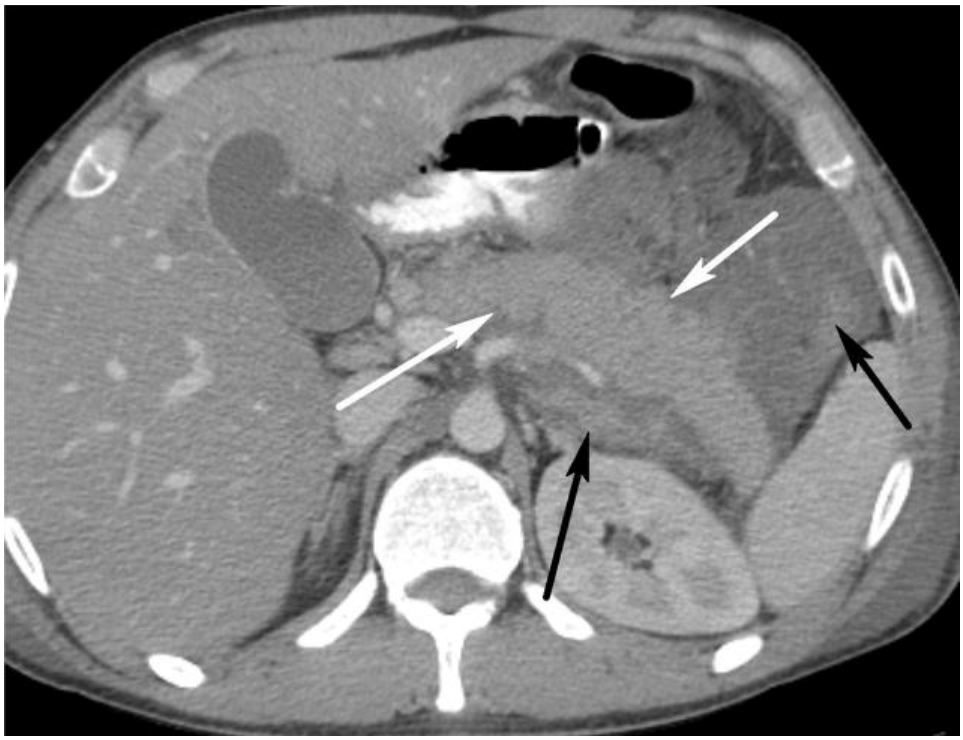

Fig. 3. CT of patient E., 24 years old, with alcoholic peripancreatic necrosis on the 5th day from the onset of the disease. Growing and swelling of the pancreas (white arrows). Around the body and tail with the transition to perinephric fiber area mentions diverse dying-peripancreatic necrosis (black arrows).

### Necrosis of pancreatic parenchyma with peripancreatic necrosis

A combination of necrosis of pancreatic parenchyma with peripancreatic necrosis is the most common type of the disease, and it can be observed in 75-80% of patients with UNP [44]. Radiological manifestations of this form of the disease are the combinations of the changes described previously for isolated necrosis of pancreatic parenchyma and actually peripancreatic necrosis (Fig. 4). It is believed that peripancreatic necrosis associated with extensive necrosis of pancreatic parenchyma, is most often associated with damage to the walls of the main pancreatic duct [19].

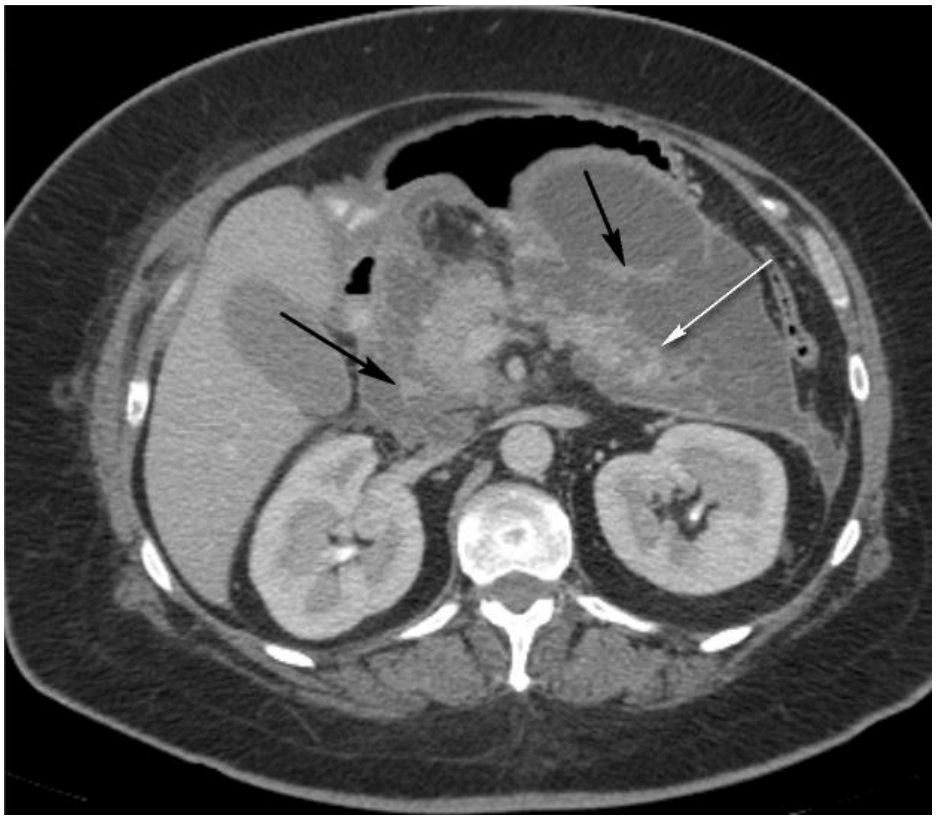

Fig. 4. CT of patient R., 48 years old, with idiopathic pancreatic necrosis. Notes the necrosis of the tail of the pancreas (white arrow). Around the head and body anterior to the pancreas marked zone of peripancreatic necrosis (black arrows).

## Pancreatic and peripancreatic accumulations

The AP may be accompanied by the formation of pancreatic (parenchymatous) or peripancreatic. In the Revision of the Atlanta classification (2012), there is a significant distinction between liquid and postnecrotic crowd, which was not in the original classification (Atlanta) 1992 [39]. Accordingly, new approaches all sharp accumulations are defined as either acute peripancreatic liquid accumulation (APLA) as either acute necrotic accumulation (ANA), depending on the absence or presence of necrosis in this cluster, respectively. IPRS could lead to the formation of APLA and, over time, to the formation of pancreatic pseudocysts. Necrotizing pancreatitis in its three forms can cause formation of ANA, and during the course of the disease is localized necrosis. Again it should be noted that all these clusters can be sterile and infected.

## Acute peripancreatic liquid accumulation (APLA)

Peripancreatic fluid accumulations occur in patients with EIP resulting from inflammation or damage one (several) of the small peripheral branches of the

pancreatic duct. APLA are formed within 48 hours at 30-50% of patients with PD [27]. These entities are usually in anatomical borders of retroperitoneal space and most often in the vicinity of the pancreas (Fig. 5), and they have no visible walls. Most often, the fluid accumulation are formed in stuffing the bag, but there are other localization: front pararenal space (usually the left), mesentery of the transverse colon, small intestine mesentery root, as well as gastro-hepatic, gastro-splenic and transverse ligaments [5, 27]. About 50% APLA dissipated spontaneously in the first several weeks, therefore, surgical interventions at this stage, this category of patients should be avoided, as drainage or suctioning could cause infection [44].

It is considered important that the liquid cluster, located in the parenchyma of pancreas (distorting or replacement part) should be regarded as necrosis (more precisely, as ANA), rather than as APLA [44]. On 1-st week AP distinguish between APLA and ANA can be difficult or even impossible, because both types of clusters appear as field without contrast dying when conducting a CT study. However, if there are non-coloured areas with variables sections weakening contrast in these clusters, assume the diagnosis peripancreatic necrosis [44]. When diagnosed with peripancreatic you cannot diagnose necrosis, but clearly detectable light heterogeneity of content peripancreatic clusters, It is recommended to conduct a CT in dynamics, in which (usually after 7-10 days of onset) are becoming clear and heterogeneity could more confidently establish a correct diagnosis.

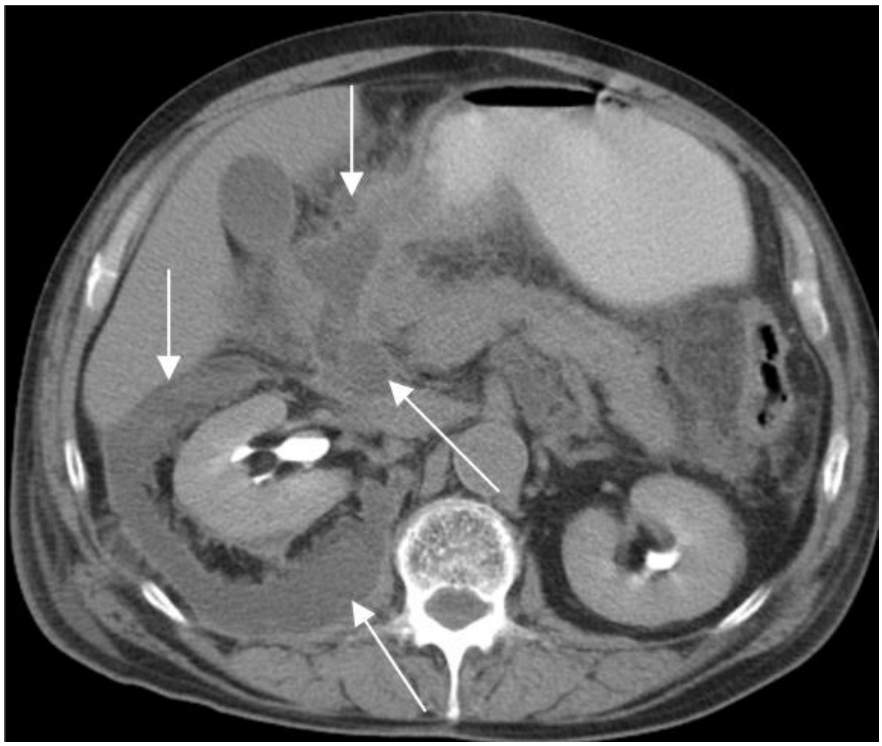

Fig. 5. Patient F., 70 years, 7 days from the onset of the disease. Notes a slight swelling of the pancreas with the formation fluid accumulations in the area of perirenal tissue and head right (White arrows).

## Pseudocyst

Within 4 weeks from the start of the EIP APLA can gradually move in pseudocyst. Pseudocyst occurs as a complication of AP about 10-20% of cases [29]. The contrast CT images of the pseudocyst misdiagnosed as well defined, usually round or oval peripancreatic liquid accumulation of homogeneous low-level dimming (about 20 HU) [24]. These liquid accumulation typically are surrounded by well defined non-coloured thin capsule up to 1-3 mm (the latter is composed of fibrous or granulation tissue) and does not contain necrotic components (Fig. 6) [8, 30]. Generally well-coloured wall fails to emerge before 4 weeks and liquid cluster with non-contrasyed wall should be classified as APLA.

It should be noted that in 58% of cases-25 pseudocyst contains liquid with a high activity of amylase and lipase through messages with pancreatic duct system [5]. In this case, 50% of patients of the pseudocyst do not cause any symptoms and often such a message with channels spontaneously aborted, with the passage of time, and liquid accumulation disappear [5, 26]. Determination of the presence or absence of a functioning communication with the main flow of the pancreas is quite important, as

it affects the further tactics of treatment. And, generally, in addressing this challenge, the most informative is cholangiopancreatography [14, 25]. The second half of patients the availability of the pseudocyst is manifest abdominal pain, secondary infection, bleeding from the arrosive receptacle, systemic inflammatory response syndrome on the background of the breakthrough of cysts in the abdomen, as well as the development of phenomena of biliary obstruction or dyspepsia on a background of compression of the duodenum [42].

Pancreatic pseudocyst are infected and sterile. Infected (suppurated) cyst is a term coined by the Revision of the Atlanta Classification AP instead of previously held notions of pancreatic abscess [3]. Infected Cyst on CT scan is determined by how well defined encapsulated education near the pancreas, while the wall of the capsule is thicker and inhomogeneous, than a sterile form of disease [12]. In 20% of cases within infected pseudocysts can be detected by gas bubbles or fluid level with gas over it [7].

However, it is known that in 30% of cases of pathological education parapancreatic zone, initially considered as a pancreatic pseudocyst are her cystous tumors [49]. It is believed that a successful differential diagnosis between these diseases should pay great attention to anamnesis the life of the patient and detailed study of CT/MRI data. If the patient has not been characteristic bouts of op for CT jumper determines whether rendering in lumen liquid accumulation or nodulous building walls, there are changes in regional lymph nodes or painting walls strengthened capsules — assume the presence of cystic neoplasm [5, 49].

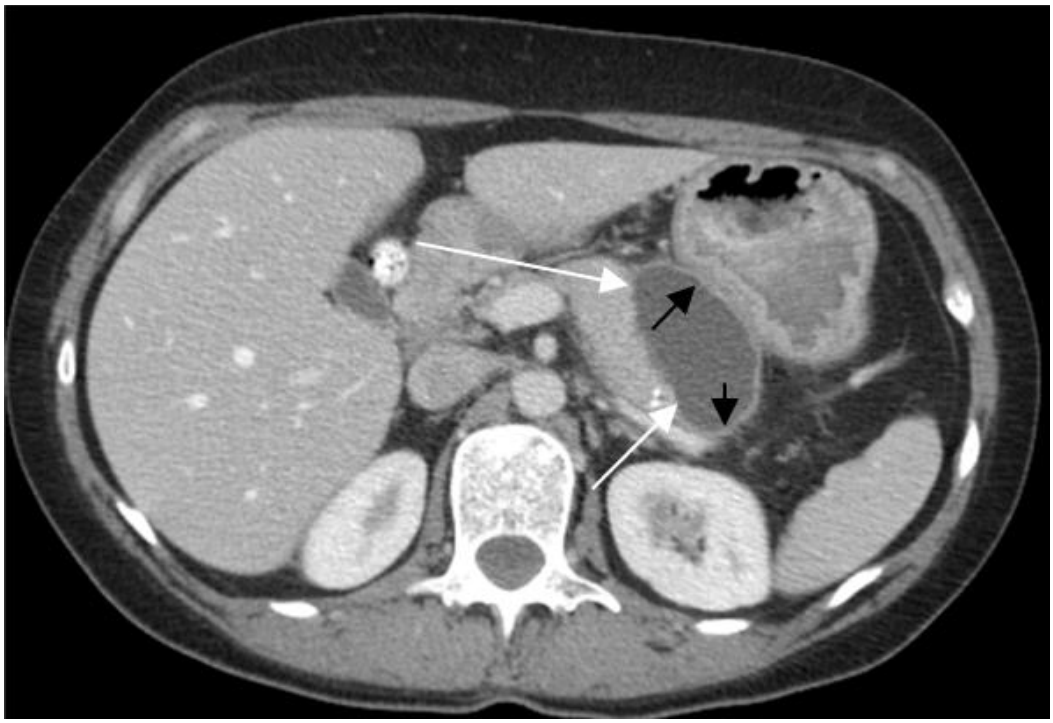

Fig. 6. Patient E., 40 years old, with chronic pancreatitis, watch CT-study without exacerbation. You can see liquid cluster in the area of the tail of the pancreas with clearly defined walls (black arrows) is a pseudocyst (white arrows).

## Acute necrotic accumulation (ANA)

In the first 4 weeks of onset clusters formed pancreatic necrosis, which contains both liquid and necrotic tissue (necrotised adipose tissue, sequesters, blood clots) in various ratios should be detected as ANA. In ONS melting necrotic tissues occurs gradually (typically within 2-6 weeks), as is their destruction [21]. It is for this reason that due to insufficient time to organize the most important fragments of the dead tissues with the formation of the corresponding heterogeneity when rendering within 1 week of APLA differentiation and ANA is extremely difficult. Both of these clusters can manifest in the form of homogeneous non-coloured areas on the CT-images [1]. As a rule, difference between the pathological accumulations of data becomes possible after 1 week of illness, because clusters containing necrotic debris, becoming more diverse (Fig. 7).

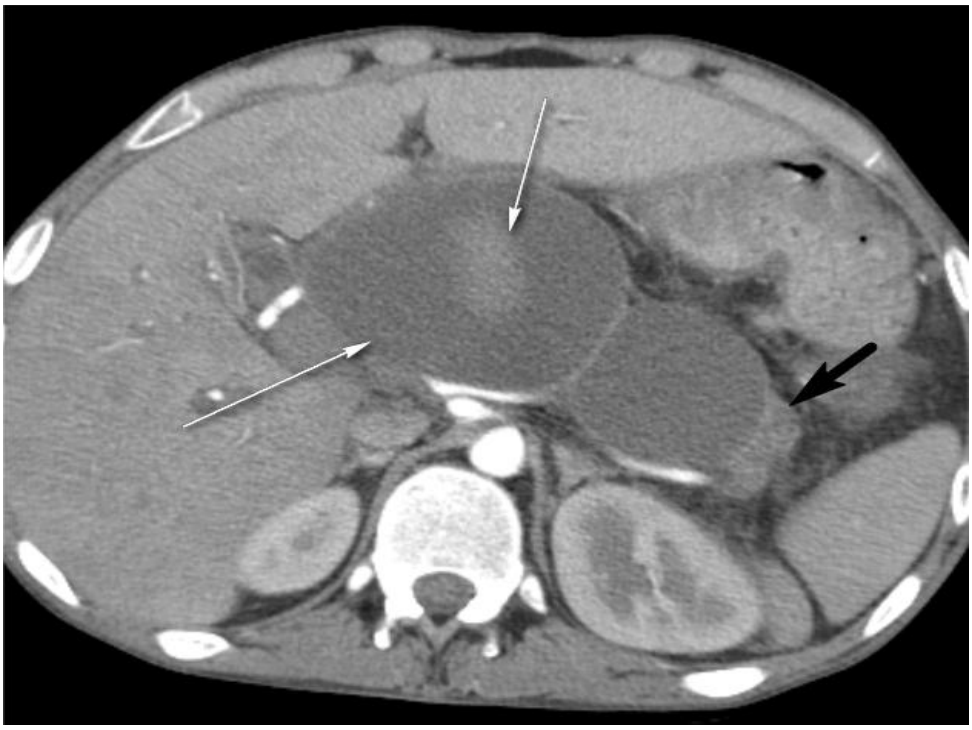

Fig. 7. Patient K., 24 years old, with acute alcoholic pancreatitis, 7th day from the onset of the disease. Defines two pancreatogenic in the area of the body and tail of the pancreas with mixed content in one (White arrows). Black arrow marked plot saved parenchyma pancreas tail (black arrow).

## Localized necrosis

As well as APLA of the pseudocyst is formed over time (usually at 4 week and later) ANA "matures" in the softness of the necrosis (walled of necrosis). This term has replaced such concepts as pancreatic pseudocyst sequestration with necrosis, necroma or organized pancreatic necrosis, which are manifestations of late stage development of ANA [9, 13]. It is believed that the softness of necrosis develops in 1-9% of the cases of complicated cases, up to 90% of such entities is localized in the body or tail pancreas [48]. As ONS softness necrosis can involve itself and pancreatic parenchyma and peripancreatic fabrics (Fig. 8), or only peripancreatic fabrics, or pancreas. Characteristic features for localized necrosis is considered to be: large size clusters, spreading it in paracolic or retrocolic space, uneven capsule education, presence of tissue components, deformation or violation of the integrity of the pancreas, as well as a lack of pancreatic hypertension (pancreatic duct diameter up to 4 mm) [13]. With the above changes are detected in approximately 80% of the cases the differential diagnosis of localized necrosis, and pseudocysts.

Prior to the introduction of the modern terminology is often viewed as necrosis softness pseudocyst with necrosis, resulting in erroneous selection tactics of patients with this pathology and, consequently, unsatisfactory results of treatment. As opposed to pseudocyst, which in most cases can be treated effectively puncture softness necrosis contains necrotic tissues that could not be evacuated from the area of interest is simple percutaneous venting [38].

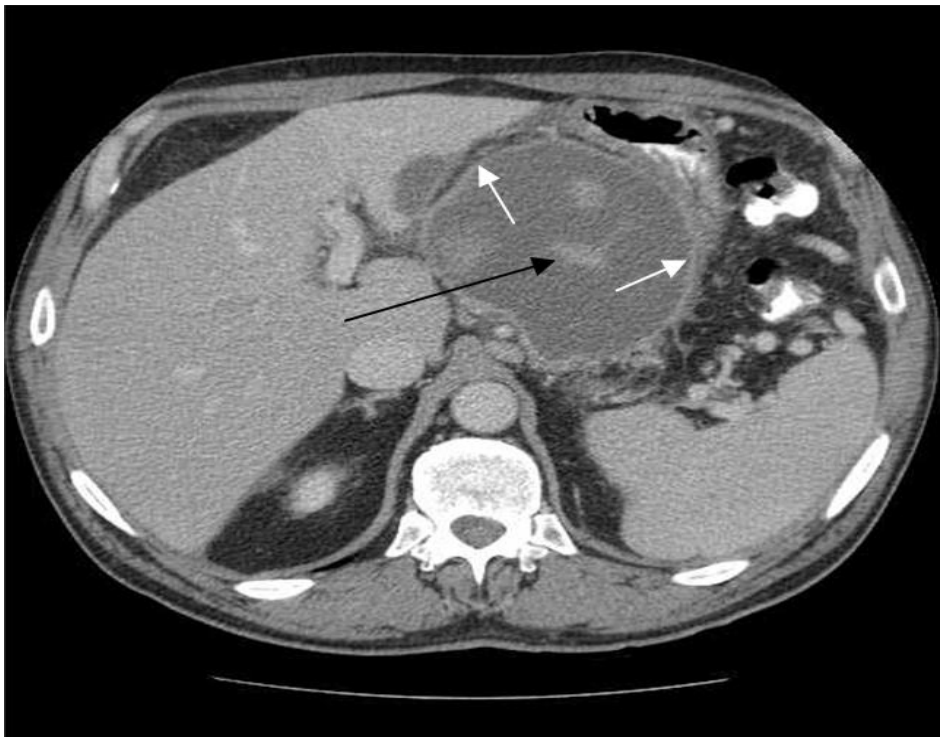

Fig. 8. Patient Sh., 58 years old, rendered softness of necrosis (black arrow) with fairly well formed walls (White arrows). 28th day from the onset of the disease.

Thus, CT-AP visualization is very important for the differentiation of the various forms of the disease, the treatment of which (conservative, mini-invasive or surgical) on various dates of onset differs dramatically. Classification of AP audited Atlanta introduces new classification definitions clarifying which can directly influence the tactics of treatment. In Table 5, all classification definitions are systematized AP indicating the preferred therapeutic tactics in their diagnosis.

Table 5

**AP morphological forms according to the Revision of the Atlanta Classification and possible treatment options**

| Type of accumulation | Time from AP | Necrosis | Localization | Manifestation | Infection | Drainage or surgery |
|---|---|---|---|---|---|---|

| | | | | | | |
|---|---|---|---|---|---|---|
| | onset | | | | | |
| **Interstitial edematous pancreatitis** | | | | | | |
| ОПЖС | ≤4 weeks | No | Adjacent to the pancreas, only extrapancreatic | Homogeneous, liquid weakening of stain, absence of liquification (sequestrum), non-encapsulated | Very rarely | Not required |
| Pseudocyst* | >4 weeks | No | Adjacent to or in the distance from the pancreas | Homogeneous, liquid weakening of stain, absence of liquification (sequestrum), encapsulated | Rarely | Rarely (upon infection or symptoms) |
| **Necrotizing pancreatitis** | | | | | | |
| Sterile ANA | ≤4 weeks | Yes | In parenchyma and/or extrapancreatically | Heterogeneous**, contains non-liquified material, of diverse form, non-encapsulated | No | Основано на клинических проявлениях, чаще чрескожное дренирование, хирургическая операция редко*** |
| Infected ANA | ≤4 weeks | Yes | In parenchyma and/or extrapancreatically | Heterogeneous**, contains non-liquified material, of diverse form, non-encapsulated | Yes | Чрескожное дренирование, хирургическая операция позже, если требуется*** |
| Sterile localized necrosis | >4 weeks | Yes | In parenchyma and/or extrapancreatically | Heterogeneous**, contains non-liquified material, of diverse form, encapsulated | No | Чрескожное дренирование основываясь на клинике, последующая хирургическая операция, если требуется*** |
| Infected localized necrosis | >4 weeks | Yes | In parenchyma and/or extrapancreatically | Heterogeneous**, contains non-liquified material, of diverse form, encapsulated | Yes | Чрескожное дренирование, хирургическая операция, если требуется*** |

* — rarely upon necrotizing pancreatitis after resection or in syndrome of disabled duct
** — rather homogeneous in the first stages
*** — or endoscopic manipulations

## AP complications

All four types of pancreatic accumulations can be sterile and infected. From the beginning of the AP to the onset of symptoms of infection most often takes place at least 2-3 weeks [34, 45]. Distinguish between sterile and infected enough accumulation is important, because treatment and prognosis have different [31]. Now it is proved that the CPA and the softness of the necrosis infected more often than

APLA or pseudocyst, referring to the presence of non-viable tissues. The fact that bacterial contamination may be suspected on contrasting CT images by the presence of gas bubbles in the crowds due to gas-forming microorganisms (Fig. 9) [36]. However, this symptom occurs in only 12-18% of cases [46]. If there is no gas in the pathological accumulation, final confirmation of the infection can be obtained by executing a fine-needle aspiration biopsy (FNAP) with the subsequent bacterioscopy and bacteriological research [32]. Due to the risk of infection of pancreatic FNAP accumulations it is recommended that you run only in cases where there is a clear clinical hypothesis of purulent-septic complications. During aspiration biopsy is not recommended gastric or duodenal access to avoid possible additional ways of infection foci of necrosis [28]. It is for this reason that retroperitoneal way rather than access through the ventrolateral wall of the abdomen. It is believed that with the aim of fluid aspiration diagnosis of infectious complications in AP 10% less false negative results [2]. Therefore, if the result is negative and clinical FNAP suspected infection process is saved, you should conduct the study.

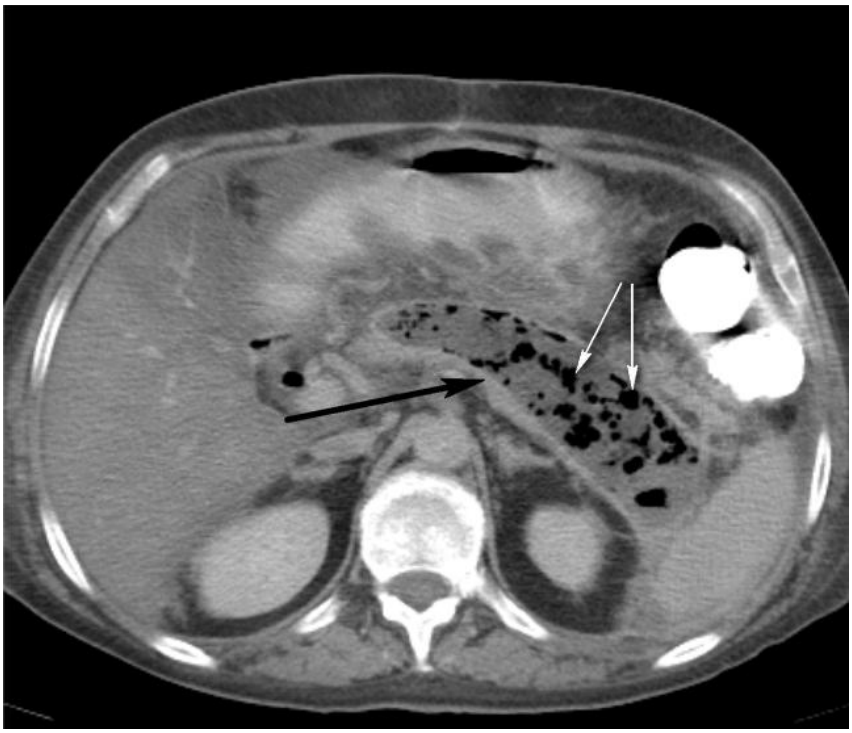

Fig. 9. Patient Ts., 51 year old, determined by the softness of the necrosis (black arrow) with indirect signs of infection — gas bubbles (white arrows).

As complicated currents necrotizing pancreatitis is considered frequent lately (thanks to improvement of CT/MRI visualization) "syndrome interrupted pancreatic duct (disconnected pancreatic duct syndrome). The essence of this syndrome is in violation of the integrity of Wirsung's duct and prolonged leakage of pancreatic juice in peripancreatic tissue that causes the formation of large aggregations of pancreatogenic [18]. In General, symptoms of pancreatic duct interrupted develops in 30% of all cases of ANP and most often is formed as a result of cervical lesions of the pancreas as the most sensitive plot parenchyma to disturbed perfusion of tissues [17]. CT-criteria are: plot of necrosis in the neck or body pancreas not less than 2 cm distal to the parenchyma of a viable plot of necrosis and pancreatic juice leaks confirmation with pancreatography (Fig. 10) [20].

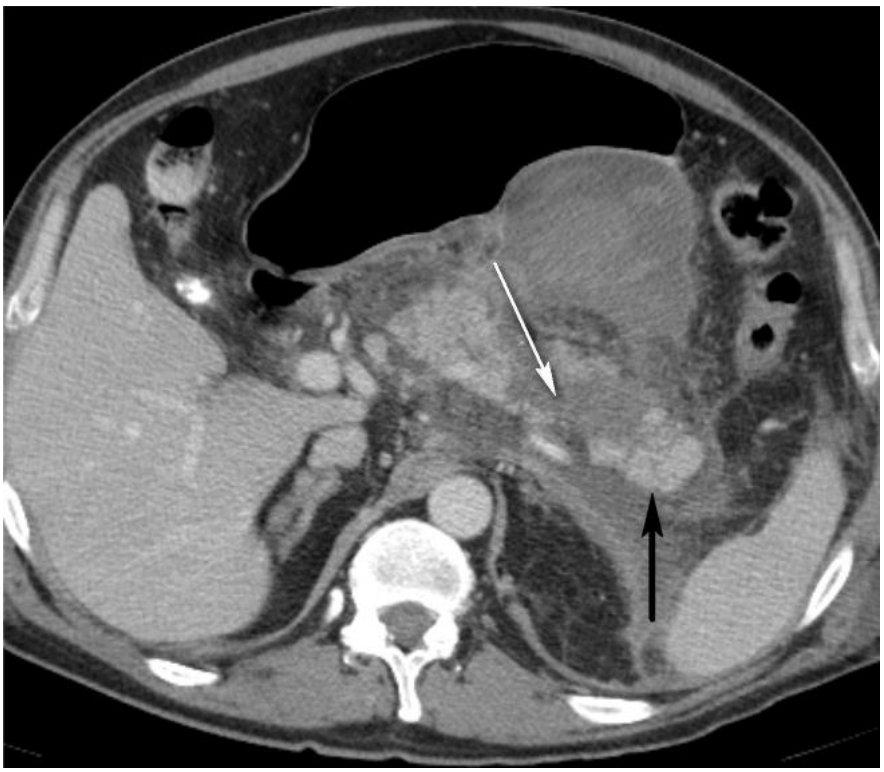

Fig. 10. Patient L., 56 years old, through 3 days of onset. Identifies major acute necrotic parapancreatic in tissue and body of the pancreas (black arrow) with mixed content. Saved plot tail pancreas (black arrow) is separated from the area of necrosis (most likely occurs syndrome of interrupted pancreatic duct).

Improved CT visualization among complications was more likely to meet the associated AP vascular disease pancreatoduodenal zone. Currently, vascular complications are detected at 25% of patients in this category [23, 47]. The most common complication of thrombosis of the splenic vein is AP (from 10 to 40%), although there are variants of thrombosis of superior mesenteric or portal veins venous thrombosis develops as a result of inflammatory vascular intimal injury or compression of the pancreatic crowd. As a result of venous outflow can later develop segmental portal hypertension, varicose veins, heart spleen [37, 38]. it is well identified with contrasting abdominal CT also arterial complications AP : damage of arterial vessels as a result of their arrosion with the development of pseudoaneurism, subsequent bleeding. The most commonly affected splenic artery (40%), gastroduodenal artery (30%) and pancreatoduodenal artery (20%) to [10, 22]. Mortality in this disease even if the diagnosis and appropriate treatment is 12%, and in their absence reaches 90% [11].

Thus, CT-AP visualization is very important for better differentiation of different variants of the disease and the selection of optimal treatment tactics. the Revision of the Atlanta Classification (2012) has a distinct focus and visualization's use of the new uniform terminology allows you to standardize approaches to diagnostics, simplify and standardize the professional communication among stakeholders, and thereby improve the results of treatment.

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# CT modern potential in acute pancreatitis visualization (part 2)

A. A. Filatau, A. A. Litvin

*Gomel Regional Clinical Hospital,*

*Gomel State Medical University, Gomel, Belarus*

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Currently, the main role in the diagnostics and stratification of acute pancreatitis is given to the CT imaging. The aim of this message (part 2) is to review the classification of modern CT-definitions of various forms of acute pancreatitis according to the Revision of the Atlanta Classification (Miami, 2012). CT imaging of acute pancreatitis is very important for a better differentiation of the various options of the disease and the choice of optimal treatment strategy. The Revision of the Atlanta Classification (2012) has a clear adherence to the CT-visualization; its use will unify approaches to the diagnostics of acute pancreatitis, standardize professional communication among interested specialists, and improve treatment outcomes.