Relationship of Arterial Changes in Acute Pancreatitis on CT Angiography with Modified CT Severity Index

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J Gastrointestinal Abdominal Radiol ISGAR 2022;5:37–42.

Abstract

Purpose To evaluate the prevalence of arterial changes in patients with acute pancreatitis (AP) on computed tomography angiography (CTA) and determine their association with etiology of AP, presence of necrosis, collections and severity of AP.

Materials and Methods A total of 50 patients (20 women, 30 men; mean age: 43.04 ± 13.98; age range: 18–77 years) with AP underwent contrast-enhanced computed tomography (CECT) scan and CTA of abdomen, which was evaluated for necrosis and fluid collection (s). On CTA, splanchnic arterial structures were assessed for vascular complications. Association between vascular changes and presence of necrosis, fluid collections, etiology of AP and severity of AP (as assessed by modified computed tomography severity index CTSI) was determined.

Results Arterial complications were seen in 28 percent (14/50). The most frequently involved artery was superior pancreaticoduodenal artery (12 percent), followed by splenic artery (8 percent) and right gastric artery (8 percent; ➤Fig. 1). No significant association was seen between arterial changes and gallstone or alcohol-induced AP. Arterial changes showed a significant association with presence of acute necrotizing pancreatitis (ANP), presence of collections and severe AP (CTSI 8–10) (p < 0.05 for each).

Conclusion Arterial changes on CTA are frequently seen in patients of AP having ANP. There is a significant association between arterial changes and presence of necrosis, collections and severe AP.

Keywords
► thrombosis
► Pancreatitis
► complications
► necrosis
► collection

Introduction

Acute pancreatitis (AP) is an acute pancreatic inflammatory disease with varying involvement of both the local tissue and/or distant organs. It is a significant medical and surgical problem, being the most common gastrointestinal reason for hospitalization.1 Mild AP has a very low (less than 1%) mortality rate.2,3 Based on the presence of sterile or infected necrosis, mortality in patients with more severe AP can range from 10 to 30 percent.4 There are multiple causes of AP. Cholelithiasis
(35–40%) is the most common cause, followed by alcohol (30%). Transabdominal sonographic assessment for choledolithiasis should thus be conducted for all patients at admission. The 2012 revision of Atlanta classification requires at least two of the following three criteria for diagnosis:

1) Abdominal pain consistent with the disease.
2) 3-fold increase in serum amylase or lipase levels.
3) Imaging findings consistent with AP.

Several scoring systems and criteria like APACHE, Ranson, and BISAP scores have been used to stratify AP. However, since 1992, the Atlanta criteria have been commonly used as a clinically based classification method for AP. The recent revision of Atlanta criteria in 2012 has further simplified the classification of AP. It incorporates organ failure and/or local complications. The pancreatic and peripancreatic fluid collections are classified into four main groups, according to the revised Atlanta classification. Either acute pancreatic fluid collections (APFCs), which represent a collection of fluid without associated necrosis, or acute necrotic collections (ANCs) involving pancreatic and/or peripancreatic necrosis can be seen during the first 4 weeks following the onset of AP. After 4 weeks of initiation, fluid collections with a formed definable wall are referred to as a “pancreatic pseudocyst,” if the collection contains only fluid with no associated solid necrotic portion, or as a “walled off necrosis” (WON) that may include both the pancreatic parenchyma and the peripancreatic tissue, or any one alone.

Contrast-enhanced computed tomography (CECT) is routinely used to diagnose AP and define its severity. Modified computed tomography severity index (CTSI), developed by Mortele et al, has shown an excellent correlation between severity of AP and duration of inpatient stay and requirement of surgical/ percutaneous intervention. Using computed tomography angiography (CTA), we can visualize venous and arterial anatomy in such patients. Vascular complications of AP are infrequent and poorly recognized. They may involve arterial as well as venous circulation. Although vascular complications associated with pancreatitis are uncommon, they can prove fatal in a really brief duration. These occur in acute as well as in chronic pancreatitis. Among arterial changes, pseudoaneurysm formation, thrombosis, hemorrhage, and ischemic changes are seen. Arterial aneurysms can cause catastrophic bleeding in some patients.

Similarly, splanchnic venous thrombosis is now being increasingly recognized as being commonly observed in AP, especially in patients with acute necrotizing pancreatitis (ANP).

While there are a few studies on splanchnic venous thrombosis in AP, studies on arterial changes are sparse.

To our knowledge, there are only a few studies on CTA, a noninvasive technique to study vascular changes in AP. This study used CTA to investigate occurrence of arterial changes in AP and correlate them with etiology, presence of necrosis, collections and severity of AP.

Materials and Methods

The study was approved by the institutional review board, and informed consent was obtained by all patients. A total of 50 patients (mean age: 43.04 ± 13.98; age range: 18–77 years) were selected on the basis of presence of at least two out of three of the following criteria:

(i) Abdominal pain consistent with the diagnosis of AP.
(ii) Serum amylase and/or lipase greater than three times the upper limit of normal.
(iii) Characteristic findings from abdominal imaging.

Patient with underlying chronic pancreatitis or those with any prior endoscopic/radiological/surgical intervention for AP were excluded from the study.

All CT examinations were performed using a 64 slice multidetector CT scan machine Ingenuity by Philips after, at least, 5 days of onset of clinical symptomatology. The CT scan included plain scan of the pancreas region, followed by contrast-enhanced scanning and CTA including arterial phase, pancreatic parenchymal phase and portal venous phase taken at 25, 40 and 60 seconds, respectively, after intravenous (IV) contrast administration. Delayed scan was taken in cases of suboptimal distal bowel distension if indicated. Contrast scan was done by administering nonionic iodinated contrast agent at 1 to 2 mL/kg body weight by pressure injector at 3.8 mL/sec for CTA. Plain water was administered as neutral oral contrast agent before the scan to achieve bowel distension for evaluation of gastrointestinal complications. Neutral rectal contrast was administered in accordance with institutional protocol if indicated. On the table, plain water (~ 300 mL) was given orally just before scan initiation as per tolerance of the patient.

The CECT was specifically evaluated for pancreatic, peripancreatic and extrapancreatic necrosis and fluid collection(s). On CTA, splanchnic arterial structures were assessed for any abnormalities, which were classified on the basis of morphology as compression, inflammatory changes, thrombosis, aneurysm and hemorrhage.

Based on the modified computed tomography severity index (CTSI), AP was graded as mild, moderate or severe (∼ Tables 1 and 2). Degree and extent of necrosis was evaluated, and site and size and nature of fluid collections noted.

Table 1 Modified CTSI

<table>
<thead>
<tr>
<th>Category</th>
<th>Findings</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pancreatic inflammation</td>
<td>Normal pancreas</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>Intrinsic pancreatic abnormalities</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>with or without inflammatory changes</td>
<td></td>
</tr>
<tr>
<td></td>
<td>in peripancreatic fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pancreatic/peripancreatic fluid</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>collection or peripancreatic fat</td>
<td></td>
</tr>
<tr>
<td></td>
<td>necrosis</td>
<td></td>
</tr>
<tr>
<td>Pancreatic necrosis</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>≤ 30%</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>≥ 30%</td>
<td>4</td>
</tr>
<tr>
<td>Extrapancreatic Complications</td>
<td>One or more of the following: asci,</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>pleural effusion, vascular</td>
<td></td>
</tr>
<tr>
<td></td>
<td>complications, parenchymal complications</td>
<td></td>
</tr>
<tr>
<td></td>
<td>and/or gastrointestinal tract involvement</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviation: CTSI, computed tomography severity index.
artery (in 8 percent), right gastric artery (in 8 percent). Vascular complications included thrombosis and compression. Pseudoaneurysm formation or hemorrhage was not seen in this study.

Prevalence of changes in superior pancreaticoduodenal artery was 12 percent, in the form of compression, and seen in 6 patients (Fig. 3). Prevalence of changes in splenic artery was calculated to be 8 percent in the form of compression in all patients (Figs. 2 and 4), one of whom also had associated splenic infarcts. Changes in celiac axis were seen in 4 percent, with compression in 2 percent and complete thrombosis in 2 percent. Prevalence in hepatic artery was also 4 percent in the form of compression. Compression of common hepatic artery was seen in 2 percent. Gastrroduodenal artery involvement was not seen in any patient in this study group. Prevalence of changes in inferior pancreaticoduodenal artery was 4 percent, in the form of compression, and seen in two patients. Concomitant involvement of multiple arteries was seen in 10 percent.

Cholelithiasis was the most common etiological factor (64.3 percent), followed by alcohol ingestion (28.6 percent). However, no significant association was seen between cholelithiasis or alcohol ingestion and arterial changes (p > 0.05).

Pancreatic bulk was increased in 47 out of 50 patients (94 percent) and was normal in 3 out of 50 (6 percent). Pancreatic necrosis was present in 39 out of 50 cases (78 percent). Of these, 25 out of 50 (50 percent) had necrosis involving < 30 percent pancreatic parenchyma and 14 out of 50 (28 percent) had necrosis involving > 30 percent of pancreatic parenchyma. Peripancreatic necrosis was present in 14 out of 50 patients (28 percent). Combined pancreatic and peripancreatic necrosis was present in 39 patients (78 percent). Peripancreatic necrosis alone was present in 2 patients (4 percent). Based on presence of either pancreatic or peripancreatic necrosis or both, 41 out of 50 patients (82 percent) had ANP. Nine out of 50 patients (18 percent) with absence of both pancreatic as well as peripancreatic necrosis had interstitial edematous pancreatitis (IEP). All 14 (100 percent) patients with arterial changes had ANP. A significant association was seen between presence of necrosis and arterial changes (p < 0.05).

Presence of pancreatic collection was seen in 14 percent (7/50) and extrapancreatic collections in 66 percent (33/50). On the whole, collections were seen in 68 percent. Collections were seen in 92.9 percent (13/14) with arterial complications, with a statistically significant association seen between presence of pancreatic/ peripancreatic collections and presence of vascular complications (p < 0.05).

Based on modified CTSI scores, 8 percent (4/50) had mild AP, 28 percent (14/50) had moderately severe AP, and 64 percent (32/40) had severe AP. All 14 patients with arterial changes had severe AP. A significant association was noted between arterial changes and severe AP.

### Table 3 Prevalence and nature of arterial changes in our study group

<table>
<thead>
<tr>
<th>Artery</th>
<th>Prevalence (in percentage)</th>
<th>Nature</th>
</tr>
</thead>
<tbody>
<tr>
<td>Celiac axis</td>
<td>4</td>
<td>Compression (n = 1), Thrombosis (n = 1)</td>
</tr>
<tr>
<td>Splenic artery</td>
<td>8</td>
<td>Compression (n = 4)</td>
</tr>
<tr>
<td>Hepatic artery</td>
<td>4</td>
<td>Compression (n = 2)</td>
</tr>
<tr>
<td>Common hepatic artery</td>
<td>2</td>
<td>Compression (n = 1)</td>
</tr>
<tr>
<td>Superior pancreaticoduodenal artery</td>
<td>12</td>
<td>Compression (n = 6)</td>
</tr>
<tr>
<td>Inferior pancreaticoduodenal artery</td>
<td>4</td>
<td>Compression (n = 2)</td>
</tr>
<tr>
<td>Right gastric artery</td>
<td>6</td>
<td>Compression (n = 3)</td>
</tr>
<tr>
<td>Left gastric artery</td>
<td>6</td>
<td>Compression (n = 3)</td>
</tr>
</tbody>
</table>

Abbreviation: AP, acute pancreatitis; CTSI, computed tomography severity index.
Discussion

The function and accuracy of CT in the diagnosis and staging of AP as well as its contribution to prognostic value has been widely documented in literature.\(^\text{10-12}\) To establish a CT severity index, Balthazar and colleagues concentrated on both the occurrence and degree of peripancreatic fluid collections and pancreatic necrosis, as seen on CECT. In this effort to improve the value of CT as a predictor of pancreatitis severity, the CTSI was described as providing an easily achieved and widely available grading system.\(^\text{12}\) Modified CTSI, developed by Mortele et al, has shown a better correlation of disease severity with outcome.\(^\text{9}\) While an excellent association has been identified in the past between the incidence of most morbidity and mortality in patients with AP and the modified CTSI score, it is uncertain if the development of such significant extrapancreatic complications is closely related to the severity of the acute inflammatory pancreatic disease. Only a few studies have reported on the occurrence of peripancreatic vascular complications with CT. Dörfel et al used the modality of color Doppler ultrasound to study vascular complications.\(^\text{13}\)

While many studies have reported on venous complications and splanchic venous thrombosis in AP, arterial changes have only been reported in a few studies. There is a significant variability in prevalence of arterial changes in AP, ranging from 1.2 to 18 percent.\(^\text{13-16}\) In a study using conventional angiography to study vascular changes in patients of AP, ischemic changes at arteriography were observed in 18 patients (66.7%).\(^\text{17}\) The prevalence of arterial changes in our study was 28 percent. Among arteries, the most frequently involved was superior pancreaticoduodenal, followed by splenic artery. There was a higher prevalence of arterial changes in our study (28 percent) compared with the aforementioned studies, likely due to the fact that most of these studies do not describe compression of vessels. When vascular spasm on conventional angiography was studied,
the prevalence of arterial changes was high (66.7%). Among arterial changes, compression was the most common in our study, followed by thrombosis. Splenic infarcts were seen in one patient in our study associated with splenic vein thrombosis and splenic artery compression. Harris et al reported arterial hemorrhage in 18 percent. Mortele et al reported arterial hemorrhage in 5 percent in their study. In the study by Dorffel et al, arterial changes in the form of arterial pseudoaneurysm were noted in 6.8 percent. Talukdar et al reported prevalence of arterial changes in their study, as 1.2 percent in the form of splenic artery pseudoaneurysm seen in two patients. Dorffel et al reported vascular complications (including both venous and arterial) to be significantly more frequent in alcohol-induced AP as compared with gallstone-induced AP. In our study, there was no significant association between arterial changes and etiology of AP. This could be explained by low sample size (n = 50) in our study. To our knowledge, no other study has correlated etiology of AP with vascular changes.

Dorffel et al reported vascular changes to be more frequent in ANP. They used color Doppler to assess vascular complications, including both splanchnic venous and arterial, and reported prevalence of splanchnic vessel thrombosis to be 60 percent in ANP and 32 percent in IEP with fluid collections. In our study as well, arterial changes were seen almost exclusively in those with ANP. There was a significant association between necrosis or ANP with presence of arterial changes, which is in accordance with previous literature.

Gonzalez et al as well as Harris et al reported a significant correlation between vascular changes and peripancreatic fluid collections. Gonzalez et al reported presence of a colocalized collection in 80 percent (16 out of 20) of those with splanchnic venous thrombosis. Harris et al reported fluid collections in 53% of the 45 patients with splanchnic venous thrombosis in their study. However, none of the aforementioned studies correlated presence of pancreatic/peripancreatic collections solely with arterial changes. In our study, collections were seen in 13 out of 14 patients (92.9 percent) with arterial complications. Thus, majority of patients with arterial changes had presence of fluid collections. There was a statistically significant association between presence of collections and occurrence of arterial changes in our study.

In our study, according to the modified CTSI, the severity of the AP was graded as mild (0–2) in 8 percent patients, moderate (4–6) in 28 percent, and severe (8 to 10) in 64 percent patients. Thus, majority of patients in our study were in the severe AP group. Previous studies had a smaller percentage of study population with severe AP (Mortele et al—11.32%). Mortele at al did not find any correlation between arterial hemorrhage and severity of AP, using CTSI to assess severity of AP. This could be explained by low prevalence of arterial hemorrhage in their study. Gonzalez et al as well as Dorffel et al also reported a positive correlation of vascular changes and severe AP. In the study by Inoue et al, using conventional angiography to study arterial changes in AP, the frequency of severe ischemic change was significantly correlated with the Ranson score and was seen more often (66.7%) in the most severe group (Ranson score of 5). In our study, according to the modified CTSI, the severity of the AP in our study was mild (0–2) in 8 percent patients, moderate (4–6) in 28 percent patients, and severe (8 to 10) in 64 percent patients. Arterial changes were exclusively seen in those with severe AP with a statistically significant association between arterial changes and severity of AP (as assessed by modified CTSI). Among the aforementioned studies, none have used modified CTSI as a measure of disease severity.

Our study population had a larger percentage of cases with severe AP as compared with the aforementioned studies, which could be a reason for higher prevalence of arterial changes in our study group.

Conclusion
Arterial complications of AP, although infrequent, can have disastrous and fatal consequences. Their association with modified CTSI, which have not been reported previously according to the authors' knowledge; is significant. CTA is a noninvasive and relatively quick diagnostic tool, which can aid in early diagnosis and treatment of these life-threatening complications, thus significantly reducing the morbidity and mortality.

Funding
None.

Conflict of Interest
None declared.

References
with patient outcome. AJR Am J Roentgenol 2004;183(05):1261–1265