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SMALL BOWEL ISCHEMIA CAUSED BY STRANGULATION IN COMPLICATED SMALL BOWEL OBSTRUCTION. CT FINDINGS IN 20 CASES WITH HISTOPATHOLOGICAL CORRELATION

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Purpose: **To analyze the CT findings in 20 cases of complicated small bowel obstruction with surgically and histopathologically proven small bowel ischemia, caused by extrinsic venous outflow obstruction of the affected bowel loops and to discuss the question, if bowel wall thickening, abnormal bowel wall enhancement, ascites and mesenteric stranding correlate with the severity of bowel wall damage.**
Methods: **CT scans of 20 patients with surgically an histopathologically proven isolated small bowel ischemia caused by strangulation (10 patients with only partial mural, still potentially reversible small bowel ischemia and 10 patients with transmural irreversible small bowel infarction) were analyzed retrospectively with special emphasis on the presence and degree of small bowel wall thickening, enhancement of the bowel wall, ascites and/or mesenteric stranding at CT and the question whether these findings correlated with the severity of ischemic small bowel wall damage in these patients.**
Results: **Small bowel wall thickening, local mesenteric stranding and ascites were equally common in both groups of patients, regardless of whether obstruction and strangulation related small bowel ischemia was transmural or only partial mural. Out of those patients who were examined by contrast enhanced studies no patient showed lack of enhancement along the ischemic bowel loops.**
Conclusion: **Although highly sensitive and specific for small bowel ischemia in complicated small bowel obstruction, the presence and degree of bowel wall thickening, ascites or local mesenteric stranding at CT do not correlate with the severity of ischemic small bowel wall damage and even the presence of bowel wall enhancement does not exclude severe and potentially transmural bowel infarction in these patients.**

Key-words: Intestines, stenosis or obstruction – Intestines, CT.

Acute bowel ischemia is a complex entity that may affect both the large and the small bowel and that can result from a variety of different conditions that critically disturb intestinal perfusion (1, 2). The clinical, radiological and histopathological presentation of bowel ischemia may show a wide range. In partial mural bowel ischemia the ischemic damage of the intestine may be limited to the mucosa, or it may be more pronounced and affect also the submucosal space or the muscularis propria. Contrarily, in transmural bowel ischemia (ie. bowel infarction) the ischemic bowel wall damage involves all layers and typically represents a full thickness necrosis of the affected bowel wall.

While mild ischemic bowel lesions involving only the mucosa are typically self limiting and reversible, more pronounced ischemic bowel wall damage involving also the submucosal space or the muscularis propria may lead to scarring and strictures as a late complication. Therefore, surgical bowel resection is not absolutely necessary in mild and only partial mural bowel ischemia, whereas transmural bowel infarction definitely requires surgical resection of the necrotic bowel segments.

Over the past years CT has become the key imaging modality for the detection of acute bowel ischemia. It is well known that bowel ischemia may present with a wide range of imaging findings at CT including bowel wall thickening, absent or heterogeneous bowel wall enhancement, hypo- or hyperattenuating bowel wall thickening, mesenteric fluid and ascites, pneumatosis or even mesenteric or portal venous gas (2-9). Although many of these CT findings are not highly specific, the diagnosis of intestinal ischemia can be made with a high accuracy by CT if there are additional radiological or clinical findings suggesting the diagnosis of acute bowel ischemia.

In cases of arterial bowel ischemia these include severe arteriosclerosis, occlusion of visceral vessels, aortic dissection, severe mural thrombosis of the aorta, infarctions in other organs such as spleen or kidneys (suggesting thromboembolic disease), or just a typical and suggestive clinical setting such as a positive medical history of prior abdominal aortic surgery, retrograde angiography, atrial fibrillation, cardiovascular disease, hypotensive episodes, hematological disorders, vasculitis or known administration of certain drugs (10-21).

However, the majority of histopathologically proven cases of small bowel ischemia in our daily routine represent venous ischemia, caused by extrinsic compression of mesenteric veins in patients with complicated small bowel obstruction, in which the presence of bowel ischemia usually represents only an additional, but surely important finding, since it strongly influences the further management of these patients. It is known, that in context with clinical and radiological findings of acute small bowel obstruction the presence of small bowel wall thickening, ascites and mesenteric stranding at CT has a high sensitivity and specificity for the diagnosis of strangulation induced venous small bowel ischemia (22-24).

However, differentiation between partial mural and transmural bowel ischemia is difficult by CT as long as perforation has not yet occurred and as long as pronounced pneumatosis or portal venous gas do not indicate a more severe and potentially

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Table I. — Results.

Imaging findings (n = 20)	Partial mural ischemia (n = 10)	Transmural infarction (n = 10)
Bowel wall thickening	5 (50%)	6 (60%)
Mesenteric stranding	8 (80%)	8 (80%)
Peritoneal fluid / ascites	7 (70%)	8 (80%)
One or two findings	5 (50%)	3 (30%)
All three findings	5 (50%)	6 (60%)
Bowel wall thickness	4-7 mm (mean 5.8 mm)	4-12 mm (mean 7.2 mm)
Bowel wall enhancement	5/5 (100%)	4/4 (100%)

transmural infarction. Furthermore, absence of bowel wall enhancement seems to be a highly specific CT finding in cases of arterio-occlusive bowel infarction but, this is probably not valid for cases of venous ischemia without thrombosis of the mesenteric veins.

Therefore the present study was initiated to evaluate retrospectively if the presence and degree of bowel wall thickening, bowel wall enhancement, mesenteric stranding or ascites correlate with the severity of ischemic bowel wall damage in cases of obstruction and strangulation related small bowel ischemia.

Material and methods

Patients

20 consecutive patients with histopathologically proven isolated small bowel ischemia were included into our study. Patients were identified by reviewing the histopathological reports of all patients who underwent small bowel resection due to small bowel obstruction and who showed ischemia or infarction. Patients in whom ascites could have had other causes, such as liver cirrhosis or peritoneal carcinomatosis and patients with focal or diffuse neoplastic or inflammatory diseases affecting the bowel, the mesentery or the peritoneal cavity were not included into our study as well as patients in whom transmural bowel infarction could be suspected by CT already on the basis of pronounced pneumatosis and/or portal venous gas or free intraperitoneal air and/or peritonitis indicating intestinal perforation. Therefore, our study population consisted exclusively of patients with non-perforated complicated obstruction of their small bowel, caused by adhesions or herniations with or without volvulus, and with subsequent also purely mechanically induced extrinsic venous small bowel ischemia.

The patients were divided into two groups based on the severity of small bowel ischemia. Group 1 consisted of 10 patients with isolated transmural small bowel infarction secondary to strangulation obstruction. Group 2 consisted of 10 patients with isolated, but only partial mural small bowel ischemia secondary to strangulation obstruction. 9 patients were male, 11 were female. The age of our patients ranged from 28 to 89 years with a mean age of 62.75 years. 15 patients underwent surgery within 12 hours after their CT examination. 2 patients of group 1 were operated the next day (within 24 hours) after their CT examination and one patient of group 1 even with a delay of 3 days. However, these patients were not excluded from our study since they had shown positive findings of bowel ischemia (wall thickening, stranding and ascites in two and pronounced ascites in one of these patients) already at the time of their CT examination. In 2 patients of group 2 the time delay between CT and abdominal surgery was also 12-24 hours, but these patients were also not excluded from our study since one of them showed wall thickening, ascites and stranding and the other pronounced ascites already at the time of their CT examination and since they both showed subacute partial mural bowel ischemia at the time of surgery. In all patients the ischemic parts of the small bowel were resected and histopathological analysis was therefore available in all cases (Table I).

Image acquisition and image analysis

CT scans were performed using a Somatom Plus 4 scanner (Siemens, Erlangen, Germany). 9 CT examinations were contrast enhanced studies, while 11 CT examinations were unenhanced studies. In contrast enhanced studies, intravenous contrast was administered with a volume of 100 ml and an injection rate of 2 ml/second. If intravenous contrast was given, image acquisition was performed at a portal venous phase with a scan delay of 70 seconds. Slice thickness and table feed ranged from 8/8 mm to 10/10 mm and window/center was 350/50. All patients received oral contrast prior to their CT examination although in the majority of cases opacification of the entire small bowel and especially of those segments that were strangulated was not reached at the time of scanning. All CT scans were analyzed retrospectively by two experienced radiologists with special interest in gastrointestinal radiology who were unaware of the surgical and histopathological results. Special emphasis was given to describe abnormal small bowel wall thickening of more than 2 mm, local mesenteric stranding and ascites if present and to determine the degree of each one of these findings. Bowel wall enhancement and presence or absence of mesenteric venous engorgement were analyzed only in nine patients according to the fact that eleven CT examinations were performed as unenhanced studies only. Finally, all surgical and histopathological reports on the resected small bowel wall segments were reviewed. Transmural small bowel infarction was defined as continuous ischemic full thickness necrosis of the affected small bowel wall with or without serositis, whereas partial mural small bowel ischemia was defined as ischemic necrosis limited to the mucosa and submucosa with or without parts of the muscularis propria but without continuous necrosis of all bowel wall layers and without serositis. Finally the presence and degree of bowel wall thickening, ascites and mesenteric stranding at CT was compared to the severity of small bowel ischemia as determined by histopathology in all patients.

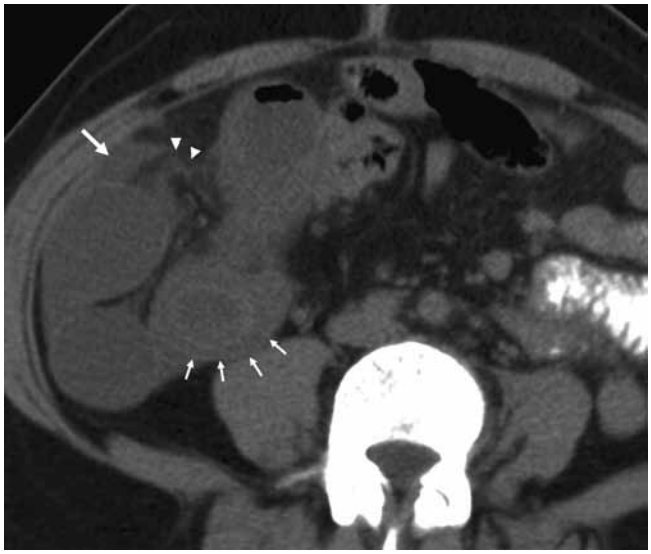

Fig. 1. — Unenhanced spiral CT of the abdomen in a patient with acute small bowel obstruction shows thickened small bowel loops (small arrows), local mesenteric stranding (arrowheads) and small amounts of free intraperitoneal fluid (large arrow). Surgery confirmed strangulation obstruction with ischemic small bowel loops and histopathological analysis showed transmural small bowel infarction with full thickness necrosis of the affected small bowel walls.

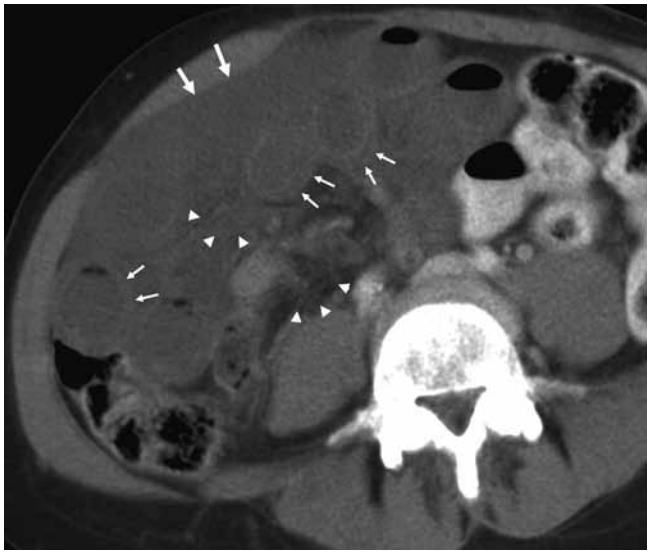

Fig. 2. — Contrast enhanced spiral CT of the abdomen in a patient with acute small bowel obstruction shows thickened small bowel loops (small arrows), local mesenteric stranding (arrowheads) and free intraperitoneal fluid (large arrows). Surgery confirmed strangulation obstruction with ischemic small bowel loops but histopathological analysis showed only early ischemia limited to the mucosa and submucosa of the affected small bowel walls.

Results

In those 10 patients who presented with transmural small bowel infarction secondary to complicated small bowel obstruction (Group 1), bowel wall thickening was present in 6 patients (60%) and ranged from 4-12 mm (mean 7.2 mm). Ascites was present in 8 patients (80%) and mesenteric stranding was present in 8 patients (80%). 6 patients (60%) showed all 3 of above findings, 1 patient (10%) showed two of above CT findings and 2 patients (20%) only one of above CT findings. One patient of group 1 showed neither wall thickening, nor mesenteric stranding nor ascites at CT. However, in all four patients from group 1 who received intravenous contrast the bowel walls showed contrast enhancement.

In those 10 patients who presented with only partial mural small bowel ischemia secondary to complicated small bowel obstruction (Group 2), bowel wall thickening was present in 5 patients (50%) and ranged from 4 mm to 7 mm (mean 5.8 mm). Ascites was present in 7 patients (70%) and mesenteric stranding was present in 8 patients (80%). 5 patients (50%) showed all 3 of above findings and 5 patients (50%) presented with only one of above CT findings. However, in all five patients from group 2 who received intravenous contrast the bowel wall showed contrast enhancement.

Overall small bowel wall thickening, bowel wall enhancement, mesenteric stranding and/or ascites were comparably common in those patients who proved to have transmural small bowel infarction secondary to complicated small bowel obstruction as compared to those patients who had only partial mural small bowel ischemia (Figs. 1-5). Differentiation between transmural and partial mural small mall bowel ischemia in strangulation obstruction was not possible by CT since some patients with mild bowel ischemia limited to the inner layers (mucosa and submucosa) showed already very pronounced bowel wall thickening, ascites and mesenteric stranding at CT, comparable to the findings of some patients with transmural ischemia, whereas others with transmural ischemia presented with much less pronounced CT findings (Fig. 5).

Discussion

Over the past years the value of CT in diagnosing acute bowel ischemia has been well established (1-9). The causes for acute bowel ischemia range from embolic, thrombotic or atherosclerotic occlusion of the mesenteric arteries (10, 11), primary or secondary thrombotic occlusions of the mesenteric veins (12, 13) certain hematological disorders (12-14), various forms of vasculitis (14-18) to various non-occlusive conditions (19-21).

Nevertheless, one of the more common reasons for small bowel ischemia encountered in our daily routine is complicated small bowel obstruction including closed loop obstruction with strangulation by adhesions, incarceration in internal or external hernias or small bowel volvulus in which the involved small bowel may show venous or hemorrhagic ischemia and/or infarction that is caused by mechanically induced extrinsic occlusion of the mesenteric veins with or without subsequent mesenteric venous thrombosis (22-23).

In cases of small bowel obstruction patients are often not able to drink enough contrast material prior to their CT – examination and administration of oral contrast is also not absolutely necessary for the CT diagnosis under such circumstances, since the orally ingested contrast material will often not reach the point of obstruction in these cases

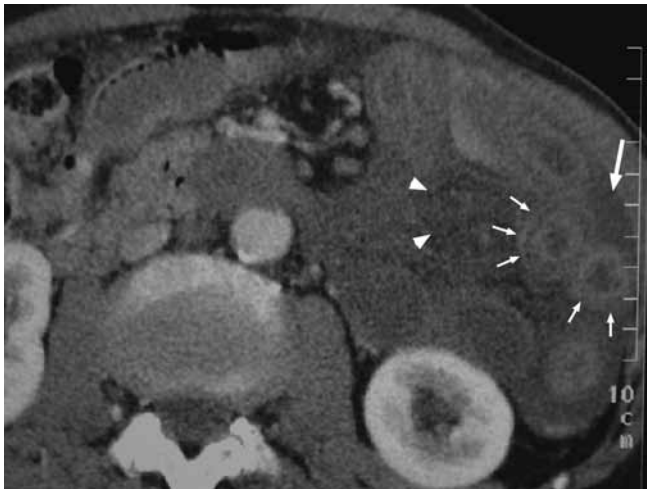

Fig. 3. — Contrast enhanced spiral CT of the abdomen in a patient with acute small bowel obstruction shows thickened small bowel loops (small arrows), local mesenteric stranding (arrowheads) and small amounts of free intraperitoneal fluid (large arrow). Surgery confirmed strangulation obstruction with ischemic small bowel loops and histopathological analysis showed transmural small bowel infarction with full thickness necrosis of the affected small bowel walls.

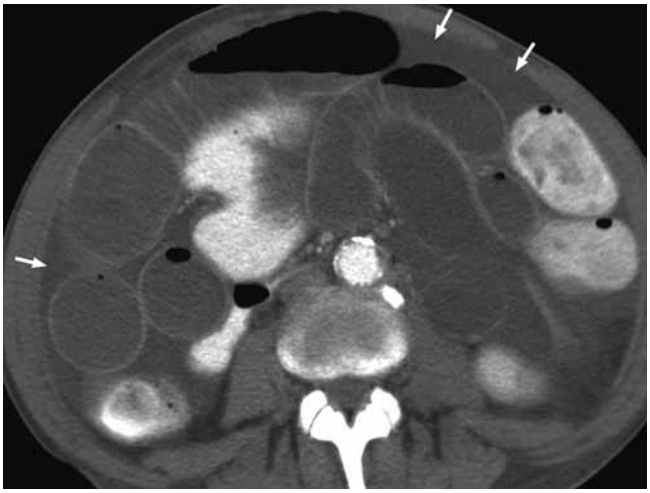

Fig. 5. — Contrast enhanced spiral CT of the abdomen in a patient with acute small bowel obstruction shows only dilated bowel loops without bowel wall thickening, but large amounts of free intraperitoneal fluid (large arrows). Surgery confirmed strangulation obstruction with ischemic small bowel loops and histopathological analysis showed transmural small bowel infarction with full thickness necrosis of the affected small bowel walls.

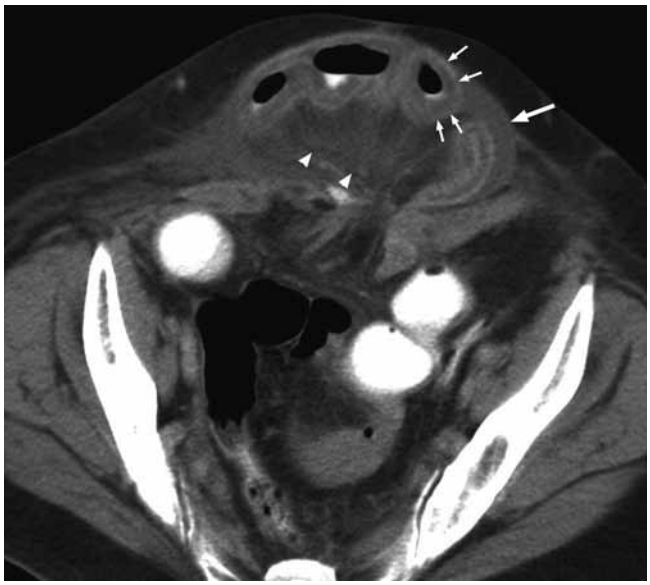

Fig. 4. — Contrast enhanced spiral CT of the abdomen in a patient with acute small bowel obstruction due to an abdominal wall hernia shows thickened small bowel loops (small arrows), local mesenteric stranding (arrowheads) and free intraperitoneal fluid (large arrow). Surgery confirmed strangulation obstruction with ischemic small bowel loops but histopathological analysis showed only early ischemia limited to the mucosa and submucosa of the affected small bowel walls.

anyway. However, since it is not easy to know in advance what`s awaiting you, administration of some oral contrast prior to the CT – examination may be helpful for detection of perforations and especially also to pronounce the prestenotic dilatation of bowel loops and to depict the exact level and degree of obstruction.

In contrast to inflammatory bowel wall thickening, in which oral opacification of thickened and hyperemic bowel walls may be even contraproductive, since it may sometimes masquerade the hyperdense thickening of the bowel walls, this problem does usually not occur in strangulation related small bowel ischemia, where the thickened bowel walls are relatively hypodense due to edema, and therefore, may be usually well depicted – regardless if the lumen of the affected bowel loops is filled with contrast material or not.

Intravenous contrast administration on the other hand is important in this setting, and although many patients of our retrospective study had been examined unfortunately by unenhanced CT only, one should at least try to perform such CT – studies as unenhanced and contrast enhanced studies with acquisition of a portal – venous scan with a time delay of around 70 seconds in order to be able to analyze the enhancement pattern of the bowel walls and also not to miss potentially important additional findings, inlcuding for example an associated secondary mesenteric venous thrombosis.

Although many CT findings may be encountered in acute bowel ischemia, unfortunately most of them are non-specific since bowel wall thickening, hypo- or hyperattenuating bowel walls, inhomogeneous bowel wall enhancement, mesenteric stranding and ascites may occur in many non-ischemic conditions as well. Therefore, acute bowel ischemia may be diagnosed by CT with a higher accuracy only if CT

also shows the primary cause of bowel ischemia (such as occlusions of mesenteric vessels or infarctions of other organs suggesting embolic disease), more specific signs of bowel ischemia (such as pneumatosis or portal-venous gas) or if there is a highly suggestive patients history or a very typical clinical setting.

Therefore, if small bowel wall thickening, mesenteric stranding and/ or ascites are encountered in a clinical and radiological setting of acute small bowel obstruction, the reported specificities of each one of these CT findings for strangulation obstruction with associated small bowel ischemia may rise as high as to 78%, 90% and 76%, respectively, and the presence of two of these CT findings may even further increase the specificity of CT for the diagnosis of bowel ischemia in small bowel obstruction to 94-99% (23).

Nevertheless, although the overall sensitivity and negative predictive value of helical CT in diagnosing strangulation obstruction and bowel ischemia in complicated small bowel obstruction has been reported to be as high as 96% and 99%, respectively, it is very difficult, if not impossible to estimate the severity of bowel ischemia as long as transmural bowel infarction with perforation has not yet occurred under these conditions and as long as there is absence of free intraperitoneal air, signs of diffuse peritonitis or pronounced pneumatosis intestinalis and portal-venous gas (24-26).

Unfortunately in cases of complicated small bowel obstruction bowel wall thickening, mesenteric stranding and ascites do result from venous congestion and transudation, which is caused by mesenteric venous outflow obstruction caused by compression and/or strangulation of mesenteric veins with or without subsequent mesenteric venous thrombosis. Therefore, and since it is impossible to estimate the degree of compromise of the mesenteric venous circulation as well as the duration of preexistent mesenteric venous outflow obstruction at the time of the CT examination the presence of above CT findings can not allow to estimate the severity of the ischemic damage to the small bowel wall according to the fact, that edema of the bowel wall, intramural hemorrhage and edema within the adjacent mesentery as well as fluid in the mesentery and in the peritoneal cavity may develop earlier under these circumstances than subsequent transmural infarction and full thickness necrosis of the involved bowel segments.

This is shown by our findings, where the presence of bowel wall thickening, mesenteric stranding and ascites at CT was equally encountered in both groups of patients, regardless whether their obstruction related small bowel ischemia was transmural or only partial mural.

The fact that the time delay between CT and surgery was more than 12 hours in three patients of group 1 and in two patients from group 2 may be a limitation of our study, but we can argue that the three cases of group 1, in whom the time delay between CT and surgery has been more than 12 hours might have had only partial mural bowel ischemia at the time of their CT examination and that their ischemic bowel wall damage just progressed to transmural bowel infarction in the meantime. This would only support our results which show that the CT findings of strangulation obstruction and subsequent bowel ischemia may be observed in earlier stages allready – at a time when the obstruction related bowel ischemia is only partial mural and, therefore, still potentially reversible.

According to the studies of Balthazar and Zalcman CT has a sensitivity of 83% and 96%, a specificity of 93% and 93%, a positive predictive value of 79% and 72% and a negative predictive value of 95% and 99% for the diagnosis of strangulation related small bowel ischemia in complicated small bowel obstruction. Although small bowel ischemia in strangulation obstruction may be missed by CT as shown by older studies including non-helical CT scans, the latest study of Zalcman et al, including only results from helical CT scans shows that the sensitivity, specificity and negative predictive value of CT in diagnosing strangulation obstruction range from 93-99%. The significantly lowest value of this study is found in the positive predictive values (72%) and according to our findings it may be speculated if this result is probably underestimated. The study of Zalcman showed that out of those 23 patients with true positive CT scans for strangulation obstruction only 14 patients needed bowel resection, which means that the remaining 9 patients with proven bowel ischemia must have had reversible ischemia which did not request bowel resection. Therefore, one may further speculate that those cases from the studies of Balthazar and Zalcman, who have shown CT findings of bowel ischemia but who did not undergo surgery and who therefore were counted as „false positive“ did probably not represent false positive cases throughout, but probably at least partially also cases with partial mural and reversible bowel ischemia.

Taking these considerations into account, the high specificity of CT in diagnosing strangulation obstruction would not further increase singificantly according to the fact that the majority of patients with small bowel obstruction do not have bowel ischemia. In contrast, the positive predictive value of CT in the detection of strangulation obstruction might increase if those cases that were not operated and therefore interpreted as false positive (5/24 patients in the study of Balthazar and 7/32 patients in the study of Zalcman) could be counted as false positive for bowel infarction but as true positive for partial mural bowel ischemia.

However, it is suggestive that the occurrence of bowel wall thickening, mesenteric stranding and ascites preceeds the occurrence of bowel ischemia in certain cases of strangulation obstruction and, therefore, some of these “false positive cases” reported by Balthazar and Zalcman could represent really false positive cases, similarily as it is also surely possible that some of our patients from group 2 might have developed partial mural bowel ischemia only in the meantime between CT-examination and surgery. Nonetheless, the bowel wall mucosa is very sensitive to ischemia and a venous outflow obstruction, that causes transudation into the bowel wall, into the mesentery and/or the peritioneal cavity will quite rapidly also induce some ischemic changes of the inner bowel wall layers. Therefore, neither the presence of bowel wall thickening nor the presence of mesenteric stranding or ascites will allow clear differentiation between transmural small bowel infarction and only partial mural small bowel ischemia in complicated small bowel obstruction.

Although absence of bowel wall enhancement is one of the most specific CT findings in arterio-occlusive small bowel infarction, this is obviously not always valid for cases of venous ischemia, where the arterial perfusion of affected bowel loops seems to be maintained for a quite long time – regardless whether

partial mural bowel ischemia has already progressed to hemorrhagic necrosis – and the crucial differentiation between potentially reversible partial mural bowel ischemia and irreversible transmural bowel infarction can obviously be made only intraoperatively under these circumstances.

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