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Nonocclusive Mesenteric Hypoperfusion Syndromes: Recognition and Treatment

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The main focus when discussing acute or chronic mesenteric ischemia is on occlusive disease, arterial or venous. This article reviews present knowledge on mesenteric nonocclusive hypoperfusion syndromes. The following three clinical entities are reviewed: (1) Intraabdominal hypertension (IAH), or abdominal compartment syndrome (ACS), is important after ruptured abdominal aortic aneurysm repair. IAH >20 mm Hg occurs in approximately 50% of patients after open repair and in 20% after endovascular repair of ruptured abdominal aortic aneurysm, but these patients are different and no randomized data exists yet. A consensus issued by the World Society of Abdominal Compartment Syndrome provides guidance. Early conservative treatment of IAH and, alternatively, abdominal closure devices for leaving the abdomen partially open temporarily are discussed and a treatment algorithm is suggested. (2) Colonic ischemia after abdominal aortic surgery, its risk factors, clinical presentation, and treatment are discussed. A significant number of such patients develop IAH and reducing the abdominal perfusion pressure affects the left colon, the sentinel organ in these patients. (3) Nonocclusive mesenteric ischemia (NOMI); most often such patients suffer from severe cardiac failure requiring massive inotropic support. The condition is difficult to define. Early diagnosis with multidetector row computed tomography is a worthwhile alternative when angiography presents difficulties. A stenosis of the superior mesenteric artery is frequently enough that it should be ruled out because endovascular treatment can be lifesaving. New knowledge on these three different mesenteric hypoperfusion syndromes is reviewed. Success in treating these difficult patients is benefited from a multidisciplinary approach.
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WHEN DISCUSSING ACUTE or chronic mesenteric ischemia, the main focus is naturally on occlusive disease, arterial or venous. Vascular occlusion is, however, not mandatory to produce intestinal gangrene. Bowel ischemia develops when the oxygen supply to the intestines is insufficient to meet metabolic needs. Particularly in the critically ill patient, when multiple interventions are performed in attempts to save the patient's life, the intestinal circulation may be compromised. Knowledge about the pathophysiology behind mesenteric hypoperfusion syndromes has recently increased and this article will review present knowledge focusing on these three clinical entities.

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Intraabdominal Hypertension and the Abdominal Compartment Syndrome

Knowledge that a tense abdomen can be a life-threatening condition is not new. The pediatric surgeon Gross described the condition in 1948 as a complication of repairing large omphaloceles.[1] However, it was as late as 1984 that the term *abdominal compartment syndrome* (ACS) was suggested by the vascular surgeon Kron.[2] The reason to include a discussion of ACS in this article is that it is the may be the most common cause of intestinal hypoperfusion in contemporary medicine.

Physiological Consequences of Increased Intraabdominal Pressure

Understanding the physiological consequences of an increased intraabdominal pressure (IAP) has emerged gradually during the last decades. The World Society on the Abdominal Compartment Syndrome is an inter-disciplinary organization of professionals with an interest in this condi-

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Table 1 Grading of Intraabdominal Hypertension[3]

Grade	IAP (mmHg)
I	12-15
II	16-20
II	21-25
IV	>25

Abbreviation: IAP, intraabdominal pressure.

tion. It has organized four World Congresses, the latest in Dublin, June 2009. Educational material is available on its Web site (www.wsacs.org). Consensus definitions[3] and recommendations for management[4] have been published.

Normal IAP in a critically ill patient is 5 to 7 mm Hg and at an IAP of 12 mm Hg, renal functional impairment develops. With increasing IAP, dysfunction develops in virtually all organ systems of the body.[3] That the intestinal circulation suffers from intraabdominal hypertension (IAH) has been demonstrated in experimental studies. Diebel et al showed a decrease in portal venous blood flow by one-third in pigs subjected to an IAP of 20 mm Hg.[5] IAH has been graded according to the consensus definition (Table 1), and ACS is defined as an IAP >20 mm Hg and organ dysfunction, thus it is not based on merely pressure measurement. If the mean arterial pressure is low, ACS can develop with an IAP <20 mm Hg. The combination of abdominal perfusion pressure (mean arterial pressure minus IAP) <60 mm Hg and organ dysfunction is also defined as ACS.[3] In the consensus documents, different methods to measure IAP are also described.

IAH/ACS can occur in a number of other clinical scenarios, including trauma and pancreatitis. It can even occur without previous abdominal pathology or surgery, for example, after massive resuscitation for sepsis.[3] Balogh et al demonstrated that use of supranormal resuscitation in general, and use of crystalloids in particular increased the risk of developing ACS after trauma.[6]

IAH/ACS after Operation for a Ruptured Abdominal Aortic Aneurysm

Because this article is written for vascular surgeons, we will focus on the risk of developing IAH/ACS among vascular surgical patients. Patients at risk are those who suffer major bleeding, particularly after operation of a ruptured abdominal aortic aneurysm (rAAA). The fact that renal impairment develops with only an IAP of 12 mm Hg is important to consider in the management of patients operated on for rAAA because it is uncommon that the IAP is <12 mm Hg in the early postoperative period.[7-10] Lower grades of IAH are important to patients suffering from low blood pressure, resulting in a critical abdominal perfusion pressure (see previous section), a rather common situation after operation of a rAAA. The IAH/ACS represents a "second hit" to the patient who was previously in preoperative shock after a rAAA. Experimental data suggest that the intestines suffer more extensive injury after such a second period of ischemia/reperfusion, suggested to be due to depletion of natural scavengers. Pathological IAP is a continuum from mild effects on urinary output and ventilation to a life-threatening condition with bowel ischemia.

According to guidelines in the consensus document, patients with two risk factors for IAH/ACS should have their IAP monitored[4] (evidence base grade 1B). Among the risk factors identified, many are prevalent after rAAA repair, eg, acidosis, hypothermia, polytransfusion, coagulopathy, sepsis, peritonitis, liver dysfunction, mechanical ventilation, use of Positive End Expiratory Pressure, abdominal surgery, and massive fluid resuscitation. Thus, it is evidence-based to monitor IAP in all patients after rAAA repair.

What are the observations among patients operated on for rAAA? In a retrospective study of 104 patients operated on for rAAA between 1978 and 1988, four patients developed overt ACS and two were left with open abdomen at the end of rAAA repair (5.8%);[11] patients were not monitored with IAP and this is probably an underestimation. In another retrospective study from the Mayo Clinic, among 223 patients operated on for rAAA during a 10-year period, 53 (24%) were treated with open abdomen.[12] In 43 of these patients, mesh was used at the primary abdominal closure, and 10 patients (4.5%) underwent a decompression laparotomy due to IAH/ACS. IAP was not measured consistently, however, and was not reported.

Akers et al reported on 23 patients operated on for rAAA.[13] Four were treated with delayed abdominal closure and two required decompression for ACS, 26% in all. No IAP measurements were reported. Oelschlager et al performed a retrospective study on 38 patients treated for rAAA at the Harborview Medical (Trauma) Center in Seattle, Washington, of which 39% died during surgery.[14] Among the 23 survivors, 15 died in the postoperative period; total perioperative mortality was 79%. In comparison, 30-day mortality after rAAA repair in Sweden 2000 to 2005 was 33%.[15] No measurements of IAP were reported, but among the 23 surviving patients, 8 (35%) were either left open or reopened. In Leicester, UK, 75 patients were studied and 22 were operated on for rAAA.[8] IAP was only measured once every 24 hours, and only if the patient was still on the ventilator. Among the patients operated on for rAAA, all of which were on the ventilator at 24 hours and thus were measured at least twice, 12 of 22 (55%) had an IAP value ≥15 mm Hg.

The Consensus definition agreed upon in 2004 was evaluated by performing a retrospective study of patients operated on for rAAA with open repair in Uppsala, Sweden.[10] Nine of 17 patients (53%) consistently monitored at 4-hour intervals for a minimum of 48 hours had an IAP >20 mm Hg at any time. Virtually all serious complications (eg, renal, pulmonary, heart failure, and colonic ischemia) occurred in this subgroup of patients where mortality was 22% compared to no deaths in the subgroup with an IAP <20 mm Hg. In a prospective study of patients with rAAA at two Swedish Hospitals, 29 patients with rAAA were monitored at 4-hour intervals throughout their intensive care unit stay, and 16 (55%) had an IAP ≥20 mm Hg.[16] Thus, if measured consistently, an IAP >20 mm Hg occurs in about half the patients after open repair (OR) of rAAA.

IAH/ACS after Endovascular Repair of rAAA

Data on prevalence of IAH/ACS after endovascular aneurysm repair (EVAR) for rAAA are scarce. In 2005, Mehta et al reported an incidence of 20% of ACS among 30 patients treated with EVAR for rAAA.[17] This, too, is probably an underestimation because they did not monitor IAP and used a more limited definition of ACS than in the World Society on the Abdominal Compartment Syndrome Consensus.[3] They identified four risk factors for developing ACS, ie, use of an aortic occlusion balloon, coagulopathy, massive transfusion requirements, and use of an aorto-bi-iliac versus an aorto-uni-iliac device. Mortality among those who developed ACS was higher (67% *v* 13%; $P = .01$). In 2006, the Albany group reported an extended experience of 40 patients treated with EVAR for rAAA with an incidence of 17%.[18] From the risk factors identified by Mehta et al,[17] a major factor affecting the incidence of ACS was treating hemodynamically unstable patients with EVAR.

In the largest series of patients with rAAA treated with EVAR, in which most patients were monitored with IAP and a proactive approach to ACS was implemented, Mayer et al reported that 20 of 102 patients (20%) developed ACS.[19] Mortality in this subgroup was 30%, compared to 8% among those who did not develop ACS. Total mortality was 13%.

Reported incidence of ACS after EVAR is thus lower than after OR. Comparing the incidence of IAH/ACS after OR with that after EVAR for rAAA in a nonrandomized design is, of course, like comparing apples with oranges. Patients selected for EVAR are often more hemodynamically stable and have a more favorable anatomy, resulting in less bleeding and, consequently, a decreased risk of developing IAH/ACS. In the ongoing Immediate Management of Patient with Ruptured Aneurysm: Open Versus Endovascular repair trial, randomizing between OR and EVAR prior to computed tomography examination in patients with rAAA, an optional protocol on IAP will be included. Hopefully, randomized data will be available for analysis within approximately 2 years. It can be concluded, however, that IAH/ACS is an important problem whether the repair is OR or EVAR.

Action and Timing When IAH/ACS Develops

As surgeons, we have a tendency to be somewhat simplistic in our approach to clinical problems. There are no yes or no answers to the complex issues of when and how to act when IAH/ACS is imminent. An advantage of routine IAP monitoring is that conservative treatment of IAH can be initiated early. The treatment algorithm used at our institution is given in Figure 1.

Treating abdominal pain is the first step and epidural analgesia reduces IAP considerably among patients with tense, painful abdomen.[4] The most powerful tool in prevention of IAH/ACS among patients on a ventilator is neuromuscular blockade (NMB).[4] In a prospective study on 10 patients with IAH, 9 reduced their IAP significantly after a single dose of cisatracurium.[20] If the patient is still on the ventilator and develops IAH 16 to 20 mm Hg, NMB is often effective in lowering IAP, increasing urinary output, and reversing the

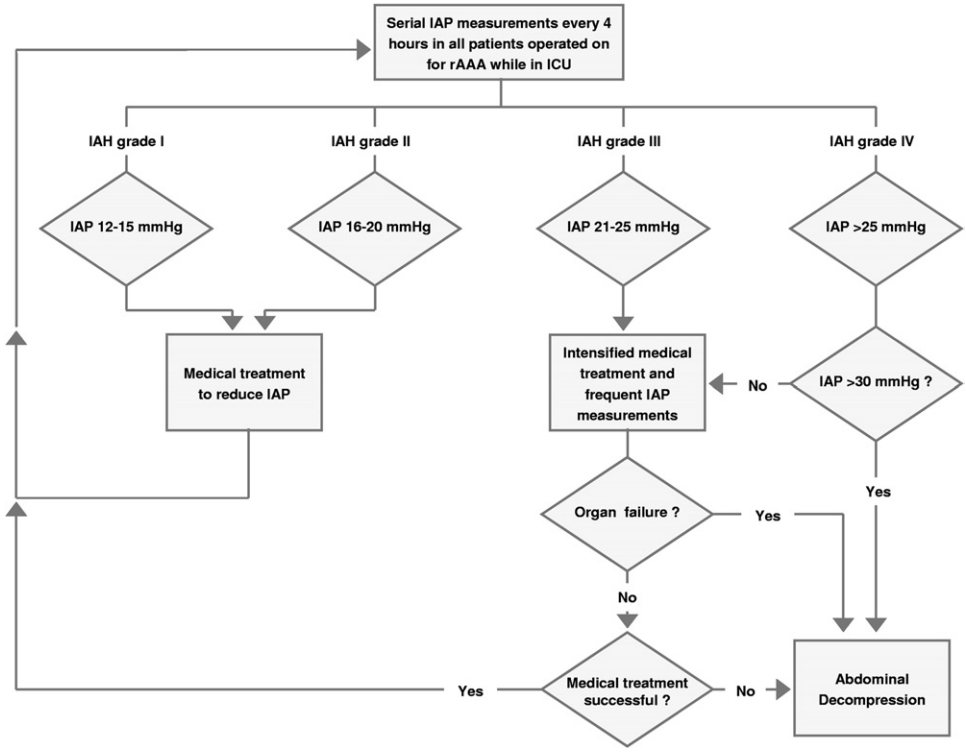

Figure 1 Proposed algorithm on how to act on different levels of intraabdominal hypertension (IAH). ACS, abdominal compartment syndrome; IAP, intraabdominal pressure; rAAA, ruptured abdominal aortic aneurysm. The combination of an abdominal perfusion pressure (mean arterial pressure minus IAP) <60 mm Hg and organ dysfunction is also defined as an ACS.[4]

progression. Although early extubation and ventilation without NMB are natural choices in a normal postoperative situation, this is not the case when the patient has imminent ACS. There are important side effects of prolonged NMB that should be considered, however, particularly the risk of atelectasis and pneumonia.

Optimum fluid resuscitation is controversial. Balogh et al[6] compared two different trauma resuscitation strategies (500 and 600 mL/min^{-1}m^{-2}, respectively). They concluded that "supranormal" resuscitation resulted in a doubled risk of IAH, ACS, organ dysfunction, and death. There are no specific studies on patients operated on for rAAA, but studies on burn and mixed nontrauma surgical patients have shown that resuscitation with isotonic crystalloids increases risk compared to resuscitation with hypertonic crystalloid or colloid solutions.[4] Patients in the early postoperative phase after operation for rAAA are sensitive to hypovolemia, and can easily develop hypoperfusion of the abdominal organs and, in particular, the left colon. This has been detected by tonometry of the sigmoid colon,[16,21,22] and can often be reversed timely with volume resuscitation. An increased IAP will be reflected in an elevated central venous pressure,[3] increasing the risk of not detecting hypovolemia.

Hypertonic colloid solutions in combination with furosemide has been our routine management in this situation and a regimen supported by the Guidelines.[4] When urinary output is not sufficient to reverse volume overload, hemodialysis/ultrafiltration treatment should be considered.

Intestinal contents also contribute to increased IAP. Unfortunately, prokinetic motility agents, such as erythromycin or neostigmine, are seldom effective, and no prospective study has been undertaken evaluating their possible effect.[4] We start enteral feeding early, but also check regularly and drain the accumulated gastric content when necessary.

Decompression Laparotomy

When IAP is >20 mm Hg and/or ACS develops, and conservative treatment is not effective, decompression of the abdomen is necessary and often life-saving (Fig 1). If IAP is >30 mm Hg, there is a risk of acute circulatory arrest and decompression should not be delayed.[5] The patient may seem beyond therapy, and the anesthesiologist may resist accepting the patient for laparotomy. However, the high risk of mortality in this situation is reduced if the patient is volume-loaded prior to decompression.[5]

Decompression laparotomy is most effectively performed through a complete midline incision, although depending on previously performed incisions, this may have to be modified. Cheatham et al have demonstrated excellent long-term physical and mental health among patients after abdominal decompression therapy.[23]

Temporary abdominal closure has to be performed while the patient is treated with an open abdomen (OA). Several different techniques have been described. In principle, the abdomen should be kept open, avoiding adhesions between the intestines and the abdominal wall, as well as lateralization (lateral retraction) of the abdominal wall. It is also important to protect the viscera, preventing fistula formation, which is a dreaded complication to OA. The strategy was summarized in a recent publication.[24]

A high frequency of primary delayed fascial closure was reported with vacuum-assisted wound closure.[25,26] This technique works quite well when a shorter period of treatment with OA is required. With patients needing OA after rAAA, longer treatment periods are often required, resulting in lateralization of the fascial edges, prohibiting delayed primary fascial closure. Different adjunctive techniques have been described to prevent lateralization, including the Wittmann patch.[27] That, however, has been reported to be associated with an increased risk of bowel erosion or fistulae. We have developed a new technique wherein a temporary Prolene mesh is sutured to the fascial edges and placed between the inner and outer bandages.[28] The innermost sponge is covered by a perforated plastic sheet to prevent direct suction and contact between the sponge and the intestines. The mesh is permeable and facilitates evacuation of intraabdominal fluids. When the bandage (V.A.C. Abdominal dressing®, KCI, San Antonio, Texas, USA) is replaced every 2 to 3 days, the mesh is opened in the midline and gradually tightened. Ultimately, the mesh can be removed, allowing primary fascial closure as long as 8 weeks after open abdomen treatment (Figs 2-4). In four Swedish hospitals, 82 patients were treated with this technique. Fascial closure was achieved after a median of 15 days and four mesh-tightening procedures in 94% (67 of 71 patients), whereas four were reconstructed with

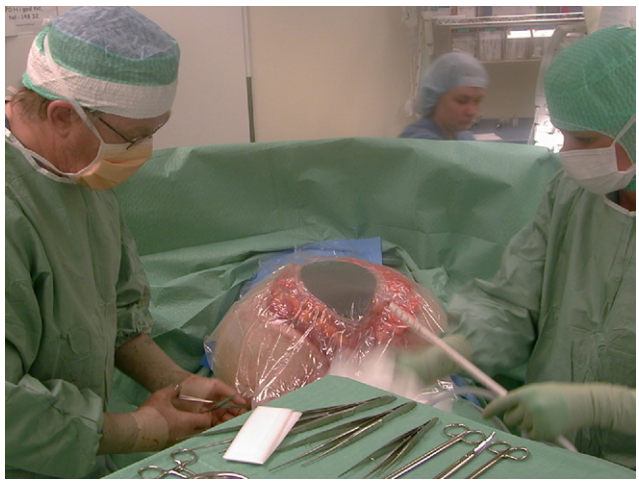

Figure 2 This patient was treated with endovascular aneurysm repair despite an unfavorable anatomy, because he was a high-risk patient for open repair due to obesity and chronic obstructive pulmonary disease. Due to a distal type I endoleak, the abdominal aortic aneurysm ruptured, the patient was transported to Uppsala by helicopter, and the leakage could be treated endovascularly. He required massive transfusions, his body weight had increased from 110 to 125 kg, and he developed abdominal compartment syndrome requiring decompression laparotomy. This figure shows how the inner layer of the KCI Abdominal VAC dressing is placed around the intestines, preventing the abdominal wall from adhering to the intestines.

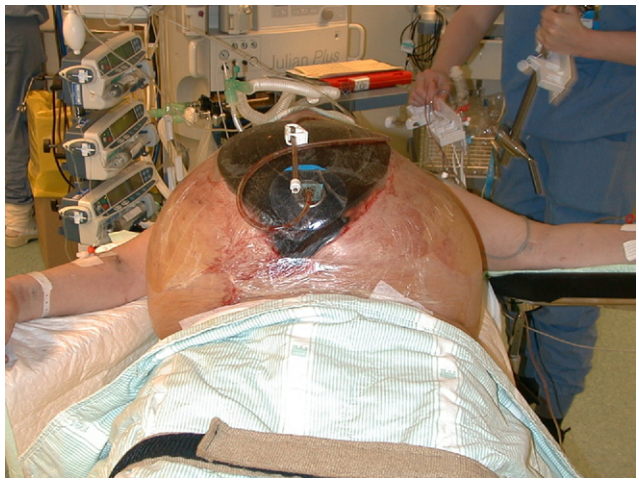

Figure 3 The same patient as in Figure 2, the dressing has now been completed. This patient's fascia could be closed after 24 days. The patient survived and no infection or hernia developed after a follow-up of 4 years.

mesh. The remaining 11 patients died before closure of the abdomen.[29]

Van Herzeele et al reported a successful decompression of a patient with ACS after operation of rAAA with EVAR without laparotomy through an 18-cm lumbotomy,[30] an alternative to OA after EVAR of rAAA. We have not used this technique, believing that decompression this way might not be as effective as with a midline laparotomy.

Primary Delayed Closure of the Abdomen

The Mayo Clinic left many patients open at the end of the primary laparotomy (43 of 223, 19%).[14] The Zürich group have also reported that they have left all their rAAA patients after OR open.[18] This issue would ideally be addressed by a randomized controlled trial. Designing such a trial is problematic, however, because the difference between leaving all patients with OA, and monitoring all patients' IAP and performing "on-demand-decompression" may not be great.

Complications to Treatment with OA

The most dreaded complications of treating a patient with OA who has a vascular graft are intestinal fistula[24] and graft infection. Graft infections can develop years after AAA repair, thus long-term follow-up is crucial to evaluate this risk. Akers et al reported development of an intraabdominal abscess in one of six patients treated with OA after rAAA[13] who was treated with OA for 51 days. Oelschlager et al reported no instances of graft infection among four survivors treated with OA after rAAA.[14] We have treated >20 patients with OA after AAA surgery, and have not yet experienced any late graft infection. To summarize, although the exact risk of graft infection after treatment with OA after AAA repair remains unknown, it is likely not very high, and has to be balanced against the risk of not decompressing a patient with IAH/ACS. Intestinal fistulae formation is another threat. Contact between the intestines and mesh or foam must be avoided. We have not yet experienced any intestinal fistula considered a complication of OA treatment.[28]

Colonic Ischemia Following Abdominal Aortic Repair

The first successful resection of an AAA and its replacement with a graft was reported by Freeman and Leeds in 1951,[31] and the first case report of colonic gangrene after AAA repair was reported by Moore the following year.[32] Since then, the complication of colonic ischemia (CI) has been a companion of aortic-iliac surgery. Longo et al reported an incidence of 1.2% among 4,957 AAA operations from the US Veterans Affairs Registry, but they did not state if any of those operations were for rupture.[33] In a study on 2,930 aortoiliac operations prospectively registered in the Swedish Vascular Registry (Swedvasc), frequency of clinically evident transmural bowel gangrene was 2.8%, but 7.3% among the 412 patients operated on for rAAA in preoperative shock and 23% of the deaths were associated with CI.[34]

Risk Factors for Postoperative CI after Aortoiliac Repair

With the previously mentioned larger series, it was possible for the first time to perform a multivariate analysis to study risk factors to develop CI after aortoiliac surgery.[35] This is of great importance because there are many confounding factors: Patients presenting with rAAA have larger aneurysms with more hostile anatomy, are older, have more comorbidities, suffer longer cross-clamping, and greater bleeding, etc. The following independent risk factors were identified: preoperative shock, renal insufficiency, emergency surgery, age, type of hospital, aortobifemoral grafting, operating, and

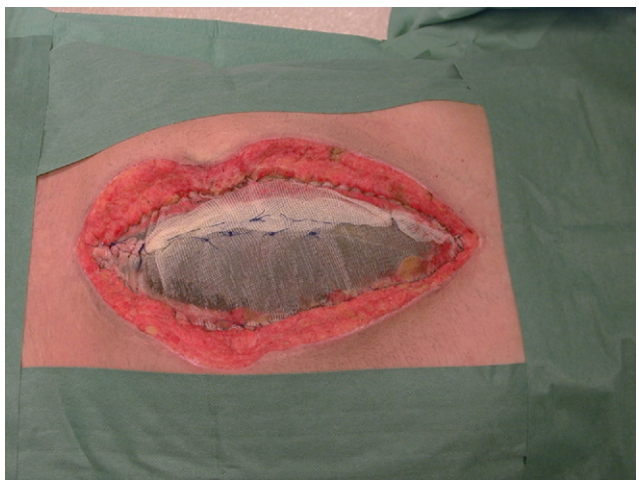

Figure 4 Another patient who developed abdominal compartment syndrome after a Thoracic EndoVascular Aortic Repair for type B aortic dissection with intestinal ischemia and an intestinal reperfusion syndrome (see also Fig 9). The Prolene mesh has been tightened in the midline over the inner dressing, approximating the fascial edges. This patient's abdomen was closed after 12 days, no infection or hernia developed after a follow-up of 2 years.

cross-clamping times, as well as ligation of the hypogastric arteries.[35] In previous publications, it had been stated that surgery for aneurismal disease was a risk factor compared to surgery for aorto-occlusive disease, but that was probably a mere effect of mixing patients with AAA rupture with those operated on electively. In this investigation, no difference in risk was found between the two groups of elective surgery. This, of course, is more of an historical note because open surgery for aortoiliac occlusive disease is quite rare now. In a more contemporary study, Becquemin et al studied 1,174 patients operated on between 1995 and 2005 (492 with EVAR, 88 for rupture) with similar conclusions for both risk factors and incidence.[36]

Higher Incidence after Routine Sigmoidoscopy

Several investigators have performed routine postoperative sigmoidoscopy after operation for rAAA, reporting CI in about half the patients studied. In this situation, however, mucosal ischemia is also detected. Champagne et al reported 36% CI at sigmoidoscopy in 62 patients who survived rAAA more than 24 hours, but only 14.5% required colonic resection.[37] Perfusion of the sigmoid colon can also be monitored by measuring the CO_2 in a balloon catheter placed there through a colonoscope. The method, referred to as tonometry or intramucosal pH measurement (pH_i), can be used to time sigmoidoscopy, thus increasing diagnostic accuracy. Two prospective studies using this technique showed that prolonged hypoperfusion of the sigmoid colon, defined as $pH_i \leq 7.1$, was strongly associated with adverse outcomes after AAA repair.[21,22]

Clinical Presentation

The classical presentation of CI after aortoiliac surgery is abdominal pain and early passage of bloody stools, often diarrhea. The diagnosis is not always that straightforward, however. In a study of 63 patients suffering the complication, only 26% had both early (defined as within 24 hours after completion of the operation) and bloody passage of stools (Fig 5),[34] although 62% had either symptom (Fig 6). Many patients have

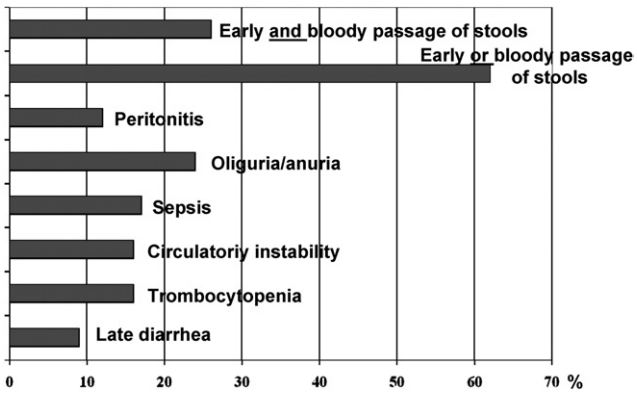

Figure 5 Presenting symptoms (%) among 63 patients suffering colonic ischemia after aortoiliac surgery.[34]

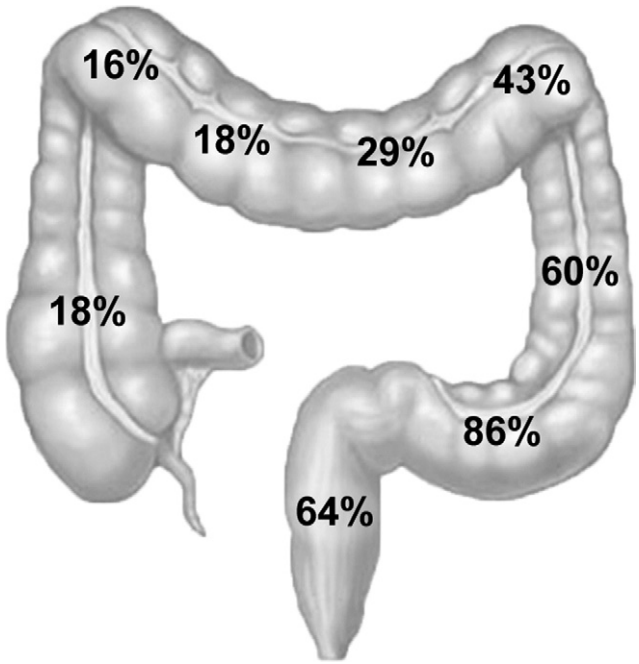

Figure 6 Distribution (%) of colonic ischemia in 63 patients suffering colonic ischemia after aortoiliac surgery.[34] Note that more than one segment could be affected, explaining why the sum exceeds 100%. Reprinted from Björck M, Bergqvist D, Troëng T: Incidence and clinical presentation of bowel ischaemia after aortoiliac surgery-2930 operations from a population-based registry in Sweden. Eur J Vasc Endovasc Surg 12:139-144, 1996,[34] with permission.

rather nonspecific indications of deterioration, such as oliguria, sepsis, and circulatory instability.

The fact that the inferior mesenteric artery is either occluded by the disease or is sacrificed during surgery makes the left colon most susceptible to ischemia. Although the gangrene can affect a larger proportion of the colon, and sometimes even the small bowel, in 95% of the 63 patients some part of the left colon was affected[34] (Fig 6). The important message is that sigmoidoscopy up to the left colonic flexure should be performed without hesitation whenever a patient does not recover as expected after aortoiliac surgery.

IAH and CI

Despite the fact that some surgical pioneers in exploring the pathophysiology of IAH were vascular surgeons,[2] it is only during the last 5 to 10 years that vascular surgeons have started to take an interest in measuring IAP. As described here, the few articles that have published data on this subject have very seldom measured IAP at close intervals during the postoperative period.

In order to investigate the association between CI and IAP among patients operated on for rAAA, we studied sigmoid colon perfusion by tonometry (pH_i; Tonocap [(GE Healthcare, Helsinki, Finland)]) and IAP.[16] IAP was measured every 4 hours. Patients with $pH_i \leq 7.1$ were treated for suspect hypovolemia with colloids and underwent colonoscopy. Patients with IAP ≥ 20 mm Hg were treated with NMB and/or

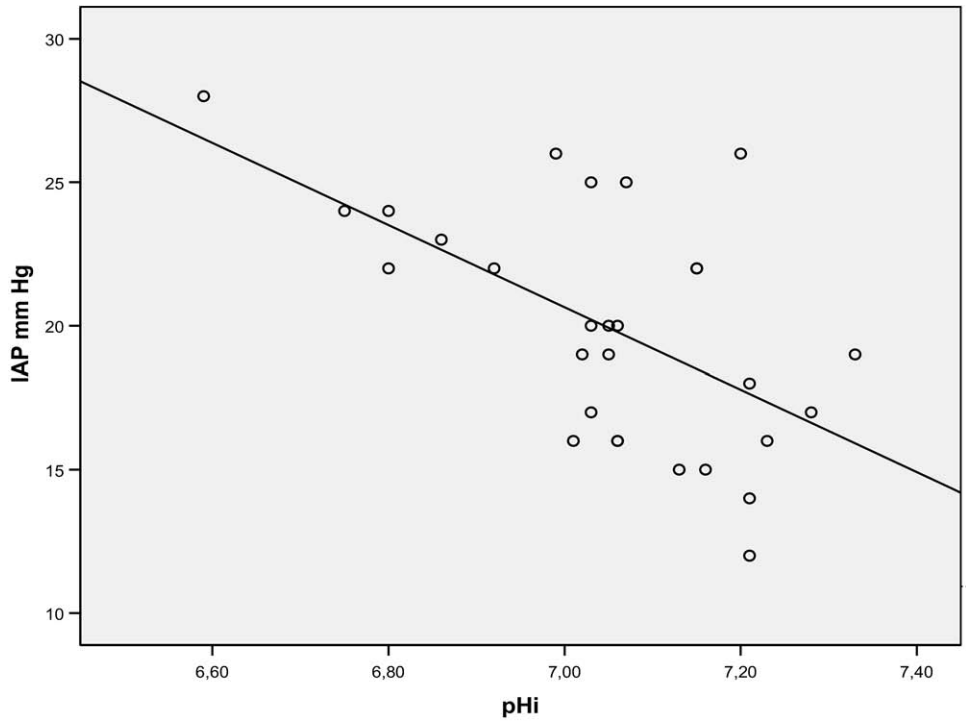

Figure 7 Patients who were monitored after ruptured abdominal aortic aneurysm (rAAA) repair, with both with intraabdominal pressure (IAP) and intramucosal pH (pHi) (colonic perfusion). The highest IAP value registered for each patient has been plotted against its corresponding pHi, with regression line.[16] Reprinted from Djavani K, Wanhainen A, Valtysson J, Björck M: Colonic ischemia and intra-abdominal hypertension following open surgery for ruptured abdominal aortic aneurysm. A prospective study. Br J Surg 96:621-627, 2009,[16] with permission.

relaparotomy. Fifty-two consecutive patients underwent open rAAA repair. Thirty-day mortality in the whole group was 27% (14 of 52). Eight patients died shortly after surgery, not permitting monitoring. Fifteen patients were not monitored because of logistic problems and mortality in this group was 33% (5 of 15). IAP and pH_i were measured throughout the intensive care unit stay in the remaining 29 patients. Monitoring resulted in volume resuscitation (n = 25), NMB (n = 16), colonoscopy (n = 19), and relaparotomy (n = 2). Mortality was 3.4% (1 of 29). Twenty-three (79%) had pH_i ≤7.1, of whom 15 (52%) had pH_i ≤6.9. An IAP ≥20 mm Hg occurred in 16 patients, of whom 10 also had pH_i <6.90. One with IAP >20 mm Hg and pHi ≤6.9 developed colonic gangrene, but survived after early recognition and colonic resection. Peak IAP values correlated with the simultaneously measured pH_i ($r = 0.39$, $P = .003$), which is illustrated in Figure 7. The higher the IAP, the more likely sigmoid colon ischemia, but there are other mechanisms behind CI, discussed here, both preventable, such as improper ligation of the inferior mesenteric artery (IMA) (see the next paragraph) or bilateral ligation of the internal iliac arteries; and unpreventable, such as preoperative renal insufficiency. We identified two mechanisms related to colonic hypoperfusion after rAAA repair in this study, ie, early hypoperfusion is often explained by hypovolemia, secondary to the inflammatory response with capillary leakage. The other mechanism, hypoperfusion due to IAH, can develop early, but is more prevalent later, as a result of aggressive volume resuscitation. In Figure 8, the temporal relationship between IAH and colonic hypoperfusion is illustrated in 10 patients. IAH and colonic hypoperfusion occurred simultaneously in these patients. Makar et al also studied these temporal relations in 30 patients operated on for rAAA (16 OR, 14 EVAR) and concluded that IAH preceded organ failure,[38] thus reaching a similar conclusion.

Prevention and Early Recognition

The keys to successfully managing the complication of CI, one that has accompanied surgery for abdominal aortic disease since the 1950s, are prevention and early recognition. Technical surgical details are important. During open surgery, the aneurysm should be opened to the right of its midline and the IMA should be suture-ligated from within the aneurysm wall. An IMA occluded flush to the aorta can allow significant blood flow distal to the occlusion.[39] The variability of the blood flow in the IMA and in the hypogastric arteries is great, but only approximately 10% of its combined blood flow is from the IMA,[39] consequently, it can be even more important to preserve the blood flow to the hypogastric arteries. Several investigators have reported that both unilateral and, even more so, bilateral ligation of the hypogastric arteries are associated with increased risk of CI,[21,22,33-35,40] so should be avoided, if possible.

As discussed here, increased IAP is an important contributor to colonic hypoperfusion after rAAA repair. By control-

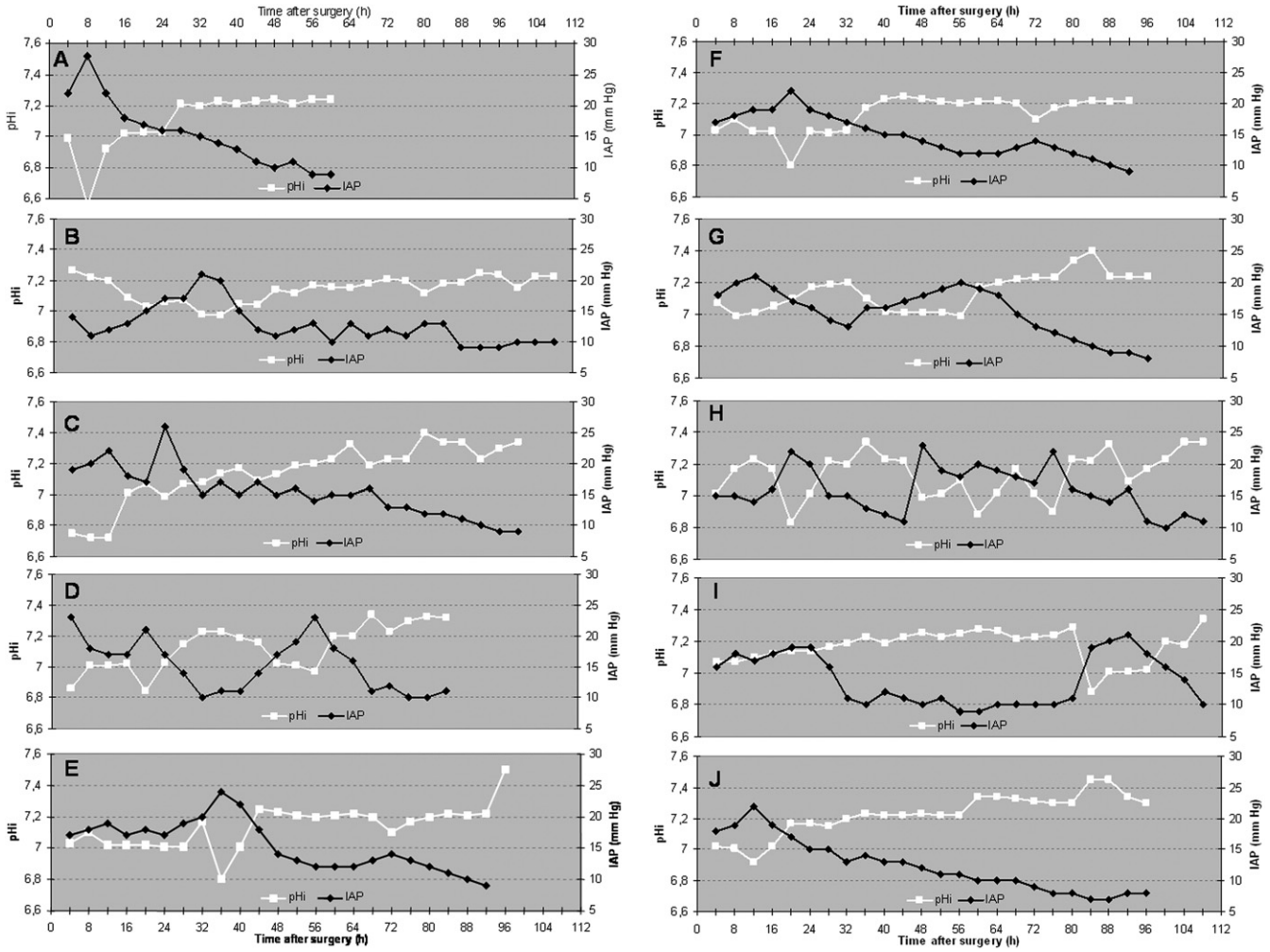

Figure 8 The temporal relationships of intraabdominal hypertension and colonic ischemia in the 10 patients who developed both intraabdominal pressure (IAP) ≥20 mm Hg and intramucosal pH (pHi) <6.90. Only patients A and E developed clinical signs of colonic ischemia.

ling these two mechanisms of hypovolemia and IAH with timely and appropriate interventions, CI and a fatal outcome because of it can be prevented. The issue of hypovolemia is of particular importance if the patient has an increased IAP because in that clinical scenario, the Central Venous Pressure (CVP) will become elevated even if the patient is hypovolemic. If CVP (or wedge pressure) is used to evaluate the volume status of the patient, 50% to 66% of the IAP should be extracted from the CVP.[4] Monitoring IAP and timely intervention makes sense and can improve outcomes.[16,19,22,38,41]

Even if all possible preventive measures are implemented, some patients will still develop CI, and early recognition and timely intervention are the keys to success. In this clinical scenario, the old surgical wisdom "It is better to look and see, than to wait and see," is still valid.

Treatment and Outcomes

Treatment of CI after aortoiliac surgery is straightforward, the gangrenous bowel must be resected as quickly as possible, and a Hartmann procedure is the choice in most cases. A "damage-control" abbreviated laparotomy is usually indicated because the patient often suffers from sepsis and hypotension. All unnecessary procedures should be avoided and postponed to a second-look operation. There is often additional bowel with mucosal gangrene that needs to be protected from further ischemic injury. With contemporary knowledge on the pathophysiological mechanism, it is important to leave the abdomen open if there is any doubt about the viability of the remaining bowel, thus contributing to better perfusion by reducing IAP. Remembering that abdominal perfusion pressure is mean arterial pressure minus IAP, even an IAP of 12 to 15 mm Hg makes a difference.

The reported mortality rates are approximately 41% to 53% among patients suffering this complication,[33-36] in contemporary series, in mixed populations with predominantly elective surgery. With a modern management algorithm it should be possible to improve on this. In fact, controlling this complication may improve overall survival after aortic surgery, particularly in the group operated on for rAAA.[10,16,22]

Nonocclusive Mesenteric Ischemia

It is an old observation that critically ill patients sometimes develop intestinal gangrene even though the three mesenteric arteries are patent. The typical patient is critically ill, with severe cardiac disease or sepsis, and on (often multiple-drug) inotropic support. This condition has been named nonocclusive mesenteric ischemia (NOMI), but it is not well-defined, which also makes estimation of its prevalence problematic. In mixed patient populations with intestinal gangrene, the proportion of patients with NOMI has been reported to be between 4% and 60%.[42,43]

As with all estimations of mesenteric vascular disease, a low autopsy rate is another methodological problem in most investigations. Acosta et al studied this disease in the population of Malmö, where 35,784 deaths occurred during the years 1970 to 1982, and the autopsy rate was 87%.[44] The disease was defined as an intestinal gangrene despite open arteries and with no signs of embolism, dissection, or strangulation. Estimated overall incidence of NOMI with intestinal infarction (verified at autopsy or operation) was 2.0/100,000 person-years, compared to 8.6 for thromboembolic occlusion of the superior mesenteric artery[45] and 1.8 for intestinal gangrene caused by mesenteric venous thrombosis[46] in the same cohort. A clinically important finding was that a large proportion of the patients (25 of 62; 40%) had a stenosis of the superior mesenteric artery, a lesion potentially treatable with endovascular measures. Those with a stenosis of the superior mesenteric artery were older ($P = .002$) and they more often had a concomitant stenosis of the celiac trunk ($P < .001$). Synchronous infarction in the liver, spleen, or kidney occurred in roughly one fifth of patients. In a nested case-control study, fatal cardiac failure (odds ratio = 2.9), history of atrial fibrillation (odds ratio = 2.2), and recent surgery (odds ratio = 3.4) were independent risk factors for fatal NOMI.[44] The fact that recent surgery was an important risk factor is thought-provoking; conceivably some of these patients had an unrecognized ACS.

When scrutinizing the contemporary literature on NOMI, it mostly consists of case reports, small case series, radiological reports (describing the angiographic diagnostic criteria), and reviews. Mitsuyoshi et al[47] report one of the few studies with a prospective study design. They report from a referral center in Kyoto, yet only 22 patients during a 13-year period were treated, <2 patients/year,[47] illustrating the difficulty studying this disease. They diagnosed and treated the first 13 patients in a more conventional manner, diagnosing the condition with angiography, and mortality was high, 9 of 13. One of the reasons for these poor results was the fact that the patient was often a poor candidate for angiography, which resulted in diagnostic delay. After introduction of multidetector row computed tomography (MDCT), which has improved the diagnostic accuracy of mesenteric occlusive disease dramatically, they established the following four criteria for early diagnosis: (1) symptoms of ileus that slowly appear and increase, such as discomfort in the abdomen or frank abdominal pain; (2) a requirement for catecholamine administration; (3) an episode of hypotension; and (4) slow elevation of the transaminase level (including Lactate DeHydrogenase). If three of these four criteria were present among patients after cardiovascular surgery, or on renal replacement therapy, an early diagnosis was attempted with MDCT. Treatment was given with intravenous high-dose prostaglandin E_1. After implementing this algorithm, only one of nine patients has died.

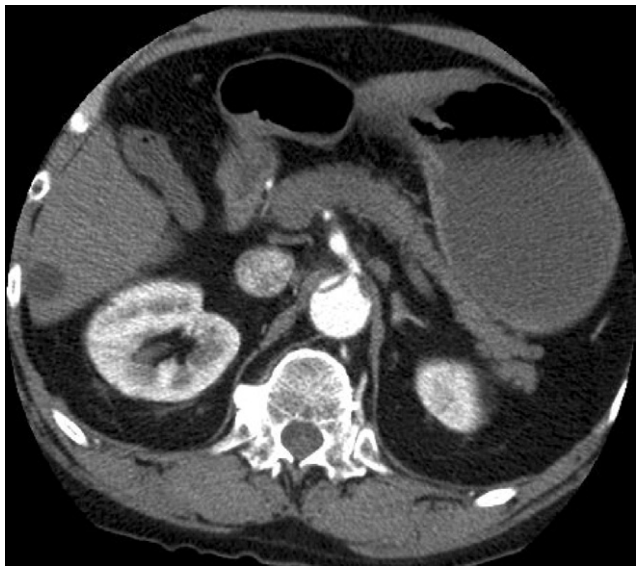

Figure 9 This computed tomography image shows how the true lumen is compressed by the pseudo lumen in a patient with acute Stanford type B dissection. The patient developed severe intestinal ischemia and reperfusion syndrome after stent grafting of the entry. This resulted in an abdominal compartment syndrome, the treatment of which is described in Figure 4.

Multiple drugs have been used to treat the condition and the alternatives were summarized in a recent review.[48] Traditional therapy consists of intraarterial administration of vasodilators, such as papaverin, nitroglycerin, or glucagon. Because of the loss of mucosal integrity, most authors suggest antibiotic treatment and at least animal experiments support that regime. As could be expected, no controlled studies comparing different pharmacological agents exist.

Replacement of angiography with MDCT and changing drug therapy from intraarterial to intravenous administration, as suggested by Mitsuyoshi et al,[47] represents a potential breakthrough in treatment of NOMI. It should be remembered, however, that the frequency of arterial stenosis is high among these patients,[44] which may be difficult to assess with MDCT if the aorta is calcified.

Aortic Dissection with Mesenteric Hypoperfusion

Finally, it should be mentioned that both Stanford type A and type B dissections are sometimes complicated by mesenteric hypoperfusion (Fig 9). This is a specific entity that is not normally considered part of NOMI. If stent grafting of the tear (or entry) does not solve the problem, the mesenteric

arteries may also need to be stented, sometimes followed by a laparotomy. We have experienced ACS secondary to reperfusion injury without previous laparotomy in this clinical scenario. Because of a lack of space, this review does not cover this important and growing patient category.

Summary

Mesenteric hypoperfusion syndromes represent a group of serious conditions from which the patient's life is almost always at stake. We tend to see different aspects of the NOMI pattern, depending on our medical specialty. One of the keys to success in treating these difficult and challenging patients is using a multidisciplinary approach. The cardiologist or intensivist might not consider ACS or an arterial stenosis, the radiologist might not consider intravenous therapy, and the vascular surgeon might not consider drug therapy.

Conflict of Interest Disclosure

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