CORRESPONDENCE

Acute Mesenteric Ischemia: a Vascular Emergency

by Prof. Dr. med. Ernst Klar, PD Dr. med. Parwis B. Rahmanian, Prof. Dr. med. Arno Bücker, Prof. Dr. med. Karlheinz Hauenstein, Prof. Dr. med. Dr. h.c. Karl-Walter Jauch and Prof. Dr. med. Dr. phil. Bernd Luther in volume 14/2012

Fatal Outcome

The authors pointed out the necessary diagnostic evaluation using biphasic contrast-enhanced computed tomography. However, nearly all patients with acute mesenteric ischemia have multiple comorbidities and vascular damage, and have correspondingly poor renal function. For this reason, radiologists often argue that contrast-enhanced CT cannot be undertaken.

This ignores the fact that the alternative to further renal damage caused by the contrast medium is death.

CT before exploratory laparotomy will therefore be possible in very few cases only.

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The author declares that no conflict of interest exists.

Differential Diagnostic Aspects Are Lacking

Acute abdomen requires emergency treatment, including emergency diagnostic evaluation. In the introduction of their review article, the authors say that "While [...] around 1% of all patients with acute abdomen have AMI [acute mesenteric ischemia], AMI is the cause of acute abdomen in up to 10% of patients aged over 70", without explaining differential diagnostic aspects.

Intestinal angioedema with massive mucosal swelling presents with similar symptoms; this seems to be neither more common nor rarer than AMI.

Intestinal angioedema can have allergic triggers but can also be caused by taking angiotensin converting enzyme inhibitors and angiotensin II receptor blockers (many cases have been documented in the literature) or be provoked by non-steroidal anti-inflammatory drugs. An individual case of intestinal angioedema has been described in a patient after taking a calcium antagonist (1).

Hereditary angioedema (HAE, a defect in C1-INH synthesis), angioedema with normal concentrations of C1-esterase-inhibitor but functional insufficiency, and angioedema based on acquired C1-esterase-inhibitor deficiency (acquired angioedema, AA) as well as idiopathic angioedema should also be included in the differential diagnostic evaluation. While a patient's medical history can provide an early indication of angioedema due to medication, diet, or hereditary causes and thus prevent unnecessary laparotomy, hereditary angioedema and other forms need to be confirmed using specific laboratory-based examination.

In case of clinical suspicion, this should include immunological determination of C1-INH, functional determination of C1-INH, measurement of C1, C2, C4, CH50, and, if required, measurement of autoantibodies against C1-INH (2).

Adding intestinal angioedema as a differential diagnosis to the well-known range of causes for acute abdomen might be worth mentioning, in order to clearly differentiate from vascular-ischemic causes.

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Digitalis Medication as the Cause of NOMI

I have read the article on the vascular emergency that is acute mesenteric ischemia with interest. Regarding the pathophysiology especially of nonocclusive mesenteric ischemia (NOMI), the authors focus on two scenarios: firstly, chronic hemodialysis, and secondly, clinical status after heart surgery. It is important to mention in this context that especially in older patients, overdosage of digitalis medication can be the cause of nonocclusive mesenteric ischemia, which is rare but none the less worth considering in the differential diagnostic considerations (1). DOI: 10.3238/arztebl.2012.0709c

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A New Approach to Early Diagnosis?

A comprehensive overview of acute mesenteric ischemia was long overdue, because awareness of this complex disease entity, which clinicians often think about too late, especially in the intensive care setting, urgently needs improving (1). The authors mention the problems in capturing symptoms in critically ill patients in intensive care: analgesic sedation, mechanical ventilation, volume replacement, and vasopressor therapy rarely allow for targeted diagnostic evaluation. On the other hand increasingly ageing and comorbid patients would lead us to expect a higher estimated number of unknown cases of mesenteric underperfusion—but exact data are currently lacking.

The authors emphasize that urgent imaging (contrast-enhanced computed tomography/angiography) is the diagnostic method of choice. For ventilated, critically ill patients in intensive care, this requires huge efforts with an inherent (transport) risk. Furthermore, the incidence of contrast-induced renal failure with subsequent need for renal substitution treatment is some 16%, associated with longer-term intensive care and hospital treatment and higher mortality (2).

Contrast-enhanced ultrasound might offer an innovative diagnostic approach: injecting a contrast medium that is free from side effects (phospholipid coated, sulphur hexafluoride gas containing microbubbles as reflectors for ultrasound waves) increases the imaging resolution of vessels many times. Encouraging reports are available for the reliable diagnosis of complex vascular structures, for example, after surgery for abdominal aortic aneurysm (3), and a convincing prospective evaluation has been undertaken for the early detection of intestinal ischemia (4). Our own positive experiences have convinced us that contrast-enhanced ultrasound could replace "traditional" imaging—which is expensive and takes time, while also having a higher side effect profile—at least in some cases.

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In Reply:

K H Haegler raises the important issue of additional, contrast medium-related, renal damage. If a patient meets the risk profile for mesenteric ischemia, rapid contrast-enhanced CT is the top priority. Existing renal damage is often subsequent to dehydration and should be treated in parallel by using infusion therapy. In patients with known compensated renal failure, possible additional damage owing to contrast medium with a deterioration in renal function should be accepted. K H Haegler weights the problem correctly: if in doubt, choose contrast-enhanced CT, even if there is a risk for the renal failure to worsen.

I thank K Lehman for her comments on intestinal angioedema as a differential diagnosis to acute mesenteric ischemia. Our article aims at providing a very clear description of mesenteric ischemia; the objective was to reduce the persistent high mortality by rapid exclusion and speedy detection of mesenteric vascular occlusion. If the patient fits the profile and clinical presentation of possible mesenteric ischemia, then triphasic, contrast-enhanced CT should be undertaken without delay. If this shows that the vessels are clear, the remaining differential diagnoses can be narrowed down without putting the patient at risk. Intestinal angioedema is one such differential diagnosis.

R Sen Gupta points out that overdosage of digitalis medication is of particular etiological relevance as a cause of nonocclusive mesenteric ischemia. We agree. In the overview "Clinical manifestation, risk factors, and classification of acute mesenteric ischemia" *(Box)* we included digitalis medication among the risks. Overdosage is not easily confirmed in the emergency setting. For us it was important to emphasize that every patient taking digitalis medication should be assessed precisely with regard to NOMI.

T Bein and colleagues in their letter focus on contrast-enhanced sonography as an innovative diagnostic approach for detecting mesenteric ischemia. The publication by Hamada et al cited in the letter (Br J Radiol 2007) has prospectively evaluated this approach. The study included 50 patients who were admitted to hospital with a diagnosis of ileus. Contrast-enhanced sonography aims to detect flow signals in the distended intestinal wall. Where intestinal wall perfusion was not confirmed, the researchers concluded that ischemia was present. The sensitivity of this approach is 94.1%. Visualizing the large mesenteric arteries and veins was not the study's objective. By contrast, the main objective of our article was to shorten the time to the eventual diagnosis of mesenteric ischemia. The triphasic contrast-enhanced CT or angiography that we recommended have the advantage of very detailed imaging of the large mesenteric vessels, together with the anatomical structure of the perfusion problem, in order to set out an optimal treatment concept consisting of radiological intervention, lysis, or surgery. By comparison, contrast-enhanced ultrasonography provides insufficient information compared with the therapeutic algorithm we presented in our article. The only way to lower the persistently high mortality of mesenteric ischemia is by shortening the time from initial manifestation to therapy. Contrast-enhanced sonography entails an extension of the time taken to diagnosis. In future, we will be successful in treating mesenteric ischemia only if we make the diagnosis in the shortest possible amount of time, by using a single, easily available diagnostic tool that enables comprehensive interpretation. Each further investigation that precedes contrast-enhanced CT or angiography is a step in the wrong direction. We dealt with the problem of contrast medium in our response to the first reader's letter.

In conclusion I thank the correspondence authors for dealing with our publication in such detail. This has helped clarify further important aspects of the symptoms.

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