scrutiny. Those based on low-certainty evidence are the least supported by current data and the most likely to be subject to change in the future. The committee also made some statements that were labeled as *ungraded good practice statements*.[15] These statements did not have direct supporting evidence, but had ample indirect evidence and would be considered by many surgeons as surgical principles.

Diagnostic evaluation

Recommendations.

1. In patients with abdominal pain, weight loss, and food fear, we recommend an expedited workup to exclude gastrointestinal malignancies and other potential causes. The expedited workup may include an esophagogastroduodenoscopy, a colonoscopy, an abdominal CT scan, and an abdominal ultrasound examination. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)
2. We recommend making a diagnosis of CMI in patients with the appropriate clinical scenario and the presence of significant stenoses (>70%) within the celiac axis and SMA. The diagnosis maybe also made in patients with the appropriate clinical scenario and a significant stenosis (>70%) in either the celiac axis or SMA alone. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)
3. We recommend using the mesenteric duplex ultrasound (DUS) examination as the preferred screening test for MAOD. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate)
4. We recommend using CTA as the preferred definitive imaging test for MAOD unless unusual anatomic features obscure the anatomy such that a catheter-based arteriogram may be required. Level of recommendation: Grade 1 (Strong), Quality of Evidence: B (Moderate) (*1B*)

Rationale and background

CMI is caused by the failure to achieve adequate postprandial intestinal blood flow. This condition usually results from the presence of atherosclerotic occlusive disease at the origin of the mesenteric arteries and is associated with the typical atherosclerotic risk factors. There is a tremendous amount of redundancy in the mesenteric circulation, and, accordingly, the symptoms of CMI do not typically develop unless both the CA and SMA have hemodynamically significant lesions (>70%). However, it is possible to have symptoms consistent with CMI and disease isolated to a single mesenteric vessel, typically the SMA. MAOD, in contradistinction to CMI, is relatively common and affects a large percentage of patients with peripheral vascular and aneurysmal disease. Patients with CMI typically present with postprandial abdominal pain, weight loss, and food fear. The differential diagnosis for this clinical presentation is quite extensive and includes gastrointestinal malignancies first and foremost. Accordingly, the diagnostic workup should exclude other gastrointestinal causes and potentially include an esophagogastroduodenoscopy, colonoscopy, abdominal ultrasound examination, and abdominal CT scan. The diagnosis of CMI requires the appropriate clinical presentation and the presence of significant MAOD. Neither physical examination nor routine laboratory studies are particularly helpful and, unfortunately, there is no well-accepted functional test that is sufficiently sensitive or specific. Mesenteric DUS examination is an excellent screening test for MAOD, and CTA is the definitive imaging test and has largely replaced catheter-based arteriography.

Detailed justification

The blood flow to the gastrointestinal tract is provided via the CA, SMA, and IMA with collateral contributions via the internal iliac artery, most notably the hemorrhoidal arteries. The gastroduodenal and pancreaticoduodenal arteries provide connections between the CA and SMA. The marginal artery of Drummond and the arc of Riolan (meandering or central anastomotic artery lying in the mesentery close to the inferior mesenteric vein) connect the inferior mesenteric (via the left colic artery) to the superior mesenteric (via the middle colic artery).[16] The disruption of these collateral networks, for whatever reason (eg, IMA ligation during exposure of the infrarenal aorta), can lead to AMI and CMI in patients with MAOD. Because of this well-developed network of collaterals, most patients usually do not develop symptoms of CMI or AMI unless there is a significant stenosis or occlusion in at least two of the mesenteric vessels (ie, CA, SMA, and/or IMA). Notably, Oderich et al[6] described the arteriographic findings of more than 200 patients with CMI and reported that 98% had significant occlusive disease in two of the three mesenteric vessels and 92% had an occlusion or critical stenosis in the SMA. In a longitudinal study of asymptomatic patients with mesenteric ischemia, only patients with significant occlusive disease in all three mesenteric vessels eventually developed symptoms.[17] However, significant occlusive disease in a single mesenteric vessel, usually the SMA, can lead to CMI if the collateral network is inadequate.[4-6] Unfortunately, there is not a direct association between the number of mesenteric vessels involved and the presence of symptoms, thereby confounding the diagnosis and underscoring the difference between MAOD and CMI.

During the fasting state, 20% of the cardiac output courses through the mesenteric arteries.[18] The gastrointestinal blood flow increases after a meal, achieving levels that exceed the fasting values by 100% to 150% over the ensuing 3 to 6 hours.[19] This hyperemic response starts with the anticipation of the meal, but the major hemodynamic effects become most evident after ingestion and movement of the food bolus into the small bowel. The vasodilation of the mesenteric vessels begins 3 to 5 minutes after ingestion and persists for 4 to 6 hours depending on the meal composition with the maximal