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Commentary: Early malperfusion syndrome—a new concept

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CENTRAL MESSAGE

ATAAD patients with early MPS could be managed differently than those with late MPS.

More and more cardiac surgeons are realizing the gravity of visceral malperfusion in acute type A aortic dissection (ATAAD) and choose to treat the visceral malperfusion first when a patient has already developed malperfusion syndrome (MPS).[1-3] The difference between malperfusion and MPS is similar to the difference between bacteremia and sepsis, or between HIV and AIDS. End-organ malperfusion is inadequate blood flow to the end organs, which can be diagnosed by clinical exam (peripheral pulses) and computed tomography angiography. MPS is the consequence of prolonged malperfusion, including cell, tissue, and organ necrosis, dysfunction, and failure, which can be clinically diagnosed based on symptoms (eg, abdominal pain), physical exam (eg, peritoneal signs), and laboratory tests (eg, serum lactate, arterial blood gases). Just like sepsis, MPS is a process of development of end-organ damage. In the early stage, because of ischemia, patients start to have cell death, tissue death, and organ dysfunction, but no organ death and failure. For example, patients with superior mesenteric artery (SMA) malperfusion can have abdominal pain, mildly elevated lactate, and mild metabolic acidosis but no bloody stools, peritoneal signs, or transmural necrosis of intestine, as in the case described by Ni and colleagues.[2] We can define this stage of malperfusion syndrome as early-stage MPS, which also includes mildly increased liver enzymes, bilirubin, creatinine, and myoglobin in early MPS due to celiac artery, renal artery, and extremity malperfusion. If the malperfusion is not resolved, patients will continue to develop extensive organ necrosis and failure, such as bloody stools, peritoneal signs, severe metabolic acidosis and shock, and multiorgan failure, as in the case described by Preventza and colleagues[1] In those patients, we frequently find dead bowel at laparotomy. We can define this stage as late MPS. In our previous studies, a majority of MPS cases are late MPS.[4,5]

For patients with late visceral MPS from either dynamic or static malperfusion, we strongly recommend first resolving the malperfusion endovascularly with fenestration/stenting[4,5] or TEVAR with aortic stent graft and, as indicated, branch artery stents,[1,3] then performing delayed open aortic repair after the patients recover from multiorgan failure, acidosis, and acute respiratory distress syndrome and can tolerate cardiopulmonary bypass and hypothermic circulatory arrest.[1,3-5]

The management of early visceral MPS can be guided based on the mechanism of malperfusion: dynamic versus static, keeping in mind that in many cases it is mixed dynamic and static. Dynamic malperfusion is caused by the aortic dissection flap covering and occluding the orifice of the aortic branch vessels, such as the SMA. The occlusion of branch vessels is affected by the blood pressure (BP) dynamically, with worse occlusion with high BP, and often (but not invariably) less or no occlusion with low BP, based on our observations with intra-aortic ultrasound. Therefore, it is very important to control the BP not only to prevent aortic rupture, but also to minimize end-organ malperfusion. The dynamic malperfusion can be solved by open aortic repair to resect the primary intimal tear and direct the blood flow into the true lumen, endovascular fenestration/stenting, or TEVAR. For patients with early MPS due to dynamic malperfusion, it is reasonable to perform open aortic repair to resolve the malperfusion and prevent rupture

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Disclosures: The author reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

Received for publication Dec 13, 2020; revisions received Dec 13, 2020; accepted for publication Dec 21, 2020; available ahead of print Dec 25, 2020.

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JTCVS Techniques 2021;7:25-6
2666-2507

https://doi.org/10.1016/j.xjtc.2020.12.018

of the ascending aorta, especially in patients with coronary malperfusion or cerebral malperfusion, which demands immediate open repair to restore blood flow to the heart and brain. Because patients are still in the early stage of visceral or extremity MPS, they most likely can recover from surgery. Static malperfusion is a constant occlusion of a branch frequently resulting from the dissection and thrombosis of the aortic branch vessels, which requires stenting or vascular bypass to restore blood flow, as described by Ni and colleagues. Open central aortic repair or TEVAR alone does not resolve static malperfusion. Therefore, for patients with early MPS from static malperfusion, it is reasonable to resolve the malperfusion first before open aortic repair; otherwise, the end organ has continuous malperfusion throughout the open operation, and the patient can develop late MPS and multiorgan failure. Ni and colleagues stented the SMA and renal artery first and performed open aortic repair in one stage with good outcomes.

The diagnosis of early and late visceral MPS is critical for management. When an aortic branch vessel, such as the SMA, is occluded, there is minimal blood flow through the mesentery, and lactate and toxic metabolites can be trapped in the tissue and not flushed into the circulatory system. No blood in, no blood out. In such cases, the serum lactate or acidosis would be deceivingly low. In those situations, other clinical evidence could be helpful in diagnosing patients in the early or late stage of MPS, such as the time since onset of abdominal pain, record of serial BP measurements (hypertension results in more severe dynamic malperfusion), severity of pain, bloody stools, and peritoneal signs.

In summary, for patients with late visceral MPS, we recommend endovascular resolution of malperfusion and delayed open aortic repair. For patients with early visceral MPS from dynamic malperfusion, immediate open aortic repair is reasonable. For patients with early visceral MPS from static malperfusion, endovascular resolution of malperfusion, followed by immediate or delayed open aortic repair, is recommended.

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