No guts, no glory: Visceral ischemia and acute type B aortic dissections

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Visceral ischemia is a catastrophic complication that afflicts 7% of patients with acute type B aortic dissection and is associated with a 9-fold increased risk of in-hospital mortality.^{1,2} Jonker and colleagues³ report an analysis of patients with acute type B aortic dissection and visceral ischemia, along with visceral ischemia, a challenging scenario that is difficult to diagnose early and is often fatal without intervention. The authors observed that the rates of surgery for the management of visceral ischemia have declined over time, from 46.5% in the early era (1996-2001) to 16.1% in the most recent era (2008-2012), whereas rates of endovascular intervention increased from 34.9% to 67.7% during the same time interval. Furthermore, mortality rates were similar after surgical and endovascular management of visceral ischemia (25.8% and 25.5%, P not significant), and medical management was the only independent predictor of mortality in multivariate analysis (odds ratio 5.91, 95% confidence interval 1.2-31.0, $P = .036$).

The generalizability of this study is its greatest strength. Rather than focusing on a single-center experience with a predilection for certain therapies, the authors report data from the International Registry of Aortic Dissection, a multicenter registry that has been enrolling patients with acute aortic dissection since 1996. By including patients regardless of their clinical status or treatment strategy, the data in this registry offer insight into the “real-world” experience of patients with acute type B aortic dissection. This analysis includes the largest unselected consecutive patient cohort who have this condition and visceral ischemia, and is therefore of great value to the reader.

Visceral ischemia can be difficult to diagnose, as many of the signs appear late in the process and may be obscured in a patient who is sedated and intubated. The diagnosis is often made by a combination of clinical signs (a distended and

tender abdomen, biochemical evidence of liver and pancreatic insufficiency, lactic acidosis) and radiographic findings (severe compression of the true lumen across the visceral segment, compromised flow to the celiac axis and/or superior mesenteric artery, ischemia of visceral organs). Diagnostic laparoscopy or laparotomy may confirm decreased viability or necrosis of the gut, although this is often a late finding.

The observation in this report that preoperative limb ischemia (28% vs 7%, $P < .001$) and acute renal failure (41% vs 14%, $P < .001$) were more common among patients with visceral ischemia is an important one.³ Clinicians should have a high index of suspicion that patients with limb or renal malperfusion may have visceral malperfusion in addition. The identification of a sensitive and specific early diagnostic marker of visceral ischemia would be very useful clinically, as early intervention is paramount for a successful outcome.

The prognostic importance of visceral malperfusion is primarily determined by the severity and duration of ischemia. A patient with mild transient organ ischemia will fare better than one with a profound lactic acidosis secondary to infarcted bowel. The lack of details regarding the severity and duration of visceral ischemia in this cohort is a limitation of this study. Furthermore, the mechanism of malperfusion may have important implications for treatment and was unavailable for reporting in this article. Two mechanisms have been identified as the primary causes of organ ischemia after aortic dissection: (1) dynamic obstruction secondary to a collapsed true lumen (Figure 1); and (2) static obstruction with branch vessel obstruction secondary to extension of the dissection/thrombus into visceral arteries or branch avulsion.^{4,5}

Treatment of visceral malperfusion has evolved from open aortic fenestration, first performed in 1935⁶ and now used only infrequently, to an endovascular-first approach. Dynamic obstruction may be relieved percutaneously by: (1) proximal stent placement to increase flow in the true lumen; or (2) fenestration to equalize pressures across the septum. Static obstruction likely needs more focal treatment of the malperfused branch by peripheral stenting. Intravascular ultrasound may be particularly helpful in determining the relative sizes of the lumina, location and size of spontaneous fenestrations, characteristics of the mobile flap, as well as the diameter of the major branch arteries and their relationship to any communications. A better understanding of the physiology and imaging of static and dynamic branch vessel obstruction will be important in directing the treatment

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FIGURE 1. Acute type B dissection with visceral ischemia. Dynamic obstruction of mesenteric arteries by a compressed true lumen with the flap prolapsing into the celiac and superior mesenteric arteries. The patient had an acute type B dissection complicated by severe visceral ischemia. *SMA*, Superior mesenteric artery.

of patients with ischemic complications of acute aortic dissection.

Jonker and colleagues³ answer several questions and prompt us to ask others: What was the severity and temporal course of the visceral malperfusion in this cohort? What was the mechanism of branch vessel compromise? Was bowel resection necessary? Which patients experienced radiologic and/or clinical resolution of the malperfusion? What surgical techniques and adjuncts led to successful patient outcomes? This important paper sets the framework for future investigations regarding the etiology and treatment of patients with acute type B aortic dissection and visceral malperfusion.

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