

REPLY: THE ART OF WINNING AN UNFAIR GAME: IMMEDIATE AORTIC REPAIR FOR MESENTERIC



MALPERFUSION SYNDROME Reply to the Editor:

We read with interest the letter¹ discussing our analysis of acute type A aortic dissection (ATAAD) complicated by malperfusion syndrome (MPS).² In that article, we report outcomes of immediate aortic repair for all-comers with ATAAD, regardless of MPS status. By contrast, Dr Ak¹ argues that patients with mesenteric MPS ought to receive a tailored interventional strategy based on the preoperative severity of mesenteric MPS. First, he cites literature suggesting that patients with mesenteric MPS who undergo immediate aortic repair (our preferred strategy³) have early mortality as high as 80%. 4 More importantly, he suggests that the severity of mesenteric MPS dictates clinical outcome (ie, early mortality), and it therefore ought to dictate the appropriate interventional strategy. Specifically, patients with severe mesenteric MPS may benefit from endovascular mesenteric revascularization before open aortic repair. He argues that profound metabolic derangements from mesenteric MPS might make separation from cardiopulmonary bypass impossible, whereas immediate mesenteric revascularization may reverse those metabolic derangements thereby improving the outcome of a delayed aortic repair.

Although this is a plausible argument, we believe the situation is far from clear. First, in our cohort, only 4 of 13 patients (30%) with mesenteric MPS did not survive to discharge, which is significantly lower than the 80% early mortality for mesenteric MPS that he cites. Indeed, other literature suggests that rates of early mortality may be as low as 41% for patients with mesenteric MPS undergoing immediate aortic repair.⁵ Yet, whatever the percentage, we certainly agree that mesenteric MPS is an extremely challenging situation that is likely associated with the highest mortality after ATAAD. Secondly, of the 4 early mortalities from mesenteric MPS in our study, only 1 patient fits his description of severe mesenteric MPS—bloody diarrhea with profound metabolic acidosis. The other 3 patients with early mortality had mild acidosis (pH > 7.3) and mild lactatemia (<4 mmol/L) at presentation, and only 2 of these 4 patients had bloody bowel movements, whereas the other 2 only had nonperitonitic abdominal pain. Conversely, 4 of the 9 patients with mesenteric MPS who survived to discharge had a severe presentation with significant lactic acidosis and bloody diarrhea, whereas the other 5 patients

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who survived to discharge had relatively normal preoperative laboratory values in the setting of abdominal pain and nonbloody bowel movements. By implication, preoperative stratification of mesenteric MPS by severity did not necessarily correlate with clinical outcome in our study, which has also been observed in prior studies. Admittedly, the small sample size and retrospective nature of our data makes these observations anecdotal. However, we do not agree that the challenge of managing mesenteric MPS implies that an endovascular approach for patients with severe mesenteric MPS is warranted.

In short, the existing data are too limited and unclear to defend a paradigm of utilizing endovascular mesenteric revascularization versus immediate open aortic repair based on severity of mesenteric MPS at presentation. Although we agree that mesenteric MPS in the setting of ATAAD is challenging to manage, especially with concomitant renal or iliofemoral MPS, we await higher-quality data to adjudicate this important issue. In the interim, these authors prefer to play the game by the existing rules; that is, to restore true lumen perfusion as soon as possible.

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Conflict of Interest Statement

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