

Predictors of septic shock following gastrointestinal anastomotic leaks: Only signposts, no destination

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Anastomotic leaks are associated with a high risk of morbidity and even mortality.^[1] Owing to this, surgeons have made considerable efforts to determine the clinico-biochemical factors that may help predict the development of anastomotic leaks or at least signal their development prior to their clinical manifestation (which usually precedes the development of septic shock if rapid action is not taken). Such parameters have been based on the markers of systemic inflammatory response syndrome (SIRS). The reason for making these efforts is the appreciation of the potential for rapid downward spiral (toward septic shock) once the anastomotic leak manifests clinically, and to thereby permit early detection which will aid the institution of effective measures, viz. “source control,” along with supportive measures such as fluid resuscitation and antimicrobials. The problem with such studies has been that surgery is in itself an insult that may be accompanied by SIRS, and hence, the factors studied tend to lack sensitivity.^[2]

In this issue of the journal, Choudhuri and Uppal^[3] present an audit from the Intensive Care Unit (ICU) of a tertiary referral care center on the predictors of septic shock following anastomotic leaks as sequelae of major gastrointestinal (GI) surgery. The study included 103 patients with anastomotic leaks following major GI surgery who were transferred to the ICU for resuscitation and intensive management. On comparing the 72 patients who developed septic shock with those who did not, the authors noted that the former had a higher Acute Physiology and Chronic Health Evaluation II (APACHE II) score, a lower

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mean arterial pressure (MAP), and tachycardia at the time of admission to ICU. The authors noted that the patients who developed septic shock received more packed red cell transfusions. Septic shock was more frequently encountered after pancreaticojejunostomy (PJ) and hepaticojejunostomy (HJ) leaks, with factors such as malignancy, chronic obstructive pulmonary disease (COPD), the need for packed red cell transfusions, bacteremia, and the leak from HJ and PJ being the independent predictors of mortality and length of ICU stay. The study has re-validated the importance of the APACHE II scoring system for predicting septic shock following anastomotic leaks.

This study included 103 patients managed over nearly 2½ years, certainly a very large number. At the outset, the biggest shortcoming of this study is the lack of information on the surgical management of these patients once the leak was detected. It is unclear as to at what time following the clinical detection of the leak were the patients transferred to the ICU and whether surgical or interventional radiologically guided therapy had been initiated or not. This information achieves significance as the failure to achieve source control will certainly alter the importance of all the other contributory factors. From the authors’ observations (low MAP, tachycardia, greater APACHE II score, and a higher base deficit at the time of ICU admission), it appears that the patients

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were shifted to the ICU after the development of septic shock.

Some negative observations on the data presented may have been because of the significant differences in the frequencies of oesophago-jejunal rather than PJ anastomotic leak between the two groups (septic shock vs. no septic shock). The authors briefly suggested a scoring system entitled SEPAL in Table 4, but never really clarified what this was and also its potential application. Another interesting observation is the higher incidence of gastrojejeunal leaks in the non-septic group. Gastrojejeunal leaks are notoriously associated with poor outcomes. This really makes the reader wonder why this is so and begs the need for more information on source control/early surgical salvage.

Addressing the individual factors detected by the authors to be associated with the risk of developing septic shock we would like to make some observations. Rather than relating the delay in diagnosis of bile duct injuries and resultant leaks to an increased propensity to develop septic shock, it would have been really useful to know how the leaks were detected and managed in the first place (surgical/conservative/interventional radiology). The relationship between malignancy associated leaks and septic shock was an interesting observation. Two factors that may be linked to malignancy and the increased risk of surgical complications have been the nutritional status and whether the surgery was performed in the emergency setting or as an elective procedure. This would be something interesting to look into. The authors have rightly pointed out the need for information on intraoperative blood loss in order to critically determine

the importance of blood transfusion as a risk factor for septic shock. No comments on the presence of pulmonary complications leading to an increased risk for septic shock following anastomotic leak. Besides, there is no mention of how many blood cultures were taken from each patient. Given the variable yield of blood cultures, in general, such information would be of great importance when interpreting the role of bacteremia in predicting septic shock.

In the end of the study, the authors have made a couple of interesting observations that need to be better studied. However, is this the first study to address the predictors of septic shock following anastomotic leak? Well it certainly is not the first and will certainly not be the last in the great struggle of surgeons and intensivists in the search for predictors of developing septic shock in patients with anastomotic leaks after major GI surgeries. The need for a more balanced, scientific approach as a joint effort between surgeons and intensivists would seem a more prudent way to approach the problem.

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