Predictors of septic shock following anastomotic leak after major gastrointestinal surgery: An audit from a tertiary care institute

Anirban Hom Choudhuri, Rajeev Uppal

Background: Anastomotic leak is a serious complication after major gastrointestinal surgery and majority of deaths occur due to septic shock. Therefore, the early identification of risk factors of septic shock may help reduce the adverse outcomes. Objective: The aim of this audit was to determine the predictors of septic shock in patients with anastomotic leak after major gastrointestinal surgery. Design: Retrospective, audit. Materials and Methods: The patients admitted in the gastrosurgical intensive care unit (ICU) of our institute between September 2009 and April 2012 with anastomotic leakage after surgery were identified. The ICU charts were retrieved from the database to identify the patients progressing to septic shock. A comparison of risk factors was made between the patients who developed septic shock (septic shock group) against the patients who did not (non-septic shock group). Results: The study sample comprised of 103 patients with anastomotic leak, of which 72 patients developed septic shock. The septic shock group had a higher APACHE II score, lower MAP, and higher HR at the time of ICU admission. They received greater transfusion of packed red blood cells during their ICU stay. Septic shock was more common after pancreaticojejunostomy and hepaticojejunostomy leaks. Conclusion: Presence of malignancy, chronic obstructive pulmonary disease (COPD), packed red blood cell transfusion, bacteremia, and hepaticojejunostomy or pancreaticojejunostomy leaks were independent predictors of mortality and length of ICU stay. To the best of our knowledge there are no available studies in the literature on the predictors of risk factors of septic shock in patients with anastomotic leakage.

Keywords: Anastomotic leakage, post-leak sepsis, septic shock

Introduction

Sepsis is systemic inflammatory response syndrome (SIRS) secondary to infection, and, when associated with organ dysfunction, produces several life-threatening complications. While the occurrence of sepsis after major surgery is uncommon in healthy patients, the risk increases after emergency surgery, greater surgical insult, blood transfusions, advancing age, and male gender.[1-4] The extent to which the co-morbid illness increases the risk of sepsis after surgery has been studied in great detail, and many scoring systems like Charlson co-morbidity score (CCS) have been derived to measure its burden.[5]

Anastomotic leak is a serious complication after major gastrointestinal surgery that considerably increases the mortality and morbidity.[6] A large number of patient-related and surgery-related risk factors are known to influence anastomotic leak, but consensus is lacking on their independent role due to their mutual interdependency.[7] However, the majority of deaths occur after anastomotic leak due to overwhelming sepsis.[8]
Since the progression from anastomotic leak to severe sepsis and septic shock is often rapid and fatal, the early identification of risk factors for this progression may help reduce the mortality and morbidity. The aim of this retrospective audit was to determine the predictors for developing septic shock in patients with anastomotic leak after major gastrointestinal surgery.

Materials and Methods

All the patients admitted in the gastrosurgical intensive care unit of our hospital between September 2009 and April 2012 with anastomotic leak after major gastrointestinal surgery were identified. The ICU charts of the patients were retrieved from the database and an audit was performed to identify the patients who progressed to septic shock. A comparison of the risk factors was made between the patients who developed septic shock against the patients who did not. The risk factors were primarily determined by literature search and collected from the information available in the ICU charts.

“Anastomotic leak” was defined as the leak of luminal contents from a surgical joint between two hollow viscera and was diagnosed on the basis of clinical suspicion and/or radiological investigation.

“Sepsis” was defined as SIRS such as fever, tachycardia, tachypnoea, or leucocytosis in response to a culture-proven or clinically suspected infection.

“Severe sepsis” was defined as sepsis with at least one additional sign of organ hypo perfusion or dysfunction such as cardiac dysfunction, acute lung injury, or altered mental status.

“Septic shock” was defined as severe sepsis in addition to a systemic mean blood pressure < 60 mmHg (or < 80 mmHg if previous hypertension) after an attempt at adequate fluid resuscitation or a need of systemic vasopressors to maintain a mean blood pressure > 60 mmHg (or > 80 mmHg if previously hypertensive).

“Bacteremia” was defined as a positive blood culture, excluding isolates believed to be contaminants.

Categorical data are presented as numbers (%). Quantitative data are presented as mean (SD). Statistical analyses were performed using SPSS software (version 12.0; SPSS Inc., Chicago, IL). Univariate analysis was conducted to determine the potential risk factors for the occurrence of septic shock. Chi-square tests or Fishers’ exact tests were used for qualitative variables. The required significant level was set at a $P < 0.05$. A multivariate analysis was performed by stepwise logistic regression with sepsis as dependent variable to identify independent risk factors for the development of septic shock. The variables that were analyzed as potential predictors were selected from the literature and from the clinical experience of the researchers. The variables consisted of patient characteristics (age, sex, and smoking habit), type of the leak (esophagogastric, colorectal, and pancreaticojejunal), and severity of condition (APACHE II score, blood transfusion needs).

Results

The study sample comprised of 103 patients with anastomotic leak in the aforesaid period. Sepsis occurred in 90 patients and septic shock in 72 patients. The details of the demographic and clinical characteristics of the septic shock and non-septic shock groups are presented in Table 1. As would be expected, compared with the non-septic shock group, the septic shock group had a significantly greater derangement of physiological state at the time of ICU admission. This was reflected by a higher APACHE II score (19 ± 6 vs. 9 ± 4; $P < 0.05$), lower mean arterial pressure (45 ± 4.2 vs. 70 ± 6.2; $P < 0.05$), higher heart rate (130 ± 9 vs. 72 ± 8; $P < 0.05$), and a greater base deficit (~4.2 ± 5.1 vs. −2.6 ± 4.6; $P < 0.05$). Although the hemoglobin level at the time of ICU admission was similar (8.2 ± 1.4 vs. 9.3 ± 2.1; $P > 0.05$) between the groups, the septic shock group received more packed red blood cell (RBC) transfusions (47.3% vs. 27.9%; $P < 0.05$) during the ICU stay. The progression to septic shock occurred more after pancreaticojejunal (22% vs. 13%; $P < 0.05$) and hepaticojejunal anastomosis than jejunojejunal anastomosis (45% vs. 37%; $P < 0.05$). Bacteremia was more common in the septic shock group (87% vs. 34%; $P < 0.05$). Multiorgan dysfunction at the time of ICU admission was more common in the septic shock group (77% vs. 14.3%; $P < 0.05$).

Univariate analysis of septic shock and outcome

Analyses were conducted to evaluate the univariate associations between septic shock and common outcomes after anastomotic leak (i.e., in hospital mortality, 30-day mortality, ICU length of stay, and hospital length of stay). Chi-square analyses were performed to determine the association between septic shock and mortality. The septic shock group had a higher stay in hospital (17.3% versus 9.1%; $P < 0.05$) and 30-day mortality rates (15.1% vs. 8.7%; $P < 0.05$). Analysis of variance (ANOVA) was performed to evaluate the association between septic shock and ICU and hospital length of stay. It was seen that the septic shock group spent a significantly longer time in the ICU (18 ± 7 days vs. 10 ± 4 days; $P < 0.05$) and
hospital (35 ± 4 days vs. 19 ± 8 days; P < 0.05). The septic shock group also had a greater number of ventilator days in the ICU (11 ± 5 days vs. 4 ± 3 days; P < 0.05).

**Predictors of septic shock in anastomotic leak**

Logistic regression analysis identified malignancy, chronic obstructive pulmonary disease (COPD), liver disease, heart disease, packed RBC transfusion, presence of bacteremia, hepaticojejunostomy anastomotic leak, colorectal fistula, and APACHE II score > 15 as significant independent predictors of septic shock in patients with anastomotic leak [Table 2].

To evaluate the predictive value of septic shock and the combined effects for outcome, the significant univariate predictors for septic shock were entered into multiple logistic regression models. Results of these analyses are presented in Table 3. Analysis indicated that the interaction of septic shock with the variables of APACHE II > 15, bacteremia, malignancy, hepaticojejunostomy leak, and packed RBC transfusions were significant predictors of in-hospital mortality. The analysis also indicated that the interaction of septic shock with APACHE II > 15, bacteremia, malignancy, hepaticojejunostomy leak, and liver disease was a significant predictor of the length of ICU stay, while the interaction of septic shock with APACHE II > 15, bacteremia, and hepaticojejunostomy leak was a significant predictor of the length of hospital stay.

### Table 1: Comparison of septic shock and non-septic shock patients on demographic, clinical, and anastomotic leak measurements

|                        | Septic shock (n=72) | Non-septic shock (n=31) | P value |
|------------------------|---------------------|-------------------------|---------|
| Age (years; mean±SD)   | 45±4.5              | 42±2.6                  | >0.05   |
| Male (%)               | 36                  | 40                      | >0.05   |
| Smoking (%)            | 40                  | 39                      | >0.05   |
| Presence of co-morbidities (%) |        |                         |         |
| Malignancy             | 23                  | 8                       | <0.05   |
| Diabetes               | 34                  | 14                      | <0.05   |
| Heart disease          | 40                  | 36                      | >0.05   |
| COPD                   | 19                  | 4                       | <0.05   |
| Liver disease          | 11                  | 3                       | <0.05   |
| APACHE II on admission (points; mean±SD) | 19±6               | 9±4                     | <0.05   |
| APACHE II mean > 15 on admission (%) | 27                  | 14                      | >0.05   |
| MAP (mmHg) at admission (mean±SD) | 45±6.2             | 70±4.2                  | <0.05   |
| HR at admission (mean±SD) | 130±9               | 72±8                    | <0.05   |
| Hb (%; mean±SD)        | 8.2±1.4             | 9.3±2.1                 | >0.05   |
| Platelet (count; mean±SD) | 0.84±0.04         | 0.95±0.07               | >0.05   |
| Prothrombin time (Quick %; mean±SD) | 72±23              | 80±22                   | >0.05   |
| Base excess at admission (mmol/L; mean±SD) | −4.2±5.1          | −2.6±4.6                | <0.05   |
| pRBC transfusion (%)   | 47.3                | 27.9                    | <0.05   |
| Fresh frozen plasma (n; mean±SD) | 2.3±0.7            | 1.1±0.5                 | <0.05   |
| Type of leak (%)       |                     |                         |         |
| Oesophagojejunal       | 22                  | 13                      | <0.05   |
| Pancreatojejunal       | 21                  | 17                      | NS      |
| Hepaticojejunal        | 45                  | 37                      | <0.05   |
| Colorectal             | 9                   | 24                      | <0.05   |
| Gastrojejunal          | 3                   | 9                       | NS      |
| Bacteremia (%)         | 87                  | 34                      | <0.05   |
| Multi organ failure (%)| 77                  | 14.3                    | <0.05   |
| Ventilation days (days; mean±SD) | 11±5           | 4±3                     | <0.05   |
| ICU LOS (days; mean±SD) | 18±7                | 10±4                    | <0.05   |
| In hospital LOS (days; mean±SD) | 35±4              | 19±8                    | <0.05   |
| 30 day mortality (%)   | 15±1                | 8.7                     | <0.05   |
| In hospital mortality (%) | 17.3              | 9.1                     | <0.05   |

APACHE II=Acute physiology and chronic health evaluation score II; LOS=Length of stay; ICU LOS=Intensive care unit length of stay; pRBC=Packed red blood cell; Hb=Hemoglobin; HR=Heart rate; MAP=Mean arterial pressure; COPD=Chronic obstructive pulmonary disease; NS=Non-significant

### Table 2: Predictors of septic shock (Logistic regression analysis)

| Variable          | OR    | CI       | P value |
|-------------------|-------|----------|---------|
| Malignancy        | 2.41  | 5.23     | <0.05   |
| COPD              | 1.15  | 3.36     | <0.05   |
| Liver disease     | 3.59  | 11.61    | <0.05   |
| Heart disease     | 1.29  | 2.37     | <0.05   |
| pRBC transfusion  | 3.31  | 5.87     | <0.05   |
| Bacteremia        | 2.41  | 8.00     | <0.05   |
| Hepaticojejunostomy leak | 3.09 | 11.23   | <0.05   |
| Colorectal fistula | 4.12  | 9.34     | <0.05   |
| APACHE II>15      | 3.19  | 7.27     | <0.05   |

CI=confidence interval; OR=odds ratio; COPD=chronic obstructive airway disease; APACHE II=acute physiology and chronic health evaluation score II; pRBC=packed red blood cells

### Table 3: Multiple logistic regression models for mortality, ICU length of stay, and hospital length of stay

#### Mortality

| Predictors                  | Co-efficient | CI       | P value |
|-----------------------------|--------------|----------|---------|
| APACHE II<15                | 9.11         | 4.63-12.28 | <0.05   |
| Bacteremia                  | 3.17         | 2.36-7.45 | <0.05   |
| Malignancy                  | 0.029        | 0.021-0.037 | <0.05   |
| Hepaticojejunostomy leak    | 5.63         | 5.17-6.28 | <0.05   |
| Colorectal fistula          | 0.58         | 0.17-0.98 | >0.05   |
| pRBC transfusion            | 0.8          | 0.24-1.37 | >0.05   |
| Liver disease               | −1.15        | −2.39-1.04 | >0.05   |

#### ICU length of stay

| Predictors                  | Co-efficient | CI       | P value |
|-----------------------------|--------------|----------|---------|
| APACHE II<15                | 20.98        | 19.35-23.64 | <0.05   |
| Bacteremia                  | 6.75         | 6.33-7.19 | <0.05   |
| Malignancy                  | 0.03         | 0.02-0.03 | >0.05   |
| Hepaticojejunostomy leak    | 7.23         | 5.12-9.67 | <0.05   |
| Colorectal fistula          | 0.97         | 0.77-1.25 | >0.05   |
| pRBC transfusion            | 1.10         | 1.02-1.87 | >0.05   |
| Liver disease               | 0.98         | 0.66-1.28 | >0.05   |

APACHE II=Acute Physiolog un and chronic health evaluation score II; pRBC=Packed red blood cells; CI=confidence interval; ICU=Intensive care unit; OR=odds ratio
significant predictor of hospital length of stay. Based on the results from univariate and multivariate analysis, a simple scoring system for septic shock after anastomotic leak (SEPAL) has been developed. The risk of mortality can be predicted from this score. This includes APACHE II > 15 during presentation, malignancy, bacteremia, packed RBC transfusion, and hepaticojejunostomy leak. Each of the variables is given a score of 1. If the total score exceeds 3, the risk of mortality is 100% [Table 4].

Discussion

To the best of our knowledge, no prior studies have evaluated the predictors of septic shock following anastomotic leak after major gastrointestinal surgery. The present study of 103 patients of anastomotic leak had a septic shock of 69.9% (n = 72) with a mortality of 45.8% (n = 33).

Our study found a greater incidence of septic shock following anastomotic leak in patients with lower mean arterial pressure, higher heart rate, greater APACHE II score, and a higher base deficit at the time of ICU admission. This is in agreement with studies showing prolonged hypotension and microvascular ischemia predisposing to tissue ischemia and anastomotic failure. Although it is known that sepsis increases the risk of microvascular ischemia due to excessive production of reactive oxygen species (ROS), it is not clear whether anastomotic failure itself leads to oxidative stress producing sepsis. However, the rapid progression to septic shock in this group of patients can be due to the inhibition of sympathetic nervous system and loss of baroreceptor reflex control of arterial blood pressure.

Our study found that the patients in septic shock group received greater packed RBC and fresh frozen plasma during the ICU stay. This suggests a more critical nature of their illness in comparison to the non-septic shock group. Telem et al., found intraoperative blood loss of 200 ml or more and intraoperative transfusion requirement as major risk factors for anastomotic leakage after colorectal surgery. It is not possible to know from our study whether or not intraoperative transfusion increases the likelihood of septic shock, because we did not include the intraoperative blood transfusion requirement for analysis. However, Perner et al., found that most patients with septic shock who required transfusion had higher disease severity and lower hemoglobin levels than their non-transfused counterparts. Despite this difference, the mortality was similar in both adjusted and unadjusted analyses. Some studies have shown that blood transfusions during surgery may be associated with with prolonged and difficult surgery and intraoperative hypotension, which may be independently associated with the development of sepsis after surgery.

In our study, both the groups had similar hemoglobin level at the time of ICU admission, but there was greater requirement of transfusion in the septic shock group during the ICU stay. This mimics the findings of previous studies, although our study design and sample size does not allow us to make inference about the treatment effects.

Our study found that the presence of associated co-morbid illnesses like diabetes, malignancy, COPD, and liver disease increases the risk of septic shock. This is in agreement with a study showing higher levels of circulating biomarkers of endothelial cell adhesion (E-selectin) and vascular endothelial growth factor (VEGF) signaling (sFLT-1) in septic shock patients with diabetes. Since many of the endothelial pathways activated during sepsis remain already upregulated in diabetic patients, they can develop organ dysfunction at an earlier stage. Similarly, malignancy has been associated with increased likelihood of post-operative sepsis and as an independent predictor of death in septic shock.

A potential reason for the increased incidence of septic shock after hepaticojejunostomy and pancreaticojejunostomy leak may be the delayed diagnosis after leak. This is possible because biliary enteric anastomosis usually involves smaller ducts that are usually multiple. If the injury or stricture is above the bifurcation of right and left hepatic ducts, a small accessory duct may be missed, leading to bile leak and the subsequent sequel.

Our study found that both 30 day mortality and in-hospital mortality were higher among the septic shock patients. The following variables: APACHE II > 15, presence of bacteremia, malignancy, hepaticojejunostomy leak, and packed RBC transfusions were found to be independent predictors of mortality. Although there are limited evidences in the literature on the influence of these factors exclusively for gastrointestinal surgery, a prognostic scoring system known as Prognostic Index (PI) developed by a multivariate probit analysis was found

---

**Table 4: Septic shock after anastomotic leakage score and mortality risk**

| Variable                                      | Score |
|-----------------------------------------------|-------|
| APACHE II > 15 during presentation            | 1     |
| Malignancy                                    | 1     |
| Bacteremia                                    | 1     |
| Packed RBC transfusion                        | 1     |
| Hepaticojejunostomy leak                      | 1     |

If the total score is >3, the mortality risk increases to 100%. RBC = Red blood cells.
to accurately predict the severity and mortality of 83 surgical patients of gastrointestinal diseases in Japan.\[20\] These include age, pulse rate, blood urea nitrogen, serum albumin, serum cholesterol, and serum potassium. Although the evolution of mortality risk identification and prediction tools are limited by stratification in clinical trials, their usefulness in clinical decision making cannot be ignored. More recently, Estimation of Physiologic Ability and Surgical Stress (E-PASS), a prediction scoring system requiring nine variables was found to predict accurately the occurrence of anastomotic leak and its prognosis in various kinds of gastrointestinal procedures.\[21,22\]

Roman-Marchant et al. found that early onset septic shock is more severe and yet has a better outcome than late-onset septic shock.\[23\] This is evidenced by a shorter duration of shock, shorter length of ICU stay, and lesser mortality. Although our study did not differentiate between the early and late-onset septic shock, our results showed a higher mortality and greater length of ICU stay in the septic shock over non-septic shock group. Roman-Marchant et al., found that the principal organism in early septic shock was Streptococcus pneumonia and in the late septic shock was Pseudomonas. Since Pseudomonas infections normally carry a higher mortality, the higher mortality in the late septic shock can be because of the same. However, another study similar to ours found that, as compared to non-septic shock patients, septic shock patients are significantly older, have higher severity scores, have longer ICU and hospital stay, and carry longer ICU and hospital mortality.\[24\] In that study, the respiratory functions, cardiovascular functions, and fungal infections were found to be strong independent predictors of death in septic shock (5.6, 4.3, and 2.0-fold, respectively). We also found similar results, although the presence of malignancy and liver disease emerged to be more important predictors in our study.

There are several limitations in our study. First, being a retrospective audit, it has the inherent drawbacks of its design. Some variables that may be of interest (e.g., procalcitonin and C-reactive protein) are not routinely recorded in our registry and could not be analyzed. Second, the study population includes 103 patients with anastomotic leak and 72 patients with septic shock. This is a small number with limited ability to detect an association between patient factors and sepsis. However, our study has a similar power to a study that has reported a strong association between postoperative sepsis and a high CCS.\[25\] Third, our study is a single-center study in a university teaching hospital and tertiary referral centre for gastrointestinal surgery. Our results may not apply to all hospitals with different case mix and different quality of postoperative care. The specialized nature of our study setting and our anastomotic leak is a highly selective population in any setting population limits the generalizability of our results. Fourth, our study does not consider the possible variations in the risk of sepsis due to genetic polymorphism. Various studies have shown that genetic polymorphism in the tumor necrosis factor-α promoter significantly increases the risk for severe sepsis and mortality.\[26\]

To conclude, the identification of predictors for septic shock after anastomotic leak may be useful in reducing mortality, length of ICU, and hospital stay and costs. Various combinations of methods including the preoperative characteristics can be used to carry out such identification. We recommend further studies over different hospital settings.

References

1. Veltkamp SC, Kemmeren JM, van der Graaf Y, Edlinger M, van der Werken C. Prediction of serious complications in patients admitted to a surgical ward. Br J Surg 2002;89:94-102.
2. Madbouly KM, Senagore AJ, Renzi FH, Delaney CP, Waters J, Fazio VW. Perioperative blood transfusions increase infectious complications after ileal pouch procedures (IPAA). Int J Colorectal Dis 2006;21:807-13.
3. Behrman SW, Zarzaur BL. Intra-abdominal sepsis following pancreatic resection: Incidence, risk factors, diagnosis, microbiology, management and outcome. Am Surg 2008;74:572-8.
4. Wiedmann JW, Ithorn D, Andress HJ, Schilling FW. Incidence and mortality of severe sepsis in surgical intensive care patients: The influence of patient gender on disease process and outcome. Intensive Care Med 2000;26:167-72.
5. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: Development and validation. J Chronic Dis 1987;40:373-83.
6. Moran BJ, Heald RJ. Risk factors for and management of anastomotic leakage in rectal surgery. Colorectal Dis 2001;3:135-7.
7. Schrock TR, Devaney CW, Dunphy JE. Factor contributing to leakage of colonic anastomosis. Ann Surg 1973;177:513-8.
8. Hyman NH, Osler T, Cataldo P, Burns EH, Shackford SR. Anastomotic leaks after bowel resection: What does peer review teach us about the relationship to postoperative mortality? J Am Coll Surg 2009;208:48-52.
9. Post HL, Verhoefen PM, Pronk A, Sfeeaan I, Houweling PL. Intraoperative blood pressure changes as a risk factor for anastomotic leakage in colorectal surgery. Int J Colorectal Dis 2012;27:765-72.
10. Boyle NH, Manifield D, Jordan MH, Mason RC. Intraoperative assessment of colonic perfusion using scanning laser Doppler flowmetry during colonic resection. J Am Coll Surg 2000;191:304-10.
11. Vignali A, Gianotti L, Braga M, Radelli G, Malevazi L, Di Carlo V. Altered microperfusion at the rectal stump is predictive for rectal anastomotic leak. Dis Colon Rectum 2000;43:76-82.
12. Telen DA, Chin EH, Nguyen SQ, Divino CM. Risk factors for anastomotic leak following colorectal surgery. A case-control study. Arch Surg 2010;145:371-6.
13. Perner A, Smith SH, Carlsen S, Holst LB. Red blood cell transfusion during septic shock in the ICU. Acta Anaesthesiol Scand 2012;56:718-23.
14. Vamvakas EC, Carven JH, Hibbert PL. Blood transfusion and infection after colorectal cancer surgery. Transfusion 1996;36:1000-8.
15. Farinas-Alvarez C, Farinas MC, Fernandez-Mazarrasa C, Llorea J, Casanova D, Delgado-Rodriguez M. Analysis of risk factors for nosocomial sepsis in surgical patients. Br J Surg 2000;87:1076-81.

16. Schuetz P, Yano K, Sorasaki M, Ngo L, St Hilaire M, Lucas JM, et al. Influence of diabetes on endothelial cell response during sepsis. Diabetologia 2011;54:996-1003.

17. Schuetz P, Castro P, Shapiro NJ. Diabetes and sepsis: Preclinical findings and clinical relevance. Diabetes Care 2011;34:771-8.

18. Danai PA, Moss M, Mannino DM, Martin GS. The epidemiology of sepsis in patients with malignancy. Chest 2006;129:1432-40.

19. Sawaya DE Jr, Johnson IW, Sittig K, McDonald JC, Zibari GB. Iatrogenic and noniatrogenic extrahepatic biliary tract injuries: A multi-institutional review. Am Surg 2001;67:473-7.

20. Matsusue S, Kashihara S, Koizumi S. Prediction of mortality from septic shock in gastrointestinal surgery by probit analysis. Jpn J Surg 1988;18:18-22.

21. Haga Y, Wada Y, Takeuchi H, Ikejiri K, Ikegama M. Prediction of anastomotic leak and its prognosis in digestive surgery. World J Surg 2011;35:716-22.

22. Koushi K, Korenaga D, Kawamura H, Okuyama T, Ikeda H, Takenaka K. Using the E-PASS scoring system to estimate the risk of emergency abdominal surgery in patients with acute gastrointestinal disease. Surg Today 2011;41:1481-5.

23. Roman-Marchant O, Orellana-Jimenez CE, De Baeker D, Melet J, Vincent JL. Septic shock of early or late onset: Does it matter? Chest 2004;126:173-8.

24. Guidet B, Aegerter P, Ganzit R, Moshaka P, Dreyfuss D. CUB-Rea Study Group. Incidence and impact of organ dysfunctions associated with sepsis. Chest 2005;127:942-51.

25. Mokart D, Leone M, Sumini A, Brun JP, Tison A, Delpiero JR, et al. Predictive perioperative factors for developing severe sepsis after major surgery. Br J Anaesth 2005;95:776-81.

26. O’Keefe GE, Hybki DL, Munford RS. The G→A single nucleotide polymorphism at the-308 position in the tumor necrosis factor α promoter increases the risk for severe sepsis after trauma. J Trauma 2002;52:817-25.

How to cite this article: Choudhuri AH, Uppal R. Predictors of septic shock following anastomotic leak after major gastrointestinal surgery: An audit from a tertiary care institute. Indian J Crit Care Med 2013;17:298-303.

Source of Support: Nil, Conflict of Interest: None declared.