



Butler University Digital Commons @ Butler University

Scholarship and Professional Work – COPHS

College of Pharmacy & Health Sciences

3-2006

Bowel necrosis associated with enteral feeding

Jane M. Gervasio

Butler University, jgervasi@butler.edu

Ryan Ednalino

Butler University

Jennifer Reynolds

Butler University

Gary P. Zaloga

Methodist Research Institute

Follow this and additional works at: http://digitalcommons.butler.edu/cophs_papers



Part of the [Pharmacy and Pharmaceutical Sciences Commons](#)

Recommended Citation

Gervasio, Jane M.; Ednalino, Ryan; Reynolds, Jennifer; and Zaloga, Gary P., "Bowel necrosis associated with enteral feeding" (2006).
Scholarship and Professional Work – COPHS. Paper 22.
http://digitalcommons.butler.edu/cophs_papers/22

This Article is brought to you for free and open access by the College of Pharmacy & Health Sciences at Digital Commons @ Butler University. It has been accepted for inclusion in Scholarship and Professional Work – COPHS by an authorized administrator of Digital Commons @ Butler University. For more information, please contact fgaede@butler.edu.

Bowel necrosis associated with enteral feeding

Jane M. Gervasio, Ryan Ednalino, Jennifer Reynolds, Gary P. Zaloga

Background: Small bowel necrosis (SBN) is a serious complication associated with a high fatality. On rare occasions, SBN is encountered in critically ill patients receiving enteral nutrition (EN) via the small bowel. The cause of SBN in patients receiving concomitant EN is unclear and likely complex. The purpose of this chart review was to determine the incidence of feeding-associated SBN, determine if delivery of EN increased a patient's risk for developing SBN, and describe the features of feeding-associated SBN. Since there has been advances made in medicine over the past few decades, we also wished to determine this information in the setting of current state-of-the-art medical therapy.

Methods: The study was a retrospective chart review from January 1999 to December 2003. Patients = 18 years with a confirmed diagnosis of SBN, diagnosed following hospital admission, were identified using the hospital's computerized database. Charts were analyzed for a variety of factors including hemodynamic parameters prior to diagnosis, vasopressors received, EN delivered and clinical outcomes.

Results: During the five-year study period, 127,301 patients were admitted to the hospital, 8,480 patients with a principal or secondary diagnosis of hypotension. EN was delivered to 5272 patients. A total of 48 cases of SBN that developed following hospital admission, confirmed by laparotomy, were identified giving an overall incidence of 0.04% of admissions. Reasons for hospital admission varied among patients and included nausea/vomiting/abdominal pain (N=9,

18%); motor vehicle accident (N=8, 16.7%); cardiovascular event (N=4, 8.3%); aortic aneurysm (N=4, 8.3%); subarachnoid hemorrhage (N=2, 4.2%); sepsis (N=2, 4.2%); and other (N=7, 14.5%). Patients were 64 ± 15.5 years (mean \pm SD) and 33 (69%) of the patients were female. Six of the 48 SBN patients (0.004% of total admissions) were identified as having received EN within 72 hours prior to diagnosis. Four of these 6 patients were switched to TPN before diagnosis due to intolerance of EN. Clinical and hemodynamic parameters 72 hours prior to diagnosis are reported in Table 1. Clinical outcomes are reported in Table 2.

Conclusion: SBN diagnosed following hospital admission is rare but life-threatening. Early clinical signs include increase heart rate and abdominal distention and a majority of patients had previous small bowel resections. Most patients admitted with hypotension did not develop SBN. Vasopressor use was common in patients with SBN but it remains unclear whether its use was a cause or a result of the infarction. Associated use of EN in patients with SBN was rare (1:1000), as was the presentation of hypotension (3:1000). Adjusting for the severity of illness, SBN occurred at similar incidences in patents receiving EN and those not receiving EN. Further study of the etiology of SBN is warranted.

Table 1.

	SBN s/ EN (N=42)	SBN w/ EN (N=6)
Tachycardia (>100 bpm)	35 (83.3%)	6 (100%)
Hypotension (SBP <90)	23 (54.8%)	4 (66.7%)
Abnormal WBC (>15k)	21 (50.0%)	6 (100%)
Temperature >100.5°F	23 (54.8%)	5 (83.3%)
Abdominal distention	34 (81.0%)	6 (100%)
Nausea / vomiting	20 (47.6%)	0 (0%)
Abdominal trauma	11 (26.2%)	1 (16.7%)
Small bowel resection	27 (64.3%)	2 (33.3%)
Non-related surgery	15 (35.7%)	5 (83.3%)
Vasopressors	18 (42.9%)	5 (83.3%)

Table 2.

	SBN s/ EN (N=42)	SBN w/ EN (N=6)		
ICU LOS (days)	8.7 ± 10.3 4 (Median)	29.8 ± 38.6 13 (Median)	T = 1.33; <i>p</i> = 0.240	MWU = 39.00; <i>p</i> = 0.017
Total LOS (days)	19.8 ± 21.1 12.5 (Median)	31.0 ± 40.1 14 (Median)	T = 1.073; <i>p</i> = 0.289	MWU = 97.00; <i>p</i> = 0.366
Death	24 (57.1%)	6 (100%)		Fisher's exact <i>p</i> = 0.071

T = Independent sample t-test; MWU: Mann-Whitney U test