

## Prevention and treatment of gastrointestinal dysfunction following severe burns: A summary of recent 30-year clinical experience

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### Abstract

**AIM:** To sum up the recent 30-year experience in the prevention and treatment of gastrointestinal dysfunction in severe burn patients, and propose practicable guidelines for the prevention and treatment of gastrointestinal (GI) dysfunction.

**METHODS:** From 1980 to 2007, a total of 219 patients with large area and extraordinarily large area burns (LAB) were admitted, who were classified into three stages according the therapeutic protocols used at the time: Stage 1 from 1980 to 1989, stage 2 from 1990 to 1995, and stage 3 from 1996 to 2007. The occurrence and mortality of GI dysfunction in patients of the three stages were calculated and the main causes were analyzed.

**RESULTS:** The occurrence of stress ulcer in patients with LAB was 8.6% in stage 1, which was significantly lower than that in stage 1 ( $P < 0.05$ ). No massive hemorrhage from severe stress ulcer and enterogenic infections occurred in stages 2 and 3. The occurrence of abdominal distension and stress ulcer and the mortality in stage 3 patients with extraordinarily LAB was 7.1%, 21.4% and 28.5%, respectively, which were significantly lower than those in stage 1 patients

( $P < 0.05$  or  $P < 0.01$ ), and the occurrence of stress ulcer was also significantly lower than that in stage 2 patients ( $P < 0.05$ ).

**CONCLUSION:** Comprehensive fluid resuscitation, early excision of necrotic tissue, staged food ingestion, and administration of specific nutrients are essential strategies for preventing gastrointestinal complications and lowering mortality in severely burned patients.

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**Key words:** Severe burn; Gastrointestinal function; Fluid resuscitation; Staged food ingestion

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### INTRODUCTION

Gastrointestinal dysfunction is a common complication of severe burns. Injury to GI function, especially to GI barrier function, is an important initiator as well as a stimulator for occurrence of systemic inflammatory response syndrome (SIRS), sepsis and multiple organ dysfunction syndrome (MODS) following severe burns<sup>[1]</sup>. With the deeper understanding of GI function and changes in the stereotype of clinical treatment in recent 30 years, a series of new therapies including fluid resuscitation, early escharectomy, continuous renal replacement therapy, and use of glutamine and growth factor has been adopted in the treatment of severe burns<sup>[2,3]</sup>. Although animal experiments have shown that these new therapies do play a positive role in the prevention and treatment of GI dysfunction following severe burns, there has been a lack of convincing clinical

data to confirm the outcome<sup>[4-6]</sup>. The present study reviewed the clinical data of 219 patients with large area burns (LAB) in recent more than 20 years, who were classified into different stages according to the therapeutic protocols used at the time. Based on the review, the outcomes of GI function protection and treatment were compared, analyzed and summarized in an attempt to propose some practicable guidelines for the effective prevention and treatment of GI dysfunction.

## MATERIALS AND METHODS

### Clinical data

This study included 219 patients with severe burns who were admitted to this burn center from January 1980 to August 2007. They were classified as LAB patients (50%-79% TBSA, or degree III burn area > 20%) and extraordinarily large area burn (ELAB) patients (80%-100% TBSA, or degree III burn area > 50%). According to the therapeutic protocols used at the time, they were assigned to three stages: stage 1 from 1980 to 1989, stage 2 from 1990 to 1995, and stage 3 from 1996 to 2007. The occurrence of GI dysfunction and mortality were analyzed statistically.

**Stage 1 (1980-1989):** Limited fluid resuscitation was advocated during the shock phase of burn patients. In other words, the total fluid input was minimized as long as the vital signs were stably maintained, and the urine output was controlled at a level of 0.5 mL/h per kg body weight. The first escharectomy was usually done 4-7 d after burn injury, and the operation area was 20%-30% TBSA in most cases. Patients were mostly starved in the early stage of burn and relied on intravenous nutrition. To prevent stress ulcer, gastric mucosal protection agents and anti-acid drugs were administered routinely.

**Stage 2 (1990-1995):** The major therapeutic changes were advancing the first escharectomy to 3-4 d after burn injury, expanding the operation area as much as possible, and excising the necrotic tissue as early as possible. When bowel sounds recovered 2-4 d after burn injury, food intake was started gradually through the gastric tube and patients were encouraged to take food orally, with administration of appropriate amounts of gastrokinetic drugs such as domperidone to promote gastrointestinal peristalsis. Oral norfloxacin and nystatin were administered routinely within 2 wk after burn. Selective decontamination of the digestive tract (SDD) was also recommended.

**Stage 3 (1996-2007):** The comprehensive resuscitation strategy was advocated for shock burn, which includes sufficient resuscitation and maintenance of urine output at 1-1.5 mL/h per kg BW; routine intravenous instillation of vasoactive drugs such as small doses of dopamine; adjustment of the gastrointestinal tract and renal blood perfusion; and use of antioxidants such as large doses of Vitamin C and E to eliminate free oxygen radicals. In addition, antibiotics were used prudently,

including shortening the duration of antibiotic administration and reducing the variety of antibiotics. So far as gastrointestinal nutrition is concerned, staged food ingestion was advocated, where small amounts of light fluids (20-40 mL/h) were instilled through the gastric tube 2 h after burn to stimulate gastrointestinal peristalsis. Once bowel sounds recovered, the amount of food was increased gradually. Usually the amount of enteral nutrition fed through the gastric tube was increased to 2000-2500 Kcal/d 3-6 d after burn. Specific nutrients were used such as oral glutamine, L-arginine, dietary fiber and subcutaneous growth factor.

Since 2003, early administration of continuous renal replacement therapy (CRRT) for 5-8 consecutive days has been advocated in patients with GI failure accompanied with sepsis. The content of endotoxin, IL-1 $\beta$ , IL-6 and IL-8 in plasma were analyzed before and after CRRT treatment. TNF- $\alpha$  content was measured by radioimmunoassay. The activity of diamine oxidase (DAO) in plasma was tested according to the previous report<sup>[7]</sup>.

### Indexes for assessing GI function

There was no uniformed criterion for assessing GI dysfunction<sup>[8]</sup>. Based on the diagnostic criteria for MODS and GI symptoms commonly seen in burned patients, GI dysfunction is summarized as follows: (1) abdominal distension: bowel sound was reduced and food intolerance exceeded more than 5 d; (2) stress ulcer: gastric fluid aspirated from the gastric tube appeared bloody macroscopically and gastric mucosa was erosive and ulcerative gastroendoscopically; (3) severe stress ulcer: blood loss exceeded 800 mL within 24 h; (4) alteration of intestinal microbiota: Gram-negative *E.coli* was amplified, and the bacillus/coccus ratio was greater than 10:1; and (5) enterogenic infection: highly suspected systemic infection occurred after ruling out wound surface, pulmonary and indwelling catheter infections<sup>[9,10]</sup>.

### Statistical analysis

Data were testified by Pearson's Chi-square test, and Fisher's two-tail exact test.

## RESULTS

Of the 219 severe burn cases analyzed (Table 1), 89 cases were LAB and 130 cases were ELAB. There was no significant difference in age distribution and burn area between the three stages of patients.

Table 2 shows that the occurrence of stress ulcer in LAB patients of stage 3 was 8.6%, which was significantly lower than 30.3% of stage 1 patients ( $P < 0.05$ ). No hemorrhage from severe stress ulcer and enterogenic infection occurred in the patients of stage 2 and 3.

Compared with LAB patients, the occurrence of gastrointestinal complications and mortality in ELAB patients were significantly higher, indicating that occurrence of gastrointestinal complications was closely

Table 1 General clinical data of 219 burned patients

Stage	<i>n</i>	LAB		ELAB		
		Age (yr)	TBSA (%)	<i>n</i>	Age (yr)	TBSA (%)
1	33	26.5 ± 18.7	65.1 ± 12.4	45	28.5 ± 13.7	91.4 ± 10.2
2	21	27.7 ± 13.8	74.6 ± 10.3	29	26.9 ± 14.6	89.3 ± 8.2
3	35	25.9 ± 16.3	69.5 ± 12.2	56	26.4 ± 13.3	93.1 ± 9.4

TBSA: Total body surface area.

Table 2 GI complications and mortality in LAB patients (%)

Stage	<i>n</i>	AE	SU	SSU	FA	EI	Mortality
1	33	12.1	30.3	3.0	15.1	3.0	12.1
2	21	4.8	19.0	-	4.8	-	4.8
3	35	2.9	8.6 <sup>a</sup>	-	2.9	-	2.9

AE: Abdominal extension; SU: Stress ulcer; SSU: Severe stress ulcer; FA: Flora alteration; EI: Enterogenic infection. Compared with the stage 1, <sup>a</sup>*P* < 0.05.

associated with the severity of burn. Table 3 shows that the occurrence of abdominal extension and stress ulcer and mortality in the stage 3 ELAB patients were 7.1%, 21.4% and 28.5%, respectively, which were significantly lower than those of stage 1 (*P* < 0.05 or *P* < 0.01), and the occurrence of stress ulcer in the stage 3 ELAB patients was also significantly lower than that of stage 2 patients (*P* < 0.05).

In the 5 patients with GI failure accompanied with severe sepsis, endotoxin, IL-1β, IL-6, IL-8 and TNF-α levels and plasma DAO activity were decreased significantly after CRRT (Table 4) (*P* < 0.01).

## DISCUSSION

Timely and effective fluid resuscitation is the basis and guarantee of curing severely burned patients<sup>[11]</sup>. Before the 1990s, the therapeutic concepts were limited to such that excessive fluid infusion would aggravate edema so that limited resuscitation was addressed. Under the bunker of stable vital signs lies the problems of GI hypoxia and ischemia, or occult GI shock<sup>[12,13]</sup>. Since the mid and late 1990s, comprehensive resuscitation strategies for maintaining the stability of vital signs and splanchnic resuscitation to restore GI blood supply as early as possible and reduce hypoxic and ischemic injuries as much as possible have been recommended<sup>[14]</sup>. It is suggested that small doses of dopamine should be administered to dilate the renal and GI vessels<sup>[15]</sup>, and free oxygen radical clearing agents to attenuate ischemia/reperfusion injury in the process of resuscitation<sup>[16,17]</sup>. These comprehensive resuscitation measures played an important role in protecting GI function, helping resume bowel sound earlier and digestive function<sup>[18]</sup>. No stress ulcer occurred during the shock phase, which laid a sound foundation for future treatment.

It has been generally accepted that early enteric nourishment plays an essential role in preventing GI dysfunction following severe burns<sup>[19,20]</sup>. But as LAB often causes serious edema of GI mucosa, there is a

Table 3 GI complications and mortality in ELAB patients (%)

Stage	<i>n</i>	AE	SU	SSU	FA	EI	Mortality
1	45	24.4	60.0	11.1	20.0	15.5	55.6
2	29	13.8	48.3	6.9	13.8	10.3	41.3
3	56	7.1 <sup>a</sup>	21.4 <sup>bc</sup>	3.6	8.9	7.1	28.5 <sup>b</sup>

Compared with stage 1, <sup>a</sup>*P* < 0.05, <sup>b</sup>*P* < 0.01; compared with stage 2, <sup>c</sup>*P* < 0.05.

Table 4 Endotoxin, IL-1β, IL-6, IL-8 and TNF-α levels and plasma DAO activity before and after CRRT

	Endotoxin (Eu/mL)	TNF-α (pg/mL)	IL-6 (pg/mL)	IL-8 (pg/mL)	DAO (U/mL)
Before CRRT	0.76 ± 0.13	272 ± 28	518 ± 64	583 ± 51	2.98 ± 0.94
After CRRT	0.045 ± 0.017 <sup>b</sup>	57 ± 15 <sup>b</sup>	98 ± 25 <sup>b</sup>	105 ± 31 <sup>b</sup>	1.27 ± 0.54 <sup>b</sup>

Compared with before CRRT, <sup>b</sup>*P* < 0.01.

concern that early food intake would give additional burden to the GI tract or even cause acute gastric dilation, resulting in vomiting and aspiration. For this reason, there is controversy over when and how to take food. Before the 1990s, food intake was usually started when patients resumed bowel sound. After the mid and late 1990s, the idea of staged food intake was advanced: a small amount of light fluid is started several hours after burn so as to not only supplement nutrition but stimulate GI peristalsis and improve GI blood supply. Once bowel sound resumes, the amount of food can be increased. Using immunoregulatory nutrients such as oral glutamine, L-arginine and dietary fiber promoted post-burn repair of GI mucosa, maintained GI barrier function, and reduced translocation of enterogenic bacteria and endotoxins<sup>[21,22]</sup>.

The microenvironment formed by GI resident bacteria forms an ecologic barrier in the intestinal lumen, preventing intestinal pathologic bacteria from colonization and substantial proliferation<sup>[23]</sup>. To maintain normal intestinal microbiota, we paid special attention to the followings: prudent use of antibiotics and routine use of SDD. In earlier treatment of severe burns, a variety of broad-spectrum antibiotics were often used concomitantly. But our clinical experiences showed that mere use of antibiotics failed to control infections effectively in severely burned patients; instead it often caused alteration of bacterial flora, resulting in superinfection. Since the mid and late 1990s, the principle of “bold use of antibiotics and bold discontinuation of them” has been advocated. In other words, the duration and variety of antibiotics should be minimized, and the use of antibiotics should be enhanced properly during the edema reabsorption phase and the perioperative period. By doing so, the incidence of systemic infection was lowered, and furthermore it avoided alteration of intestinal microbiota effectively. The use of SDD within two weeks of burn injury inhibited the growth of Gram bacteria and fungi and maintained the stability of intestinal microbiota, which may also be beneficial to reducing superinfection

of intestinal bacteria<sup>[24]</sup>.

Early excision of necrotic tissue and closure of the wound surface are essential in the treatment of severe burns<sup>[25]</sup>. Positive surgical treatment has become a generally accepted idea. Our practice is that surgery is started 3-4 d after burn injury and the area of escharectomy at a time is much larger than before, usually reaching 60%-75% TBSA. The wound surface is covered with heterogeneous skin, which plays an important role in preventing systemic inflammatory reaction and protecting organ functions<sup>[26]</sup>.

In some patients in whom fluid resuscitation was not implemented effectively for various reasons, wound surface infection often caused severe injury to the GI function, or even toxic paralytic ileus palsy, greatly increasing toxin absorption and bacterial superinfection. Toxins absorbed in the blood act on the GI tract, which in turn lowers the GI kinetics, resulting in a vicious cycle<sup>[27]</sup>. Treatment of this kind of critically severely burned patients is a real challenge, in whom the mortality rate is usually high. Apart from the above mentioned routine treatments, we also used CRRT to filtrate inflammatory mediators and toxins in the body, which significantly lowered the content of endotoxins and inflammatory factors and DAO activity. As the vicious cycle was broken off, the therapeutic outcome was usually good<sup>[28]</sup>.

In summary, post-burn GI dysfunction is caused by multiple factors, and therefore maintaining GI function is a systematic engineering project. The therapeutic strategy should not rely on a single treatment or a single drug<sup>[29,30]</sup>. Furthermore, as severe burn itself may cause serious injury to various functions of the body, prevention of multi-organ functions should be addressed. Clinical experiences in recent 30 years have demonstrated that comprehensive fluid resuscitation, early excision of necrotic tissue, staged food ingestion, and administration of specific nutrients are essential strategies for preventing gastrointestinal complications in severely burned patients. Once severe GI dysfunction and sepsis occur, individualized comprehensive treatment should be implemented without delay. CRRT developed in recent years appears to be a promising strategy in the treatment of severe burns<sup>[31]</sup>.

## COMMENTS

### Background

Gastrointestinal dysfunction is a common complication of severe burns. Injury to GI function, especially to GI barrier function, is an important initiator as well as a stimulator for occurrence of systemic inflammatory response syndrome (SIRS), sepsis and multiple organ dysfunction syndrome (MODS) following severe burns.

### Research frontiers

The study analyzed and summarized the authors' clinical experiences in recent 30 years in the prevention and treatment of gastrointestinal dysfunction in severely burned patients in an attempt to propose some practicable guidelines for the effective prevention and treatment of GI dysfunction following severe burns.

### Innovations and breakthroughs

Comprehensive fluid resuscitation, early excision of necrotic tissue, staged food ingestion, and administration of specific nutrients are essential strategies for

preventing gastrointestinal complications and lowering the mortality in severely burned patients.

### Applications

The study provided some practicable guidelines for the effective prevention and treatment of GI dysfunction following severe burns.

### Peer review

The paper analyzed and summarized some practicable experience for the effective prevention of GI dysfunction. It is valuable to see the actual results from the therapies by the authors over the last 30 years.

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