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Pathophysiology of the gastrointestinal tract in burn disease

O. V. Kravets, V. V. Yekhalov, V. V. Gorbuntsov

Dnipro State Medical University

✉ Vasyl Yekhalov: sesualiy@gmail.com

O. V. Kravets, http://orcid.org/0000-0003-1340-3290
V. V. Yekhalov, http://orcid.org/0000-0001-5373-3820
V. V. Gorbuntsov, http://orcid.org/0000-0001-7752-0993

The review of foreign publications resulted in a generalisation of medical reports on the pathological changes of the gastrointestinal tract in burn disease. Burn disease produces an immediate reaction in all organs and systems, which are not always able to maintain homeostasis and frequently suffer pathophysiological and morphological damage. One of those target systems is the gastrointestinal tract. Only in very rare cases do severe (mainly electrical) burns cause direct injury to the abdominal cavity organs, thus resulting in a very severe clinical course and high mortality. Patients of all ages who have experienced a burn injury have an increased overall risk of developing gastrointestinal diseases, which include pathology of the esophagus, stomach, and intestines, as well as lesions of the gallbladder, biliary tract, and pancreas. With a burn area of 40—95 %, 5.7 % of the victims were diagnosed with pathology of the abdominal organs. Among them, 26.0 % had an abdominal catastrophe (infarction or perforation), 37.0 % had bleeding from the upper parts of the gastrointestinal tract, 32.0 % had paralytic intestinal obstruction, and 5.0 % developed pancreatitis and acute necrotizing cholecystitis. Large burns are usually associated with a significant decrease in splanchnic perfusion. After severe burns, intestinal ischemia and hypoxia disrupt the intestinal epithelial barrier and enteric bacterial translocation, leading to serious complications such as systemic inflammatory response syndrome, sepsis, and multiple organ failure. Peritonitis or gastrointestinal bleeding accounted for 88.2 % of deaths among patients with gastrointestinal dysfunction. In general, gastrointestinal dysfunction was more common in patients with inhalation injuries, burn shock, large burn areas, and high analgesic requirements.

Keywords
burn disease, gastrointestinal tract, pathophysiology, review, treatment.

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According to the results of statistical research by the Institute for Health Metrics and Evaluation (Washington), Ukraine has one of the highest rates of burn mortality in the world (4.55 per 100 thousand), second only to ten countries in the post-Soviet area and the African continent, which emphasises the relevance of combustiological research in the country. Burn disease (BD) produces an immediate reaction in all organs and systems, which are not always able to maintain homeostasis and frequently suffer pathophysiological and morphological damage. One of those target systems is the gastrointestinal tract (GIT). [50, 54].

Only in very rare cases do severe (mainly electrical) burns cause direct injury to the abdominal cavity organs, thus resulting in a very severe clinical course and high mortality [16]. Therefore, our review focused on the mediated damaging dynamic effect of burn stress / shock and BD on the digestive system in general and its specific components, leading to the emergence of severe life-threatening syndromes and conditions.

Burn patients of all ages who have experienced a burn injury are at increased risk for gastrointestinal disease, including:

- esophageal, gastric, intestinal lesion;
- noninfectious enteritis and colitis;
- gallbladder, biliary tract, and pancreatic lesion.

The pediatric burn cohort had higher rates of hospitalisation and length of hospital stay than patients with abdominal pathology without burns [4, 50].

Stress ulcers are a well-known clinical phenomenon with a variety of features, ranging from asymptomatic superficial lesions and occult

gastrointestinal bleeding to overt clinically significant blood loss. Disruption of the intestinal barrier, where the mucosal glycoprotein is destroyed by increased concentrations of refluxed bile salts or uremic toxins, often leads to ulcer formation. Stress-induced hyperproduction of gastric acid due to increased secretion of gastrin is also likely [21, 24, 32]. Ulcers caused by burn stress / shock are mostly seen in the acid-producing regions of the body and the pyloric part of the stomach, but they can also be located in the antrum and duodenum [24, 32, 56]. Burn shock leads to splanchnic hypoperfusion and ischemia of the gastric mucosa, causing its atrophy, reduced ability to neutralise hydrogen ions, and impaired healing [8, 38, 56]. Initially, it can manifest itself in the form of erosive gastritis, starting with asymptomatic superficial lesions [24, 32, 37].

J. Swan (1823) first demonstrated the relationship between skin burns and damage to the GIT mucous membrane [41]. T. B. Curling (1842) described acute duodenal ulcers (which later became known as Curling's ulcers) in a group of burn patients [13, 42, 56]. Stress-induced gastritis can be called:

- diffuse mucosal damage;
- stress ulcer;
- hemorrhagic gastritis;
- erosive gastritis;
- Curling's ulcer;
- Cushing ulcer.

What they have in common is the presence of multiple surface erosions, beginning in the proximal acid-secreting part of the stomach and spreading distally. Cushing ulcers develop in BD as a result of damage to the central nervous system. Morphologically, Cushing ulcers are single and deep, can affect the esophagus, stomach or duodenum, emerge within a few hours after the injury, and appear as wedge-shaped hemorrhages on the mucous membrane with superficial necrosis. If these erosions continue to progress and spread to the submucosa, significant life-threatening bleeding may result [9, 24, 32, 51]. Most cases of Curling's ulcers in the current literature are secondary to severe thermal burns. There is a known casuistic case of their formation as a result of sunburn [21]. The risk of ulceration was higher in patients with burn area over 20 % total body surface area (TBSA) compared to patients with burn area less than 20 % TBSA (odds ratio 4.31). In addition, the incidence of ulcers was higher in patients with epigastric pain compared to patients without this symptom (odds ratio 4.55) [24, 56]. In burn patients with TBSA no less than 70 %, the frequency of Curling's ulcer was 40 % [8]; the risk of their development increased significantly under sepsis or septic shock [24, 50].

According to the diagnostic criteria for symptoms related to the gastrointestinal tract in BD, the dysfunction of the digestive system is characterised as follows [56]:

- flatulence: intestinal noise decreases, and food intolerance persists for more than 5 days;
- stress ulcer: gastric fluid aspirated through a gastric probe macroscopically has bloody impurities, and gastroendoscopically, the gastric mucosa is affected by erosions and ulcers;
- severe stress ulcer: blood loss exceeds 800 ml within 24 h;
- change in the intestinal microbiota: gram-negative *E. coli* are amplified, and the bacillus/coccus ratio is greater than 10 : 1;
- suspected systemic infection is confirmed after exclusion of the primary focus of the wound surface, pulmonary infection, and catheter-related sepsis.

Although stress ulcers can lead to perforation, this occurs with an incidence of less than 1 % [21, 24, 32]. Prevention of stress ulcers with the help of proton pump blockers, histamine-2 (H2)-receptors and early enteral nutrition has significantly reduced the incidence of Curling's ulcer and the frequency of its complications in recent decades [8, 9, 15, 23, 38, 39, 56].

Abdominal compartment syndrome

In patients with large burns, there is a significant risk of increased intra-abdominal pressure (IAP) in the presence of thermal damage to the abdominal cavity and capillary leak syndrome due to the systemic inflammatory response and aggressive fluid replacement [31]. The abdominal cavity has a certain tolerance for increased intraperitoneal volume without any marked increase in IAP, the increase of which does not necessarily cause abdominal compartment syndrome (ACS). The higher the IAP and the more trigger factors, the greater the risk of developing ACS [19, 53]. An increase in IAP causes progressive hypoperfusion and ischemia of the intestine and other peritoneal and retroperitoneal structures. The effect of IAP is not limited to the organs of the internal abdominal cavity but directly or indirectly affects each of the organ systems (dysfunction of the pulmonary, cardiovascular, kidney, liver, central nervous and gastrointestinal systems) [53].

ACS is a severe complication of BD with a mortality rate of (40—100) %. The percentage of burns on the total surface of the body is the main risk factor for the development of ACS:

- the prevalence of intra-abdominal hypertension reached 53 % in burn patients with TBSA over 15 % [48];
- ACS develops in every second patient with TBSA over 20 % in adults and with TBSA over 25 % in children;

- with TBSA over 60 %, ACS occurs always;
- morbidity in adults is higher than in children [7, 18, 19, 22, 31, 48, 52, 53].

A recent systematic review demonstrated that the infusion volume in particular was directly responsible for the development of ACS and was associated with 97 % mortality with TBSA over 60 % [53]. The development of ACS in a burn patient undergoing infusion therapy is quite difficult to identify [37]. Active fluid resuscitation in BD causes capillary leak syndrome and exacerbates splanchnic edema, which increases intestinal permeability, bacterial translocation, and elevated IAP. In patients with burn shock, it is necessary to try to quickly restore microcirculation using the minimum amount of fluid. Liquid resuscitation is usually used for BD, according to the Parkland formula 4 ml/Percentage of the total body surface per 1 kg within 24 h.

Due to diffusion and estimated compartment volumes, it is assumed that approximately two-thirds of the infused crystalloid solution enters the interstitial space [22, 44].

Both insufficient and excessive amounts of injected fluid lead to dysfunction of organs and tissues and the development of multiple organ failure syndrome (MOFS). An infusion volume of no less than 250 mL/kg administered within the first 24 h is a major risk factor for ACS. In case of fluid overload, splanchnic edema increases the intraperitoneal volume, and the abdominal wall is stretched to such a degree that further stretching becomes impossible. Combined with a systemic inflammatory response, this can lead to significant capillary leakage and diapedesis of fluid into the abdominal cavity (edema and ascites). The elasticity of the tissues of the abdominal wall decreases, which is associated with circular burns of the abdomen or chest (density of the burn scab). As a result, the ability of the abdomen to distend is limited, the critical point of the IAP growth is reached with a smaller increase in the intraperitoneal volume, and ACS can occur with smaller infusion volumes [2, 7, 19, 22, 26, 28, 53]. ACS is characterised by organ failure as a result of a long-term rise in IAP over 20 mm Hg [26, 29, 48].

The phenomenon of capillary leakage contributes to:
- ischemia-reperfusion injury;
- release of vasoactive substances;
- interstitial edema;
- activation of free radical oxidation.

Secondary intra-abdominal hypertension usually develops within 48 h of a burn, and ACS usually occurs with septicotoxemia. Patients with inhalation poisoning from combustion products have an increased risk of fluid sequestration [19, 31].

With TBSA less than 20 %, the prevalence of ACS is estimated at (4.1—17.0) %, with (65—75) % of the patients having a further risk of developing intraperitoneal hypertension without organ dysfunction [2, 19].

Elevated IAP leads to certain systemic disturbances [19, 22, 31]:

- over-upward displacement of the diaphragm, cardiac and pulmonary compression, decreased venous return, and subsequently hypoxemia, hypercapnia, atelectasis, and ventilation/perfusion mismatch;
- renal vasoconstriction, activating sympathetic innervation and the renin-angiotensin system; oliguria at an IAP over 15 mm Hg, and anuria at an IAP of no less than 30 mm Hg; in burn patients, the kidneys are particularly vulnerable to damage associated with elevated IAP, and 69.9 % develop acute kidney injury (AKI); the average period of development of AKI is about 3 days; individual analysis of the risk factors for AKI determined the relationship between the IAP, the use of glycopeptides and vasopressors;
- decreased perfusion of the intestinal mucosa at an IAP of 10—20 mm Hg; disturbed blood flow through the abdominal and superior mesenteric arteries at an IAP of no less than 40 mm Hg;
- compression of the mesenteric veins, impairing drainage and aggravating ACS, ultimately leading to further intestinal hypoperfusion, decreased intramural pH, and worsening lactic acidosis. Released inflammatory cytokines activate capillary permeability, which increases edema and IAP. This is a vicious cycle in which swelling leads to hypoperfusion of tissues, which, in turn, increases their swelling.

Careful monitoring of the IAP is recommended for every burn patient receiving an infusion of 250 ml/kg or more [53]. It is advisable to measure IAP every 2—4 h with TBSA at no less than 20 % [19]. IAP can be measured using an intraperitoneal catheter or indirectly by measuring pressure in the rectum, stomach, inferior vena cava, or bladder. The most acceptable and practical method is the registration of the IAP in the bladder. Physiological IAP ranges from 5 to 7 mm Hg. High systemic arterial pressure can maintain the perfusion of abdominal organs even in conditions of elevated IAP. Determination of abdominal perfusion pressure (APP) according to the formula

APP = mean arterial pressure (MAP) – IAP

is considered the best marker of perfusion of abdominal organs. Patients with IAP less than 10 mm Hg have no risk of developing ACS, while those with IAP no less than 25 mm Hg mostly develop it, and abdominal perfusion pressure less than 60 mm Hg

is associated with higher mortality after initiation of ACS [22, 53].

Clinicians should anticipate the risk of potential intestinal ischemia under ACS with the complication of late intestinal obstruction and recognise that the final pathological consequences of ischemic damage may occur many months after the first episode of ACS [22]. The TBSA percentage is directly correlated with ACS. The combination of:

- AKI;
- positive (daily and cumulative) fluid balance;
- high IAP;
- high extravascular lung fluid index (EVLWI);
- low APP

indicates a negative prognosis [45].

ACS can be treated using fluid resuscitation with continuous venous dialysis and ultrafiltration [17]. The current management of ACS consists of:

- decompression laparotomy using temporary abdominal closure techniques, including the Bogotá pouch;
- skin closure over open fascia;
- using Wittmann patch and vacuum techniques.

These methods are reliable, but temporary [28]. A circular burn of the abdominal area creates a tourniquet effect, and an urgent decompression escharotomy of the abdominal wall ensures a rapid decrease in the IAP. This improves ventilation, hemodynamic parameters, and oxygen exchange and can reduce mortality and treatment times. If simple escharotomy and decompression necrectomy do not lead to a decrease in the IAP, laparotomy decompression is performed [39, 53]. Fascial closure within 48 hours was associated with improved survival compared with later fascial closure [36]. The survival rate of burn patients with ACS after laparotomy is quite low (only 16 %). Better results can be achieved by following the strategy of immediate laparotomy and early fascial closure [36]. Although decompression laparotomy can save the lives of many patients with ACS, this procedure is associated with significant complications. Intestinal-cutaneous or intestinal-atmospheric fistula occurs in (2—45) % of patients after laparotomy and open abdomen. These fistulas are quite difficult to repair, and in about 40 % of cases, they can lead to electrolyte imbalance, hypotrophy, and death [28].

Decrease in splanchnic perfusion

Large burns are usually associated with a significant decrease in splanchnic perfusion. In animal model studies, mesenteric blood flow typically decreases shortly after burn injury by more than 50 % after burn at TBSA equal 40 %. This is associated with an increase in the resistance of mesenteric vessels, which is due to the action of [5]:

- vasoactive mediators:
  1) angiotensin II;
  2) vasopressin;
  3) vasoactive intestinal polypeptide 4;
- inflammatory mediators released from affected tissues:
  1) thromboxanes;
  2) leukotrienes.

Burns lead to a significant decrease in blood flow in the small intestine, which subsequently leads to intestinal mucosal dysfunction and villus tip ischemia [23]. Patients with burns have a high risk of developing ischemic enterocolitis (IE) in the immediate post-burn period. If intestinal pneumatosis is identified radiographically, diagnostic efforts should attempt to identify ischemia and necrosis, as these cause high mortality. IE is a rare condition characterised by gas formation in the gastrointestinal tract; its treatment and prognosis depend on the specific cause. The true incidence of IE is unknown, as most cases are asymptomatic. In burn patients, IE has an incidence of 0.5 % at clinical diagnosis; however, postmortem studies show that up to 50 % of burn patients have some degree of ischemic bowel injury. No patient developed IE earlier than 7 days after the injury [3].

Acute mesenteric ischemia (AMI) is a rare, severe complication of BD with a high mortality rate, the incidence of which is 1 % to 2 %. The main reported cause of AMI is a non-occlusive one, which is consistent with the hypothesis of intestinal perfusion limitation in the early phase of shock [29, 50]. Burn patients are at risk for intestinal ischemia due to massive fluid shifts, cardiac output limitations, and decreased regional organ perfusion due to reduced intravascular volume. In addition, there are reports that early enteral nutrition may play a fixed role in the development of intestinal ischemia after a severe burn injury [3].

Violation of the perfusion of the intestines, kidneys, and other internal organs leads to rapid liver and kidney failure, intestinal ischemia, and a decrease in the diaphragm mobility [14]. After a severe burn, blood flow is redistributed in favour of vital organs such as the heart and brain, due to which the blood flow to the intestine is noticeably reduced. Intestinal ischemia and hypoxia disrupt the intestinal epithelial barrier with subsequent enteric bacterial translocation, greatly contributing to the development of systemic inflammatory response syndrome, sepsis, and multiple organ dysfunction syndrome, which are common causes of burn mortality [15]. Hypercatabolism in BD is combined

with a decrease in regional oxygen delivery to a level insufficient to meet the metabolic needs of the intestine. Burn-induced intestinal ischemia leads to oxidative stress and hypoperfusion, resulting in limited nutrient supply [23, 29].

The initial response is intraperitoneal vasodilatation, but prolonged ischemia leads to vasoconstriction with redistribution of blood flow away from the splanchnic organs, which persists even after normalisation of intestinal blood flow. Venous congestion can lead to gut dysoxia and hypoperfusion, especially in the presence of right ventricular dysfunction. This early damage can lead to changes in intestinal integrity and can induce bacterial translocation from the lumen with the activation of systemic inflammatory pathways. Redistribution of blood flow also occurs at the microcirculatory level [29].

Recently, parameters of hemodynamics and use of catecholamines were not evaluated as potential triggers of AMI in BD [50]; only TBSA and sepsis were identified as potential risk factors, including severely burned patients with different study designs [29]. After a severe local thermal injury, approximately 25 % of all patients develop ulcers, bleeding, spontaneous perforations, and acute mesenteric infarction, which is the most severe complication. The most common causes of acute intestinal ischemia are:

- arterial embolism;
- arterial thrombosis;
- venous thrombosis;
- non-occlusive disease.

Although there is no precise data on the incidence and consequences of acute intestinal necrotizing ischemia in patients with severe burns, this complication is well known in specialised burn centres [47]. Burn patients with massive intestinal infarction have over 75 % mortality. Non-occlusive mesenteric ischemia is the most frequent cause of gastrointestinal infarction in burn patients compared to other ones, in which acute mesenteric ischemia is the result of occlusive mesenteric ischemia in 80 % of cases [3].

In the general population, acute mesenteric ischemia is found in only 1 % of patients with acute abdomen; most often, its cause is embolism and thrombosis in the area of the mesenteric vascular bed. In almost all cases, the upper mesenteric artery is affected; less often, the abdominal trunk and lower mesenteric artery. Only about 20 % of all cases of acute mesenteric infarction are caused by non-occlusive mesenteric ischemia (NOMI). In all cases of NOMI, there is a drop in perfusion in the area of the mesenteric arteries, which leads to hypoperfusion and necrosis of the intestine. Decreased mesenteric blood flow may be caused by hypotension, decreased cardiac output, vasospasm of the mesenteric arteries, or sepsis. Diagnosis of this vascular emergency in severely burned victims who are unable to report pain or nausea is difficult, so it is not surprising that this dramatic event is diagnosed in only 0.5 % of living patients. At autopsy, the diagnosis of acute mesenteric infarction increases to [29, 48, 50]:

- 2 % — in children;
- 7 % — in adults.

In critically ill patients, the feasibility of physical examination is uncertain; increased abdominal distension, peritoneal symptoms, or hematochezia require radiological evaluation [3]. The development of metabolic acidosis and increased serum lactate may also play a role as early markers of NOMI. Mortality and the risk of NOMI are higher among patients with high lactate levels and severe base deficiency. In this case, relapse of acidosis after successful resuscitation of burn shock combined with significant pneumatosis within 48 hours is an indication for urgent surgical intervention [3].

Disruption of the intestinal epithelial barrier and enteric bacterial translocation

After severe burns, intestinal ischemia and hypoxia lead to disruption of the intestinal epithelial barrier and enteric bacterial translocation (EBT), leading to serious complications such as [15]:

- systemic inflammatory response syndrome;
- sepsis;
- multiple organ failure.

Increased permeability of the intestinal mucosa and decreased expression of junctional complex proteins in the intestinal epithelium were observed after a significant burn. Cystic fibrosis transmembrane conductance regulator (CFTCR) is a protein that participates in the transport of chlorine ions across cell membranes and is widely expressed in epithelial cells. The function and expression of CFTCR in the intestinal epithelium after a severe burn are inhibited by hypoxia. The mutation causes a protein defect, a genetic disorder, and cystic fibrosis affecting the lungs, pancreas, liver, kidneys, and intestines [15].

The clinical picture of the intestinal form of cystic fibrosis is manifested by secretory insufficiency. Patients complain of dryness in the mouth, which is due to the increased viscosity of saliva. Putrefactive processes are activated in the intestine with flatulence and cramp-like pain, mostly in the epigastric area, which is due to insufficient duodenal neutralisation of gastric juice. This can cause the development of duodenal ulcers or the formation of small intestinal ulcers. Hepatomegaly is caused by cholestasis; there is a high risk of developing [15]:

- intestinal obstruction;
- secondary urolithiasis and pyelonephritis;

• disaccharidase deficiency and latent diabetes due to damage to the insular apparatus of the pancreas.

One of the mechanisms underlying the pathological process is excessive inflammation in the small intestine. Loss of CFTCR by the intestinal epithelium leads to increased release of proinflammatory cytokines, including interleukin-1β (IL-1β) and interleukin-8 (IL-8). In addition, tumour necrosis factor α (TNF-α), IL-1β, and IL-8 are known to promote denaturation of tight junction proteins (ZO-1, occludin) and induce increased intestinal barrier permeability, which may lead to intestinal inflammation, which becomes the source o a f systemic inflammatory response. In vitro studies of intestinal cells have shown that mitogen-activated protein kinase signalling is an important modulator of intestinal inflammation. Pentoxifylline attenuates burn-induced intestinal permeability and subsequent intestinal inflammation [6, 15]. Increased permeability of the mucous membrane of the GIT as a result of a burn occurs due to:

• a decrease in tight junction proteins in the intestinal epithelium;
• increased apoptosis of intestinal cells;
• impaired cell proliferation.

These effects also lead to inhibition of intestinal barrier function and bacterial translocation. A decrease in splanchnic blood circulation causes ischemia-perfusion injury of the intestines and sepsis [44].

Fibrous changes, mediated by the inflammatory state after a severe burn, contribute to a decrease in peristalsis of the small intestine [49]. Proliferation of smooth muscle cells correlates with poor tissue compliance. The proliferation of smooth muscle cells leads to the activation of the secretion of extracellular matrix (ECM) proteins. In inflammatory bowel disease, fibrous remodelling with the deposition of excess ECM proteins causes disturbances in colonic motility and absorptive function [49]. A number of studies have demonstrated that vitamin D3 exerts a protective anti-inflammatory effect on the intestinal epithelial barrier and prevents IE [15].

Syndromes of general gastrointestinal dysfunction

A severe burn is accompanied by inhibition of gastric emptying and contractility of the antrum, ileum, and colon [40, 43, 49]:

• global suppression of GIT motility is observed already 24 hours after the burn;
• the rate of gastric emptying decreases by 37—42 % after a burn already 6 h after the injury;
• small intestinal motility is reduced by 24—42 % after 6 h;
• colonic motility is reduced by approximately 34 %.

In critical burns, there are a number of syndromes of general gastrointestinal dysfunction, which include [44, 48]:

• delayed defecation;
• opioid-related bowel dysfunction;
• acute colonic pseudo-obstruction.
• In the gastrointestinal system, a burn leads to:
• increased gastric secretion;
• decreased intestinal peristalsis;
• decreased absorption of nutrients;
• increased permeability of the GIT mucosa;
• bacterial translocation;
• increased IAP.

It also increases the likelihood of mucosal ulceration and gastrointestinal bleeding. These effects cause constipation, sepsis and ACS [35, 44, 48]. In BD, 45.4 % of patients developed gastrointestinal dysfunction with various problems, including [48]:

• gastrointestinal bleeding or ulcers in 30.2 % of patients;
• nausea and vomiting in 22.1 %;
• delayed defecation in 43.0 %;
• abdominal distension in18.1 %
• diarrhea in 5.4 %.

Gastrointestinal dysfunction was involved in 88.2 % of deaths if it manifested as gastrointestinal bleeding or nausea and vomiting, but if functional symptoms such as constipation and/or diarrhea did not predominate. In general, gastrointestinal dysfunction was more common in patients with [48]:

• inhalation injury;
• burn shock;
• large burn area;
• high opiate requirement.

Constipation in burn patients is very common and multifactorial in its etiology. A high prevalence (36.1 %) of late defecation (absence of defecation within 6 days after hospitalisation in the intensive care unit) in critically ill adult patients with thermal injury was described. Late defecation may reflect global gastrointestinal motility dysfunction, as demonstrated by more frequent episodes of constipation after the first defecation, feeding intolerance, and total parenteral nutrition [44].

Gastrointestinal-related sepsis and MOFS may still occur, even if the initial GIT dysfunction is relatively mild and reversible. These effects may be related to dysbacteriosis, with the overgrowth of harmful bacteria due to disruption of the normal intestinal barrier and / or altered immune response [48].

Dysfunction of the GIT leads to acute colonic pseudo-obstruction (ACPO), which is also known as Ogilvie syndrome. Acute non-toxic megacolon is a functional obstruction of the lower parts of GIT; first described by W. H. Ogilvie (1948) in burn

patients [11], it is a rare state characterized by acute dilatation of the colon in the absence of mechanical obstruction. This manifestation of intestinal pseudo-occlusion is associated with several etiological factors [29, 38, 48, 50]. Burns and antipsychotic drugs have been identified as risk factors. Opiates, such as fentanyl, are the drugs most commonly implicated in Ogilvie syndrome; their effect on intestinal transit is diverse:

- increasing the tone of the ileocecal and anal sphincters;
- reducing the peristalsis of the small and large intestines;
- reducing the sensitivity to stretching;
- changing the defecation reflex.

All these factors contribute to the slowing of transit and lead to constipation.

Ogilvie syndrome is a more serious complication than those that can develop in some patients after the use of opiates. Sedatives, such as midazolam, can also disrupt intestinal motility through inhibition of vagal tone. Studies of patients receiving analgesia with benzodiazepines have demonstrated abnormalities in intestinal motor activity, including increased contractions, which may lead to pseudo-obstruction syndromes. These abnormalities were reversible after drug discontinuation [25]. The incidence of this syndrome ranges from 0.5 to 1 % in burn centres in patients with more than 15 % of the body surface affected by burns [25, 33]. Functional obstruction leads to a colonic reflex that impairs motility and increases dilation.

- ACPO is characterised by [50]:
- massive distension of the colon in the absence of mechanical obstruction (80—90 %);
- abdominal pain and hypertympanism (80 %);
- nausea and / or vomiting (60 %);
- constipation (40 %);
- fever (37 %),
- and also by tachycardia and leukocytosis.

Analgesics and sedatives used in burn patients may also be associated with this state and are thought to be secondary triggers for vagal depression [33]. The course of ACPO in burn patients is significantly different and often more complicated than in abdominal ones without thermal injuries [12]. The clinical picture of ACPO is often vague, no sign is pathognomonic for the disease. Abdominal bloating, with or without pain, is the most common symptom. These patients often have constipation, although the ability to defecate or pass gas is preserved in 40 % of patients [12]. Contrast-enhanced abdominal tomography is the ideal imaging study to evaluate a possible site of occlusion or complication [33].

Patients with Ogilvie syndrome are usually treated conservatively if the diameter of the distended bowel is less than 12 cm and there is no evidence of intestinal ischemia or perforation. The treatment is carried out with the help of a nasogastric and transrectal probe for decompression within 48—72 hours along with correction of hydroelectrolyte imbalance, withdrawal of sedatives and treatment of infections. Conservative treatment during the first two to six days is effective in (83—96) % [33]. In case of deterioration or expansion of the intestine by more than 12 cm, medical or surgical treatment is indicated. Intravenous administration of neostigmine, which is a reversible acetylcholinesterase inhibitor that indirectly inhibits muscarinic receptors and promotes colonic motor activity, has been recommended [12, 33]. Bowel perforation is the most serious complication of HPTC, the risk of which increases significantly with the duration of bowel distention. General guidelines indicate that a cecal diameter over 9 cm is abnormal, and a diameter over 12 cm is at high risk of perforation. The thin-walled cecum is the least adapted to a sharp increase in intraluminal pressure (according to Laplace law) [12]. Ulceration and necrosis of the colon in burn patients are rare and usually occur in the cecum with the formation of stercoral peritonitis with a negative prognosis. The risk of perforation increases with age and is higher in men [5, 25]. Among the complications [20, 35, 37]:

- 26 % of patients had an abdominal catastrophe (infarction / perforation);
- 37 % had bleeding from the upper parts of the GIT;
- 32 % had paralytic intestinal obstruction;
- 5 % developed pancreatitis and acute necrotizing cholecystitis.

Small bowel intussusception is a rare clinical state in BD with symptoms of intestinal obstruction such as:

- abdominal pain;
- vomiting;
- rectal bleeding;
- food intolerance.

It requires an emergency laparotomy to eliminate intestinal obstruction; otherwise, ischemia, necrosis, and intestinal perforation may develop [25, 46].

Violation of the GIT barrier function is an important initiator and stimulator of the systemic inflammatory response syndrome, sepsis, and MOFS in BD [54]. The functionality of the gastrointestinal barrier can be impaired along with increased intestinal permeability and subsequent translocation of bacteria/endotoxins through:

- autophagy in the visceral organs;

- expression of tight junction proteins and inflammatory cytokines;
- disruption of the gut microbiota;
- low blood flow;
- septic shock;
- administration of catecholamines;
- stress of the endoplasmic reticulum,
- mucosal atrophy;
- lack of enteral nutrition.

A disturbed intestinal epithelial barrier is responsible for systemic inflammatory response syndrome, sepsis, multiple organ dysfunction syndrome, and other severe complications [58]. The translocation of endotoxins from the intestine has been identified as a factor in mortality [30]. An increase in the growth of intestinal bacteria after a burn injury has been noted as a result of decreasing intestinal immunity, hypoperfusion, and impaired intestinal motility. An increase in intestinal permeability has been documented for several hours to several days after burn injury, allowing intestinal bacteria to enter extraintestinal sites (mesenteric lymph nodes, liver, and lungs). The process recurs when additional triggers of intestinal hypoperfusion occur (blood loss, sepsis). The faecal microbial communities of healthy people are dominated by the following families:

- *Bacteroidaceae*;
- *Lachnospiraceae*;
- *Ruminococcaceae*.

Faecal samples from patients with burn injuries show a marked decrease in the relative abundance of these three families, but a dramatic increase in the relative abundance of *Enterobacteriaceae* [10].

Diarrhea often occurs in BD, which is explained by a violation of intestinal integrity and dysbacteriosis. The prevalence of nosocomial diarrhea in BD was 120/10 thousand, and 21 % of these were caused by *C. difficile* infection [54]. Soluble fibre can improve intestinal barrier function and prevent bacterial translocation. Soluble fibre is rapidly fermented by commensal bacteria and produces short-chain fatty acids [34, 55].

The hypermetabolic response to burn injury is highest among critically ill patients [1]. In patients with burns, gastrointestinal stasis leads to impaired digestion and absorption; this increases concern about protein nutrition, increasing the burden on the kidneys [27]. As a result of insufficient nutrition, the intestinal mucosa is damaged (autophagy), which leads to bacterial translocation and gram-negative sepsis [39]. In the early stage of BD, intestinal adynamia may occur, with a high risk of acute gastric ulceration, which can be reduced by early enteral feeding aimed at maintaining body weight and endocrine homeostasis [3, 37, 39, 40, 43].

Dysphagia of various degrees was found in 27.78 % of people; its risk factors include [57]:

- TBSA over 18 %;
- older age;
- head and face injuries;
- the presence of inhalation trauma.

Patients with a single residual gastric volume of no less than 250 mL were classified as having gastrointestinal motility disorders. There are three main problems with enteral nutrition [39, 40]:

- gastric ulcer;
- intestinal obstruction;
- mucosal damage.

Post-pyloric administration of nutritional mixtures is a modern standard method of enteral feeding [3, 43]. Parenteral nutrition should be used selectively for specific conditions such as paralytic ileus, pancreatitis, intestinal obstruction, or contraindications to enteral feeding, as it is associated with a higher rate of infection due to prolonged access to central veins [39]. A combination of [13]:

- early optimal infusion resuscitation;
- early initiation of enteral nutrition (within 24—48 hours after injury);
- prevention of gastric ulcers;
- use of prokinetic drugs;
- avoidance of NSAIDs and steroids
- allows, in most cases, to prevent gastrointestinal dysfunction in patients with severe burns.

Conclusions

Unfortunately, very little attention is paid to the pathophysiology of the digestive system in case of skin burns in available domestic literary sources.

Based on the analysis of foreign publications, we conducted a certain generalisation of medical reports on the pathological changes of the GIT in burn disease.

The authors hope that the collected information will be useful for combustiologists, gastroenterologists, and doctors of other specialties.

Declaration of interests

The authors declare that there is no conflict of interest and their own financial interest in the preparation of this article.

Authors contributions

O. V. Kravets, V. V. Yekhalov: conceptualization, writing the original text; V. V. Gorbuntsov: editing, translation.

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Патофізіологія шлунково-кишкового тракту при опіковій хворобі

О. В. Кравець, В. В. Єхалов, В. В. Горбунцов

Дніпровський державний медичний університет

На основі аналізу зарубіжних публікацій проведено узагальнення медичних повідомлень щодо патологічних змін шлунково-кишкового тракту (ШКТ) при опіковій хворобі. Опікова хвороба від самого початку супроводжується негайною реакцією всіх органів та систем, які не завжди здатні підтримувати гомеостаз і здебільшого набувають патофізіологічних та морфологічних ушкоджень. До таких органів-мішеней належить ШКТ. Лише в дуже рідкісних випадках тяжкі (переважно електричні) опіки спричиняють пряму травму органів черевної порожнини, яка супроводжується тяжким клінічним перебігом та високою летальністю. Пацієнти всіх вікових груп, які перенесли опікову травму, мають підвищений загальний ризик розвитку шлунково-кишкових захворювань (патологію стравоходу, шлунка, кишечника, ураження жовчного міхура, жовчних шляхів і підшлункової залози). При площі опіку 40—95 % у 5,7 % постраждалих було діагностовано патологію органів черевної порожнини, серед них у 26,0 % виникла абдомінальна катастрофа (інфаркт/перфорація), у 37,0 % — кровотеча з верхніх відділів ШКТ, у 32,0 % — паралітична кишкова непрохідність, у 5,0 % — панкреатит та гострий некротичний холецистит. Великі опіки зазвичай асоціюються зі значним зниженням спланхічної перфузії. Ішемія кишечника та гіпоксія після тяжкого опіку спричиняють порушення кишкового епітеліального бар'єра та ентеральної бактеріальної транслокації, що призводить до серйозних ускладнень — синдрому системної запальної відповіді, сепсису, поліорганної недостатності. Дисфункція ШКТ спричинила близько 88,2 % смертей, якщо вона ускладнювалася перитонітом або шлунково-кишковою кровотечею. Загалом дисфункція ШКТ була більш поширеною в пацієнтів з інгаляційною травмою, опіковим шоком, великою площею опіку та високою потребою в знеболювальних препаратах.

Ключові слова: лікування, огляд, опікова хвороба, патофізіологія, шлунково-кишковий тракт.

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