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## Tumor intestinal obstruction as a cause of ischemic enterocolitis (review and case report)

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**ABSTRACT** *AIM: to analyze, based on literature data, a clinical case of total necrotizing obstructive enterocolitis in a patient with CO.*

*CLINICAL CASE: a stable patient with no comorbidities developed septic shock within hours following tumor stenting for CO. The underlying cause was ischemia of both the small and large intestines. The patient required subtotal colectomy and prolonged treatment in the intensive care unit to achieve recovery.*

*CONCLUSION: colon ischemia in the context of CO remains an under-researched condition, with undefined diagnostic criteria and a high postoperative mortality rate. This issue warrants close attention and broader discussion within the medical community.*

**KEYWORDS:** colonic obstruction, obstructive colitis, enterocolitis, ischemia of the colon

**CONFLICT OF INTEREST:** the authors declare no conflict of interest

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## INTRODUCTION

Colorectal tumor obstruction (CO) develops in about 20% of patients with colon and rectal tumors, is accompanied by high mortality and morbidity, and requires long-term inpatient treatment. In 2019–2020, CO was detected in 20,653–22,211 patients in the Russian Federation and was followed by a postoperative mortality rate of 15.39% — 17.05%. In uncomplicated tumors, mortality was about 5% [1,2]. The main causes of death include infectious complications (progressive peritonitis), multiple organ failure, and intestinal ischemia (II) [3]. Increased intraluminal pressure, excessive bacterial growth in the intestinal lumen, overgrowth and ischemia of the intestinal wall are accompanied by translocation of microorganisms and toxins into the lymphatic system and systemic bloodstream [4], which is considered as the causes of infectious complications

and sepsis. In 1.6–4.1% of patients, CO is followed by perforation [5,6], in which mortality reaches 62% [7]. The risk of perforation increases with dilation of the cecum greater than 10 cm [8,9]. To describe ischemic changes in patients with CO, many authors use the term of “obstructive colitis” (OC) or enterocolitis (OEC) [10], which is defined as ulcerative–inflammatory lesion of parts of the small or large intestine located proximal to the site of obstruction [11]. In addition to CO, ischemia of the colon (IC) has been described in: heart diseases (arrhythmias, heart failure, shock), vascular diseases (embolisms, thrombosis, vasculitis), infectious diseases (E. coli, hepatitis B, cytomegalovirus), iatrogenic effects (surgeries on the aorta), under the influence of pharmacological drugs and physiological conditions (running for long distances) [12,13]. IC has been described as a rare complication after coronary angiography [14], myocardial infarction [15], colonoscopy, etc.

At the same time, it is rarely possible to identify the true origin of an ischemic event in each individual patient [16].

## AIM

To analyze the clinical case of total necrotizing obstructive enterocolitis in a patient with tumor intestinal obstruction.

### Terminology

The term of "OC" has been used for over 30 years [10]. Despite this, the term has not become generally accepted among clinicians and pathologists, although PubMed offers 2,085 publications on the topic [17]. "OC" is not identified in the e-library, English-language and Russian clinical guidelines on CO, and most publications on the topic are descriptions of clinical cases or small samples. Apparently, OC should be considered as a special case of IC [16]. The number of publications on IC is significantly higher than on OC, and includes, in addition to individual cases, significant clinical series, population-based studies, which in 2015 were summarized in the clinical guidelines of the American Society of Gastroenterology (SAGES) [18]. It should be noted that along with ILI the term of "ischemic colitis" (IC) is used. Formally, this term reflects the inflammatory response to ischemic injury, although it is often used as a synonym for ILI.

Ischemic enterocolitis (IEC), like obstructive enterocolitis (OEC), is an even rarer condition [19]. There is almost no information about the extent of lesion of the small intestine, the mechanism of lesion, and the outcomes of IEC and OEC. It is reported that ischemic lesions in the small intestine in OEC are localized mainly in the mucosal-submucosal layer and spread 10–150 cm proximally from the ileocecal valve [20,21].

### Epidemiology of IC

The detection of IC, regardless of the causes, has been on an increasing trend over the past 30 years, primarily due to improved diagnosis. In the period

between 2005 and 2009, the detection rate of IC was 22.9 cases per 100,000 population [16]. These data may not fully reflect the actual indicators, because most patients have mild IC, which is why patients rarely seek medical help, and even when they do, IC is not diagnosed in all [22]. The incidence of OC among patients with CO is indicated at 0.3–14% [17,23–25].

### Etiology and Pathogenesis of IC and OC

**IC.** The concept of IC pathogenesis is based on the hypothesis of hypersensitivity of the colon to ischemia. As indicated, the blood supply to the colon has areas vulnerable to ischemia, in particular, at the level of the hepatic, splenic bends of the colon and sigmoid colon [16].

At the same time, a violation of the main blood flow is usually not the main cause of IC; only 20% of patients develop IC due to the cessation of blood flow in the main vessels. In most patients, IC develops due to incoming (temporary) hypoperfusion of the intestinal wall due to spasm, vasoconstriction, thrombosis of the microcirculatory course against the background of critical conditions, hypovolemia, hypercoagulation [21,26]. Ischemia, followed by restoration of blood flow and reperfusion injury, is accompanied by the release of free radicals, apoptosis of cells of the intestinal mucosa and microcirculatory course, translocation of microorganisms and endotoxins. The severity of the injury depends on the duration of ischemia and the extent of the involved vascular basin [16,27–29].

Gordeeva A.E. et al. consider reperfusion playing a leading role in the pathogenesis of ischemic intestinal injury. Pronounced edema of the intestinal wall due to lesion of the microcirculatory course and endotheliocytes is a characteristic feature of reperfusion injury [30]. Knowledge of these features can be useful for the diagnosis of IC.

**OC.** Experimental studies have established significant changes in blood circulation in the intestinal wall in OC, in particular, a two-fold increase in blood supply [31] against the background of hypovolemia, hypotension, and compression of

intramural vessels. An increase in pressure in the intestinal lumen above 35 mm of water column for several hours was accompanied by ischemic injury to the intestinal wall [32]. In the experiment, OC occurred significantly more often and affected a larger area with full than with partial OC [33]. There is an unconfirmed hypothesis that the severity of intestinal lesion may be related to the rate of reperfusion. It is suggested that rapid decompression of the large intestine may contribute to reperfusion lesion.

### **Characteristics of Patients. Symptoms**

The mean age of 3,241 patients with IC included in a recent systematic review was 70 (19–98) years, of whom 58.2% were women [34]. Usually, IC and OC are initially manifested by bleeding from the rectum and abdominal pain, as well as nausea and vomiting, i.e. symptoms that are clinically indistinguishable from colorectal cancer [35]; however, they are grounds to suspect IC and conduct a targeted check up.

### **Diagnosis of IC**

Laboratory changes are not specific to IC, but they are, nevertheless, useful for assessing the severity of the disease and choosing treatment approach. Organ dysfunction and sepsis are indicated as criteria for severe IC requiring urgent measures, and abnormal laboratory tests are indicated as a predictor of an unfavorable outcome [16]. Of the instrumental methods, CT and colonoscopy are the most informative, the results of which, complementing each other, make it possible to assess the severity and danger of ischemic injury.

For dynamic CT, a phasicity of changes can be detected depending on the predominant damaging mechanism: ischemia or reperfusion. In the phase of *ischemia*, dilatation of the intestine is observed with a characteristic expansion of its lumen and thinning of the wall to the “thickness of paper” with the formation of diffuse fluid of the abdominal cavity or retroperitoneally. Pneumatosis and a decrease in intestinal wall density, pneumoperitoneum, dilatation of the lumen greater than 5

cm are indicated as signs of irreversible intestinal injury, although the specificity of these signs is quite low.

*Reperfusion*, due to injury to the endotheliocytes of the microcirculatory course and impaired permeability of the intestinal wall, is accompanied by extravasation of fluid from the vascular lumen into the tissue. As a result, the intestinal wall looks thickened (more than 1 cm). The mucosa may have increased density due to hyperemia and hemorrhagic phenomenon (the double halosign). The intestinal lumen, unlike ischemia, decreases. Diffuse fluid increases around the intestine. Intestinal fragments with predominant ischemic lesion are often combined with areas where reperfusion injury prevails [36]. CT may be important as a screening method for diagnosing obvious irreversible injury or perforation. Subsequent colonoscopy makes it possible to assess the degree and prevalence of ischemia, especially during the first 48 hours after the onset of IC symptoms [18]. The following signs of IC were found in 297 patients during colonoscopy: hyperemia (83.7%), edema (69.9%), slight injury (42.6%), superficial ulceration (57.4%), deep ulceration (21.7%), narrowing and stenosis of the lumen (8.4%), intraluminal blood (8.4%), and blue-black nodules on a dark background, indicating gangrene (5.5%) [37]. It is emphasized that colonoscopy in case of suspected IC is carried out without preparation, which is not generally accepted. Angiography is rarely used, as it is almost never possible to identify blood flow disorders in large vessels [38].

### **Diagnosis of OC**

Colonoscopy, CT scan, barium or water-soluble enema are useful for the diagnosis of primary tumors and IO, but rarely allow to establish OC and, moreover, the degree of ischemic lesion to the intestinal wall. Assessment of the viability of dilated thinned intestinal wall on the background of IO is a significant difficulty [24]. CT scans of signs of non-viability (intramural gas, impaired accumulation of contrast agent, gas in the portal vein system) did not show high accuracy in diagnosing

irreversible ischemic lesion of the intestine. For example, transmural necrosis of the cecum on CT scan was detected only in 26% of patients with intramural gas [39]. Nevertheless, Ko G.Yu. et al. (1997) managed to differentiate ischemic segments of the intestine (ISI) from those affected by a tumor based on CT assessments of their thickness, heterogeneity, and the nature of blood supply [40]. In a study involving 308 patients, PETCT demonstrated a higher informative value than CT, which allowed to establish OC in 9.4% of patients with CO [25].

### ***Intraoperative Diagnosis of IC and OC***

Intraoperative visual assessment is not informative in case of lesion of the mucosal-submucosal layer; obvious changes on the serous layer are detected only in case of transmural irreversible injury [26,34,41].

Changes corresponding to OC are often detected as an accidental finding when examining a removed specimen [23,42]. New technological tools such as Near-infrared indocyanine green (NIR — ICG) angiography have potential advantages [43]. A recent study has shown the possibility of more accurately determining the extent of ischemic injury and the boundaries of resection in OC, since the hypoperfusion area established by ICG angiography was 8–25 cm longer than according to the results of the visual assessment [44].

### ***The Depth and Extent of the Lesion with OC***

In OC and OEC, changes are more often detected in the mucous and submucosal layers of the large or small intestine [32]. Toner, M. et al. found focal changes in the form of limited ulcers with a diameter of 0.5–2 cm or in the form of merging circular lesions with a length of 8–25 cm, well separated from the normal mucosa [10]. Transmural lesion is rarely described [41]. Emoto, S. et al. found no cases of transmural lesion among 40 patients with OC [24].

As a characteristic feature of OC, most authors indicate the preservation of a portion of the normal mucous layer between the margins of colitis and

the tumor [17,23]. The length of this section can range from 2.5 to 60 cm [10, 23]. Because of this feature, even a targeted examination of the mucosa along the resection line may be uninformative [42].

### ***Histological Changes with OC***

Regardless of the severity and nature of the disease, a diagnostic sign is the presence of normal (non-inflamed) sections of the intestine of variable length separating the obstruction zone from the OC area. In the absence of an obvious heart attack, inflammation of the mucous layer and hemorrhage into the submucosa develops. Fibrin thrombi may occur, other than those resulting from ulceration. Usually, partial loss and degeneration of crypts with characteristic opaque eosinophilia of its own plate is observed in the ischemic mucosa. Macrophages saturated with hemosiderin may be an additional clue in the diagnosis of OC [23].

### ***Surgical Approach***

**IC.** About 25% of patients with IC undergo surgery, more often with right-sided injury than with ischemia of other segments, respectively: 53.6% and 14.5% [34]. The choice of treatment method and prognosis are related to the severity of IC. Male age, hypotension (blood pressure < 90 mmHg), tachycardia (heart rate > 100 beats/min, abdominal pain without rectal bleeding, and urea > 20 mg/dl, Hb < 12 g/dl, LDH > 350 U/L,  $\text{Na}^+ < 136$  mEq/L (mmol/L),  $\text{WBC} > 15 \times 10^9/\text{L}$ ) are indicated as risk factors for an unfavorable outcome. The IC of a mild course is established in the absence of risk factors, moderate — in the presence of 3 or fewer risk factors, and severe — more than 3 risk factors [18]. In moderate to severe cases of IC, antibacterial therapy and consultation with a surgeon are recommended. Severe course is the basis for admission to the ICU [34]. Irreversible lesion (gangrenous colitis), as well as progressive organ dysfunction, is indicated as an indication for surgery [16]. There is evidence of a more severe course of right-sided colitis, in particular, a higher incidence of gangrenous changes [34]. The

ACG recommendations indicate peritonitis, massive bleeding, fulminant colitis with toxic dilation of the colon (TDC), portal and intramural gas on CT, and worsening of the condition [18]. Intestinal dilation on CT scan, organ dysfunction, and high lactate levels are indicated as predictors of irreversible intestinal ischemia [45].

**OC.** Unlike IC of other etiology, where conservative treatment is effective in most patients, single-stage or staged surgeries are the main type of treatment in patients with OC. At the same time, there is no obvious evidence of the effect of OC on outcomes. In one study involving 56 patients with OC, “total or subtotal colectomy” with primary anastomosis was not accompanied by serious complications. A staged treatment with stenting or probe decompression at the first stage is indicated as an acceptable option in patients without total ischemic injury and perforation [21,24].

In the series, which included 43 patients, the second stage was successfully resected, usually with primary anastomosis, which was accompanied by the only postoperative complication — bleeding from the anastomosis [24]. Successful probe decompression of CO during 10 days and subsequent successful resection was reported by Matsuda, T. et al. During the operation, severe edema and ulceration of the terminal ileum were detected [21].

### ***Mortality and Adverse Events with IC and OC***

**IC.** Organ dysfunction and sepsis are indicated as the main causes of fatal IC outcomes [34]. Mortality among patients with IC who underwent colectomy is indicated at the level of 25–79% [46–49]. In a study from the USA, which included 4,548 patients with colectomy for all types of IC in the period between 2010 and 2015, the 30-day postoperative mortality was 25.3%. Age, comorbidity index, functional status, preoperative septic shock, acute renal failure, preoperative hemotransfusion, and surgery delay of more than 3 days after hospitalization were identified as factors of 30-day mortality [50]. According to a recent systematic review, right-sided or total colitis

was the main predictor of the need for surgery and an unfavorable outcome.

With right-sided IC, 19.7% of patients died, while 9.1% of the rest died. The mortality rate was also significantly higher after surgery than after non-surgical treatment, respectively: 39.3% and 6.2% [48, 51].

**OC.** The analysis of the OC studies revealed several other indicators. Although cases of OC with septic shock and fatal outcomes have been described [41,52,53], several studies have shown that OC have no effect on the outcome of CO.

In the largest of them, mortality among patients with OC did not differ from the other patients with CO and was less than 4% [17]. Among the rare adverse events associated with OC, failure and bleeding in the anastomosis area and colostomy dysfunction are indicated [24,54]. We offer clinical observation of a patient with OEC and septic shock. This observation of OC obviously went beyond the disease, which “does not affect outcomes.” At the same time, OC was not diagnosed by CT, colonoscopy, intraoperative and primary histological examination.



**Figure 1.** Computed tomography of the abdominal cavity with intravenous reinforcement revealed a tumor of the sigmoid colon and intestinal obstruction during hospitalization



**A typical start.** A 60-year-old man was hospitalized with a 3-hour history of abdominal pain, nausea and repeated vomiting. The physiological status was satisfactory. Hypersthenic. The BMI was 28.5 kg/m<sup>2</sup>. The abdomen was swollen, painful, without peritoneal symptoms. The laboratory parameters were within subnormal values. A CT scan revealed a tumor of the sigmoid colon, intestinal obstruction: the diameter of the colon was 7 cm, the small intestine was 1–1.5 cm (Fig. 1). Without technical difficulties, the patient underwent stenting of tumor stenosis with a self-expanding metal stent.

**Something went wrong: sepsis, septic shock.**

After waking up, the patient stopped making contact (in a retrospective survey, the patient remembered only the stage of transportation for a colonoscopy). Due to the increasing multiple organ failure (lactate — 5 mMol/L, procalcitonin — 9.3 ng/ml), the patient was transferred to the ICU. On the background of infusion, antibacterial, and renal replacement therapy (RRT), by day 3, the patient's condition corresponded to septic shock and required catecholamine support, ALV, and RRT. The growth of *Escherichia coli* was detected in the blood.

**CT.** On a series of CT scans, the dilatation of the colon remained up to 6 cm in dynamics (Fig. 2). From

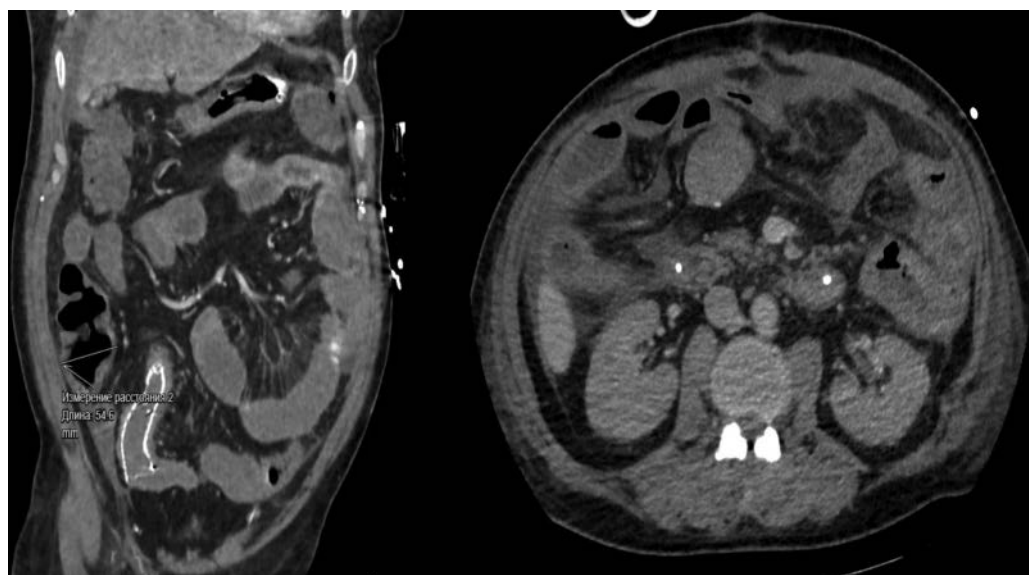
day 2, dilatation of the small intestine appeared, and by day 3, edema of the walls of the small and large intestines with diffuse fluid accumulations had grown. Sigmoidoscopy performed 52 hours after stenting showed the patency of the stent and the absence of any significant changes, except for dilatation of the proximal colon and hyperemia of the mucosa immediately proximal to the stent.

**A toxic megacolon? Colectomy.** By day 3, toxic dilation of the large intestine was suspected.

The council decided on a colectomy. During surgery, there was a serous effusion in the abdominal cavity, a stenosing tumor of the sigmoid colon, and the correct location of the stent.

The adducting sections of the colon were dilated to 6 cm, visually viable, although the intestinal wall was duratively altered, which visually and palpationally corresponded to hypertrophy of the intestinal wall. The small intestine was up to 4 cm in diameter, and its serosa was not visually altered. Subtotal colectomy with D2 lymphodissection, 25 cm resection of the ileum and ileostomy was performed. The wall of the small intestine along the resection line was dense, the mucous layer was pink.

**The results of the examination of the surgical specimen.**



**Figure 2.** By the 3rd day of inpatient treatment, dilation of the colon persists with a properly functioning stent. Dilation and swelling of the small intestine wall appeared. Significant diffuse fluid accumulations appeared in the abdominal cavity.

**Seeding from the abdominal cavity:** sterile.

**The result of the histological examination:**

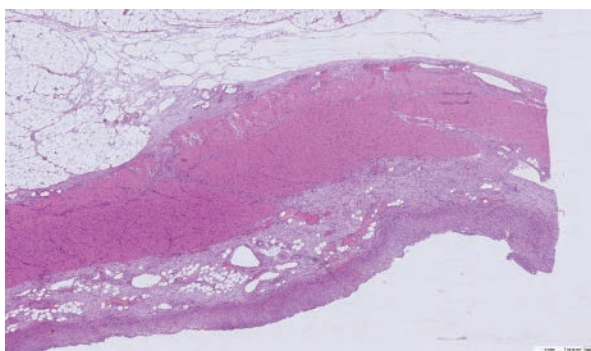
Highly differentiated adenocarcinoma of the sigmoid colon (G2, pT2, pN2a (5/36), L1, V0, Pn0, R0). In the mucous layer of the small and large intestines, proximal to the tumor, total necrosis of the mucous and submucosal layers and extensive ulceration with mild or moderate leukocyte infiltration, edema and hemorrhages in the submucosal layer, vascular dilatation, unevenly expressed fullness of veins, multiple fibrin and red obturating thrombi in the lumen of capillaries and small veins were found; signs of myolysis in the muscle layer with its thinning were detected. Similar changes were found in the ileum (Fig. 3). Due to the fact that the etiology of necrotic changes was not established, the specimens were consulted at the Russian Federal Coloproctology Center.

The changes in the adductor sections of the large intestine were regarded as ischemic enterocolitis on the background of a stenosing tumor of the sigmoid colon.

**Postoperative Treatment**

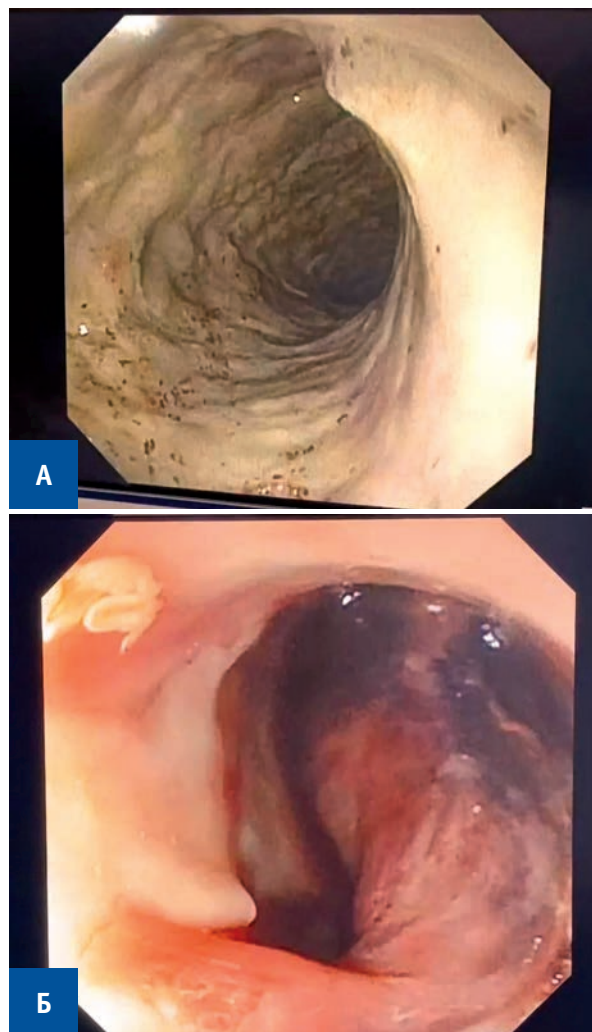
The patient underwent the surgery satisfactory — inotropic support with norepinephrine did not exceed 0.3–0.5 mk/kg/h. However, the surgery did not lead to an improvement in the patient's condition. The patient did not absorb enteral nutrition for more than 30 days. The stoma did not function for 14 days, after which recurrent intestinal

bleeding appeared. Enteroscopy (through a stoma) showed massive lesion of the mucous layer — ulcerative necrotic changes, which decreased, but remained significant by day 20 (Fig. 4a, b). At the same time, although the stoma looked viable, a pronounced edema of most of the wall remained on the CT scan. Against the background of sepsis-induced coagulopathy, several episodes of intra-abdominal bleeding occurred after surgery (the source was the vessels of the large omentum), for which relaparotomy was performed twice. By **day 20**, several hematomas with a total volume of about 1 liter had formed in the abdominal cavity, which were treated conservatively. A cava



**Рисунок 3.** Микрофотографии участка восходящей ободочной кишки. Увеличение  $\times 200$ , шкала 600 нм. Окраска гематоксилин-эозином. Тотальный некроз слизистой с геморрагической имбицией.

**Figure 3.** Micrographs of the ascending colon section. Magnification  $\times 200$ , scale 600 nm. Hematoxylin-eosin staining. Total necrosis of the mucous membrane with hemorrhagic imbibition.



**Figure 4.** A — Enteroscopy on the 9th day after surgery. Total necrosis of the ileum mucosa within 15 cm of the ileostomy. Б — Enteroscopy on the 20th day after surgery. Fibrinous overlays on the ileum mucosa. The intestinal lumen is formed by a blood clot.

filter was installed in connection with ileofemoral thrombosis. ALV and RRT continued for 2 months. The total duration of ICU treatment was 105 days. The dynamics of procalcitonin levels during the treatment is shown in (Fig. 5).

**Discharge from hospital.** The patient was discharged to the rehabilitation center on the 137th day from the moment of hospitalization with a loss of 40% of body mass.

**What is going now?** 6 months after the discharge, weight gain was about 10%, renal failure that does not require RRT persists, anemia (Hb 100 g/l), hypoproteinemia (60 g/l), albumin level — 30 g/l. There are cicatricial changes in the terminal part of the ileum, requiring periodic bougie. There are no signs of progression of the tumor and infectious process. The issue of constructive surgery is being discussed.

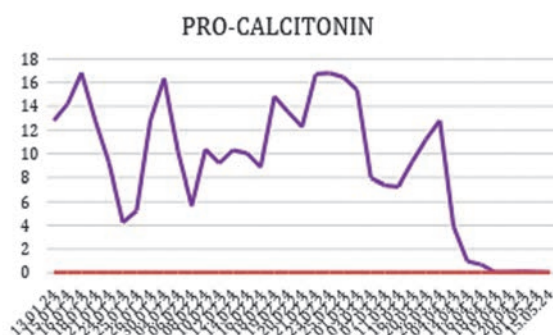
### **Discussion of the Problem Status and Surgical Tactics Used in the Patient**

The problem of IC has been actively discussed for several decades, but there have been no breakthrough discoveries to solve it. Since the release of the only recommendations in 2015, dozens of new publications have appeared, which, however, have not fundamentally changed the state of the problem and have not led to an update of the guidelines. A significant part of the questions of etiology, diagnosis, treatment and prevention of

IC remain unanswered. Existing diagnostic methods: CT, colonoscopy and morphology in reality can detect only non-specific signs of lesion of the large intestine, similar to colitis of infectious origin and Crohn's disease, in connection with which Carlson, R.M., et al.'s doubts were expressed about the generally accepted ischemic theory of IC. In particular, the authors found no evidence of "a special vulnerability of the colon to ischemia," and questioned whether IC was associated with critical conditions and patient instability [55]. It was impossible to assess the prevalence and actual clinical significance of OC based on the literature studied. The opinions of experts range from "it does not matter" to "deadly dangerous", as in the case of our patient. Several studies have shown a significant incidence of ulcerative lesions of the adductor intestine in obstruction, but these changes were not life-threatening, and their treatment was not accompanied by complications. We could not find any analytical data on gangrenous diseases.

For surgeons in Russia, the problem of IC and OC is obviously not considered among the priorities. According to the established clinical practice of emergency units, the cause of segmental or total necrosis of the large intestine is usually indicated by a violation of the arterial circulation, and non-transmural IC is not detected at all. The incidence of IC in Russia has not been studied [56]. Based on statistics from international studies, the annual number of patients with IC in Russia may exceed 30 thousand. Erosive colitis of unknown etiology is indicated as the cause of intestinal bleeding in 40% of patients [57]. At the same time, according to the English-language literature, most of them have colitis of ischemic origin. In the treatment of this patient, IC was also not considered as the cause of the rapid deterioration of his condition, and the genesis of the lesion was not established even by the results of the initial morphology. At the same time, an early surgery (up to 3 days) with necrotizing OC is indicated as life-saving.

A retrospective CT scan showed a typical picture of ischemia — reperfusion of the large intestine



**Рисунок 5.** Динамика изменения уровня прокальцитонина (нг/мл). За период с 13.01.24 по 14.04.24 уровень прокальцитонина оставался значительно выше нормальных показателей.

**Figure 5.** Dynamics of procalcitonin levels changes (ng/ml). During the period from 01/13/14 to 04/14/14, procalcitonin levels remained significantly higher than normal.



with increasing swelling of the intestinal wall, dilatation of the large and small intestines. At the same time, sigmoidoscopy on the 3rd day after stenting did not reveal any anomalies, which may be due to the peculiarities of lesion of the intestinal wall with the preservation of an unchanged mucosal area between the tumor and the lesion. Examination of the large intestine during surgery did not allow us to fully assess the extent of lesion of the intestinal wall. Surgeons found an intact serous layer, which is usually described with non-transmural lesion. If such a bowel was found, without an appropriate preoperative examination, most surgeons would hardly have decided on a colectomy. Admittedly, the surgery did not eliminate sepsis, which had been fighting for several months. A possible source of sepsis could be the small intestine, the volume of gangrenous changes in which became clear after the surgery. In the available literature, we have not been able to find successful cases of treatment of such extensive enterocolitis. Was a more extensive surgery necessary, including resection of the small intestine? This surgery volume was discussed after the surgery, when the lesion extent became clear. Although extended resections are indicated as a risk factor for an unfavorable outcome in IC [58], it is clear that such surgeries are required by patients with an appropriate lesion scope. The question of the validity of colectomy also cannot be considered unambiguously resolved, since transmural lesion has not been established. A recent publication described a patient with total necrotic OC and septic markers, although without organ dysfunction, who was successfully cured by repeated endoscopic decompression of the large intestine [59]. Finally, there is currently a question about the feasibility of reconstructive surgery. Anemia, hypoproteinemia, and persistent body mass deficiency cannot

be corrected for 6 months, probably due to lesion of the small intestine, malabsorption syndrome, and persistent renal failure.

## CONCLUSION

Obstructive ischemic colitis (enterocolitis) has no generally accepted explanation, is not recognized by pathologists and clinicians as an independent disease, and is not considered in most guidelines on intestinal obstruction. Perhaps that is why the unexplained severity of the patient's condition after successful tumor stenting came as a complete surprise to the attending surgeons. The emergency measures taken to save the patient may not have been fully timely and/or sufficient, including due to insufficient awareness among doctors. Therefore, the problem of the large intestine ischemia requires close attention and broad discussion not only by surgeons, but also by doctors of other specialties.

## AUTHORS CONTRIBUTION

Concept and design of the study: *Aleksandr E. Tyagunov, Aleksandr V. Sazhin*  
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