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Cytomegalovirus Colitis: A Case Report

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Abstract
We report a case of cytomegalovirus (CMV) colitis. The patient (a woman with a provisional diagnosis of Crohn disease) presented with severe abdominal pain, diarrhea, and weight loss. Despite difficulties in differential diagnosis, we were able to make the correct diagnosis based on findings of the histological examination of the biopsy specimens obtained during colonoscopy: intranuclear inclusions characteristic of CMV and specific CMV-associated changes in the intestinal mucosa.
This case highlights the importance of histopathological examination in differential diagnosis of inflammatory diseases of the gastrointestinal tract.
Keywords: cytomegalovirus, cytomegalovirus colitis, immunosuppression, pathology
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Цитомегаловирусный колит (клинический случай)

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Резюме
В данной статье представлен клинический случай цитомегаловирусного колита. У пациентки – молодой женщины с предварительным диагнозом «болезнь Крона» – наблюдались сильная боль в животе, диарея и потеря веса. Несмотря на трудности дифференциальной диагностики, удалось поставить правильный диагноз по результатам гистологического исследования биоптата, полученного при колоноскопии: наличие внутриядерных включений, характерных для цитомегаловируса, и специфические изменения слизистой оболочки кишки, связанные с цитомегаловирусом.
Данный клинический случай подчеркивает важность патогистологического исследования в дифференциальной диагностике воспалительных заболеваний желудочно-кишечного тракта.
Ключевые слова: цитомегаловирус, цитомегаловирусный колит, иммуносупрессия, патология
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Introduction

Human cytomegalovirus (CMV) is a widespread DNA virus and member of the Herpesviridae family (herpesviruses). CMV infection can be asymptomatic, manifest as local lesions (most commonly as sialadenitis), or have a generalized nature, including life-threatening damage to the liver, adrenal glands, and kidneys.[1,2]

The routes of CMV transmission are airborne, contact, sexual, transplacental (intrauterine), and via transfusion of blood or its products and organ transplantation.[3]

There are 3 different types of CMV infection: primary infection (infection in individuals who have never encountered the virus before) with an asymptomatic or mildly symptomatic course in immunocompetent individuals, although the virus remains indefinitely in the genome; CMV reactivation due to the compromised immune system and emergence of T-cell immune response; superinfection, ie, infection of CMV-seropositive individuals.[4]

In 1881, changes characteristic of CMV infection were first observed by Hugo Ribbert, German pathologist. In 1956, 75 years later, Margaret Gladys Smith, American pathologist, discovered and described the virus, which is called *Human herpesvirus 5* and has an affinity for salivary gland tissue.[5,6]

The virus has tropism for various types of epithelial cells, particularly salivary glands and renal tubules, alveolar cells, vascular endothelium, blood cells, and fibroblasts. Once inside the human body, CMV persists indefinitely, with a possibility of superinfection.[7]

The broad tissue tropism of CMV accounts for the polymorphism of the clinical course of the infection and high probability of secondary immunosuppression.[1,8]

During CMV replication, it acts cytopathically, forming giant cells with characteristic intranuclear and cytoplasmic inclusions. Tissue reactions in CMV infection have a fairly uniform 2-component nature, consisting of cytomegalic cell transformation and interstitial lymphohistiocytic infiltration. Nodular infiltrates are usually found in cases of acute onset of the disease and rapid fatal outcome. In the localized form of CMV infection, changes are detected in the salivary glands, predominantly parotid, less commonly submandibular and sublingual glands. In its generalized form, specific morphological changes are observed: "owl's eye" (characteristic changes in the nuclei of epithelial cells) in the epithelium of many organs and systems.[9,10]

According to epidemiological data in the UK and the USA, 40% to 60% of the adult population with a middle and high socioeconomic status and up to 80% of the population with a low socioeconomic status are CMV-seropositive. In developing countries, the prevalence of CMV infection is even higher: up to 80% in children and 100% in adults.[11]

CMV infection of the gastrointestinal tract is a relatively common manifestation among patients receiving immunosuppressive therapy, HIV-infected patients, or organ and tissue recipients.[2] However, in some cases, CMV infection with intestinal mucosa involvement may also develop in immunocompetent individuals. Recent studies identified a potential link between CMV colitis and various forms of chronic inflammatory bowel diseases (IBD). Two diseases, Crohn disease and ulcerative colitis, fall into the IBD category.[3] Patients with IBD are at risk of CMV infection for several reasons as they often receive immunosuppressive therapy (corticosteroids, azathioprine, 6-mercaptopurine, cyclosporine A, or methotrexate).[3,12] Moreover, inflammation itself is a predisposing factor as CMV has a clear tropism for proliferating cells and granulation tissue due to pronounced neoangiogenesis with endotheliocyte proliferation in the focus of inflammation.[13,14]

The clinical course of CMV infection involving the colon is generally nonspecific. CMV colitis can have a wide range of symptoms of varying severity, including diarrhea, fever, weight loss, abdominal pain, rectal bleeding, and colonic perforation. The average duration of CMV colitis symptom manifestation is 13 days.[14,15]

Based on complete blood cell count findings, patients with CMV colitis typically have low levels of hemoglobin and albumin, leukocytopenia, and thrombocytopenia associated with immunodeficiency.[16]

In patients with IBD, symptoms of CMV colitis tend to mimic the corresponding intestinal disease, thus complicating the differential diagnosis.[2,3,14] Also, CMV is the most likely cause of steroid resistance in IBD, with the resistance rate reaching 70% in CMV-positive IBD patients compared with 35% in CMV-negative IBD patients.[17]

Objective

To describe a variant of CMV colitis course and features of its pathomorphology based on a case from our clinical practice.

Methods

We studied 6 biopsy specimens from the ileum, colon (ascending, transverse, descending, and sigmoid), and rectum of a female patient.

The raw material was fixed in 10% neutral formalin solution and dehydrated through ascending concentrations of alcohol. The tissues were embedded in paraffin blocks, sectioned using a microtome, and subjected to histological processing using the following methods: hematoxylin and eosin staining, Periodic Acid–Schiff staining with iodine (PAS reaction).

Histological examination was conducted to assess the preservation of histological architecture, such as flattening and atrophy of ileal villi, as well as branching, dilation, and atrophy of crypts in the colon. We also evaluated the presence or absence of neutrophilic infiltration in the lamina propria, epithelium, and crypt lumens, as well as the presence or absence of granulomatous inflammation and changes characteristic of viral infection.

Microscopic examination of the slides was performed using Leica DM2500 optical microscope.

Clinical data were obtained from the patient's medical records.

Results and Discussion

Patient A., born in 1988, was admitted to the medical ward at a regional hospital for further examination, diagnosis clarification, and treatment adjustment.

Medical history: The patient considers herself ill for 2 years. During this period, she was examined in local hospitals and republican health care institutions in Minsk, Republic of Belarus. She consulted a gastroenterologist at the Republican Gastroenterology Center and was diagnosed with Crohn disease A2L3B1 (Montreal classification of Crohn disease, 2006)[18], involvement of the small and large intestines, moderate activity with clinical response to 5-aminosalicylic acid, glucocorticoids (June 2019); Crohn Disease Activity Index, 130 points. She took mesalazine (Salofalk) (10 tablets), prednisolone (currently 3 tablets, started with 8 tablets). She noted worsening condition over the past 2 months, with minimal effect from medication.

Upon admission, the patient complained of frequent loose stools with blood and mucus up to 30 times a day, elevated temperature rising to 38 °C in the evening, weight loss (10 kg) over the past few months, and irregular menstrual cycles.

On initial examination, the patient's condition was moderate, with clear consciousness. She had an asthenic build, with a body mass index of 15, vesicular breath sounds, respiratory rate of 16 breaths per minute, heart rate of 88 beats per minute, rhythmic pulse, blood pressure of 130/80 mm Hg, temperature of 36.0 °C. The tongue was moist; no hyperemia of the pharynx and no vomiting were observed. Upon abdominal palpation, the abdomen was soft, tender in the right and left iliac regions. No peritoneal signs were present.

Preliminary diagnosis: Crohn disease of the large intestine.

Upon admission, the following comprehensive diagnostic workup was performed.

Complete blood cell count: erythrocyte sedimentation rate, 35 mm/hour; leukocytes, 9.97×10^9/L; erythrocyte count, 3.49×10^{12}/L; hemoglobin, 108 g/L; hematocrit, 0.33; platelet count, 365×10^9/L; band neutrophils, 18%; segmented neutrophils, 67%; eosinophils, 0%; monocytes, 6%; lymphocytes, 9%.

Urinalysis: physical properties (straw color, acidic reaction), chemical properties (protein, 0.04 g/L; glucose negative), microscopic properties (erythrocytes, 0-2 per field of view; leukocytes, 3-5 per field of view).

Biochemical blood analysis: total protein, 62.3 g/L; urea, 3.7 mmol/L; creatinine, 81.0 μmol/L; C-reactive protein, 186.2 mg/L; total bilirubin, 4.3 μmol/L; alanine aminotransferase, 8 U/L; aspartate aminotransferase, 10 U/L; glucose, 4.8 mmol/L; ferritin, 268.9 μg/L.

Coagulation profile: activated partial thromboplastin time, 30.4 s; prothrombin ratio, 0.94; fibrinogen, 6.1 g/L; thrombin time, 14.5 s.

Acid-base balance: pH, 7.46; potassium, 3.2 mmol/L; sodium, 129 mmol/L; calcium, 0.97 mmol/L; chloride, 98 mmol/L.

Stool analysis: macroscopic examination (formed stool, brown color, positive reaction for blood); microscopic examination (muscle fibers, 2-4 per field of view; neutral fat, 2-4 per field of view; undigested fiber 3-4 per field of view; digested fiber, 0-1 per field of view; starch grains, 0-2 per field of view; numerous leukocytes; no helminth eggs or Clostridium difficile detected; calprotectin positive).

Enzyme-linked immunosorbent assay (ELISA) for antibodies to Chlamydia trachomatis, Treponema pallidum, HIV negative.

Electrocardiogram: sinus rhythm; heart rate 97 beats per minute.

The patient consulted a rheumatologist due to the numbness in the fingers and toes, as well as cyanosis of the hands and feet under cold conditions. She was diagnosed with limited systemic sclerosis and Raynaud syndrome. ELISA was performed to detect specific autoantibodies in the blood serum, particularly antibodies to topoisomerase I (Scl-70). The findings were as follows: 2.6 U/mL (normal range, 0.0-15.0 U/mL). Based on clinical data, specific antibodies for systemic sclerosis were within the normal range during this period, possibly due to the high doses of glucocorticoids.

Following esophagogastroduodenoscopy, the patient was diagnosed with erythematous gastritis and duodenogastric reflux. To refine the diagnosis, the patient underwent elective fibrocolonoscopy. Visually, the perianal area had no abnormalities, and the sphincter tone was normal. The colonoscope was inserted beyond the splenic flexure. The Boston Bowel Preparation Scale score was 3-3-2. Extensive ulcers (some circular) were observed. The extent of ulcerative involvement was over 30%, with inflammatory changes present in over 75% of ulcers. Some unaffected mucosa was observed in the left colon. In the lower rectum, the ulcer base was very deep and irregular, with some areas not clearly visible, suggesting a possible fistula. The ileocecal valve was competent, but ulcers and pseudopolyps were present. Beyond the valve, there was a significant narrowing and an almost circular ulcer. The colonoscope was not able to pass past this narrowing. Stepwise biopsies were performed during the examination. Based on the endoscopic findings we came to the following conclusion: Crohn disease, total colonic involvement, terminal ileum stricture.

The material obtained during fibrocolonoscopy was sent to the department of pathology.

Microscopic Examination of the Histopathological Specimens

The mucosa of the terminal ileum showed atrophy of some villi, with scattered neutrophilic infiltration and occasional eosinophils in the lamina propria. The mucosa of the cecum, transverse and sigmoid colon exhibited architectural distortion with branching and dilation of crypts, neutrophilic infiltration in the lamina propria, epithelium, and crypt lumina, ulceration, cryptitis, and crypt abscess formation. In the submucosal layer, focal angiomatosis was present with foci of granulation tissue proliferation, multiple hypertrophied endothelial cells with granular cytoplasmic inclusions, and multiple Cowdry type B inclusion bodies. The mucosa of the descending colon and rectum showed architectural distortion with branching crypts but without neutrophilic infiltration in the lamina propria.

Thus, the following histopathological diagnosis was formulated: chronic diffuse intermittent active colitis with ulceration, predominantly affecting the cecum, transverse and sigmoid colon. According to the optical microscopy data, the morphological pattern most closely corresponded to CMV colitis.

Following this diagnosis, a republican council was held: the diagnosis was revised, and recommendations for additional diagnostics and treatment were provided. The analysis of CMV DNA in blood by polymerase chain

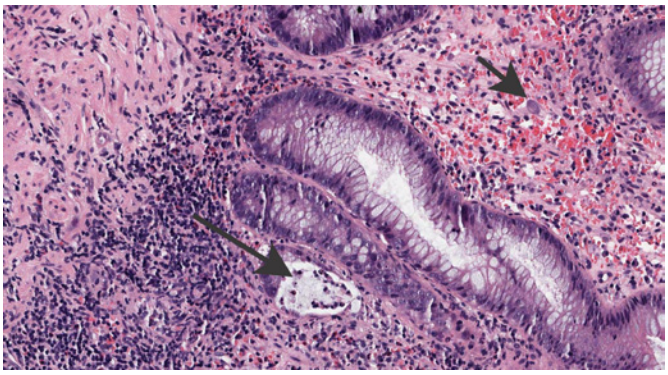

Figure 1. Cowdry type B body (short arrow). Crypt abscess (long arrow). Hematoxylin-eosin, ×150
Рисунок 1. Тельце Каудри типа В (короткая стрелка). Крипт-абсцесс (длинная стрелка). Окраска гематоксилином и эозином, ×150

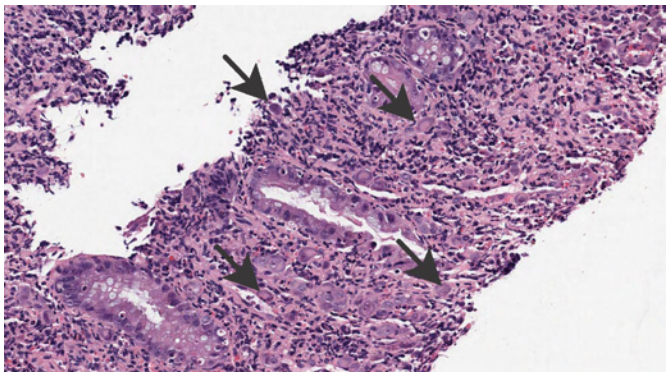

Figure 2. Multiple Cowdry type B bodies (arrows). Hematoxylin-eosin, ×200
Рисунок 2. Множественные тельца Каудри типа В (стрелки). Окраска гематоксилином и эозином, ×200

reaction (PCR) method (positive result) played a significant role in refining the diagnosis. Considering the patient's age, extensive involvement, recurrent course of the disease with steroid refractoriness, malnutrition syndrome, and concomitant rheumatic disease, biological therapy was prescribed: infliximab intravenously at a dose of 5 mg/kg of body weight per infusion according to the 0-2-6-14 scheme. In addition, it was recommended to continue taking mesalazine (Salofalk) (3 g daily, orally) and mesalazine suspension (Salofalk) (2 g every night, rectally).

After the treatment, we conducted a comprehensive follow-up involving repeated diagnostic procedures, such as laboratory tests (including blood tests, stool analysis) and endoscopy with histopathological evaluation of tissue samples, to thoroughly assess the treatment response and ensure that there were no signs of disease recurrence or persistence.

The absence of clinical symptoms coupled with negative findings on diagnostic tests indicated a favorable outcome of the therapeutic interventions. This not only signifies the effectiveness of the treatment regimen but also underscores the importance of vigilant monitoring and follow-up care in managing CMV colitis.

The misdiagnosis of chronic colitis (initially thought to be Crohn disease but later identified as CMV colitis) highlights the difficulties in accurate diagnosis of gastrointestinal conditions, especially in case of overlapping pathologies.

The key histopathological features of CMV colitis that differentiate it from Crohn disease include characteristic intranuclear inclusions known as "owl's eye" or "cytomegalic cells" within epithelial cells. CMV colitis often involves the submucosal layer, displaying focal angiomatosis and granulation tissue proliferation. Presence of eosinophilic cytoplasmic inclusions called Cowdry type A or type B bodies is indicative of CMV infection. Ulcers in CMV colitis are typically shallow and irregular, unlike the deep, serpiginous ulcers observed in Crohn disease. Moreover, inflammation in CMV colitis tends to be patchy, involving all layers of the intestinal wall, in contrast to the transmural granulomatous inflammation seen in Crohn disease. All of these distinctive histopathological features characterize the classical appearance of CMV colitis and contribute to its differentiation from Crohn disease.

Although the histopathological examination gave valuable insights into the patient's condition, we have to acknowledge the inherent limitations of biopsy-based diagnoses, including sampling bias and variability in interpretation. Furthermore, the absence of specific histopathological features characteristic of CMV colitis may pose challenges in distinguishing it from other forms of colitis. Thus, it is important to incorporate the histopathological examination and molecular diagnostics, such as PCR testing for CMV DNA, in order to refine a diagnosis and guide treatment decisions effectively.

Conclusions

CMV colitis presents a spectrum of clinical and morphological features, often leading to misdiagnosis without comprehensive diagnostic assessment. Contemporary diagnostic and therapeutic approaches for CMV colitis can significantly influence the disease trajectory, improve outcomes, and enhance patient's quality of life. This case highlights the critical need for meticulous diagnostic evaluations, especially in refractory cases characterized by extensive disease and steroid refractoriness, to ensure that appropriate treatment strategies are used effectively.

The presented case underlines the informative value of morphological diagnosis in establishing accurate diagnoses, as well as the importance of interdisciplinary collaboration and continuity of care among pathologists, gastroenterologists, and endoscopists in routine practice. Future research should prioritize elucidating the role

of molecular diagnostics, such as PCR testing for viral pathogens, in refining diagnoses and guiding treatment decisions in refractory IBD cases. Moreover, longitudinal studies are necessary to evaluate the efficacy and safety of biological therapies, particularly in patients with concurrent rheumatic conditions, to optimize treatment strategies, and improve patient outcomes. Additionally, prospective cohort studies investigating the impact of multidisciplinary care approaches on long-term disease outcomes and healthcare utilization in complex IBD cases are warranted.

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Manuscript drafting and revising: Tishchenko, Hlavatskaya
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Конфликт интересов

Авторы заявляют об отсутствии конфликта интересов.