ISSN 2786-6661
eISSN 2786-667X

UDC: 616.345-002-018.73-092.19:616.12-008.331.1]-073.7-092
https://doi.org/10.32345/USMYJ.1(149).2024.137-145

Received: June 26, 2024
Accepted: September 09, 2024

Ulcerative colitis and concomitant arterial hypertension (literature review)

Khairnasova Alina

[1]Shupyk National University of Health Care of Ukraine, Kyiv, Ukraine
[2]Ukrainian-German antiulcer gastroenterological center «Byk-Kyiv», Kyiv, Ukraine

Address for correspondence:

Khairnasova Alina

E-mail: dockhairnasova@gmail.com

Abstract: *ulcerative colitis is a complex disease that results from a complex interaction of genetic predisposition, environmental factors, which leads to dysregulation of the immune system, chronic intestinal inflammation with damage to the mucous membrane. Systemic chronic inflammation can cause the development of arterial hypertension, thereby complicating the course of the primary disease. However, the cause-and-effect relationship between these diseases is not fully understood. Early detection of high blood pressure in patients with ulcerative colitis, a multidisciplinary approach to diagnosis makes it possible to achieve optimal treatment results in a timely manner and prevent the occurrence of intestinal complications. The aim of this study was to provide a comprehensive review of diagnostic procedures such as colonoscopy, inflammatory biomarkers (faecal calprotectin, faecal lactoferrin, serum zonulin), and to study the features of the colon structure in ultrasound diagnostics in patients with ulcerative colitis and concomitant hypertension. The data from PubMed and Ukrainian scientific sources on the comorbidity of these diseases, the possibility of using non-invasive diagnostic methods to assess the condition of the intestine in patients with ulcerative colitis and arterial hypertension were analysed. The literature review includes 50 scientific sources. The main focus is on the pathophysiological relationships of diseases, peculiarities of non-invasive diagnosis of ulcerative colitis during follow-up and early detection of arterial hypertension, which makes it possible to prevent complications in ulcerative colitis. Given the literature review, it can be concluded that the problem of timely diagnosis of hypertension in patients with ulcerative colitis is relevant today. For a rational approach to the management of patients with ulcerative colitis and concomitant hypertension, it is necessary to carry out a set of diagnostic procedures, including outpatient blood pressure measurement, ultrasound examination of the intestinal wall, and the use of non-invasive inflammatory biomarkers.*

Keywords: Blood Pressure; Essential Hypertension; Inflammatory Bowel Diseases; Ulcerative Colitis; Ultrasonography.

Introduction

At the turn of the XXI century, inflammatory bowel disease (IBD) is becoming increasingly common in industrialised countries due to the increase in morbidity and the possibility of timely diagnosis (Ng et al., 2017). IBD is a group of chronic systemic inflammatory conditions with a tendency to affect the gastrointestinal tract and includes ulcerative colitis (UC) and Crohn's disease (Fabián et al., 2022). UC is a recurrent and remitting IBD characterised by mucosal inflammation that starts distally and can spread proximally to affect the entire colon (Segal et al., 2021). Worldwide, the incidence of UC is on the rise with the

ISSN 2786-6661
eISSN 2786-667X

annual incidence of UC ranging from 8.8 to 23.1 per 100,000 person-years in North America, 0.6 to 24.3 per 100,000 person-years in Europe (Du et al., 2020). In 2023, the global prevalence of UC was estimated at 5 million cases, and the incidence is increasing worldwide (Le Berre et al., 2023).

It has been described that IBD may be one of the causes of a prolonged inflammatory process (Kirchgesner et al., 2018). Inflammation is a complex and necessary component of the body's response to biological, chemical or physical stimuli. Numerous studies have shown that chronic inflammation contributes to a variety of diseases, including atherosclerosis, autoimmune diseases, asthma, diabetes, cancer, and others (Germolec et al., 2018).

Chronic inflammation plays an important role in the development and progression of cardiovascular disease (CVD). CVD is and remains the leading cause of morbidity and mortality worldwide. IBD can lead to atherosclerosis, increase the risk of CVD, especially arterial hypertension (AH) (Rungoe et al., 2013), (Choi et al., 2019).

UC and AH are two different diseases that can interact with each other, thereby complicating the course of the primary disease. However, the cause-and-effect relationship between these diseases has not yet been sufficiently studied, so international research in this area continues. Thus, there is a need to study the relationship between UC and AH due to the high degree of their comorbidity.

Aim

The aim of the study is to provide a comprehensive review of diagnostic procedures such as colonoscopy, inflammatory biomarkers (calprotectin, lactoferrin, zonulin), and to study the features of the colon structure during ultrasound diagnostics in patients with UC and concomitant hypertension.

Materials and methods

The article analyses PubMed data and Ukrainian scientific sources on the comorbidity of UC and AH, the possibility of using non-invasive diagnostic methods to assess the condition of the intestine in patients with UC and concomitant hypertension. The literature review includes 50 scientific sources. The main focus is on the pathophysiological links between UC and AH, the peculiarities of non-invasive diagnosis of UC during follow-up and early detection of hypertension, which makes it possible to prevent complications in UC.

Review and discussion

1. Pathogenetic mechanisms of the association between UC and AH

At present, reliable pathophysiological mechanisms that may link these two diseases have not yet been fully studied, but there are several theories and potential mechanisms in this regard.

One of these is genetic predisposition: some genes may be shared by UC and AH. Studies show that genes related to inflammation and blood pressure regulation may play a potential role in the development of their comorbidity (He et al., 2023). Although genetics play an important role in the pathogenesis of the disease, incomplete penetrance suggests that environmental and lifestyle factors, including diet, psychological stress, medication and smoking, also contribute to comorbidity (Abegunde et al., 2016).

As UC is a chronic disease and is accompanied by emotional stress, the latter can cause activation of the sympathetic nervous system and increase levels of stress hormones such as catecholamines. This can lead to vasoconstriction and increased blood pressure. Emotional overload associated with sympathicotonus is more likely to lead to relapse in patients with UC (Sauk et al., 2023).

Stress increases intestinal permeability and ion secretion through a mechanism that includes nerve stimulation and depletes goblet cells. Subsequently, UC is characterised by a chronic inflammatory process in the colon. Inflammation can lead to systemic immune activation and increased levels of cytokines such as interleukin-6 (IL-6) and tumour necrosis factor alpha (TNF-α). These cytokines can also contribute to the development of hypertension. In patients with UC, vascular endothelial damage occurs, and endothelial dysfunction is characterised by a decrease in the synthesis and release of nitric oxide (NO), which leads to vasoconstriction and increased blood pressure. Patients with UC demonstrate a depletion of the ileal barrier, which is manifested by a decrease in goblet cells containing mucin and the production of mucin itself (Alipour et al., 2016). Goblet cells are more susceptible to stress due to their role in protecting the intestinal epithelium from microbes and harmful substances (Chotikatum et al., 2018).

In normal condition, intestinal reactive oxygen species have bactericidal effects, participat-

ISSN 2786-6661
eISSN 2786-667X

ing in the intestinal defensive function. However, oxidative stress derived from excessive reactive oxygen species production over the buffering capability of antioxidant defense in the host would cause lipid peroxidation, intestinal mucosal barrier damage, bacterial translocation, and inflammatory response. UC is a type of chronic IBD, UC is essentially an immune-inflammatory disease, which oxidative stress plays a critical role in its pathogenesis and malignant progression to colorectal cancer. Inflammation is a process that consists of a series of protective responses, such as immune cell infiltration and cytokine expression, to eliminate pathogens/insults and initiate damage repair of the tissue (Wang et al., 2016).

The role of bacteria in atherosclerosis progression have been extensively studied for more than two decades. Low levels of bacteria could be found in the circulation in many chronic metabolic diseases, including obesity, type 2 diabetes and atherosclerosis, and is commonly referred to as "metabolic endotoxemia" (Neves et al., 2013). Several studies have reported an association between enteric bacteria and atherosclerosis. Bacterial 16S ribosomal RNA (rRNA) gene belong to *Enterobacteriaceae* have been detected in atherosclerotic plaques (Li et al., 2016). One potential mechanism for the association of AH with UC may be that elevated blood pressure can alter tight junction proteins and increase intestinal permeability, thus worsening the course of UC. For example, elevated levels of zonulin, a regulator of intestinal epithelial tight junction protein, were found to be strongly correlated with systolic blood pressure ($R^2 = 0.530$, $p < 0.0001$) and may also lead to the release of proinflammatory cytokines in the gut (Kim et al., 2018). Zonulin reversibly modulate intestinal permeability, the circulating zonulin levels were increased in diabetes, obesity, all of which are risk factors for atherosclerosis (Li et al., 2016).

The mechanisms, regarding the contribution of bacteria in atherosclerosis, are complicated. Previous studies mainly focused on the indirect mechanisms. Microbial activation of these innate immune receptors promotes inflammation that dampens reverse cholesterol transport, which in turn augments insulin resistance, hyperlipidemia, and vascular inflammation. Recent studies revealed that, in addition to digestion and absorption of many nutrients, intestinal microbial flora may play an active role in the development of complex metabolic disease by the production of metabolites (Wang et al., 2011).

Stress leads to vasospasm, triggering the mechanism of the renin-angiotensin-aldosterone system and increasing blood pressure; on the other hand, stress leads to an increase in bacterial adhesion and a decrease in the number of lactobacilli in the intestinal lumen. As a consequence of all these changes in the intestinal lumen, antigens can gain access to the epithelium, causing and prolonging inflammation in UC; intestinal permeability to large antigenic molecules leads to goblet cell activation, degranulation, mucin depletion in the colon and inflammation (Sui et al., 2022).

Therefore, the intestinal barrier, which is the link between the external and internal environment of the body, plays an important role in human health. A functional intestinal barrier allows for the absorption of nutrients and fluids, but at the same time prevents harmful substances, such as toxins and bacteria, from crossing the intestinal epithelium and entering the body (Schoultz et al., 2020).

The intestinal barrier interacts with and responds to various stimuli. It consists of several elements, such as the surface mucus, the epithelial layer and the immune defence. In the lumen, bacteria and antigens are degraded by bile, gastric acid and pancreatic juice, as well as by commensal bacteria, which inhibit the colonisation of pathogens by producing antimicrobial substances. The next element of the barrier is the microclimate, consisting of an unmixed layer of water, glycocalyx and mucus layer, which prevent bacterial adhesion through the secretion of immunoglobulin A (IgA) and the physical barrier created by glycocalyx and mucus (Camilleri, 2019). Immune regulators, such as antimicrobial proteins and IgA molecules, are released in the mucosal gel in a gradient from the epithelium to the lumen, thereby enhancing protection against lumen microbes (Johansson et al., 2016). The gut microbiota contributes to blood pressure homeostasis and prevents the development of hypertension by producing, modifying and degrading various microbial bioactive metabolites (Katsi et al., 2019). The relationship between intestinal bacteria and the mucosal barrier is well balanced in a steady state; intestinal bacteria cannot contact the intestinal epithelium. However,

ISSN 2786-6661
eISSN 2786-667X

when the mucosal barrier is not functioning, intestinal bacteria can approach intestinal immune cells and cause IBD (Okumura et al, 2018). Chronic inflammation plays an important role in the initiation and progression of cardiovascular disease, and one of the causes of a prolonged inflammatory process may be UC. Studies have shown that in patients with UC, hypertension occurred in 40% (Kristensen et al., 2013), (Ghoneim et al, 2020), and high blood pressure can worsen the course of UC (Wu et al., 2017). Thus, patients with IBD had a higher chance of developing hypertension compared to patients without IBD (odds ratio [OR] 1.71, 95% confidence interval [CI] 1.39-2.09) (Xu et al, 2022).

Scientists conducted a cohort study of the UK Biobank, which found that UC increases the risk of developing AH. A total of 281,064 participants were enrolled in the study, 2,376 (0.8%) were diagnosed with IBD at the start of the study, and 20,129 (7.2%) of the entire cohort developed hypertension with a median follow-up duration of 8.1 years (interquartile range [IQR] 7.3-8.8 years). Patients with IBD had a higher cumulative risk of hypertension compared with general population (10.9% in UC, 7.7% in Crohn's disease, and 9.3% in IBD unclassified vs. 7.1% in non-IBD, $p < 0.001$). Multivariate Cox regression analysis identified that UC, rather than Crohn's disease or IBD unclassified, was independently associated with subsequent occurrence of hypertension (HR 1.30, 95% CI: 1.11–1.52, $p = 0.001$). In propensity matching analysis, UC also showed its robustness as a risk factor for the prediction of AH (HR 1.56, 95% CI: 1.21–2.03, $p = 0.001$) (He et al., 2023).

One of the mechanisms of AH development in UC may be the use of corticosteroids. Corticosteroids are relatively inexpensive and commonly used to treat a variety of conditions, and long-term use is known to be associated with some toxicity (Rice et al., 2017). That is, in addition to the existing diagnosis of UC, the use of steroids and immunomodulators has been identified as risk factors for the development of hypertension with a comparable risk ratio (Burisch, 2023).

2. Clinical features of comorbidity of UC and AH

UC and hypertension are two different diseases that, when occurring simultaneously, can significantly affect the patient's quality of life. The polymorphism of the clinical manifestation of UC, the variety of forms depending on the location of the lesion, severity of the course, the presence of extraintestinal manifestations and complications, and concomitant hypertension require a collective decision on the treatment strategy in patients with UC with hypertension with the participation of gastroenterologists, proctologists, cardiologists and family doctors (Степанов, Псарьова, 2017). The assessment of persistent symptoms in a patient with UC should begin with a detailed medical history, including an examination of the intestinal structure, taking into account the clinical spectrum of the patient's manifestations, as the severity of symptoms may not always directly correlate with the degree of inflammatory activity (Colombel et al., 2019).

UC is a chronic disease that causes inflammation and ulcers or sores on the lining of the rectum and colon. The main symptom of UC is bloody diarrhoea with or without mucus. Associated symptoms also include urgency or tenesmus, abdominal pain, malaise, weight loss, and fever, depending on the extent and severity of the disease. The onset of the disease is usually gradual, with symptoms progressing over several weeks, and patients may experience periods of remission and subsequent relapses on their own (Lynch et al., 2023).

Patients with predominantly distal involvement may experience constipation with frequent discharge of blood and mucus. Symptoms may be preceded by an isolated episode of rectal bleeding that occurred weeks or months earlier. In severe cases, patients may also experience fever and signs of dehydration (Peppercorn et al., 2023).

Hypertension is a long-term condition in which blood pressure in the arteries is constantly rising. When it comes to hypertension, men are more likely to be diagnosed with cardiovascular disease. The onset of CVD in men with high blood pressure is observed as early as 25-44 years of age, indicating the need for preventive measures in adolescence and closer monitoring of treatment at a young age (Song et al., 2020). Hypertension is the leading modifiable risk factor for all-cause mortality and early CVD in women, and uncontrolled hypertension remains more common in older postmenopausal women than in men (Tamargo et al., 2023).

ISSN 2786-6661
eISSN 2786-667X

Hypertension is often asymptomatic until it leads to serious complications. If symptoms do occur, they may include headache, shortness of breath, dizziness, chest pain, palpitations, and vision problems.

Patients suffering from both UC and hypertension often experience a wide range of symptoms. They can stem from either disease or result from their interaction. When both diseases coexist, patients often report an increase in the severity of their UC symptoms. The additional stress associated with existing hypertension can exacerbate UC exacerbation, potentially through the interplay between stress, inflammation and the immune response (Alipour et al., 2016), (Fung, 2020). Similarly, inflammation from UC can contribute to high blood pressure by increasing arterial stiffness (He et al., 2023).

3. Minimally invasive methods for assessing the condition of the intestine in UC with concomitant AH

Ultrasonography of the intestine

Patients with UC require clinical, biochemical, endoscopic, and cross-validation studies to confirm the diagnosis and exacerbations of the disease, identify complications, and choose treatment. To date, endoscopy with biopsy remains the gold standard for the diagnosis of UC (Maconi et al, 2018), but still the primary differential diagnostic procedures could be minimally invasive diagnostic methods such as transabdominal ultrasound of the intestine, inflammatory biomarkers (calprotectin, lactoferrin, zonulin), Transabdominal ultrasound of the gastrointestinal tract provides a unique opportunity to non-invasively examine the intestine and including extraintestinal elements such as abdominal vessels, mesentery, cecum and lymph nodes (Nylund et al., 2017).

According to the EFSUMB Recommendations and Guidelines for Intestinal Ultrasound in Inflammatory Bowel Diseases, published in 2017, bowel ultrasound is a non-invasive method for assessing disease activity in IBD, well tolerated, radiation-free, accessible and inexpensive (Maconi et al., 2018). Bowel ultrasound can be performed at a regular patient appointment, without special preparation, to facilitate clinical decision-making regarding further follow-up (Dolinger et al., 2023).

Ultrasound procedures are generally considered to be very safe and there is no evidence to date to suggest a harmful effect of ultrasound in adults (Nylund et al., 2017). Intestinal ultrasound is primarily used to assess the condition of the intestinal wall, namely its thickness. It should be noted that most studies report intestinal wall thickening > 4 mm in adults and > 3 mm. in children, which may indicate the presence of an inflammatory process in the intestinal wall (Maconi et al., 2018). In practice, bowel ultrasound requires the use of a low-frequency convection transducer (3-5 MHz) and a high-frequency linear transducer (5-15 MHz), which allows visualisation of all layers of the intestinal wall and measurement of the intestinal wall thickness down to 0.1 millimetre (Dolinger et al., 2023).

A number of studies have shown that intestinal wall thickness correlates well with the clinical activity of UC, non-invasive inflammatory markers, and endoscopy findings (Kucharzik et al., 2017), (Rajagopalan et al., 2019).

The loss of the layered structure (stratification) of the intestinal wall (hypoechoic pattern) correlated with moderate (55% of cases) and severe (100% of cases), while normal layered structure of the intestinal wall was present in 87% of mild cases of UC. Increased vascularisation of the intestinal wall (increased Doppler signals with low resistance) was also associated with clinical and endoscopic indicators of UC activity (Maconi et al., 2018).

Biomarkers of inflammation

Colonoscopy is still an invasive procedure, and patients usually find it difficult to repeat it from time to time to assess the condition of the bowel. Endoscopic procedures are unpleasant and sometimes painful, time-consuming and expensive. Therefore, these disadvantages have led to the search for non-invasive markers of gastrointestinal inflammation, which should be sensitive, rapid, reliable, and cost-effective. In clinical practice, inflammatory biomarkers have gained considerable popularity over the past few decades (Núñez et al., 2021). In recent years, clinical guidelines have recommended the determination of faecal calprotectin as part of the diagnostic work-up of UC (Pathirana et al., 2018). One study demonstrated that faecal calprotectin levels significantly correlate with

ISSN 2786-6661
eISSN 2786-667X

clinical disease activity, endoscopic findings, and serum inflammatory biomarkers in patients with UC. Faecal calprotectin is a highly predictive indicator of complete mucosal healing in UC (Lee et al., 2017).

A number of other studies have shown that not only faecal calprotectin, but also lactoferrin, is a promising non-invasive indicator of mucosal healing (Boon et al., 2015). The potential usefulness of faecal lactoferrin for predicting the risk of UC recurrence has been investigated: an increase in faecal lactoferrin may occur before clinical outbreaks. In addition, some studies have demonstrated that faecal lactoferrin is a sensitive and specific marker of inflammation in children with UC; faecal lactoferrin correlates with both clinical disease activity and serum inflammatory markers (Szymanska et al., 2023).

In recent years, it has been suggested that intestinal permeability disorders are an early stage preceding the onset of IBD. Since zonulin has been proposed as a biomarker of intestinal permeability, studies have shown that serum zonulin is highly sensitive for assessing intestinal permeability in patients with UC (Caviglia et al., 2019).

Conclusions

Given the literature review, it can be concluded that the problem of timely diagnosis of hypertension in patients with ulcerative colitis is relevant today. For a rational approach to the management of patients with ulcerative colitis and concomitant hypertension, it is necessary to carry out a set of diagnostic procedures, including outpatient blood pressure measurement, ultrasound examination of the intestinal wall, and the use of non-invasive inflammatory biomarkers.

Financing

This study did not receive any funding.

Conflict of interests

The author declares that there is no conflict of interest in the preparation of this article.

Consent to publication

The author has read the text of the manuscript and agreed with its publication.

ORCID ID and author's contribution

0009-0005-9472-5567 (A, B, C, D, E, F) Khairnasova Alina

A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article.

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eISSN 2786-667X

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Степанов Ю.М., Псарьова І.В. Роль біомаркерів у діагностиці хронічних запальних захворювань кишечника ДУ «Інститут гастроентерології НАМН України», м. Дніпро, Україна. Журнал "Гастроентерологія" Том 51, №1, 2017

Неспецифічний виразковий коліт та супутня артеріальна гіпертензія (огляд літератури)

Хайрнасова Аліна

[1] Національний університет охорони здоров'я України імені П. Л. Шупика, м.Київ, Україна

[2] Українсько-німецький противиразковий гастроентерологічний центр «Бік- Київ», м. Київ, Україна

Address for correspondence:

Khairnasova Alina

E-mail: dockhairnasova@gmail.com

Анотація. *неспецифічний виразковий коліт — це складне захворювання, яке є результатом складної взаємодії генетичної схильності, факторів навколишнього середовища, що призводить до порушення регуляції імунної системи, хронічного запалення кишечника з пошкодженням слизової оболонки. Системне хронічне запалення може викликати розвиток артеріальної гіпертензії, ускладнюючи тим самим перебіг первинного захворювання. Однак, причинно-наслідкові зв'язки між цими захворюваннями не вивчені в повній мірі. Раннє виявлення підвищеного артеріального тиску у пацієнтів з неспецифічним виразковим колітом, мультидисциплінарний підхід в діагностиці дає можливість своєчасно досягти оптимальних показників при лікуванні, попередити виникнення ускладнень з боку кишечника. Метою цього дослідження було зробити комплексний огляд діагностичних процедур таких як колоноскопія, біомаркери запалення (фекальний кальпротектин, фекальний лактоферин, зонулін в сироватці крові), вивчити особливості структури товстого кишечника при ультразвуковій діагностиці у пацієнтів з неспецифічним виразковим колітом та супутньою артеріальною гіпертензією. Було проаналізовано дані PubMed та українських наукових джерел щодо коморбідності цих захворювань, можливості застосування неінвазивних методів діагностики для оцінки стану кишечника у пацієнтів з неспецифічним виразковим колітом та артеріальною гіпертензією. Огляд літератури включає 50 наукових джерел. Основна увага приділена патофізіологічним зв'язкам захворювань, особливостям неінвазивної діагностики неспецифічного виразкового коліту в ході спостереження та ранньому виявленні артеріальної гіпертензії, що дає можливість попередити ускладнення при неспецифічному виразковому коліті. З огляду на проведений огляд літератури можна зробити висновок, що проблема своєчасної діагностики артеріальної гіпертензії у пацієнтів з неспецифічним виразковим колітом є актуальною на сьогоднішній день. Для раціонального підходу до ведення пацієнтів із неспецифічним виразковим колітом та супутньою артеріальною гіпертензією, необхідно проводити комплекс діагностичних процедур, який включає амбулаторне вимірювання артеріального тиску, ультразвукове дослідження стінки кишечника, застосування неінвазивних біомаркерів запалення.*

Ключові слова: артеріальний тиск; есенціальна гіпертензія; запальні захворювання кишечника; неспецифічний виразковий коліт; ультразвукове дослідження.