

Putowski Maciej, Padala Olga, Krupa Adrianna, Konopelko Michal, Piasek Ewa. Musculoskeletal manifestations of inflammatory bowel disease. Journal of Education, Health and Sport. 2019;9(9):690-695. eISSN 2391-8306. DOI <http://dx.doi.org/10.5281/zenodo.3457374>  
<http://ojs.ukw.edu.pl/index.php/johs/article/view/7492>

The journal has had 5 points in Ministry of Science and Higher Education parametric evaluation. § 8. 2) and § 12. 1. 2) 22.02.2019.

© The Authors 2019;

This article is published with open access at Licensee Open Journal Systems of Kazimierz Wielki University in Bydgoszcz, Poland  
Open Access. This article is distributed under the terms of the Creative Commons Attribution Noncommercial License which permits any noncommercial use, distribution, and reproduction in any medium, provided the original author (s) and source are credited. This is an open access article licensed under the terms of the Creative Commons Attribution Non commercial license Share alike. (<http://creativecommons.org/licenses/by-nc-sa/4.0/>) which permits unrestricted, non commercial use, distribution and reproduction in any medium, provided the work is properly cited.

The authors declare that there is no conflict of interests regarding the publication of this paper.

Received: 10.09.2019. Revised: 19.09.2019. Accepted: 22.09.2019.

## Musculoskeletal manifestations of inflammatory bowel disease

**Maciej Putowski, Olga Padala, Adrianna Krupa, Michal Konopelko,  
Ewa Piasek**

[1putowski.maciek@gmail.com](mailto:1putowski.maciek@gmail.com)

ORCID:0000-0002-7575-2456

Department of Experimental Hematooncology, Medical University of Lublin, Chodźki 1 Street, 20- 093  
Lublin, Poland

[2olga.padala@gmail.com](mailto:2olga.padala@gmail.com)

ORCID:0000-0003-1469-0877

1st Department of Psychiatry, Psychotherapy and Early Intervention Medical University of Lublin,  
Gluska Street 1, 20-439 Lublin, Poland

[3adriannakrp@gmail.com](mailto:3adriannakrp@gmail.com)

ORCID:0000-0003-0866-3952

Department of Human Anatomy, Medical University of Lublin, Jaczewskiego 4 Street, 20-090 Lublin,  
Poland

[4mm.konopelko@gmail.com](mailto:4mm.konopelko@gmail.com)

ORCID:0000-0003-4103-7400

Department of Otolaryngology and Laryngological Oncology, Medical University of Lublin,  
Jaczewskiego 8, 20 954, Lublin, Poland

[5ewa.piasekk@gmail.com](mailto:5ewa.piasekk@gmail.com),

ORCID:0000-0003-3344-4022

I Clinic of Anaesthesiology and Intensive Therapy, Medical University of Lublin, Jaczewskiego 8, 20  
954, Lublin, Poland

## **Abstract**

Musculoskeletal manifestations are one of the most frequent extraintestinal manifestations of inflammatory bowel disease (IBD). Inflammatory of joints in IBD, both peripheral and axial arthropathies, belong to the spondyloarthritis group (SpA). The prevalence of the arthritis varies in different studies concerning around 5-14% patient with UC and 10-20% in CD. According to the Assessment in the Spondyloarthritis International Society, SpA are divided into axial and peripheral SpA based on the predominant symptoms. Two main patterns of peripheral arthritis of IBD were distinguished with different clinical presentation. Type 1 is characterized by acute and self-limiting symptoms such as pain, swelling or effusion affecting less than five, preferentially large joints, usually correlating with IBD flares. Management of the underlying disease is treatment of choice. Type 2 is characterized by polyarticular symmetric arthritis mainly affecting small joints of upper limbs. Symptoms of type 2 are often persistent for months or even years, independent of disease activity, requiring long-term treatment. Both types should be differentiated from commonly occurring arthralgia also associated with corticosteroid withdrawal. In addition to SpA, enthesitis, tenosynovitis and dactylitis may be diagnosed in IBD ranging from 7% to 50% of cases. Osteoporosis is also important complication observed in IBD with multifactorial pathogenesis i.e., corticosteroid and immunosuppressive treatment, extensive small-bowel disease or resection, age, low physical activity, nutritional deficiencies. The overall prevalence of osteoporosis in IBD is approximately 10-20%.

**Keywords:** arthropathies; osteoporosis; inflammatory bowel disease

## **Introduction**

Inflammatory bowel disease (IBD), including Crohn disease and ulcerative colitis are chronic relapsing disorders of the digestive tract. The etiology of the disease involves genetic, immunological and environmental factors including intestinal microbiota, however pathogenesis is still unknown [1]. The course of the disease is highly heterogeneous, including periods of inflammatory activity alternating with periods of remission, but also a progressive disease course, requiring urgent surgery [2]. Recent studies have demonstrated a rising incidence of IBD all over the world, especially in newly industrialized countries [3]. The most common symptoms of IBD are related to chronic inflammation of the gastrointestinal system such as diarrhea and abdominal pain. Besides the gastrointestinal symptoms, patients with IBD

can develop extraintestinal manifestations commonly affecting the joints, skin and eyes, but can the hepatobiliary system, lungs, heart and vascular system [4]. Up to 50% of patients with IBD develop at least one extra-intestinal manifestation (EIM) which can present before diagnosis. The probability of experiencing EIM increases with disease duration and also in patients who already have one EIM [5].

Musculoskeletal manifestations are one of the most frequent extraintestinal manifestations of IBDs. Inflammatory of joints in IBD, both peripheral and axial arthropathies, belong to the spondyloarthritis group (SpA) [5]. The prevalence of the arthritis varies in different studies concerning around 5-14% patient with UC and 10-20% in CD [6]. According to the Assessment in the Spondyloarthritis International Society, SpA are divided into peripheral and axial SpA based on the predominant symptoms [7].

### **Peripheral arthropathies**

The peripheral arthritis of IBD, unlike other inflammatory arthropathies, is generally non-erosive, with no persistent joint damage. Two main patterns of peripheral arthritis of IBD were distinguished with different clinical presentation. Both types should be differentiated from commonly occurring arthralgia also associated with corticosteroid withdrawal [8,9].

Type 1 (pauciarticular) is characterized by acute symptoms such as pain, swelling or effusion affecting less than five, preferentially large joints. This type of arthropathy usually correlating with activity of the underlying disease and is self-limiting with a maximum duration around 10 weeks [9]. Due to the association with bowel activity, the management of the IBD is the treatment of choice in affected patients [10].

Type 2 (polyarticular) is characterized by polyarticular symmetric arthritis mainly affecting small joints of upper limbs. This type of arthropathy is independent of the disease activity and symptoms often persist for months or even years, requiring long-term treatment [9]. Diagnosis is based on clinical symptoms with and exclusion of other forms of arthritis. Imaging using ultrasonography and radiographic imaging supports assessment [4].

In addition to SpA, enthesitis, tenosynovitis and dactylitis may be diagnosed in IBD ranging from 7% to 50% of cases [6].

### **Axial arthropathies**

Axial arthropathies including isolated sacroiliitis, inflammatory back pain and ankylosing spondylitis are associated with IBD. Diagnosis is based on magnetic resonance imaging [MRI] or radiographic features of sacroiliitis with clinical symptoms such as back pain and morning

stiffness. Radiological evidence of sacroiliitis is common in both UC and CD, occurring in 20–50 % of patients, however more frequently in CD [5,8]. Human leukocyte antigen [HLA]-B27 is found in 25–75% of patients with IBD but, due to a low prevalence it is not recommended as a diagnostic test in IBD [4,5].

Physiotherapy is a main strategy for management of axial SpA. Moreover, conventional treatment such as non-steroidal anti-inflammatory drug [NSAIDs] may be useful for short time, however it should be avoided as long-term treatment due to increased risk for relapse or higher disease activity. The use of COX-2 inhibitors such as celecoxib may be safer than conventional NSAIDs. In cases of refractory or intolerant to physiotherapy or conventional treatment, anti-TNF therapy is recommended. Biological treatment has shown improvement in arthropathies in patients with IBD [4,5,11].

### **Metabolic bone disease**

Osteoporosis is also important complication observed in IBD with multifactorial pathogenesis i.e., corticosteroid and immunosuppressive treatment, extensive small-bowel disease or resection, age, low physical activity, nutritional deficiencies. The overall prevalence of osteoporosis in IBD is approximately 10-20% [5,12]. The diagnosis of osteoporosis should be based on clinical symptoms, physical examination, bone mineral density (BMD) measurements, and laboratory results [13]. The gold standard is assessment of BMD by dual-energy X-ray absorptiometry [DXA] and osteoporosis is defined as a BMD value at least T-score lower than  $-2.5$ . Moreover, FRAX algorithm concerning other risk factors quantify the 10-year risk of experiencing a fragility fracture [14]. BMD measurement should be performed as in general population involving risk factors such as postmenopausal state, corticosteroid treatment, corticosteroid use  $> 3$  months, history of low-trauma fracture, and age [5]. However, there is no strongly supporting evidence confirming advantages of treatment and prevention of osteoporosis in young patients. The prolonged steroid treatment must be avoided to prevent bone loss [14,16].

## Literature

- [1] Khor B, Gardet A, Xavier RJ. Genetics and pathogenesis of inflammatory bowel disease. *Nature* 2011; 474(7351): 307–317.
- [2] Burisch J, Munkholm P. The epidemiology of inflammatory bowel disease. *Scand J Gastroenterol. England* 2015;50(8): 942–951.
- [3] Ng SC, Shi HY, Hamidi N, et al. Worldwide incidence and prevalence of inflammatory bowel disease in the 21st century: a systematic review of population-based studies. *Lancet* 2018; 23;390(10114): 2769-2778.
- [4] Ott C, Schölmerich J. Extraintestinal manifestations and complications in IBD. *Nat. Rev. Gastroenterol. Hepatol.* 2013; 10: 585–595.
- [5] Harbord M, Annese V, Vavricka SR, et al. The First European Evidence-based Consensus on Extra-intestinal Manifestations in Inflammatory Bowel Disease. *J Crohns Colitis* 2016;10(3): 239-254.
- [6] Colia R, Corrado A, Cantatore FP. Rheumatologic and extraintestinal manifestations of inflammatory bowel diseases. *Ann Med* 2016;48(8): 577-585.
- [7] Rudwaleit M, van der Heijde D, Landewé R, et al. The development of Assessment of SpondyloArthritis international Society classification criteria for axial spondyloarthritis (part II): validation and final selection. *Annals of the rheumatic diseases* 2009; 68: 777-783.
- [8] Fornaciari G, Salvarani C, Beltrami M, et al. Musculoskeletal manifestations in inflammatory bowel disease. *Canadian journal of gastroenterology* 2001;15: 399-403.
- [9] Sheth T, Pitchumoni CS, Das KM. Musculoskeletal manifestations in inflammatory bowel disease: a revisit in search of immunopathophysiological mechanisms. *J Clin Gastroenterol* 2014;48: 308–317.
- [10] Orchard TR, Wordsworth BP, Jewell DP. Peripheral arthropathies in inflammatory bowel disease: their articular distribution and natural history. *Gut* 1998;42: 387–391.
- [11] Herfarth H, Obermeier F, Andus T, et al. Improvement of arthritis and arthralgia after treatment with infliximab (Remicade) in a German prospective, open-label, multicenter trial in refractory Crohn's disease. *Am J Gastroenterol* 2002;97: 2688–2690.
- [12] Targownik LE, Bernstein CN, Leslie WD. Inflammatory bowel disease and the risk of osteoporosis and fracture. *Maturitas* 2013;76: 315-319.
- [13] Ali T, Lam D, Bronze MS, Humphrey MB. Osteoporosis in inflammatory bowel disease. *Am J Med* 2009;122: 599-604.
- [14] Kanis JA, Johnell O, Oden A, Johansson H, McCloskey E. FRAX and the assessment of fracture probability in men and women from the UK. *Osteoporos Int* 2008; 19: 385-397.

- [15] Scott EM, Gaywood I, Scott BB. Guidelines for osteoporosis in coeliac disease and inflammatory bowel disease. British Society of Gastroenterology. Gut 2000; 46 Suppl 1: i1-i8.
- [16] Van Staa TP, Laan RF, Barton IP, et al. Bone density threshold and other predictors of vertebral fracture in patients receiving oral glucocorticoid therapy. Arthritis Rheum 2003; 48: 3224-3229.