Opinion
Volume 8:2, 2022

Journal of Vasculitis

ISSN: 2471-9544 Open Access

Stomach Related and Hepatic Aspects of the Rheumatic Diseases

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Introduction

Gastrointestinal signs of rheumatoid arthritis are uncommon but can have serious consequences for sufferers. Some GI processes are directly related to, whereas others may be medication side effects or the result of coexisting autoimmune disorders. The importance of the tract in pathogenesis is discussed in this article, as well as the presentation, prevalence and diagnosis of RA related manifestations, concomitant GI autoimmune illnesses that may impact persons with and GI side effects of treatment. We emphasise the need of including illnesses unrelated to RA in the differential diagnosis when examining new GI symptoms in RA patients [1].

Description

The role of the gastrointestinal tract in the development of autoimmunity in humans is becoming increasingly recognised. Many autoimmune disorders, such as inflammatory bowel disease, Celiac disease and other autoimmune liver diseases, primarily affect the GI tract or the liver. Rheumatoid arthritis is a multi-organ autoimmune disease. Understanding the scope and prevalence of GI symptoms associated with, related autoimmune illnesses and RA therapies is critical for rheumatologists and other physicians caring for RA patients. All GI organs can be damaged by directly, through linked autoimmune disorders, or as a result of treatment [2]. This article will go over the presentation, epidemiology and diagnosis of GI disorders in children. RA is a common rheumatologic illness that affects. The prevalence of is estimated to be between and individuals. Lifetime risk has been found to be for men and women. Since the early, the prevalence of appears to be declining. Only in older age groups was seen a rise in prevalence, implying an increase in chronicity and a decrease in incidence. Several factors, including female sex, smoking and some infectious agents, such as GI pathogens, have been linked to an elevated risk of RA, as detailed below [3].

The same epitope a sequence of five amino acids in the hypervariable section common to multiple HLA DRB chains has also been linked to an increased risk of. However, even within identical twins, there is significant discordance in the development of RA, suggesting that environmental risk factors may play a role. There are other additional mutations that predispose to RA, but none as significantly as the common epitope and none that are essential or sufficient for disease development. A rising body of evidence suggests that the GI tract may play a significant role in the aetiology of RA. This idea was based on an epidemiologic relationship between RA and periodontitis, which led early researchers to postulate a causative role for periodontitis in the development of RA.

There are other clear incidences of GI infections inducing arthritis in people. Pathogens such as *Campylobacter, Chlamydia* and *Salmonella* cause reactive arthritisIntestinal bacterial overgrowth after ileojejunal bypass has been linked to a high rate of inflammatory arthritis, occurring in up to 50% of patients [4]. Whipple's illness is a classic example of inflammatory arthritis that occurs when a single bacterium species colonises the intestine of a vulnerable host. Modern molecular research tools have revealed a complicated interaction between microorganisms, particularly those found in the gut and the human immune system. Bacteroides fragilis and certain Clostridia species have been shown to directly upregulate T-regulatory cell activity, inducing an anti-inflammatory effect via IL-10 production [5].

Conclusion

Segmented filamentous bacteria have been shown to induce cell activity; these cells have been implicated in the pathogenesis of RA and secrete pro-inflammatory cytokines such as IL-17, TNF alpha and GM-C Dybiosis, or a shift in the homeostatic balance of commensal bacteria, is thought to result in an altered balance of anti- and pro-inflammatory interactions, leading to dysregulation of a local immune response. Local T cells may move to distant lymphatic tissue as a result of this dysregulation, allowing them to exert effects far beyond the site of activation in the intestine.

References

1. Brewer, Brian N and Diane L. Kamen. "Gastrointestinal and hepatic disease in systemic lupus erythematosus." *Rheu Dis Clin* 44 (2018): 165-175.
2. Popov, Yevgeniy and Karen Salomon Escoto. "Gastrointestinal and hepatic disease in Sjogren syndrome." *Rheu Dis Clini* 44 (2018): 143-151.
3. Craig, Ethan and Laura C. Cappelli. "Gastrointestinal and hepatic disease in rheumatoid arthritis." *Rheu Dis Clin* 4 (2018): 89-111.
4. Racanelli, Vito, Marcella Prete, Carla Minoia and Elvira Favoino, and Federico Perosa. "Rheumatic disorders as paraneoplastic syndromes." *Autoimm Revi* 7 (2008): 352-358.
5. Zizic, Thomas M., John N. Classen and Mary Betty Stevens. "Acute abdominal complications of systemic lupus erythematosus and polyarteritis nodosa." *Ame J Medi* 73 (1982): 525-531.

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Received: 10 March, 2022; Manuscript No. JOV-22-64941; **Editor Assigned:** 14 March, 2022; PreQC No. P-64941; **Reviewed:** 21 March, 2022; QC No. Q-64941; **Revised:** 24 March, 2022, Manuscript No. R-64941; **Published:** 31 March, 2022, DOI: 10.37421/2471-9544.2022.8.152.

How to cite this article: Chandler, Joseph. "Stomach Related and Hepatic Aspects of the Rheumatic Diseases." *J Vasc* 8 (2022): 152