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Induction or Exacerbation of Psoriatic Lesions During Anti-TNF- α Therapy

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Abstract

Paradoxical cases of psoriatic lesions induced or exacerbated by anti-tumor necrosis factor (TNF)-α therapy have been reported more frequently in recent years, but data related to inflammatory bowel disease (IBD) are rare. A systematic literature review was performed to provide information about this adverse effect in patients with IBD who receive anti-TNF therapy. Paradoxical cases of psoriatic lesions induced or exacerbated by anti-tumor necrosis factor (TNF)-α therapy have been reported more frequently in recent years, but data related to inflammatory bowel disease (IBD) are rare. A systematic literature review was performed to provide information about this adverse effect in patients with IBD who receive anti-TNF therapy. In the last few years, tumor necrosis factor-alpha (TNF-α) antagonists such as infliximab and adalimumab have revolutionized the treatment of inflammatory bowel disease (IBD) and psoriasis.[2]However, as the use of these biologic agents has increased, reactivation of latent infections, cutaneous reactions (eczematous, neoplastic, granulomatous, and psoriatic lesions), and other side effects have been documented.

Keywords: Eczematous • Granulomatous • Infections

Introduction

These adverse cutaneous events include an increasing number of paradoxical cases of psoriatic lesions (typical psoriasis and psoriasiform lesions). Because some patients may present with severe manifestations that require discontinuing the use of biologic agents and subsequently risk aggravating the underlying disease, physicians who treat these patients should understand the clinical manifestations of this side effect and therapeutic approaches.This paradoxical phenomenon has been described in the literature. However, studies reporting psoriatic lesions induced or exacerbated by TNF-α antagonists are largely heterogeneous regarding the therapeutic agent involved, underlying disease, treatment duration, personal and family history of psoriasis, type of cutaneous eruption, and therapeutic approaches and outcomes.[4] Furthermore, in the largest study available on this topic, patients with IBD comprised only 19.80% of the study population, whereas rheumatologic patients comprised 73.91%.We therefore conducted a systematic literature review to better understand the induction or exacerbation of psoriatic lesions (typical psoriasis or psoriasiform lesions) by TNF-α antagonists in patients with IBD. We also discuss the possible pathogenesis of this phenomenon.We performed a systematic literature review by searching the Medline (PubMed), Embase, Cochrane, SciELO, and LILACS databases for articles published from January 2004 to October 2011 (the final literature review was performed on October 30, 2011).

To identify all relevant articles published in English (clinical trials, case series and reports, and letters to the editor) about psoriasis or psoriasiform lesions induced or exacerbated by TNF-α antagonists (infliximab, adalimumab, and certolizumab) in patients with IBD, we used the following search terms: "adalimumab", "anti-TNF-α", "biological", "certolizumab", "Crohn", "inflammatory bowel disease", "IBD", "infliximab", "TNF inhibitor", "tumor necrosis factor alpha inhibitor", and "ulcerative colitis" combined with the terms "adverse event", "cutaneous adverse effects", "exacerbated", "guttate", "new-onset", "paradoxical", "plaque", "pustular" "psoriasis", "side effect", and "skin reactions". Relevant secondary references including abstracts published in the annals of national and international congresses were also included. Additionally, the reference lists of these articles were examined to identify additional studies. Repeated studies were considered only as a search source. Studies were selected based on their titles (and abstracts if they were available) and retrieved for more detailed analysis. Theoretical review articles that did not include additional cases were excluded, as were studies that did not present information about IBD separately from other diseases. Principles of the PICO strategy were adopted to ensure quality. Two authors independently extracted data from each article, and disagreements were resolved by consensus. Each study was individually reviewed to identify data concerning age, gender, personal and family history of psoriasis, biological medication administered, duration of clinical latency, lesion type (typical psoriasis or psoriasiform lesions), performance of cutaneous biopsy, therapeutic approaches and outcomes, and clinical IBD development. Nonspecific or unavailable information was designated as unknown or unstated data. The selected data were compiled in Microsoft Excel. Because this information did not provide sufficient data evidence or meta-analysis data, a simple descriptive analysis was performed. Management of the IBD following withdrawal of TNF-α antagonistIn the studies describing IBD development after discontinuation of TNF-α antagonists, gastrointestinal symptoms were controlled after the reintroduction of infliximab or with the introduction of adalimumab, certolizumab, azathioprine, methotrexate, mesalazine and corticosteroids, or methotrexate and corticosteroids demonstrating the considerable heterogeneity of approaches used after discontinuation of anti-TNF-α therapy. Psoriatic lesions recurred in cases in which etanercept (used to treat associated spondyloarthritis) and adalimumab were given. Recognition of the role played by the proinflammatory cytokine TNF-α in the pathogenesis of autoimmune inflammatory diseases (e.g., rheumatoid arthritis, IBD, and psoriasis) led to the development of TNF-α antagonists, which enabled important advances in the treatment of these disabling chronic diseases. However, paradoxical cases of psoriatic lesions induced or exacerbated by anti-TNF-α agents have increasingly been reported worldwide.

Although the first case of infliximab-induced psoriasis was described in a patient with Crohn's disease most of the evidence concerning this phenomenon was obtained from the rheumatologic literature. An increasing number of IBD patients have developed this cutaneous reaction, as described in subsequent

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reviews, and the latest review included 120 IBD patients. In the present review, 102 additional cases were included and analyzed for a total of 222 IBD patients with this reaction. Five studies were not included in this review, because it was not possible to extract only the data regarding the clinical development of patients with IBD [1,2].

Discussion

The present review showed that this adverse event was more frequently reported in adult patients who did not have a personal or family history of psoriasis, which is consistent with the results of previous studies.Pustular psoriasis was the most frequently described form of psoriasis (56%) in a recent review[7] that included patients with several underlying diseases; however, among IBD patients, plaque-type psoriasis was the most common form (61%). In contrast, psoriasiform lesions were the most frequently reported cutaneous lesions (55.86%) in our study. Because there is no clear definition of psoriasiform lesion, many lesions classified as psoriasiform may actually be the classic type of psoriasis.The review that analyzed the largest number of cases (207 patients with different underlying diseases) reported that the latency between anti-TNF-α administration and onset of psoriatic lesions was extremely variable, similar to what was observed in the present review. These cases involved different TNF-α inhibitors (infliximab, adalimumab, certolizumab, and etanercept); therefore, this paradoxical effect is a reaction to a pharmacologic class of drugs rather than a reaction to a specific drug. The present review and previous reviews found that more cases involved infliximab than any other anti-TNF-α agent, most likely because this was the first biological agent available on the market.The incidence of psoriasis among IBD patients can reach 11%, whereas it is only 1.5% in the general population [3-5].

Conclusion

In addition, the same genes (interleukin [IL]23R, IL12B, and tyrosine kinase 2) predispose for both IBD and psoriasis, which may account for the high rate of psoriatic lesions in IBD patients treated with anti-TNF-α agents.However, several aspects of this phenomenon provide evidence for the idea that it is a side effect of anti-TNF-α agents. These include the absence of a personal or family history of psoriasis in most reported cases, the increasing number of reported cases, the temporal relationship between anti-TNF-α treatment and the appearance of cutaneous lesions, and the clinical improvement observed after discontinuation of therapy and subsequent recurrence of psoriatic lesions after switching to another anti-TNF-α agent.

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Conflict of Interest

No potential conflict of interest was reported by the authors

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