HYPEREOSINOPHILIA DURING LATE INFLIXIMAB THERAPY

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BACKGROUND

Eosinophilia has several causes but anti-TNF therapy is not a usual one. We describe the case of a rheumatoid arthritis (RA) patient who developed high levels of eosinophilia due to infliximab therapy.

CASE REPORT

A 58-year-old white woman with a medical history of established atherothrombotic disease was diagnosed with seropositive RA in 2009. She had previously failed treatment with conventional synthetic DMARDs, abatacept and tocilizumab, and was started on infliximab 5 mg/kg/dose and leflunomide since 2013. Disease activity remained low with this last treatment, but a hypereosinophilic syndrome emerged after 7 years, in August 2020, and the patient was hospitalized with a hypertensive crisis and acute headache, with leukocyte counts of 31,440 cells and 24,770 eosinophils. Eosinophilia usual causes were excluded. An extensive hematological investigation was performed with two normal bone marrow biopsies and negative biomarkers (JAK2, BCR/Abl p190, p210, FIP, CHIC, PDGFRA). No primary hematologic abnormality was found and she remained on clinical surveillance, still using infliximab since there was no evidence of drug-induced eosinophilia. By December 2020, eosinophilia increased to 34,840 eosinophils and infliximab therapy was discontinued. After 3 months, the eosinophils count returned to the normal range and she is currently on therapy with golimumab and leflunomide.

CONCLUSION

Few case reports describe clinical and laboratory abnormalities induced by Anti-TNF therapy, mainly related to cutaneous and pulmonary syndromes. However, to our knowledge, a high eosinophil count such as the one seen in our patient, as well as hypereosinophilic syndrome, had not been described before. Japanese cohorts suggest that eosinophilia is an uncommon manifestation seen in about 0–0.35% of patients treated with anti-TNF. Most of them will not develop more than 1,000 eosinophils in peripheral blood and this abnormality will be seen mainly in up to 1 year of treatment initiation. Small studies propose that hypereosinophilia induced by infliximab occurs by late hypersensitivity reactions and/or high levels of IL-5. This cytokine is related to eosinophil growth and regulation and is derived from a Th2 response, which is enhanced due to the Th1 response blockade induced by Infliximab. Our patient developed high levels of eosinophilia after more than 7 years, findings not described so far in the infliximab literature. After stopping this medication, complete normalization of eosinophil count occurred. In this context, when more common causes are excluded, anti-TNF therapy should be suspected as a rare cause of hypereosinophilia.

KEYWORDS

Eosinophilia, Hypereosinophilic syndrome, Drug-induced eosinophilia.