Edema of the eyelids and sclera after rituximab infusion for orbital MALT lymphoma

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A 60-year-old man with heavily pretreated and refractory mucosa-associated lymphoid tissue (MALT) lymphoma presented with bilateral orbital swelling and lymphadenopathy. (Figure 1) Given the lack of standard chemotherapy for refractory MALT lymphoma, rituximab (375 mg/m^2) therapy in combination with cladribine was initiated.[1,2] The patient received intravenous diphenhydramine (50 mg) and oral acetaminophen (650 mg) 30 minutes before his rituximab infusion.[3] Within 2-hours of the rituximab infusion (started at 25 mg/h for 1 hour, subsequently 100 mg/h), the patient developed painful edema of the bilateral eyelids and sclera. (Figure 2) Therapy was interrupted, and he received intravenous hydrocortisone. Therapy was restarted at the rate of 25 mg/h, and he completed the total dose. One week later, treatment was again repeated with diphenhydramine and acetaminophen prophylaxis. This treatment was well tolerated, without recurrent edema. The patient received 6 additional rituximab treatments without any adverse events. Rituximab, a chimeric antibody directed against CD20, is widely used against B-cell non-Hodgkin's lymphoma (NHL). In previous reports, rituximab therapy was often associated with an infusion-related toxicity consisting of fevers, chills, and rigors, usually during the first infusion.[3,4] This toxicity usually is self-limited and generally subsides with temporary interruption or slow infusion of the rituximab concurrent with initiation of supportive care measures (such as acetaminophen and diphenhydramine administration). Subsequent treatments with rituximab are generally well tolerated and usually associated with a reduction in the infusion-related toxicity. Severe infusion-related reactions or overt bronchospasm requiring medical intervention was noted in only 2% of patients receiving rituximab during the first treatment cycle. The primary mechanism of action of rituximab in vivo remains unresolved, with several potential provocative mechanismas, including antibody-dependent cellular cytotoxicity, complement-mediated cytotoxicity, and direct induction of apoptosis through an incompletely characterized CD20-mediated signaling pathway.[5,6] Gradual tumor destruction by immune effector cells leads to local cytokine release and accumulation (tumor necrosis factor alpha, interferon gamma, and interleukin 6)[7,8] and may have resulted in local edema in our patient.

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Figure 1.

Figure 2.

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