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Keeping An Eye On Bisphosphonates: Drug Induced Uveitis
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Introduction: Bisphosphonates are considered the treatment of choice for osteoporosis. They have strong anti-osteoclastic activity and are potent inhibitors of bone resorption. Ocular inflammation, including conjunctivitis, uveitis, episcleritis and scleritis, is a rare side effect of bisphosphonates. A few cases in the literature have reported ocular inflammation secondary to bisphosphonate use. But largely, it has been an underrecognized cause of uveitis. We report a case of unilateral uveitis secondary to bisphosphonate therapy that was completely resolved after discontinuation of the medication.

Case report: A 68-year-old female with a history of osteoporosis diagnosed in 2019 on routine bone density scan presented for treatment evaluation. She had a history of multiple fractures after falling from standing height but no history of kidney stones, hypercalcemia, or GERD. She was started on alendronate 70mg once a week in 2019 with stabilization in bone density on repeat scan in 2020. After several weeks of treatment, she developed left eye uveitis and was treated with phenylephrine 2.5% and prednisolone acetate 1% ophthalmic solution. Because of these symptoms, the patient had diagnostic studies done which revealed positive ANA. She then had an extensive rheumatology workup, including serum SM antibody, ANCA screen, RNP antibody, Sjogren’s antibodies, DNA (DS) antibody, and HLA-B27 DNA typing, which were all negative. Due to some case reports citing an association of bisphosphonate use with eye inflammation, the bisphosphonate was stopped with gradual improvement in her eye symptoms. An attempt to restart alendronate resulted in worsening eye symptoms, so the therapy was ultimately switched to denosumab.

Discussion: The incidence of ocular inflammation after bisphosphonate initiation is 0.08%. Symptoms usually develop 24 hours to a few weeks after starting treatment. The potential pathogenesis of bisphosphonate-induced ocular inflammation is thought to be secondary to bloodstream absorption and secretion of the medication into tears from the lacrimal gland causing irritation to the eyes’ mucous membranes. Originally it was thought that ocular inflammation only occurred with nitrogen-containing bisphosphonates, due to the nitrogen group possibly playing a role in activating gamma delta T cells leading to the release of acute phase reactants, such as cytokines. More recently, both nitrogen and non-nitrogen containing bisphosphonates have been associated with reported cases of ocular inflammation. Presentation can be either unilateral or bilateral, but the majority of reported cases were unilateral. All patients receiving bisphosphonates who develop signs and symptoms of ocular inflammation should be referred promptly to an ophthalmologist. The drug should be discontinued once the diagnosis is made to prevent further progression. In conclusion, clinicians should be aware of the potential for ocular inflammation upon bisphosphonate initiation. Patients may benefit from switching to a different class of medication, such as denosumab, if they experience symptoms of ocular inflammation with bisphosphonates.

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