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Incomplete Vogt-Koyanagi-Harada Syndrome in a 30 year old Black Male

Abstract Text:

Young black male presents to ER with blur vision, headaches and tinnitus for 3 days. Exam reveals multiple serous retinal detachments, PEDs and choroidal thickening. Diagnosis: incomplete Vogt-Koyanagi-Harada. Treatment: high dose oral steroids.

I. 30 year old black male
CC: Heavy lids, bilateral blurred vision, photophobia for three days with new onset bilateral severe head pain
No ocular or medical history
No medication
Weakness lower extremities and ringing of ears.

II. Clinical: BCVA 20/25+ right eye, 20/25- left eye. Entrance exams and anterior segment exam within normal limits. IOP 17,18mmHg @6:10pm. Optic nerves well perfused, 0.30 C/D ratio right eye 0.45 C/D ratio left eye. DFE reveals bilateral serous retinal detachments and pigment epithelial detachments with multiple areas of subretinal fluid, all within posterior pole in both eyes. Peripheral retina was within normal limits both eyes. A contact B-Scan and diagnostic A scan: Thin dome shaped membrane in the macula left eye greater than right eye. Diffuse low reflective thickening of the fundus at the posterior pole and thickness is 2.8mm left eye, 3.3mm right eye. Minimal infiltration of tenons space. FA: Pooling in pigment epithelial detachments, no leakage both eyes. ICG: multifocal choroidal drop out both eyes, areas of blocking from PEDs. OCT: multifocal PEDs and serous retinal detachments in both eyes both peripapillary and in the posterior pole. Physical: New onset tinnitus, headaches, abdominal pain and weakness in lower extremities.
Labs: PRP, FTA-ABs, ESR, CBC, ACE-1, HIV, P-ANCA, C-ANCA all normal. PPD Positive. Chest X-ray normal MRI normal

III. VKH*PCV

B cell lymphoma Sympathetic ophthalmia CSR Posterior Scleritis Bilateral diffuse melanocytic hyperplasia SLE Sarcoidosis APMPPE

IV. The patient was diagnosed with Incomplete Vogt-Koyanagi-Harada (VKH) during the uveitic phase of the condition based on clinical diagnosis. VKH is a multisystem autoimmune condition that targets pigmented cells which can affect the eyes, ears, skin and meninges. Presentations vary with race. Can reveal bilateral panuveitis with diffuse choroidal thickening, optic disc hyperemia and serous retinal detachments.

Clinical course is separate by phases of the disease; prodromal, uveitic, convalescent, and recurrent phases. Complete VKH includes ocular, neurological and integumentary signs, Incomplete VKH involves ocular and neurological or integumentary signs while probable VKH has isolated ocular signs. Additional criteria include: No history of trauma or ocular surgery, negative laboratory results without secondary disease and bilateral ocular signs. Early ocular signs: diffuse choroiditis (serous retinal detachments, focal areas of subretinal fluid, diffuse choroidal thickening) using ultrasound with FA changes. Late ocular signs: history prior uveitis and ocular depigmentation, chorioretinal scars, anterior uveitis Immunogenetic
component: HLA gene V. VKH treated with high dose systemic steroids. Immunomodulators often needed in recurrent disease that may respond poorly to steroids.

2. Sakata, Viviane M. "Diagnosis and Classification of VKH." *Diagnosis and Classification of VKH*. Autoimmune Reviews, 15 Jan. 2014.

VI. The Patient has incomplete VKH with no skin signs. Tx at early uveitic phase before inflammation was anterior with oral prednisolone, resolution ocular signs at 2 weeks.