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Case Report *Volume 5 Issue 2*

The Efficacy of Steroidal Therapy on Solitary Sarcoid Choroid Granuloma

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Abstract

Choroidal granuloma presenting with solitary choroidal lesion with ocular signs and intraocular inflammation is majorly associated with systemic disease. The case presented an Asian middle-aged female with the ocular sign of a solitary small choroidal lesion. Ocular and physical examinations with serum tests were used as prime diagnostic tools to tackle the disease as acute uveitis sarcoid. B-scan ultrasonography was the only diagnostic test that showed the choroidal small mass elevation. The effect of systemic steroidal therapy improved visual acuity and the appearance of lesions.

Keywords: Ocular Dextrus; Ocular Sinister; Posterior Subcapsular Cataract

Abbreviations

ACE: Angiotensin-Converting Enzyme; BHL: Bilateral Hilar Lymphadenopathy; CSF: Cerebrospinal Fluid; HLA: Human Leukocyte Antigen; VKH: Vogt-Koyanagi-Harada.

Introduction

Sarcoidosis is an inflammatory disease characterized by noncaseating granulomas in affected organs. The prevalence is expected to be 10 times greater in dark-skinned Africans than whites [1]. The etiology of the disease is enigmatic and affects various organs such as the Lungs, skin, Lymph nodes, eyes, and liver. Histology shows granuloma comprises epithelioid histiocytes, Lymphocytes, and multinucleated giant cells. Ocular symptoms include Blurring of vision, photophobia, redness, and pain. General symptoms include fever, cough, dyspnea, fatigue, and erythema nodosum [2]. Diagnostic test of sarcoidosis includes testing for a raised angiotensin-converting enzyme (ACE) level and chest x-ray to evaluate the presence of hilar lymphadenopathy. However, a definitive diagnosis of sarcoidosis requires a positive biopsy of the conjunctiva, lacrimal glands, and bronchoscopy.

Case Report

A 45-year-old Asian female presented with decreased vision, mild discomfort for distance and near, redness, and photophobia in OS for two weeks. She was also complaining of mild irritable eyes. She denied ocular tearing, discharge, and headache. Her previous ocular history showed photorefractive keratectomy for twelve years of -5.00DS and presbyopia of +1.75DS. She dismissed past ocular infections and trauma. Her past medical history showed normal general health. She denied any metastases breast and spine history. She denied any ocular history of skin and chest problems. Family ocular history was no ocular associations. The Social history was normal. She had known dust allergies but denied any drug reactions and a history of previous medication. She was oriented to time, place, and person.

Uncorrected distance visual acuity was 20/25p OD and 20/32p OS. Manifest spectacle correction revealed was 20/20 OD with -0.50DS and -1.50/-0.50*90 vision was 20/25 OS. Near acuity was N5 OD and reduced N8 OS at 40cm. Pupils were equal, round, and reactive. No afferent pupillary defect was noted. Confrontation fields were full of finger counting in both eyes. Extraocular muscles were normal in all gazes without pain or diplopia. The ocular alignment showed Orthophoria with the cover test at a distance and near. Goldman application tonometry measured 14 mmHg OD, and 12 mmHg OS at 11:48 am. Anterior segment examination revealed normal adnexa, lids, lashes, puncta, and palpebral of both eyes, while mild bulbar conjunctival congestion was noted in the left eye. Both eyes showed mild dry eyes degree 1. Siedal test was normal. Anterior chambers appeared remarkable in OD and OS showed evidence of Flare cells estimation of the chamber angles was 4/4 via the Von Herrick method. Both irides were flat and brown. There were marked lenticular changes PSCC grade 2 in both eyes and vitreous activity of vitritis OS. Dilated fundoscopic exam Mydriacyl with 1% left eye revealed a slightly elevated sectoral small mass yellow spot 1.5mm*1.5mm and 1DD superior to disc. Macula showed normal contour in both eyes. Detailed disc assessment was a normal optic nerve contour with a cup-to-disc ratio of 0.30 OD 0.2 OS. There was a normal neuroretinal rim.

Diagnosed Procedure

The best screening test for sarcoid uveitis is a chest X-ray and in normal tests, an alternative CT scan of the chest may be valuable. This patient denied any chest problem so ACE (Serum angiotensin enzyme) and laboratory tests were used as tools to monitor the disease activity. The patient was advised for OCT and B-scan to visualize the loci of the disease. Depending on the severity the case was acute with a slight visual effect so it was easily manageable. However, in chronic disease with visual morbidity, a complete examination with biopsy is required to approach the ultimate disease and reduce the complication. According to the IWOS, individuals with clinical signs of ocular sarcoidosis should receive the following diagnostic tests:

- Negative tuberculin skin test in a BCG-vaccinated patient or a patient having had a positive tuberculin skin test previously.
- Elevated serum ACE levels and/or elevated serum lysozyme.
- Chest x-ray revealing bilateral hilar lymphadenopathy (BHL).
- Abnormal liver enzyme tests.
- Chest CT scan in patients with a negative chest X-ray result.

Studies have shown that the definitive diagnosis of sarcoid is biopsy. Various biopsies to the most accessible site conjunctival follicles and lacrimal gland biopsy can be done in case of lacrimal gland involvement. Where depending on the severity of the disease, pulmonary biopsy with fiber optic bronchoscopy can biopsy 80% of the disease [3]. Laboratory Tests of the patient showed raised ACE (Angiotensin converting enzyme) and abnormal liver enzyme test. Serum enzyme greater was found to be specific 76% for sarcoidosis uveitis [4] (Figures 1-4).

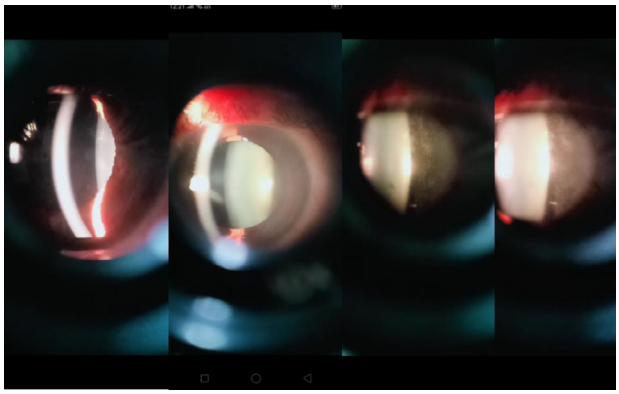

Figure 1: Photo courtesy Rubina Shah National Eye Centre.

Note: Slit lamp biomicroscopy demonstrated normal cornea, lenticular changes, and vitritis.

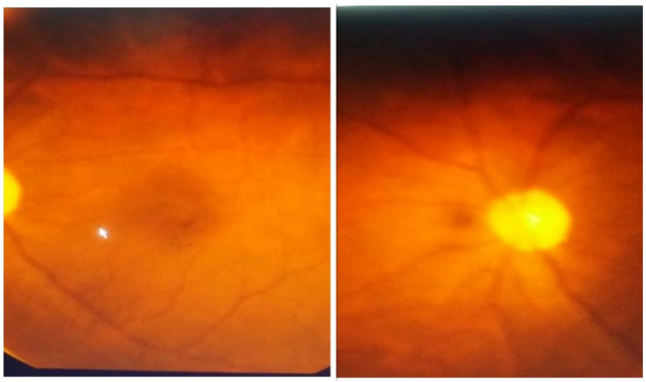

Figure 2: Photo courtesy Rubina Shah National Eye Center.

Topcon Fundus camera, Media haze as lenticular changes Vitritis impeded the image quality where granuloma is hardly seen.

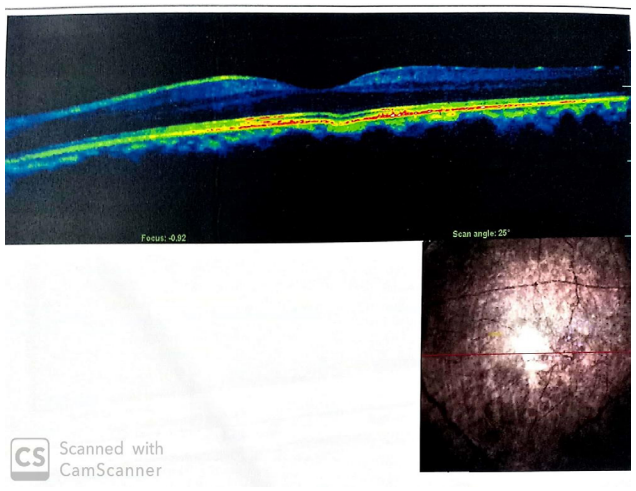

Figure 3: Photo courtesy Rubina shah National eye center.

Note: OCT line scan macula of the left eye shows intact IS/OS layer and ELM. The RPE layer shows a homogenous arrangement. The central foveal thickness profile shows 250 microns without any odema.

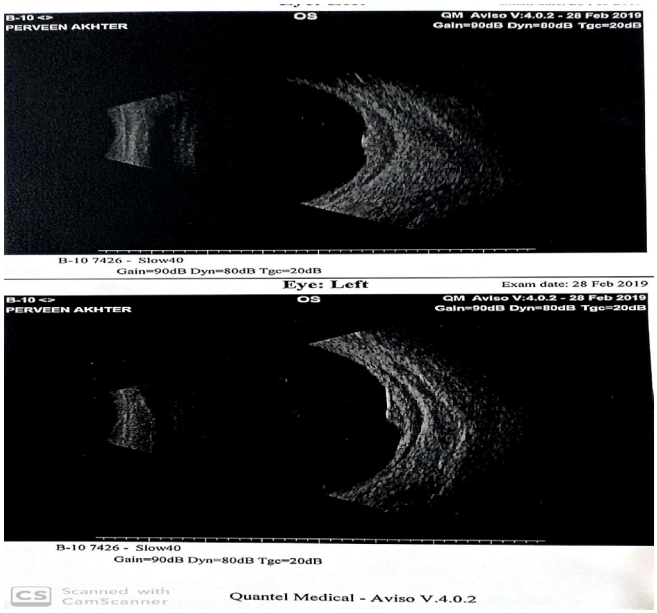

Figure 4: Photo courtesy Rubina Shah National Eye Center.

Note: Ultrasonography of the Left eye showed a two-dimensional scan that was the only helpful diagnostic test to locate the lesion. It showed cataracts with vitreous echogenicity. The retinal profile showed a slightly raised area near the optic disc.

Differential Diagnosis

The differential diagnoses of autoimmune and infectious causes of sarcoid uveitis are syphilis, Lyme disease, tuberculosis, Behcet's disease, Vogt-Koyanagi-Harada disease, and sympathetic ophthalmoplegia.

Syphilis: Syphilis is a sexually transmitted disease caused by treponema pallidum. Syphilis uveitis is a major threatening disease. The risk factor that suggests syphilis is HIV, a history of unprotected sex, and substance abuse. It affects the skin, lymph nodes, palm and sole maculopapular rashes, and oral gum cavity. Ocular findings may present conjunctivitis, scleritis, keratitis, uveitis, iritis, multifocal choroiditis, and papilledema, extraocular motility deficits. The serological test is the mainstay to diagnose the disease. Treatment includes a course of antibiotics for neurosyphilis and topical, oral steroidal therapy.

Lyme Disease

Lyme borreliosis is a tick-borne bacterial infection that was named in 1975 due to cluster or arthritis and skin rashes and subsequently proved to be transmitted by the tick of genus Exodus. This unknown spirochete has 40 distinct species causing Lyme disease and its variants are grouped in species known as B. burgdorferi. Burgdorferi are associated with different forms of the disease as European borreliosis is caused by B. garinii and B. afzelii, and American Lyme disease by B. burgdorferi sensu stricto. Lyme disease causes about 4% of uveitis in Finland. It follows a three-stage form as seen with other spirochetal infections. B. burgdorferi is inoculated via a tick bite, which forms a small papule. The most common sites are the thigh, groin, or axilla, where the hidden ticks more often remain for days as they are often missed. After 3 days to 3 weeks, the typical primary lesion, erythema chronicum migrans, starts as a slowly intensifying, erythematous rash, characteristically Associated with intense itching or sometimes pain. Vesiculation or even necrosis and ulceration of the center may occur. Eventually, the center clears to leave an annular erythema which will ultimately fade. In second-stage disease may develop 60% recurrent pauciarticular arthritis within a few months of infection. This clinical ocular manifestation shows intraocular inflammation is rather less common, but the majority presenting with uveitis have associated fever or lymphadenopathy. Anterior uveitis may be granulomatous. Neuroretinitis causing disk swelling with partial or complete macular star formation is the most common ocular manifestation. An intermediate uveitis with cell clumping and panuveitis may manifest. Peripheral retinal vasculitis is often seen that may manifest as angiomatous lesions. Patients's disease management can be observed

without treatment. Those with severe lymphadenopathy or pyrexia will require antibiotic treatment. The organism is sensitive to a variety of antibiotics including tetracycline's (typically doxycycline), and aminoglycosides including azithromycin, rifampicin, and co-trimoxazole. Those with uveitis involving the posterior pole should be treated. Topical steroids are helpful where required but the use of oral steroids is uncertain. The differential diagnosis of those with Neuroretinitis and focal retinitis includes toxoplasmosis and therefore the use of co-trimoxazole or another antibiotic with dual efficacy is reasonable. Most immunocompetent patients recover good vision [5,6].

Toxoplasmosis

Toxoplasmosis is a zoonotic infection, and its causative organism is Toxoplasma gondii, a crescentic intracellular protozoon. It is found most commonly in subtropical areas. The primary host of T.gondii is present in cats, which excretes oocytes and remains robust bodies in feces for several days, or in water for weeks or more and resists standard disinfection. Humans may ingest it from an intermediate host. This direct route of infection may be common in childhood especially in tropical areas, whereas adults commonly ingest tissue cysts infecting other intermediate hosts, by eating undercooked meat, especially pork in Western countries. It can also be acquired by drinking unpasteurized goats‟ milk or cheeses. The risk factors prevalence varies with climatic changes, dietary habits, and standards of hygiene. It may cause posterior uveitis in humans. Systemic association in some 20% of affected live-born babies has significant systemic effects, including microcephaly, hydrocephalus, microphthalmia, optic neuropathy, deafness, learning disabilities, fits, and failure to thrive. Further, Longitudinal studies have shown ocular and neurological involvement in up to 80% of cases. Manifestation of systemic causes some systemic upset, often a mild flu-like illness. Sometimes significant lymphadenopathy, particularly affecting the posterior cervical nodes, may occur. Pyrexia, malaise and fatigue, sore throat, maculopapular rash and sometimes hepatosplenomegaly may be seen. More severe effects are rare in immunocompetent individuals, but in immunocompromised individuals‟ meningoencephalitis, pneumonitis, myocarditis, and hemolytic anemia may be seen. Ocular manifestation shows a degree of active anterior uveitis in association with their active posterior segment disease. It may range from a few anterior chamber cells to a very severe uveitis with multiple posterior synechiae. The intraocular pressure is raised in 40% of patients.

Typical recurrent retinochoroiditis ocular toxoplasmosis is atrophic Chorioretinal scars of variable size with pigment clumping. An associated retinal vasculitis can be noticed. Moreover, the optic disk augurs poorly for permanent visual field loss. Neuroretinitis usually presents with marked visual loss, optic nerve head swelling, and Peripapillary exudate, and a macular star. Retinochoroiditis may cause panuveitis, and associated scleritis has been reported. Pragmatically, features that can help diagnose acute ocular toxoplasmosis are unilateral inflammation, the presence of a single white inner retinal active focus, and old Chorioretinal scarring. Few other conditions cause unifocal, localized retinitis. Episodes of active toxoplasma retinochoroiditis are self-limiting. During the period of symptomatic toxoplasmosis, symptoms will depend on both the degree of vitreous opacification and the location of the lesion. Necrosis and scarring of the active retinal focus don't require any treatment. However, treatment can limit the size of the resultant scar and bind the intensity and duration of vitreous opacification which may eradicate free intraocular tachyzoites. Anti-protozoal therapy and anti-inflammatory medication create a rationale for treatment for the disease. However, studies of the evidence in humans for the efficacy of either treatment remain questionable and drug-induced side effects may affect 40% of patients. Quinolone therapy such as pyrimethamine is a 4-aminoquinoline derivative similar to quinine. It inhibits dihydrofolate reductase, which converts folic to folinic acid. Another option is Clindamycin inhibits protein synthesis by acting on microbial ribosomes. It is taken orally in a dose of 300 mg four times daily. Ocular toxoplasmosis in pregnancy is crucial for the development of acute toxoplasma retinochoroiditis during pregnancy and is a substantial and complex management challenge. Acquired toxoplasmosis during pregnancy often leads to fetal abnormalities fluctuating from spontaneous abortion or major abnormality in the first trimester, to a range of abnormalities characterized by the TORCH syndrome. In an immunodeficient mother, there is a fetal risk even if the ocular lesion is recurrent, but this risk is difficult to quantify [6-9].

Vogt-Koyanagi-Harada (VKH) syndrome: Vogt first reported poliosis and uveitis in (Vogt 1906), later Koyanagi reported the coexistence of vitiligo and alopecia with uveitis (Koyanagi 1929) and Harada described serous retinal detachment in association with cerebrospinal fluid (CSF) abnormalities. These signs were grouped as components of a single entity known as VKH syndrome. It is common in Asian, Hispanic, and indigenous Native American individuals. There is a significant association of VKH with HLA-DR4 and HLA-DRB1 (various alleles in different ethnic groups, but most commonly HLA-DRB1*0405). The prevalence of disease is more common in women than men [10].

Histologically, VKH syndrome is a granulomatous panuveitis with diffuse infiltration of giant cells, epithelioid cells, and lymphocytes similar to sympathetic uveitis. The epithelioid cells may contain melanin pigment. Systemic association shows viral illness with malaise, pyrexia, headache, and

sometimes dizziness or meningism. Other rare symptoms of focal neurological signs may occur at this stage, even including dysphasia, cranial nerve palsy, or hemiparesis. CSF pleocytosis is usually found in the central nervous system CNS investigation. It is associated with inflammatory bowel disease, thyroiditis, and diabetes. Ocular manifestation shows always bilateral uveitis. It is usually a symmetrical onset, but sequential involvement of the second eye may be delayed by a week. Hyperemia is markedly seen with diffuse choroiditis and papillary edema. Multifocal lesions may be seen and a substantial breakdown of the RPE, and serous elevations of the retina may occur. Anterior uveitis with KPs and iris nodules may be seen but are not essential for diagnosis. Raised intraocular pressure is common which is associated with anterior chamber shallowing that indicates forward displacement of the iris/lens diaphragm caused by ciliary body swelling. The diagnostic procedure is entirely clinical and CSF cerebrospinal Fluid analysis is essential for diagnosis. VKH syndrome is likely to improve with high-dose systemic steroid treatment which is beneficial for early panuveitis. Treatment therapy may comprise 100 mg/day orally and an alternative with intravenous therapy of methylprednisolone. Gradually treatment may be tapered once control of the disease is established. Inflammation may reactivate if steroids are tapered too quickly and such episodes are felt to increase the risk of steroid-resistant disease in the future. For those with treatment-resistant disease, an alternative infliximab has been used successfully in a small number of patients [11]. Belligerent early management with systemic steroids may require additional immunosuppression management of VKH syndrome has shown a favorable outcome, but the prognosis for eventual visual function must be guarded [12,13].

Behcet's Disease

(Behcet's 1937) Turkish dermatologists described the association between intraocular inflammation and oral ulceration. A classic triad of uveitis, oral and genital ulceration, and other manifestations of the body system may become involved. Behcet's disease typically affects young adults. Their geographic distribution shows prevalence in Japan which is as high as 13.5/100000. It accounts for 20% of patients in some uveitis clinics. The manifestation of Behcet's disease is necrotizing vasculitis, but the etiology cause is unknown. A viral cause such as herpes simplex virus or a bacterial etiology (streptococci) has been suggested. The pathophysiology shows typical inflammatory foci commence with lymphocytic infiltration, followed by polymorph migration and fibroid necrosis of blood vessels. Enormous polymorph extravasation is a typical feature, although there is substantial evidence that the disease is T-cell driven, and this notion is strongly supported by the effectiveness of cyclosporine. In men, ulcers on the genitals specifically the anterior scrotum are affected. However, lesions on the posterior scrotum or perineum may also occur which may cause severe discomfort. Skin involvement shows Erythema nodosum is a non-specific panniculitis that may be associated with uveitis. Further, Folliculitis, papulopustular eruption, or acneiform nodules are also seen. Neurological involvement of the disease occurs in perhaps 10% of most cohorts and can be life-threatening.

Presentations can include ataxia, oculomotor disturbance, and sensor neural deafness. Intraocular inflammation occurs in about 80% of patients but is more common in men than in women. Posterior segment involvement and macular lesions are more likely to develop in males. Uveitis is not usually the first manifestation of the disease. Studies show that those who develop the disease aged <25 years have a higher incidence of eye inflammation. Uveitis is typically recurrent, with extremely rapid exacerbations. The disease is almost always eventually bilateral but individual attacks are usually unilateral or asymmetrical. Anterior uveitis is symptomatic, with pain and photophobia and Hypopyon is frequently seen, but is usually grade 0 and sometimes visible only on gonioscopy of the inferior angle. The posterior segment lesions progress to rapid onset vitreous opacification, the smoky appearance being caused by myriads of free-floating inflammatory cells. Through this hazy medium, areas of white focal retinitis may be punctate, and often multifocal is hardly visible. Behcet's disease treatment depends on the severity of symptoms within the areas of expertise of several specialists and close liaisons are required to optimize treatment. Anterior uveitis in Behcet's disease is often hyperacute and requires intensive topical steroid therapy, sometimes combined with subconjunctival administration. High-dose systemic steroids are given in acute posterior uveitis and particularly in severe involvement, intravenous methylprednisolone is administered. Long-term maintenance with systemic steroids alone is highly unsatisfactory at preventing recurrences, associated with substantial side effects and, in the long term, ineffective. Immunosuppressive is therefore usually required. An enthusiastic way has been introduced currently for the use of anti-TNF-α (tumor necrosis factor α) monoclonal antibody treatment in both combined immunosuppression-resistant Behcet's disease and some other forms of severe uveitis. Infliximab in particular has been widely beneficial, but anecdotally used but no controlled trial has been published yet.

Treatment

Treatment of anterior uveitis of sarcoidosis generally responds well with intensive corticosteroid therapy to suppress the inflammation. Where posterior sarcoidosis responds well with systemic oral corticosteroid therapy. The patient was referred to a medical doctor for management of the disease.

Topical Maxidex eye drops every 2 hours were prescribed for uveitis inflammation. The patient was advised oral steroids Tab Xanax (Alprazolam) bid for a week then tapperd to one dose, tab Deltacortil 5mg twice a day of five tablets then tapperd, tablet Myrim and tablet Vita-6. The patient had raised blood pressure so a repressor half dosage bid was prescribed. A follow-up examination was necessary to guard against the disease. The patient was called after 3 weeks. Moreover, other management suggested advanced posterior segment lesions such as perivascular sheathing and peripheral Chorioretinal nodules that are not associated with visual loss may not need oral corticosteroid therapy. However, major complications like vascular occlusion, neovascularization, macular mass lesions, and optic disc lesions may deteriorate vision and require corticosteroid therapy. Rarely, corticosteroid-sparing agents, such as hydroxychloroquine, methotrexate, azathioprine, mycophenolate mofetil, or ciclosporin, may be useful in patients with corticosteroids.

Follow-Up (Three Weeks)

On the date of examination patient's uncorrected visual acuity was 20/25 OU. The best corrected visual acuity was 20/20 OD with -o.50/-0.50*35 OD and -0.75/-0.50*95 with 20/20p in OS. The left eye had improved acuity with reduced AC activity. The pupil was reactive and round lenticular changes and remission of solitary granuloma near the disc. The patient was advised to follow-up for another three weeks.

Follow-Up (Three Weeks)

On the date of examination, the patient was 20/20p with spectacle correction of mild error and N5 for near with an addition of + 1.50DS. Early lenticular changes and pupils were equally round and reactive. 1-2 cells and Fundi were reasonable OU. Their blood pressure was 130/80. The patient was satisfied and Maxidex eye drop was advised to tap gradually.

Karma and co-workers classified the course of ocular sarcoidosis as monophasic, relapsing, or chronic. The three different courses of uveitis correlated with the visual outcome. Patients with monophasic uveitis retained 20/30 or better visual acuity in 88% of eyes, with relapsing uveitis in 72% of eyes, and with chronic uveitis in none of six eyes. Similarly, those with monophasic uveitis had a visual acuity of 20/70 or worse in 12% of eyes, with relapsing uveitis in 28% of eyes, and chronic uveitis in 67% of eyes. Hence the course of uveitis appears to correlate with the long-term visual outcome [14,15].

Discussion

Sarcoidosis is a multisystem granulomatous disease of idiopathic etiology characterized by intrathoracic involvement. Ocular involvement occurs in approximately 15% to 25% of patients with sarcoidosis [16,17]. Posterior segment manifestations seen in sarcoid may account for up to 28% of the lesions. The largest case series of patients with uveitis report that approximately 5% of patients with uveitis have biopsy-confirmed association with systemic sarcoidosis [18-20].

Epidemiology of the disease shows that all races are affected, but its prevalence is higher in blacks than whites. The frequency is slightly higher in females approximately 60%, where generally both sexes are affected. It is a disease of young adults, with almost three-fourths of cases occurring in those younger than 40 years of age. The prevalence in children is the least [21-24]. The etiology of sarcoidosis is unknown. Rather, multiple theories have been shown which include a variety of infectious agents, allergy to pine pollen and peanut dust, chewing pine pitch, and hypersensitivity to certain chemicals such as beryllium or zirconium. To this point, there is no conclusive evidence to implicate any of these as an etiologic agent. Studies have shown a Familial association of human leukocyte antigen (HLA) typing which suggests a possible genetic predisposition, but these studies lack any conclusion [1].

The clinical manifestations of affected organs by sarcoidosis are the lungs, lymph nodes, and spleen, skin, eyes, nervous system, and bones and joints [1,2,8,9,25]. Intrathoracic sarcoidosis has demonstrated involvement in 90% of patients. The chest radiograph shows the abnormality of the chest is the key point of diagnosis of the onset of sarcoidosis in almost all patients. Chest radiograph abnormalities have been classified into various stages depending on the severity. Stage 0 is characterized by a normal chest radiograph. Stage 1 is characterized by bilateral hilar lymphadenopathy without pulmonary infiltration and is seen in 65% of patients. Stage 2 is characterized by hilar lymphadenopathy associated with pulmonary infiltration and is seen in 22% of patients with sarcoid. Stage 3 sarcoid shows pulmonary infiltration with fibrosis but without bilateral hilar adenopathy and occurs in 13% of patients. Extrapulmonary involvement of the reticuloendothelial system, specifically the extrapulmonary lymph nodes spleen, or both occurs in 23% to 37% of patients with sarcoidosis. A palpable lymph node is often used for histologic confirmation of the diagnosis of sarcoidosis through biopsy. Further crucial skin lesions include erythema nodosum, lupus pernio, maculopapular rashes, cutaneous plaques, and subcutaneous nodules [26]. Neurosarcoidosis accounts for 2% to 7% of patients with sarcoid. The most frequent manifestation is Facial palsy. Other appearances include cranial nerve palsies, papilledema, peripheral neuropathy, meningitis, space-occupying cerebral lesions, cavernous sinus syndrome [27] and endocrine disorders such as hypopituitarism. Musculoskeletal involvement includes

bone cysts in patients with chronic sarcoid, polyarthralgia, and periarthritis in patients with acute sarcoid, and, less commonly, myopathy from granulomatous lesions within muscles [1,2,10,25,27,28].

The chest film is the best test to evaluate patients with suspected sarcoidosis. It can be often biopsied including the lungs, mediastinal lymph nodes, skin, peripheral lymph nodes, liver, and conjunctiva. Biopsy of clinically evident skin lesions or palpable lymph nodes is frequently performed because of the high yield and low morbidity [29,30]. The (ACE) serum angiotensin-converting enzyme level has been considered as a useful measurement in the diagnosis of disease. It is frequently abnormal in patients with active Sarcoid [31,32]. However, it is not only a true diagnosis of sarcoidosis as sometimes it appears to be limited due to possible active sarcoid uveitis but a normal chest film.

Studies by Hunter & Foster [33] reported that only 3% of patients with uveitis having sarcoid after the initial evaluation for a systemic disease revealed no diagnosable systemic disorder. Two distinct paradigms of sarcoidosis are prominent in the course of the disease, acute and chronic, depending on the onset, natural history, course, prognosis, and response to treatment. Acute sarcoidosis tends to have sudden, explosive onset in young patients and to go into impulsive remission within 2 years. Acute iritis is often seen in acute disease. The prognosis to respond with systemic corticosteroids is generally quite effective and shows the least long-term complications [1,2,25].

Ocular involvement manifestation of sarcoidosis intensifies specifically in keratoconjunctivitis sicca which is sought implicitly and included as evidence of lacrimal involvement in sarcoidosis [33]. The diseases described in sarcoidosis include anterior uveitis, iris and conjunctival nodules, scleral nodules, and corneal disease with either band keratopathy or interstitial keratitis [34]. Posterior-segment diseases are dominant like chorioretinitis, periphlebitis, Chorioretinal nodules, vitreous inflammation, and retinal neovascularization. Orbital disease is the disease of the lacrimal gland, nasolacrimal duct, optic nerve, and orbital granulomas. The most common ocular manifestation is anterior uveitis which is approximately two-thirds of patients with ocular sarcoid. It may be an acute iridocyclitis or a chronic granulomatous uveitis. Acute iridocyclitis is prominent mostly in patients with acute sarcoid. The prognosis is poor for those with chronic disease, as it may be more prone to lethal intractable complications such as secondary glaucoma, band keratopathy, cataracts, macular edema, and visual loss. Conjunctival and corneal lesions in patients with sarcoidosis are the least common. Orbital granuloma, independent of the lacrimal gland, occurs uncommonly [35,36]. Massive lacrimal gland enlargement mimicking a lacrimal gland tumor may occur and necessitate biopsy. Another hallmark of vitreous infiltration within sarcoidosis can appear as cellular infiltration, a nonspecific vitritis. Nevertheless, the lesions demonstrate clumping and an accumulation of vitreous fibroid, called either “snowballs” or a “string of pearls. Optic nerve association, particularly numerous granulomas of the optic nerve head occurs in 0.5% to 7.0% [7,10,25,31,37]. Chronic uveitis is associated with optic disc edema without granulomatous invasion of the optic nerve head or with papilledema [38]. Infrequently, sarcoid optic neuropathy (optic atrophy, optic neuritis, optic disc edema) can be seen in Neurosarcoidosis [39-42].

Advance complication neovascularization of optic disc peripheral neovascularization It is present in less than 5% of patients with ocular sarcoid but can cause significant visual loss due to vitreous hemorrhage. It is predominantly associated with a defined vaso-occlusive disorder such as a branch retinal vein occlusion. Neovascularization of the disc may also be due to branch or central vein occlusion. The development of secondary glaucoma in association with sarcoid uveitis appears to be a very poor prognostic sign and is associated with severe visual deterioration. It develops panuveitis with both anterior- and posterior-segment that have destructive secondary glaucoma. Treatment should be bound for immediately suppressing the inflammation and minimizing any potential ocular advance intractable complications.

Sarcoidosis in children is unfamiliar. Two clear divisions of children appear to be identified such as older children (onset age 5 years or over) almost invariably have lung involvement, with eye, liver, skin, and spleen also commonly affected. Very less patients typically develop uveitis, arthropathy, and skin rash. The disease course is familial rather than sporadic and is likely to demonstrate the same mutant gene loci CARD15. In the child with uveitis and, juvenile chronic arthritis is the prime evidence to diagnose the disease. Behcet’s disease is even less common and older children with sarcoidosis are more likely to have raised levels of ACE, but the test cannot be relied on in those less than 5 ages. Distended lymph nodes, affected skin, or conjunctiva are therefore major sites for consideration of biopsy. The prognosis and the outcome after management of the disease and its complications have not been well reported yet. Glaucoma and cataracts may occur and surgery for either will require special safeguards. The risk factors deleterious to the success of drainage surgery will be challenging for Glaucoma surgery for childhood uveitis.

Conclusion

Sarcoidosis is a disease of multisystem pathologies that affect various organs other than the eyes such as skin, lymph

nodes chest, and nervous system. It can be chronic or acute depending on the severity of the disease. The case studied the effect of steroidal therapy in the acute stage with minimal visual symptoms and improved visual prognosis. Moreover, a chronic stage can have a very poor prognosis so a patent diagnostic approach is required such as a biopsy of affected organs and laboratory tests can diagnose sarcoidosis. Anterior uveitis with solitary sarcoid granuloma responded very well with steroidal therapy. The extravagant clinical picture creates a crucial role for an eye physician to show a level of competency with medical practioner to plan disease management for the patient. Some ocular complications like cataracts and secondary Glaucoma with chronic sarcoid uveitis treatment are as straightforward as surgery. Hence a medical care provider, being aware of the characteristics of sarcoidosis will usually devise a good treatment and appropriate diagnosis.

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