Diffuse Unilateral Subacute Neuroretinitis—DUSN

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Diffuse unilateral subacute neuroretinitis (DUSN) is an ocular infectious disease caused by one of 2 different sized and as of yet unidentified nematodes capable of infiltrating the subretinal space.[1–4] It occurs mainly in children and young adults and the clinical course is characterized by periods of activity and remission. The intraocular inflammation tends to be diffuse and in the acute phase is accompanied by swelling of the optic disc, focal retinitis, and choroiditis. In the chronic phase, optic nerve atrophy occurs if the nematode is not destroyed.

History

The first case of this intraocular disease was described by Wilder[5] when she examined enucleated eyes of children diagnosed with retinoblastoma. Later, Nichols[6] identified these parasites as being *Toxocara* larvae. Parsons[7] provided the first report of an ocular syndrome associated to a subretinal mobile live worm and accompanied by a diffuse chorioretinitis in 1952. The parasite was located near the macular area, measured 1500 μm in length, and was considered an immature ascaris. Raymond and colleagues[8] observed the presence of a nematode in

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2 patients with unilateral retinal degeneration and proposed the inclusion of this ocular syndrome in the differential diagnosis of unilateral pigmentary retinosis.

Gass and Scelfo[9] described the natural history of ocular nematode infections as seen in otherwise healthy young patients from the southern portion of the United States and in the Caribbean. These authors described the condition as being characterized by significant unilateral visual loss, vitritis, optic nerve atrophy, narrowing of the retinal vessels, and peripheral pigmentary degeneration of the retina.[9] They adopted the term Wipe-Out syndrome and divided it into an early phase, characterized by decreased visual acuity, optic disc swelling, and multifocal retinitis, and a chronic or terminal phase. In 1978, Gass and colleagues[10] named the syndrome DUSN. Gass and colleagues,[10] studying 36 DUSN cases, confirmed the presence of a subretinal mobile worm in 2 patients. Gass and Braunstein[1] initially concluded that *Toxocara* was a cause of DUSN, but later ruled this out, based on negative serology in many of the patients.

In 1983, it was established that DUSN could be caused by 2 different sized nematodes, the smaller measuring 500 μm and the larger measuring between 1500 and 2000 μm. Gass[11] suspected that the smaller worm was *Ancylostoma caninun*, a dog parasite that causes cutaneous larva migrans in humans.[11] Kazacos and colleagues[12,13] identified the larger worm as being *Baylisascaris procyonis*, found in the intestine of raccoons and skunks.

McDonald and colleagues[14] described 2 DUSN cases caused by *Alaria mesocercaria* (Trematoda), a parasite found in frogs. These patients were likely infected in Asia by the ingestion of poorly cooked frog.

In Brazil, Oréfice and colleagues[15] described 2 *Toxocara canis* cases that were later diagnosed as DUSN. Other cases were subsequently documented with and without the presence of a worm.[16–18]

Souza and Nagashima[19] surgically extracted an intact subretinal worm. Morphologic characteristics suggested *Ancylostoma caninum*, but some parasitologists believed it was a third-stage *T. canis*. Although evidence suggests that most patients with DUSN will not develop the disease in the fellow eye, bilateral cases have been reported. Hence, a more appropriate term for this ocular condition might be *diffuse subacute neuroretinitis*.[7,20]

■ Etiologic Agent

Parasites of different sizes and several species of nematodes have been reported as etiologic agents of DUSN (Fig. 1), including *T. canis, A. caninum*, *Strongyloides stercoralis*, and *Ascaris lumbricoides*, the smaller nematoids, and *B. procyonis,* the larger one, but most of these reports do not present conclusive evidence about the specific agent.

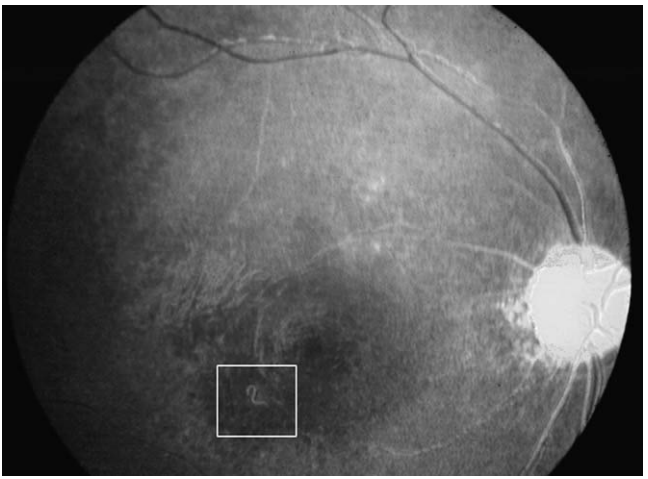

Figure 1. *Note the optic atrophy, narrowing of the retinal arteries, and widespread mottled depigmentation of the retinal pigmentary epithelium. The subretinal worm was located (detail).*

In the southeastern United States, the Caribbean, and Latin America the nematode varies in length from approximately 400 to 700 μm. In the other endemic area, such as the north and midwestern United States, it measures approximately 1500 to 2000 μm in length.[21] However, Moraes and associates[22] reported the first South American case of DUSN caused by the larger nematode and recently Vasumathy[23] described the first case of a large worm in India. In earlier reports, serologic testing was negative in most of the patients with viable intraretinal nematodes, which led Gass and Braunstein[1] to suggest that *Toxocara* was not the causative nematode in most patients with DUSN. They proposed that the nematode less than 1000 μm in length was the dog hookworm, *A. caninum*.

Retinal biopsy for DUSN via the transscleral approach has been performed by Blumenkrans and de Souza; however, precise identification of the nematode was not made.[19,24]

Because none of the nematodes described in DUSN patients have been recovered intact, identification must therefore be based on a combination of careful measurement of the parasite's dimensions, serologic testing, and epidemiologic studies, all of which have their limitations.[2]

T. canis: (1) There is a lack of serologic evidence; (2) the small size of the infective second stage larval form of *T. canis* makes it difficult to be visualized biomicroscopically; (3) the clinical picture is unlike that associated with what is considered to be more traditional ocular toxocariasis; and (4) the worldwide prevalence and distribution of *T. canis* is not in keeping with the geographic occurance of DUSN.[1]

A. caninum: The association of cutaneous larva migrans months, several years, or immediately preceding the onset of DUSN in some patients, suggests that *A. caninum* may be the small nematode that causes the syndrome.[21,25] *A. caninum* is a frequent cause of cutaneous larva migrans in the southeastern United States and in Brazil.[26] Garcia reported a prevalence of 35% cutaneous larva migrans in an epidemiologic study in schoolchildren. The infective third stage larva of *A. caninum* is approximately 650 µm in length and is capable of surviving in host tissue, including that of humans, many months and probably years without changing size or shape.[25]

Strongyloides stercoralis: Another nematode that penetrates the skin, provokes a lesion similar to that caused by larva migrans (larva currens). It is a universally distributed nematode most commonly found in tropical regions. The risk of infection is high for persons with frequent soil contact. The worm can penetrate multiple extraintestinal sites such as the brain, liver, and urinary tract. It can lie dormant for years.[27]

B. procyonis: Some controversy exists because most DUSN patients have no history of exposure to raccoons[4]; however, most patients with large nematode DUSN were from areas of the United States where raccoons are not only common, but commonly infected with *B. procyonis*.[28] Other cases involving a large worm have been published in other countries.[2,23,29] Future studies using techniques that identify nematode DNA could aid in etiologic diagnosis.

■ Diagnosis

DUSN is most frequently seen in healthy children or young adults with no significant past ocular history. The main clinical findings in the early and late stages are evanescent, multifocal, white-yellowish lesions at the level of the outer retina and choroids.[30]

Early Stage

Central or paracentral scotomas and visual acuity decrease are the principal complaints of symptomatic patients in the early stage.[9,31] Patients with acute visual loss during early stages of the disease usually present with mild to moderate vitritis, mild optic disc edema, and recurrent crops of evanescent, multifocal, white-yellowish lesions at the level of the outer retina and choroid. These lesions typically are clustered in only 1 segment of the fundus.[25] Less frequent symptoms and signs include ocular discomfort, congestion, iridocyclitis, perivenous exudation, subretinal hemorrhages, and serous exudation.[25,32] Garcia, in a series of 70 cases, found 4 patients in the early stage, all of whom presented with a live, mobile worm. All had unilateral disease and presented with vitreous cells, multifocal chorioretinal lesions, and

papilitis.[32] The intraocular worm was seen as a white, mobile, often glistening nematode that is gently tapered at both ends and varies in length from 400 to 2000 μm (Fig. 1). The worm can be seen during any stage of the disease, typically in the vicinity of active white-yellowish lesions when present. The active white-yellowish lesions (Fig. 2), which are probably caused by substances left in the wake of the nematode, disappear in 1 to 2 weeks as the nematode moves elsewhere in the eye, but often reappear at another retinal location.[25]

Late Stage

Most patients with clinical suspicion of DUSN are in the chronic phase. This is likely owing to late diagnosis or absence of specific treatment.[31] The presence of the worm in the subretinal space might affect the external retina by releasing toxic substances, followed by a diffuse tissue reaction. A diffuse degeneration of the retinal pigmentary epithelium occurs along with progressive loss of ganglionar cells with subsequent optic nerve atrophy and permanent low visual acuity.[30]

Visual acuity in late stages is profoundly decreased, with 80% or more showing vision 20/200 or worse.[31,33] Over a period of weeks or months, diffuse and focal depigmentation of the retinal pigmentary epithelium (RPE) occurs, usually most prominent in the peripapillary and peripheral retina.[21] Optic atrophy and severe retinal arteriole narrowing seems to define the late stage best. Retinal arteriole narrowing may vary by quadrant, and in conjunction with optic nerve atrophy, usually accompanies the progressive changes in the RPE[33] (Fig. 3). Other important signs for late-stage DUSN diagnosis are: an increased

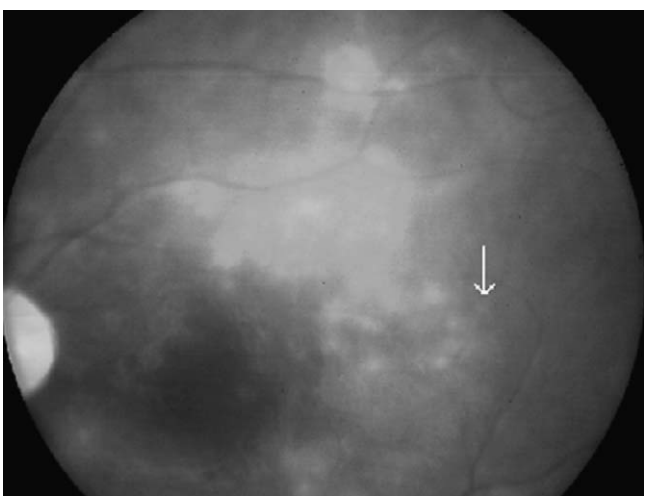

Figure 2. *Note the active white-yellowish evanescent lesions in the early stage.*

retinal inner limiting membrane reflex (Oréfice's sign), evidence of white-yellowish subretinal tunnels suggestive of remnants of larva migration in the subretinal space, and the presence of small white spots (Garcia's signs).[30]

■ Clinical Examination

Serologic Test

Serologic testing, stool examinations, and peripheral blood smears are of little value in diagnosing DUSN,[10] and no serologic test is currently available for *Ancylostoma*.[25] When a worm is identified within the eye of an otherwise healthy person, unless a peripheral eosinophilia is present, no further evaluation seems warranted to make the diagnosis. Oréfice et al[34] studied 23 DUSN cases and found 47.62% enzyme-linked immunosorbent assay (ELISA) positive, and in 2 of these with live worm, the ELISA was negative. Souza[35] reported that ELISA anti-*T. canis* was negative in most of the 39 DUSN cases he studied.

Fluorescein Angiography

In the early stage, there is hypofluorescence of the focal white-yellowish lesions of active retinitis followed by staining. Dye leakage is seen from the optic disc capillaries. Occasionally, there is evidence of prominent perivenous dye leakage (Fig. 4). In more advanced stages of the disease, angiography shows greater evidence of RPE pigment loss,

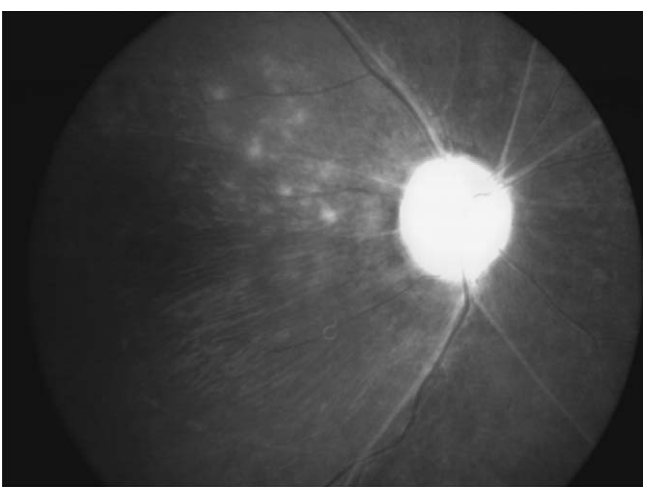

Figure 3. *Fundus findings in the late stages of diffuse unilateral subacute neuroretinitis. Note the optic atrophy and arteriolar attenuation.*

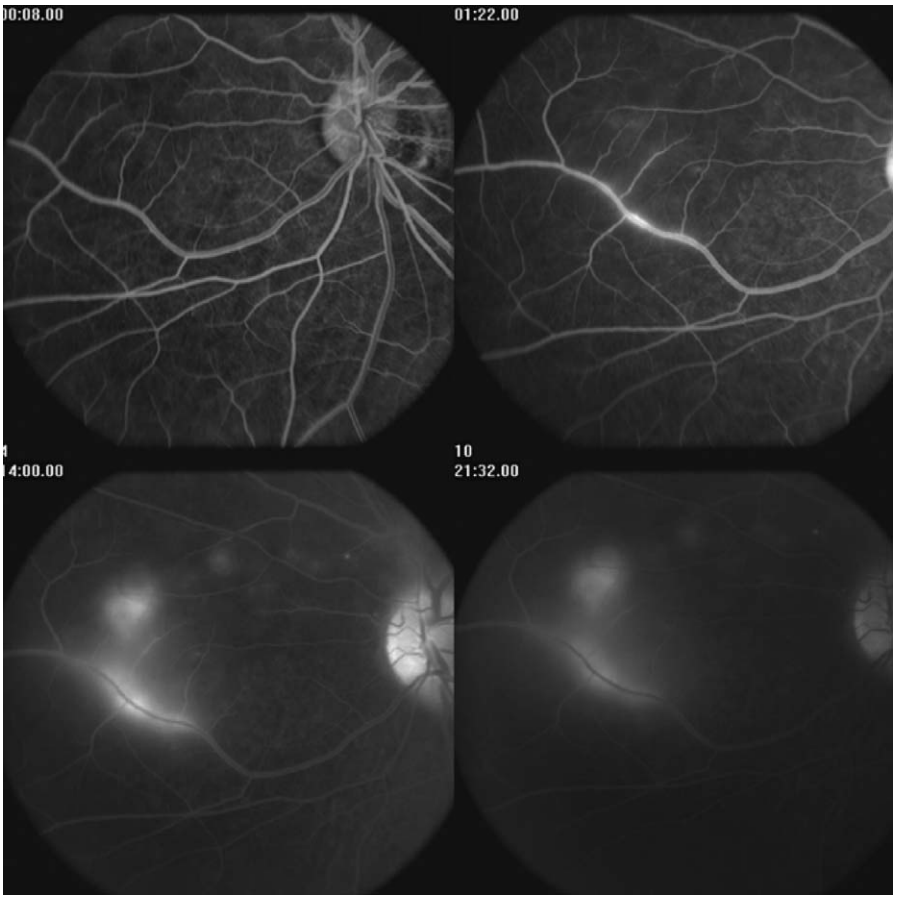

Figure 4. *Note in fluorescein angiography: there is evidence of perivenous dye leakage.*

manifested as an irregular increase in background choroidal fluorescence.[25]

Indocyanine Green Angiography

Features suggest that the choroid is also involved in early stage DUSN. Choroidal infiltration, which prevents normal choroidal indocyanine green impregnation, is most likely the physiopathogenic explanation for the hypofluorescent dark spots seen in the affected eye. The dark spots present in the initial indocyanine green angiography (ICG-A) phase seem to either disappear or persist in the late phase of the examination. Hypofluorescent dots persisting in the late phase are interpreted as full-thickness lesions allowing no ICG diffusion, whereas dots becoming isofluorescent in the late phase are interpreted as partial-thickness lesions progressively surrounded by ICG fluorescence.[36]

Electroretinogram

DUSN is characterized by a retinal diffuse inflammation that causes significant electrophysiologic alterations. Gass and colleagues[10] showed electroretinogram (ERG) damage in 36 DUSN patients in both the cone and rods systems. B wave was more affected than A wave, showing a negative ERG.[10] Oréfice and colleagues[30] described electroretinographic alterations in 6 patients and negative ERG in 5.

DUSN is not the only type of uveitis in which an abnormal ERG can be found; Birdshot chorioretinopathy and multiple evanescent white dot syndrome, among other conditions, can also have an abnormal ERG. Therefore, it is essential that the ERG findings be interpreted in the context of the overall clinical picture.[30]

The ERG in the affected eye is usually abnormal even if tested early.[21] Half of the patients generally have normal electro-oculogram and the finding of normal electro-oculogram and abnormal ERG suggests a neuroepithelium disease.[37] It is important to point out that the ERG is rarely extinguished completely, which differentiates it from some inherent retinal degeneration.[30,38]

Visual Field Studies

The visual field studies show different lesion patterns that generally do not correspond to ocular fundus changes.[25] Goldman perimetry can be useful for evaluating the visual field before and after treatment.[38]

Automated visual field can be useful for following DUSN patients with visual acuity of 20/100 or better (Garcia, personal communication).

Nerve Fiber Layer Thickness Study

There is no ancillary test to follow DUSN patients, with or without live worm, who undergo clinical treatment or photocoagulation. Garcia and colleagues[39] observed that patients with late stage DUSN had decreased nerve fiber layer thickness, as shown by GDx (Laser Diagnostic Technologies Inc). Retinal zones with a larger amount of nerve fibers had a greater decrease in the delay of the deflected light measured by the nerve fiber analyzer. Gomes and Garcia (personal communication), in a 38-patient preliminary study using optic coherence tomography (OCT stratus, Zeiss, Dublin), found a direct relation between decreased nerve fiber layer thickness in the chronic phase and decreased visual acuity.

■ Differential Diagnosis

Early signs of DUSN are often mistaken for multifocal choroiditis, acute posterior multifocal placoid pigment epitheliopathy, multiple

evanescent white dot syndrome, or nonspecific optic neuritis and papillitis. The late stage of DUSN is often mistaken for posttraumatic chorioretinopathy, occlusive vascular disease, sarcoidosis, or toxic retinopathy.[22,38,40]

■ Management

Treatment options are limited in case of DUSN.

Laser Treatment

According to Gass, laser treatment of the nematode at any disease stage can be highly effective when the worm is visualized, and may improve visual acuity and inflammatory ocular signs[17,37]; if the worm is localized in the macula, it can be induced to migrate toward the periphery by directing a bright light at the worm. Photocoagulation, the treatment of choice for DUSN, is considered an effective means of destroying the worm,[12,17,41] the search for which can nevertheless be a frustrating task.[42] It seems to offer the best chance for halting worm mobility and for resolution of the active white-yellowish lesions without causing significant intraocular inflammation or toxic damage to the eyes.[32] In a series of 70 patients diagnosed with DUSN, Garcia and colleagues[31,32] found a live worm in 4 patients in the early stage and in 22 in the late stage. After photocoagulation treatment all the patients had improved visual acuity in the early stage but photocoagulation of the worm does not improve visual acuity in the late stage.[31,32] One of them had decreased visual acuity, 2 improved and 19 remained unchanged. Apart from the round hyperpigmented retinal scar located in the photocoagulated area, ophthalmoscopy of these eyes showed no difference from the previous picture seen before laser treatment. Nevertheless, photocoagulation of the worm impedes the progression of RPE and optic nerve atrophy. In fact, diagnosis is relatively easy; however, finding a live worm in the retina is laborious and time consuming, often requiring many visits. But, we believe that every patient with this disease must be carefully evaluated to locate and destroy the worm by laser, as this approach may avoid further visual loss.[20] The larva in the subretinal space seems to release toxic substances before and after laser treatment. Garcia (personal communication) performed photocoagulation of a live worm located near the macula in a patient with 20/30 visual acuity. One month after the worm was destroyed the patient had a visual acuity of finger counting at 1 m and temporal optic nerve atrophy.

Oral Treatment

The search for an oral treatment for DUSN started in 1980 with Gass and colleagues. After several case series studies with oral antihelmintic

drugs such as thiabendazole and diethylcarbamazine, these authors and others[18,42,43] found that the tested drugs would only destroy the subretinal worms in some of their patients.[42]

Souza and colleagues[42] described 12 Brazilian patients who improved visual acuity, visual field, and active ocular inflammatory signs after treatment exclusively with high-dose oral albendazole (400 mg/d) for 30 days. In addition, during the first weeks of treatment, they found worm inactivation in 4 patients in which the worms were visible. No adverse drug side effects were observed in any of their cases during follow-up.

Cortez treated 6 patients with an identifiable, mobile nematode, using systemic albendazole rather than photocoagulation, and immobilized the nematode in only 3 cases. In all 3 cases, the focal retinitis decreased and the nematode was slowly absorbed. No new inflammation or pigment derangement was noted. Immobilization occurred after approximately 7 days of treatment. In the remaining 3 patients, the nematode was subsequently photocoagulated. No adverse effects from albendazole treatment were noted in any patient.[20]

As yet there is no clinical examination for following patients treated with antihelmintic drugs. Gold standard DUSN treatment is based on the finding of a mobile, live worm after repeated, lengthy, and exhaustive examinations aimed at its destruction through photocoagulation. Clinical treatment must be limited to cases in which no worm is found despite repeated examinations. Further multicenter studies are needed to verify the effectiveness of clinical treatment when a live worm is not found.

■ Conclusions

DUSN represents a spectrum of ocular nematode infections that can be found in many parts of the world. In Brazil, DUSN is increasingly considered an important cause of posterior uveitis in children and young healthy adults. Accurate diagnosis of DUSN is important because destruction of the worm in the early stages of the disorder can halt the progression of visual loss. It is important, therefore, to consider DUSN in patients with suggestive symptoms and signs, not only in endemic areas, but also in regions not yet identified as being endemic.[44]

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