
Diffuse Unilateral Subacute Neuroretinitis (DUSN)

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Abstract

Diffuse unilateral subacute neuroretinitis (DUSN) is a usually unilateral inflammatory disease characterized by an insidious, usually severe, loss of peripheral and central vision. Clinical characteristics are manifested in early and late stages. Parasites of different sizes and several species of nematodes have been reported as the etiology of DUSN without conclusive evidence about the specific agent. Because serologic testing has been variable, the definitive diagnosis is made when the clinical characteristics of DUSN are found in conjunction with an intraocular worm. Laser photocoagulation, pars plana vitrectomy, thiabendazole, and albendazole have been used to treat DUSN with variable success.

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Keywords

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Introduction

Diffuse unilateral subacute neuroretinitis (DUSN) was described by Gass in 1977 [1], who called it “unilateral wipe-out syndrome.” The term diffuse unilateral subacute neuroretinitis was first used by Gass in 1978 [2]. He described 29 patients seen with consistent features that included insidious, usually severe, loss of peripheral and central vision with associated findings of vitreous inflammation, diffuse and focal epithelial derangement with relative sparing of the macula, narrowing of the retinal vessels, optic atrophy, increased retinal circulation time, and subnormal electroretinographic findings (Fig. 2.1). However, the cause of the inflammation in DUSN was still unknown. In May 1978, Gass et al. reiterated his definition because the progressive unilateral visual loss was believed to be secondary to inflammation of the retina, retinal vessels, retinal pigment epithelium (RPE), and optic nerve head [3]. Later in 1983, Gass and Braunstein observed a nematode in two patients with DUSN [4]. On further searching of the literature, Gass was able to identify previously reported cases of similar nematodes that produced the same clinical picture appearing as early as 1952 [5]. Hence, a syndrome of initially unknown cause that was classified only by clinical description was later found to be related to a nematode in the subretinal space [6]. Although evidence suggests that most patients with DUSN will not develop it in the fellow eye, bilateral cases have been reported; therefore, a more appropriate term for this ocular condition might be *diffuse subacute neuroretinitis* [7]. Cortez et al. described the clinical features and management in the largest reported series to date of patients with DUSN [8]. The charts of all patients coded as having DUSN in a vitreoretinal clinic in Caracas, Venezuela, between July 1979 and August 2000 were retrospectively reviewed. They identified 82

eyes of 78 patients with DUSN. The mean age at diagnosis was 16.7 years. Thirty-three (42.3%) of the patients were female. The presenting visual acuity was 20/400 or worse in 69 eyes (84.1%). The subretinal nematode was identified in 33 eyes (40.2%), and all nematodes were small, approximately 400 μ (m) in length [8].

Etiologic Agent

Parasites of different sizes and several species of nematodes have been reported as the etiologic agent of DUSN, including *Toxocara canis*, *Baylisascaris procyonis*, and *Ancylostoma caninum*, and most of these reports do not present conclusive evidence about the specific agent. In the southeastern United States, the Caribbean islands, and South America, the nematode varies in length from approximately 400–700 μ m. In the other endemic area, the north Midwestern United States, it measures approximately 1,500–2,000 μ m in length [9]. However, Cialdini et al. reported the first South American case of DUSN caused by the larger nematode [10]. In earlier reports, serologic testing was negative in most of the patients with viable intraretinal nematodes, which led Gass and Braunstein to suggest that *Toxocara* was not the causative nematode in most patients with DUSN [4]. They suggested that the nematode less than 1,000 μ m in length was the dog hookworm, *Ancylostoma caninum*, and Kazacos et al. suggested that the larger nematode was the raccoon ascarid, *Baylisascaris procyonis* [11].

Retinal biopsy for DUSN via transcleral approach has been performed by Blumankranz and Culbertson [12]. However, precise identification of the nematode was not made [13]. Gass transclerally extracted one nematode from beneath the retina after killing it with cryotherapy; histologic details were poor, and he was

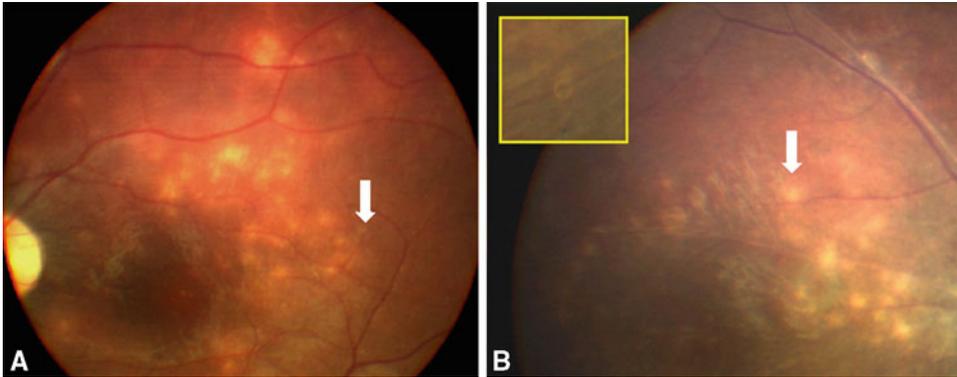


Fig. 2.1 (a and b) Patients during early stages usually present mild to moderate vitreitis, mild optic disk edema, and recurrent crops of evanescent, multifocal, gray-white lesions at the level of the outer retina (arrows). These lesions

typically are clustered in only one segment of the fundus. The intraocular worm is seen as a motile, white, often glistening nematode that is gently tapered at both ends and varies in length from 400 to 2,000 μm (inset in b)

unable to identify the nematode [14]. Via trans-vitreous approach, de Souza et al. recovered the nematode intact and motile [13]. Several parasitologists in São Paulo, Brazil, examined the nematode; the measurement of body size and the morphologic features were more consistent with a third-stage *Toxocara* larva, but because of poor fixation, definitive identification of the worm was not possible. However, Bowman recently reviewed the pictures of the worm removed by de Souza and concluded that it is most likely *Ancylostoma caninum* [9]. Because none of the nematodes described from patients with DUSN have been recovered intact, identification must, therefore, be based on a combination of careful measurement of the parasite, serologic testing, and epidemiological studies, all of which have their limitations [15].

Toxocara canis

Gass et al. initially concluded that *Toxocara* was a cause of DUSN [3] but, however, discarded this possibility based on negative serology in many of the reported patients [4]. In addition, Gass and Olsen later suggested that *T. canis* was not the cause based on the following: (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 ocular toxocariasis, and (4) the worldwide prevalence of *T. canis* is not in keeping with the endemic distribution of DUSN [16]. However, Goldberg et al. reported that low or nondiagnostic serum titers are well described in cases of *Toxocara* ocular larva migrans and suggested a similarity with the overall reduced sensitivity of serodiagnostic tests for DUSN [15, 17, 18]. Oppenheim et al. reported a case of *Toxocara* DUSN in which the patient's positive ELISA titer decreased fourfold over a 2-year period [19]. Therefore, the lack of serologic confirmation of toxocaral infection in some patients may be a reflection of the timing of the serology in relation to the onset of the disease or the immune status of the patient.

Ancylostoma caninum

The association of cutaneous larva migrans months, several years, or immediately preceding the onset of DUSN in some patients suggests that *Ancylostoma caninum* may be the small nematode that causes the syndrome [9, 16]. *A. caninum* is a frequent cause of cutaneous larva

migrans in the southeastern United States. In addition, 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 [16].

Baylisascaris procyonis

In 1984, it was suggested by Kazacos that the larger worm in patients with DUSN living in more northern climates was *Baylisascaris procyonis*, a nematode found in raccoons [20]. He proposed that *B. procyonis* larvae produce ocular larva migrans with a clinical picture that is similar to that of early DUSN in subhuman primates and other experimental animals after oral infection [11]. Additionally, the *B. procyonis* larvae may grow while they are within the eye and would account for the range of lengths of larvae seen, such as those that are 400–2,000 µm. The large nematode variant of DUSN matches the size range of *Baylisascaris*. Nevertheless, some controversy exists because most patients with DUSN have no history of exposure to raccoons [7]; 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* [21]. Significant morphometric, serologic, and epidemiologic support for *Baylisascaris* as the causative agent of DUSN was published by Goldberg [15]. A large worm of 1,500 µm length presenting in a German patient was thought to be consistent with *Baylisascaris* species [22]. In humans, the organism is capable of causing visceral larva migrans, eosinophilic meningoencephalitis, and ocular larva migrans. In addition, Mets et al. have reported two patients with eye manifestations of DUSN, both with severe neurologic degeneration and indirect immunofluorescence assays on serum and cerebrospinal fluid positive for *B. procyonis* in one and serially positive and increasing in the second [23]. In addition, Goldberg et al. suggest that ocular larva migrans and DUSN can occur without evidence of visceral larva migrans or central nervous system dysfunction [15].

Trematodes

McDonald et al. encountered two cases of human intraocular infection with mesocercariae of *Alaria* (Trematoda) in the eyes of two unrelated Asian men with signs of DUSN in which the probable source of infection was ingestion of undercooked frogs' legs containing the trematode [24]. The worm in their case 1 was analyzed from projected fundus photographs and diagnosed as an *Alaria* mesocercaria on the basis of its shape, size (500×150 µm), and movement. The worm in their case 2 was removed surgically from the vitreous and identified as *Alaria* mesocercariae, 555×190 µm in size, most likely *A. americana*. They concluded that *Alaria* mesocercariae could be a cause of DUSN.

Mode of Transmission

Baylisascaris procyonis, a parasitic infection of raccoons in the United States, causes severe neurologic and ocular disease in humans when infectious eggs from raccoon feces are ingested. However, *Ancylostoma caninum*, a parasitic infection of dogs (or sometimes a fox infection) in South America, causes cutaneous larva migrans in humans when infectious eggs from dog feces are ingested or from larvae entering through the skin (usually the foot) migrate through the bloodstream to the lungs and trachea, and are coughed up and swallowed. They attach themselves to the intestinal wall and thus complete the life cycle.

Diagnosis and Pathogenesis

Because serologic testing has been variable, the diagnosis is made when the clinical characteristics of DUSN are found in conjunction with an intraocular worm (Table 2.1). Clinical characteristics are manifested in early and late stages. DUSN most frequently is seen in healthy children or young adults with no significant past ocular history.

Table 2.1 Diffuse unilateral subacute neuroretinitis (DUSN) diagnosis

DUSN diagnosis	
Test	Findings
Ocular fundus signs	<i>Early stage:</i> mild to moderate vitreitis, mild optic disk edema, and recurrent crops of evanescent, multifocal, gray-white lesions at the level of the outer retina typically clustered in only one segment of the fundus. Others: iridocyclitis, perivenous exudation, subretinal hemorrhages, serous exudation, and subretinal neovascularization <i>Late stage:</i> progressive optic atrophy, mild or moderate vitreitis, multifocal choroiditis episodes, increase in the internal limiting membrane reflex (Oréface's sign), presence of small white spots suggestive of calcifications, tunnels in the subretinal space (Garcia's sign), narrowing of the retinal arteries, and marked focal and diffuse degenerative changes in the RPE and retina <i>Early or late disease:</i> in 25–40%, the worm is visualized
Serologic test	Unless a peripheral eosinophilia is present, no further evaluation seems warranted to make the diagnosis
FA	<i>Early stage:</i> hypofluorescence of the focal gray-white lesions followed by staining. Leakage from the capillaries on the optic disk. Perivenous leakage of dye <i>Advanced stages:</i> irregular increase in the background choroidal fluorescence
ICG-A	Dark spots present in the initial ICG-A phase that seem to either disappear or persist in the late phase of the examination
ERG	b-wave of maximum combined response is flat, with below-normal response and a decrease in relation to b/a
EOG	One-half of patients can have a normal electrooculogram
Multifocal-ERG	Variable changes as decreased foveal response density and increased parafoveal and perifoveal waveform amplitudes
Visual field test	Different lesion patterns that cannot be explained with the findings of the ocular fundus changes
SLO	High-contrast image facilitating visualization of the nematode
OCT	Decreased RNFL thickness
GDX®	<i>Early disease:</i> increase in thickness due to transitory edema <i>Chronic phase:</i> decrease in RNFL thickness

FA fluorescein angiography, ICG-A indocyanine green angiography, ERG electroretinogram, EOG electrooculogram, Multifocal-ERG multifocal electroretinogram, SLO scanning laser ophthalmoscopy, OCT optical coherence tomography, GDX® nerve fiber analyzer, RPE retinal pigment epithelium, RNFL retinal nerve fiber layer

Early Stage

Central or paracentral scotoma is the principal complaint of symptomatic patients in the early stage [2]. Visual loss is rarely reversible and usually less than 20/200 in about one-half of patients [4]. Patients with acute visual loss during early stages of the disease usually present mild to moderate vitreitis, mild optic disk edema, and recurrent crops of evanescent, multifocal, gray-white lesions at the level of the outer retina. These lesions typically are clustered in only one segment of the fundus (Fig. 2.1a) [16]. Less frequently, symptoms and signs include ocular discomfort, congestion, iridocyclitis, perivenous exudation, subretinal hemorrhages, serous exudation, and

evidence of subretinal neovascularization [16]. In approximately 25–40% of cases, a worm is visualized during eye examination [8, 25]. The intraocular worm is seen as a motile, white often glistening nematode that is gently tapered at both ends and varies in length from 400 to 2,000 μm (Fig. 2.1b). It can be seen during any stage of the disease, and if active gray-white lesions are present, the nematode usually will be found in their vicinity. The examining light may cause the worm to move by a series of slow coiling and uncoiling movements and less often by slithering snakelike movements in the subretinal space [9]. Gass and Braunstein reported that there is a greater likelihood of the longer worm leaving a tract of coarse clumping of RPE in the wake of its travels [4].

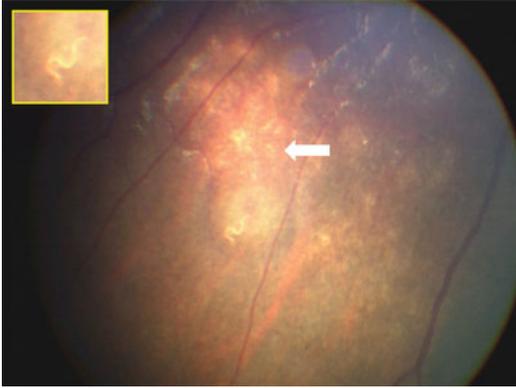


Fig. 2.2 There is a greater likelihood of the longer worm leaving a tract of coarse clumping of RPE in the wake of its travels. The shorter worm (*inset*) tends to leave focal, chorioretinal atrophic scars (*arrow*)

The shorter worm tends to leave focal, chorioretinal atrophic scars (Fig. 2.2). The focal pigment epithelial changes seen are easily explained by the location or the travel pattern of the worm. It is speculated that focal chorioretinal white spots are an immune response to a secretion or excretion from the worm [3]. The diffuse pigment epithelial changes are somewhat more difficult to explain except as a toxic reaction [26]. The active gray-white evanescent lesions, which probably are caused by substances left by the nematode in its wake, disappear in 1–2 weeks as the nematode moves elsewhere in the eye [16].

Late Stage

The clinical picture of late-stage disease usually demonstrates progressive optic atrophy with the subsequent afferent pupillary defect, mild or moderate vitreitis, multifocal choroiditis episodes, increase in the internal limiting membrane reflex (Oréface's sign), presence of small white spots suggestive of calcifications, evidence of tunnels in the subretinal space (Garcia's sign), retinal narrowing of the retinal arteries, marked focal as well as diffuse degenerative changes in the RPE and retina, and severe permanent loss of vision (Fig. 2.3) [16, 27]. Visual acuity in late stages is profoundly decreased, with 80% or more showing vision 20/200 or worse [26]. Over a

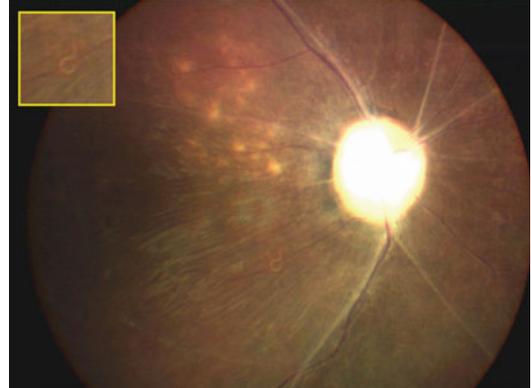


Fig. 2.3 The clinical picture of late-stage disease usually shows progressive optic atrophy, narrowing of the retinal arteries, marked focal as well as diffuse degenerative changes in the pigment epithelium and retina, and severe permanent loss of vision. The intraocular worm is shown in the *inset*

period of weeks or months, diffuse as well as focal depigmentation of the RPE occurs, usually most prominent in the peripapillary and peripheral retina, and less prominent in the central macular area [9]. Optic atrophy and severe retinal arteriole narrowing seem to define the late stage best. Retinal arteriole narrowing may vary by quadrant and, in conjunction with optic atrophy, usually are accompanying the progressive changes in the RPE. Choroidal neovascularization can occur usually in the periphery [26]. Although information about the pathogenesis of the disease is speculative, toxic products released by the larva in the subretinal space would locally affect the external portion of the retina and a diffuse tissue reaction would lead to external and internal retinal damage. Over the years, vascular narrowing and progressive ganglionar cell loss would occur until optic atrophy resulted [28].

Ancillary Tests

Serologic Test

Serologic testing, stool examinations, and peripheral blood smears are of little value in making the diagnosis of DUSN [3], and no serologic test currently is available for *Ancylostoma* [16]. When a

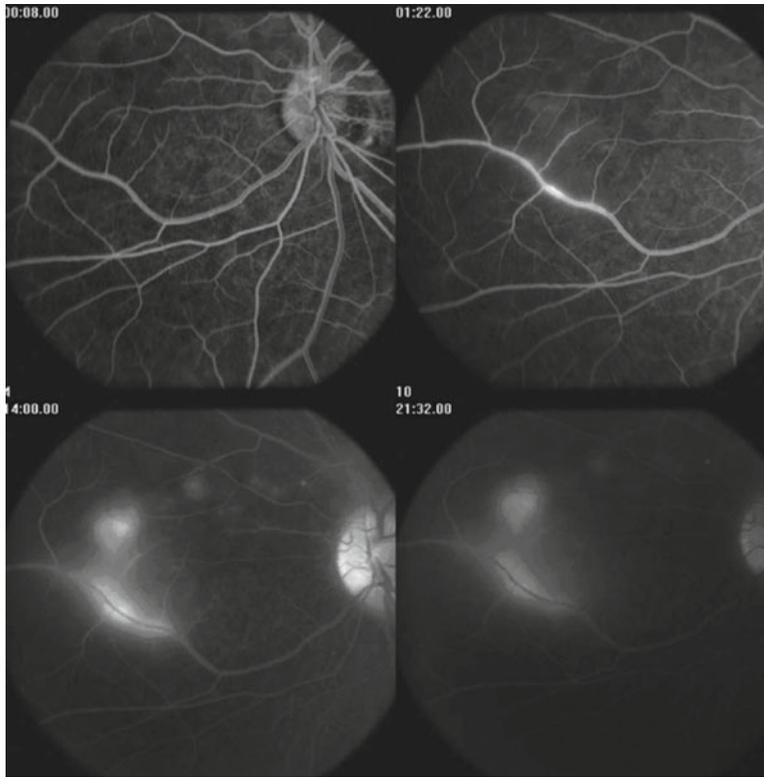


Fig. 2.4 Serial fluorescein angiogram performed on a patient with early-stage DUSN showing areas of both vascular and retinochoroidal leakage and staining

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.

Fluorescein Angiography

In the early stage, there is hypofluorescence of the focal gray-white lesions of active retinitis followed by staining. Leakage of dye is seen from the capillaries on the optic disk. Occasionally, there is evidence of prominent perivenous leakage of dye (Fig. 2.4). In more advanced stages of the disease, angiography shows greater evidence of loss of pigment from the RPE manifested angiographically as an irregular increase in the background choroidal fluorescence (Fig. 2.5) [16].

Indocyanine Green Angiography (ICG-A)

Indocyanine green angiography (ICG-A) features suggest that the choroid is also involved in early-stage DUSN. Choroidal infiltration, which prevented normal choroidal indocyanine green impregnation, most probably is the physiopathogenic explanation for the hypofluorescent dark spots seen in the affected eye. The dark spots present in the initial 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 (Figs. 2.6 and 2.7) [29].

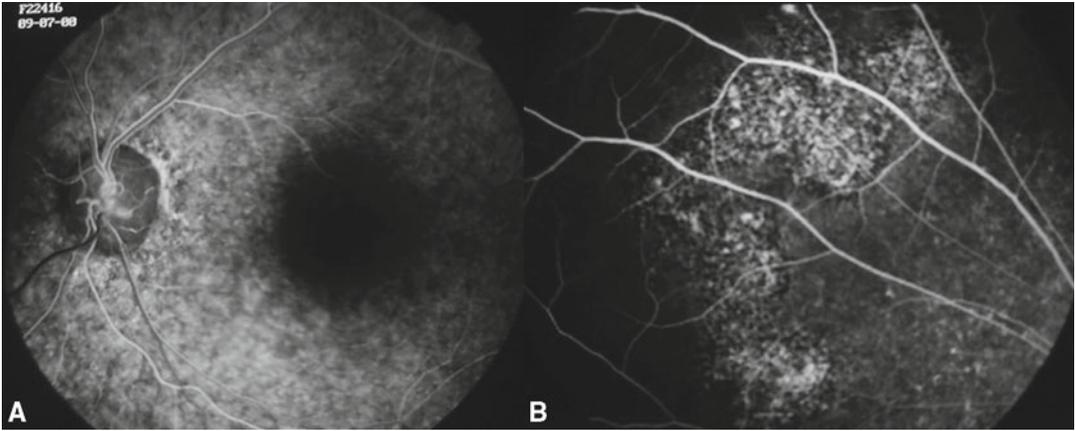


Fig. 2.5 (a) In the early stage, there is hypofluorescence of the focal gray-white lesions of active retinitis followed by staining. (b) In more advanced stages of the disease, angiography shows greater evidence of loss of pigment

from the RPE manifested angiographically as an irregular increase in the background choroidal fluorescence (Courtesy of Dario Fuenmayor-Rivera, M.D.)

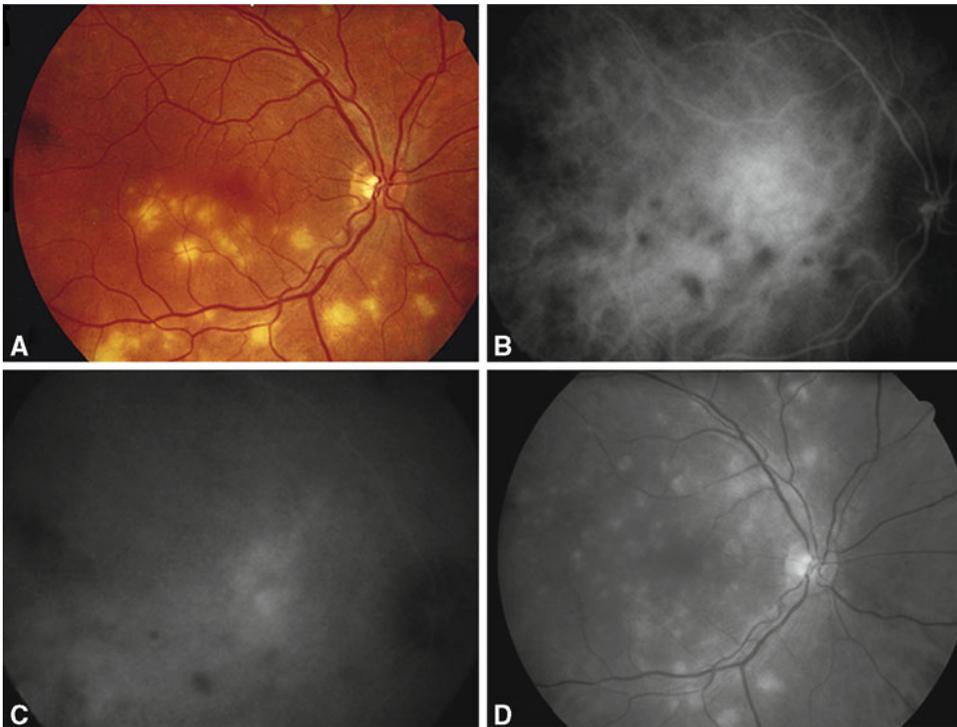


Fig. 2.6 Early-stage DUSN. (a) The affected eye revealed multiple yellow-white subretinal lesions at the posterior pole. (b) Early-phase ICG-A shows hypofluorescence of the lesions. (c) Late-phase ICG-A reveals few hypofluorescent dots and a fuzzy hyperfluorescence in the macular region. (d) After

1 month, the superior subretinal lesions increased in number and became more evident (Reprinted with permission from Vianna RN, Onofre G, Ecard V, Muralha L, Muralha A, de A Garcia CA. Indocyanine green angiography in diffuse unilateral subacute neuroretinitis. *Eye*. 2006;20:1113–1116)

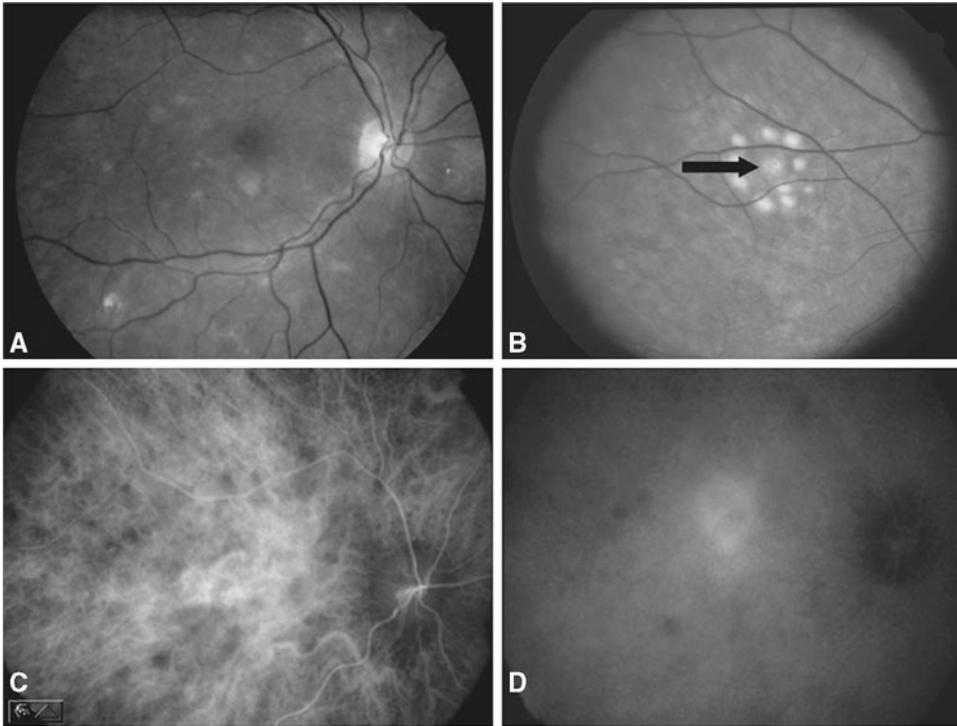


Fig. 2.7 Late-stage DUSN. (a) Observe many round hypopigmented lesions throughout the posterior pole as well as mild optic disk atrophy, discrete narrowing of the retinal vessels, and diffuse RPE degeneration. (b) The located worm surrounded by laser spots. (c and d) Early- (c) and late-phase ICG-V (d) revealed

hypofluorescent spots and an area of hyperfluorescence in the macular region (Reprinted with permission from Vianna RN, Onofre G, Ecard V, Muralha L, Muralha A, de A Garcia CA. Indocyanine green angiography in diffuse unilateral subacute neuroretinitis. *Eye*. 2006;20:1113–1116)

Electroretinogram (ERG), Electrooculogram (EOG), and Multifocal Electroretinogram

Electroretinographic changes include a mild to moderate decrease in rod and cone function, with the b-wave being more affected than the a-wave. DUSN presents a very characteristic and reproducible electroretinographic picture also found in ischemic retinal cases: negative electroretinogram (b-wave of maximum combined response is flat, with below-normal response and a decrease in relation to b/a). The mechanism of this interesting phenomenon is explained by Oréface et al. as being a consequence of a possible autoimmune, inflammatory, and/or toxic aggression toward retinal bipolar cells [27, 28].

The ERG in the affected eye is usually abnormal even if tested early in the course of the disease [8]. The more common one-half of patients can have a normal electrooculogram (EOG), and the finding of normal EOG and abnormal ERG suggests a neuroepithelium disease [25]. It is important that the ERG is rarely extinguished completely, which differentiates it from some tapetoretinal degeneration [30]. According to Martidis et al., multifocal electroretinography findings before laser treatment showed decreased foveal response density and increased parafoveal and perifoveal waveform amplitudes. Two months after laser photocoagulation of a subretinal nematode, multifocal electroretinography showed full recovery of normal findings and visual acuity remained 20/20 [31].

white dot syndrome, nonspecific optic neuritis, and papillitis. The late stage of DUSN is often mistaken for posttraumatic chorioretinopathy, occlusive vascular disease, or sarcoid or toxic retinopathy [16].

Management

Laser Treatment

At present, treatment of a visible worm with photocoagulation seems to offer the best chance for halting worm motility and resolution of the active gray-white lesions without causing significant intraocular inflammation or toxic damage to the eye. Some improvement in vision and visual field may occur after laser treatment of the worm [33]; however, in late stages of the disease, laser treatment does not improve the visual acuity of affected patients [34]. Previous studies have demonstrated the photosensitivity of different species of ocular infecting parasites, and this may be utilized in luring the target organism away from the macula. In some patients with the worm very close to the center of the fovea in which heavy photocoagulation may damage the remaining central vision, it may be possible to use low level of illumination or very light applications of the laser to chase the worm into the midperiphery, where it may be destroyed with less retinal damage [35].

Oral Treatment

Usually thiabendazole and corticosteroids have not been successful for the treatment of DUSN, except in patients with vitreous inflammation. Gass et al. reported that thiabendazole could be effective in some patients when the worm cannot be found and when DUSN is accompanied by moderate degrees of vitreous inflammation that is associated with a breakdown in the blood-retinal barrier [16]. Similarly, in this group of patients without visible worm and the typical migration of the evanescent lesions, Gass proposed the use of moderately intense scatter photocoagulation in the vicinity of the white lesions to break down the

blood-retinal barrier before the administration of thiabendazole. Observation of new white retinal lesions 4–7 days after medical treatment may indicate death of the nematode. Souza et al. reported 12 Brazilian patients who improved visual acuity, visual field, and active ocular inflammatory signs after treatment exclusively with high-dose oral albendazole (400 mg/day) for 30 days [36]. In addition, during the first weeks of treatment, they observed worm inactivation in four patients in which the worms were visible. No adverse drug side effects were observed in any of their cases during follow-up.

Pars Plana Vitrectomy (PPV)

Pars plana vitrectomy is not the standard of treatment for DUSN when the nematode is found because it can be eradicated in cooperative patients with laser. However, as previously stated, de Souza et al. recovered the nematode intact with a PPV approach and in an uncooperative young patient to standard laser treatment [13]. In addition, Meyer-Riemann et al. demonstrated that when a nematode larva is near the posterior pole, surgical extraction of the worm using vitrectomy techniques may be favorable compared to photocoagulation [37].

Controversies and Perspectives

Diffuse unilateral subacute neuroretinitis is a usually unilateral inflammatory disease characterized by an insidious, usually severe, loss of peripheral and central vision with associated findings of vitreous inflammation, diffuse and focal epithelial derangement with relative sparing of the macula, narrowing of the retinal vessels, optic atrophy, increased retinal circulation time, and subnormal electroretinographic findings. Parasites of different sizes and several species of nematodes have been reported as the etiologic agent of DUSN, including *Toxocara canis*, *Baylisascaris procyonis*, and *Ancylostoma caninum*, and most of these reports do not present conclusive evidence about the specific agent.

Clinical characteristics are manifested in early and late stages, but pathogenesis of the disease is speculative including autoimmune, inflammatory, and/or toxic mechanism of aggression as a possible cause of retinal damage. Laser photocoagulation offers the best chance for clinical resolution of the disease; however, in only 25–40% of cases, the worm is visualized during eye examination. In those patients who cannot receive laser, other treatments including pars plana vitrectomy, thiabendazole, and albendazole have been used with variable success. Probably, nowadays, the best protocol option for oral treatment is albendazole; however, the optimal dosing and duration of treatment for DUSN has still not been determined, and the suggestion to use 400 mg for 30 consecutive days is on the basis of the good results observed applying this protocol to patients with neurocysticercosis [36].

Focal Points

1. In order to avoid diagnostic mistakes, it is important to notice that patients may not manifest evidence of systemic disease and stool shedding. Eosinophilia is infrequently detected, and by the time the worm reaches the subretinal space, systemic markers may not be informative as there is likely to be a time lapse between systemic infestation and intraocular involvement, so the definitive diagnosis is made when the clinical characteristics of DUSN are found in conjunction with an intraocular worm.
2. Whenever the nematode is detected, immediate laser photocoagulation of the worm is necessary as the migratory worm may be difficult to identify later on. The aim of laser therapy is to achieve death of the worm without inflicting collateral damage to the macula. The leading end of the nematode in forward movement will be the head, and this can be identified by using a low level of illumination to shepherd the nematode away from the macula, with a posterior vertical slit beam, before laser application to the head with a single laser shot. However, it may not be easy to distinguish the

head from the tail—especially for small worms [38]. Parameters for laser treatment include spot size ranged from 200 to 300 μm . Power settings range from 150 to 200 mW with an exposure time of 0.2 s. However, Schatz et al. reported a case in which the area of the worm was treated with 200-mW, 200- μm argon green laser spot for 0.2 s with unsuccessful results. They required 0.5 s with 300 mW and a 200- μm spot to kill the worm in the inner retina [39].

3. In patients in whom the worm is visualized and treated with laser, pretreatment immunosuppression with corticosteroids has reduced retinal inflammation (sometimes increased after laser treatment). In the majority of patients in whom laser treatment cannot be done, corticosteroids have uncovered the small worm and made it easy to identify. Different and variable doses have been tested, and one of the schemes includes oral prednisone 40 mg/day for 1 week with or without previous intravenous methylprednisolone at a dose of 1 g for three consecutive days.

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