Immune Recovery Vitritis Associated With Inactive Cytomegalovirus Retinitis

A New Syndrome

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Objective: To describe a syndrome of posterior segment intraocular inflammation that causes visual loss in patients with acquired immunodeficiency syndrome and cytomegalovirus retinitis. This syndrome was associated with immune recovery mediated by combination antiretroviral treatment including protease inhibitors.

Design: A case-control study at 2 university medical centers.

Participants: One hundred thirty patients with acquired immunodeficiency syndrome and cytomegalovirus retinitis were examined at 2 medical centers for 15 months. In addition, the medical records of 509 patients examined at 1 center for 11 years before the initiation of protease inhibitor therapy were analyzed retrospectively.

Results: Five patients with symptomatic vitritis and papillitis with cystoid macular edema or epiretinal membrane formation were documented. In each patient there was inactive cytomegalovirus retinitis that had not caused visual decrease before the onset of inflammation. All patients had elevated CD4+ T lymphocyte levels (median increase, 86×10^6/L [86 cells/mm^3]) after combination treatment including protease inhibitors. Two patients with cystoid macular edema were treated with corticosteroids and had resolution of the cystoid macular edema and an increase in visual acuity without reactivation of the retinitis. Retrospective analysis failed to disclose similar patients with intraocular inflammation in the era before the introduction of protease inhibitors.

Conclusions: This newly described syndrome of posterior segment inflammation related to cytomegalovirus retinitis is a cause of visual morbidity in patients with acquired immunodeficiency syndrome. It is associated with increased immune competence as a result of combined antiretroviral treatment with protease inhibitors and may be amenable to corticosteroid therapy without reactivation of retinitis.


The introduction of human immunodeficiency virus (HIV)-specific protease inhibitors in the treatment of acquired immunodeficiency syndrome (AIDS) has resulted in a significant improvement in immune status in many patients, as indicated by increased levels of CD4+ T lymphocytes and decreased levels of plasma HIV messenger RNA. The 4 protease inhibitors currently available are ritonavir, indinavir sulfate, nelfinavir mesylate, and saquinavir sulfate. All 4 competitively inhibit the protease-mediated cleavage of viral polyproteins, preventing the maturation of infectious virions. The result is inhibition of HIV replication.

Cytomegalovirus (CMV) retinitis in patients with AIDS is associated with profound immunodeficiency and a CD4+ T lymphocyte count below 100×10^6/L (100 cells/mm^3; usually <50×10^6/L [<50 cells/mm^3]). It is typically characterized by necrotizing retinitis with little or no intraocular inflammatory response, even in the presence of extensive retinal involvement.

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Macular inflammation or exudation in patients with CMV retinitis is uncommon. Serious macular exudation associated with posterior active CMV retinitis in patients with AIDS has been described by Gangan et al. Palestine and Frishberg described a patient with AIDS-related macular edema with cotton-wool spots, other microvascular abnormalities, and, eventually, a macular star, which was attributed to HIV microvasculopathic complications. A patient with cystoid macular edema (CME) associated with AIDS and

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PATIENTS AND METHODS

After the introduction of HIV-specific protease inhibitors for the treatment of patients with AIDS, we examined several patients with decreased visual acuity due to posterior segment inflammatory complications and inactive CMV retinitis. Accordingly, we reviewed the medical records of all patients with AIDS and CMV retinitis from December 1995 to March 1997 at 2 institutions: the AIDS Ocular Research Unit (AORU) of the University of California, San Diego, and the Cleveland Clinic, Cleveland, Ohio, to determine the incidence of this phenomenon.

At both institutions, a full ophthalmic examination was performed on all patients at each visit, including bilateral indirect ophthalmoscopy. Diagnosis of CMV retinitis and assessment of activity was based on typical ophthalmoscopic appearance and evidence of progression of retinitis borders in subsequent examinations. This was recorded with retinal drawings and confirmed with wide-angle (50°-60°) fundus photographs at each visit. Vitreous activity was evaluated according to the grading system (1-4) proposed by Nussenblatt et al. In addition, the levels of CD4+ T lymphocytes and plasma viral HIV messenger RNA were checked every 2 to 3 months as indicated to monitor antiretroviral therapy.

To evaluate the role of protease inhibitor therapy in the occurrence of inflammatory complications, an additional computer search was performed at the AORU using the database of CMV retinitis patients from January 1986 to December 1995, before the introduction of highly active antiretroviral treatment (HAART) at this institution.

We also evaluated 2 control groups from the AORU database for incidence of intraocular inflammation as follows: (1) HIV-positive patients without a history of CMV retinitis who were receiving HAART and had elevated CD4+ counts (CD4+ T lymphocyte count, $\geq 50x10^6$/L [$\geq 50$ cells/mm$^3$]; n=31) and (2) HIV-positive patients with a history of CMV retinitis who were receiving HAART but whose CD4+ counts remained low (CD4+ T lymphocyte count, $\leq 30x10^6$/L [$\leq 30$ cells/mm$^3$]; n=29).

At the Cleveland Clinic, all patients of a single physician (C.Y.L.) were analyzed retrospectively during the same period. Participation was voluntary, and we received informed consent from all patients.

CMV retinitis was described by Weinberg and Moorby. This patient developed anteroposterior segment intraocular inflammation with CME. He was in a severely immunosuppressed condition (CD4+ T lymphocyte count, $3x10^6$/L [3 cells/mm$^3$]) and was not being treated with protease inhibitors. However, he was treated concomitantly with rifabutin, which may have been the cause of the inflammation. This is, to the best of our knowledge, the only documented patient with inflammatory CME. We postulated that the profound immunodeficiency in patients with AIDS has a protective effect against CME and against other vision-threatening inflammation-induced complications of necrotizing retinitis, such as papillitis and epiretinal membrane formation.

We evaluated 5 patients undergoing treatment with protease inhibitors who had rising CD4+ T lymphocyte levels and inactive CMV retinitis. All demonstrated moderate vitritis and papillitis, some with CME. We attribute these inflammation-induced complications to the increased immunocompetence of these patients. We term this new syndrome immune recovery vitritis.

REPORT OF CASES

CASE 1

A 32-year-old man (patient 3 in Table 1 and Table 2) in whom HIV infection was diagnosed in January 1989 was diagnosed as having AIDS in March 1994 when he developed Pneumocystis carinii pneumonia. The patient developed CMV colitis in April 1996 and was treated with intravenous ganciclovir. He was receiving zidovudine and lamivudine at the time. Treatment with a protease inhibitor (indinavir) was initiated in June 1996. In October 1996, while receiving maintenance intravenous ganciclovir therapy, he presented at another institution for ophthalmic evaluation and was found to have clinically inactive CMV retinitis of the left eye. In December 1996, on initial examination at the AORU, the patient complained of blurring of vision and floaters in the left eye. Visual acuity was 20/25 OD and 20/63 OS. Ophthalmoscopic examination results disclosed inactive CMV retinitis in the nasal periphery (4.0 disc diameters from the center of the fovea) of the left eye with moderate vitritis (2+) and CME. The border of the retinitis showed no opacity and appeared completely healed. There was no anterior segment inflammation. The right eye was healthy. The CD4+ T lymphocyte count was $108x10^6$/L (108 cells/mm$^3$). Fluorescein angiography was performed, and results showed moderate leakage in a petaloid pattern consistent with CME, with mild leakage from the optic disc (Figure 1).

A posterior subtenon injection of repository methylprednisolone (80 mg) was administered on January 10, 1997, and the patient continued maintenance therapy with ganciclovir. Another injection was given 8 weeks later and resulted in partial resolution of the CME ophthalmoscopically 2 weeks after the second injection. Visual acuity improved to 20/40 OU. Cytomegalovirus retinitis remained inactive.

CASE 2

A 32-year-old man (patient 3 in Tables 1 and 2) was diagnosed as having AIDS 34 months after being diagnosed as having HIV infection. Cytomegalovirus retinitis of the right eye was diagnosed in December 1994 and was treated with ganciclovir followed by combination ganciclovir and foscarnet after reactivation of the retinitis during ganciclovir therapy. Highly active antiretroviral treatment with a protease inhibitor (ritonavir initially, then indinavir) was initiated in January 1996, and elevation of the CD4+ T lymphocyte count occurred 3 months later (22-63x10^6$/L [22-63 cells/mm$^3$]) and the patient con-
continued therapy with combination ganciclovir and foscarnet. Posterior segment intraocular inflammation manifested initially as moderate vitritis (2+) with floaters. The patient developed CME and papillitis with a decrease in visual acuity (20/40 OD), as documented by indirect ophthalmoscopy, fundus photography, and fluorescein angiography. Cytomegalovirus retinitis was inactive clinically for 9 months at the onset of CME.

Posterior segment inflammation was treated with systemic corticosteroids (60 mg of prednisone for 2 weeks, decreased by 20 mg every 2 weeks). The CME improved clinically as shown by fluorescein angiography. Visual acuity improved to 20/30 OD. Cytomegalovirus retinitis did not reactivate after corticosteroid treatment.

**CASE 3**

A 44-year-old man (patient 5 in Tables 1 and 2) was diagnosed as having AIDS in February 1995 with the diagnosis of Kaposi sarcoma. The patient was diagnosed at another institution as having CMV retinitis of the right eye in December 1995 and was treated with an induction dose of intravenous ganciclovir followed by oral ganciclovir therapy for maintenance. The retinitis reactivated in March 1996 and again in June 1996, and the patient was reinduced. Treatment with a protease inhibitor (indinavir) was initiated in September 1996.

In October 1996, on initial examination at the AORU, the patient had no visual complaints. Visual acuity was 20/25 OD and 20/15 OS. There was active CMV retinitis of the superior midperiphery (3.5 disc diameters from the center of the fovea) of the right eye without anterior segment inflammation. The left eye was normal. The patient received a single intravitreal injection of cidofovir (15 µg), and oral maintenance ganciclovir therapy was continued. There was no iritis or hypotony after the injection. Resolution of the retinitis was noted 4 weeks after the injection, and retinitis remained inactive through the most recent examination. In November 1996, oral ganciclovir therapy was discontinued, and

### Table 1. Medical Data in Patients With Immune Recovery Vitritis*

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Date of Diagnosis</th>
<th>Antiretroviral Treatment</th>
<th>Anti-CMV Treatment</th>
<th>Protease Inhibitors: Initiation Date</th>
<th>CD4⁺ Cell Count at CMV Diagnosis, ×10⁶/L</th>
<th>CD4⁺ Cell Count at Onset of Inflammation, ×10⁶/L: Date</th>
<th>Plasma HIV mRNA at Onset of Inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>12/92</td>
<td>1/15/93</td>
<td>5/10/94</td>
<td>Ganciclovir</td>
<td>Indinavir: 12/12/95</td>
<td>17</td>
<td>300: 5/7/96</td>
</tr>
<tr>
<td>4</td>
<td>1/15/95</td>
<td>3/15/96</td>
<td>10/15/96</td>
<td>Zidovudine</td>
<td>Indinavir: 1/15/96</td>
<td>18</td>
<td>104: 8/15/96</td>
</tr>
<tr>
<td>5</td>
<td>2/15/95</td>
<td>2/15/95</td>
<td>12/15/96</td>
<td>Stavudine</td>
<td>Indinavir: 5/16/95</td>
<td>28</td>
<td>79: 12/15/96</td>
</tr>
</tbody>
</table>

*HIV indicates human immunodeficiency virus; AIDS, acquired immunodeficiency syndrome; CMV, cytomegalovirus; mRNA, messenger RNA; PCP, Pneumocystis carinii pneumonia, and VZV, varicella-zoster virus.

### Table 2. Ocular Findings in Patients With Immune Recovery Vitritis

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Iritis: Date</th>
<th>Vitritis: Date</th>
<th>Macular Changes: Date</th>
<th>Visual Acuity</th>
<th>Preinflammation</th>
<th>Worst During Inflammation: Duration, wk*</th>
<th>Posttreatment</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>1+ cells: 8/6/95</td>
<td>2+ cells: 7/30/96</td>
<td>ERM: 10/22/96</td>
<td>20/25</td>
<td>20/80</td>
<td>20/80</td>
<td>No treatment</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>2+ cells: 8/20/96</td>
<td>Cystoid macular edema: 9/13/96</td>
<td>20/20</td>
<td>20/80: 16</td>
<td>20/25</td>
<td>No treatment</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>1+ cells: 3/15/96</td>
<td>2+ cells: 13/15/96</td>
<td>Macular edema: 12/15/96</td>
<td>20/20</td>
<td>20/40: 10</td>
<td>20/30</td>
<td>Oral prednisone repository corticosteroid injections†</td>
<td>None</td>
</tr>
<tr>
<td>4</td>
<td>None</td>
<td>2+ cells: 12/10/96</td>
<td>Cystoid macular edema: 12/10/96</td>
<td>20/30</td>
<td>20/63: 23</td>
<td>20/40</td>
<td>No treatment</td>
<td>None</td>
</tr>
<tr>
<td>5</td>
<td>None</td>
<td>2+ cells: 1/3/97</td>
<td>ERM: 1/17/97</td>
<td>20/25</td>
<td>20/40</td>
<td>20/40</td>
<td>No treatment</td>
<td>None</td>
</tr>
</tbody>
</table>

*Weeks of decreased vision before therapy or before spontaneous vision recovery. ERM indicates epiretinal membrane.
the patient was followed up for reactivation of retinitis. The CD4+ T lymphocyte count was $59 \times 10^3$ (59 cells/mm$^3$). In January 1996, 4 months after the initiation of protease inhibitor therapy, the patient complained of floaters and blurring of vision in the right eye. Visual acuity had decreased to 20/32 OD. On examination, CMV retinitis remained inactive, and there was 2 vitritis. On January 17, 1997, the patient complained of increasing blurring of vision in the affected eye. The visual acuity was 20/40 OD and funduscopy disclosed 2 vitritis and macular changes consistent with epiretinal membrane and diffuse macular edema, and the retinitis remained inactive. Results of fluorescein angiography showed diffuse leakage of fluorescein in the macula, with mild leakage from the optic disc (Figure 2). The CD4+ T lymphocyte count was $79 \times 10^3$ (79 cells/mm$^3$). The epiretinal membrane has not been treated surgically.

At the AORU between December 1995 and March 1997, 108 patients (174 eyes) with AIDS and CMV retinitis were examined, 66 of whom had bilateral and 42 of whom had unilateral disease. During the study period, 102 of the 108 patients received protease inhibitors. Four (3.7%) of 108 patients and 4 (2.3%) of 174 eyes had significant posterior segment intraocular inflammation. At the Cleveland Clinic during the same period, 22 patients with AIDS-related CMV retinitis were evaluated, 7 of whom received protease inhibitors. One patient developed posterior segment inflammation. All of these 5 patients (5 eyes) had moderate-to-severe vitritis (2-3 on a 1-4 scale) and papillitis; 3 developed CME and the other 2 developed epiretinal membranes. The patients included 4 men and 1 woman whose ages ranged from 31 to 48 years (median, 43 years). The diagnosis of AIDS preceded the di-
agnosis of CMV retinitis by a median of 17.5 months (range, 7-36 months) in 4 patients, and CMV retinitis was the AIDS-defining diagnosis in the other patient.

All 5 patients were initially treated for CMV retinitis with ganciclovir sodium (induction dosage, 10 mg/kg per day; maintenance dosage, 5 mg/kg per day) or combination ganciclovir and foscarnet sodium. Two patients received a single intravitreal injection of cidofovir (15 µg). This therapy was not associated with iritis in either patient and preceded the onset of posterior segment inflammation by 3 months in patient 5 and 1 month in patient 2. The inflammatory complications in these patients, therefore, were not attributed to cidofovir therapy. One patient was treated with intravenous cidofovir, which was initiated 2 months after the onset of posterior segment intraocular inflammation with CME and therefore was not implicated in causing the inflammation.

All of the medications that the patients were receiving were reviewed (Table 3), and medications known to cause intraocular inflammation were not included. None of the patients were receiving treatment with rifabutin within 2 months of the onset of vitritis.

Between December 1995 and June 1996, before the onset of uveitis, all 5 patients initiated HAART with a combination of 1 or 2 reverse transcriptase inhibitors (lamivudine, zidovudine, or stavudine) and 1 protease inhibitor (indinavir or ritonavir). As a result of this treatment, all patients experienced a marked increase in CD4+ T lymphocyte levels (range of increase, 41-283×10^3/L [41-283 cells/mm^3]; mean increase, 130×10^3/L [130 cells/mm^3]; median increase, 86×10^3/L [86 cells/mm^3]) and a reduction of plasma HIV messenger RNA to undetectable levels. The time between the initiation of HAART and the elevation of CD4+ T lymphocyte levels was 2 to 5 months (median, 2 months).

Posterior segment inflammatory complications manifested as a painless decrease in visual acuity (20/40-20/63) and floaters, which occurred 2 to 16 weeks (median, 4 weeks) after the elevation of CD4+ T lymphocyte counts. At the onset of posterior segment inflammation, all of the patients had inactive CMV retinitis in zones 1 or 2.2-4,14 That had not caused visual inflammation, all of the patients had inactive CMV retinitis. The inflammatory complications in these patients, therefore, were not attributed to cidofovir therapy.13 One patient was treated with intravenous cidofovir, which was initiated 2 months after the onset of posterior segment intraocular inflammation with CME and therefore was not implicated in causing the inflammation.

To determine whether a similar entity existed before the advent and use of protease inhibitors, we reviewed the medical records of all of the patients examined at the AORU between August 1986 and December 1995, before the initiation of HAART at this institution. The records revealed 509 patients (830 eyes) with AIDS-related posterior segment ocular disease during this period. Review of these records failed to identify any patients with 2 to 3+ vitritis related to CMV retinitis. Papillitis was found in 8 eyes, 2 of which were not associated with CMV retinitis but were related to syphilis. In the remaining 6 eyes (0.1%) there was involvement of the peripapillary retina with active CMV retinitis. This was not the case with any of the patients of the present series.

Three patients with CME were documented, but these were not related to CMV retinitis; 1 was a patient with endogenous endophthalmitis and the other 2 were patients with reticulitis. Thirty-two patients (35 eyes) with serous macular exudation related to active CMV retinitis were documented, as described by Gangan et al.6 These patients manifested neurosensory retinal detachments with lipid exudates rather than CME. Finally, this review revealed 3 patients (3 eyes, 0.4%) with epiretinal membranes. All of these were related to CMV retinitis of an area of the retina adjacent to the membrane and were not associated with significant intraocular inflammation. In the patients in the present report there was no CMV retinitis adjacent to the fovae.

Regarding the 2 control groups outlined in the “Patients and Methods” section, we found no incidence of intraocular inflammation in any patient from either control group: those without CMV retinitis with elevated CD4+ counts (0 of 31) or those with CMV retinitis with low CD4+ counts (0 of 29).

We describe 5 patients with clinically significant posterior segment intraocular inflammation associated with AIDS and inactive CMV retinitis, as evaluated at 2 institutions between December 1995 and March 1997. All of these patients had marked increases in CD4+ T lymphocyte levels (63-300×10^3/L [63-300 cells/mm^3]; mean,

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**Table 3. Medications Taken by Patients With Immune Recovery Vitritis**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Patient No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trimethoprim and sulfamethoxasole</td>
<td>1, 4, 5</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>1</td>
</tr>
<tr>
<td>Prochlorperazine</td>
<td>1</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>2, 5</td>
</tr>
<tr>
<td>Azithromycin</td>
<td>2, 5</td>
</tr>
<tr>
<td>Pentamidine</td>
<td>2</td>
</tr>
<tr>
<td>Estrogen</td>
<td>2</td>
</tr>
<tr>
<td>Cephalexin</td>
<td>2</td>
</tr>
<tr>
<td>Amoxicillin</td>
<td>4</td>
</tr>
<tr>
<td>Clarithromycin</td>
<td>3</td>
</tr>
<tr>
<td>Vancomycin</td>
<td>3</td>
</tr>
</tbody>
</table>

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**COMMENT**

We describe 5 patients with clinically significant posterior segment intraocular inflammation associated with AIDS and inactive CMV retinitis, as evaluated at 2 institutions between December 1995 and March 1997. All of these patients had marked increases in CD4+ T lymphocyte levels (63-300×10^3/L [63-300 cells/mm^3]; mean,
ytomegalovirus retinitis in patients with AIDS typically manifests as a progressive necrotizing retinitis with little or no intraocular inflammation. Vitritis is not uncommon, but, when present, it is mild and minimally symptomatic, unlike that seen in our patients, who were very symptomatic. Macular inflammation or exudation is rare and has been associated with AIDS microvasculopathy or active CMV retinitis involving the optic disc, macula, or perimacular region. This was not the case in the present series.

Intraocular inflammation in this setting also has been attributed to concomitant treatment with rifabutin or cidofovir. We evaluated all patient medications and excluded patients undergoing concomitant treatment with rifabutin or cidofovir within 1 month of the onset of inflammation and visual decrease. Rifabutin use in our institution has decreased because of adverse interactions with protease inhibitors. There were no other medications being taken by these patients that cause intraocular inflammation.

The pathogenesis of the inflammatory syndrome we describe is unclear. To shed some light on this we evaluated a series of control groups, as follows. The medical records of 509 patients examined at the AORU between January 1986 and December 1995, in the pre-HAART era, were analyzed. All ocular findings at the AORU are entered into a database contemporaneously with the patient examination results, allowing for accurate data analysis. Similar patients with intraocular inflammation associated with inactive CMV retinitis were not identified, suggesting that HAART, or the resultant elevation of CD4+ T lymphocyte levels, plays a role in this phenomenon.

It is possible that the protease inhibitors themselves (indinavir and ritonavir in this series) could directly mediate this inflammation. To evaluate this, a second control group of patients from the AORU with CMV retinitis who had not responded to HAART with elevation of CD4+ T lymphocyte levels (<50×10^6/L [<50 cells/mm^3]) was reviewed (n=29). There were no patients with intraocular inflammation in this group. This suggests that increased immune competence, as indicated by elevation of CD4+ T lymphocyte levels, may be directly related to the inflammation. It is unclear why only some patients with healed retinitis and evidence of immune restoration develop intraocular inflammation.

It is also possible that the inflammatory complications we describe could be due to an ocular pathogen other than CMV. Intraocular inflammation in patients with AIDS has been described in the setting of luetic as well as tuberculous uveitis and therefore should be considered in all patients with intraocular inflammation. However, the lack of active retinitis or choroiditis in our series, and the positive response to corticosteroid therapy, makes this possibility unlikely.

Finally, to assess the role of CMV, we examined a third control group of patients who were receiving HAART, including protease inhibitors, and had high (>50×10^6/L [>50 cells/mm^3]) levels of CD4+ T lymphocytes but did not have CMV retinitis (n=31). We did not find any patients with intraocular inflammation in this control group.

It has been postulated that the absence of significant inflammatory response to CMV retinitis in patients with AIDS is due to the severe immunodeficiency associated with AIDS. With the advent of combined antiretroviral therapy, including HIV-specific protease inhibitors, many patients with AIDS may be experiencing immune reconstitution, with rising CD4+ T lymphocyte levels and decreasing HIV messenger RNA levels. It is as yet unclear whether this represents full recovery of immune function because there have been indications that these CD4+ T lymphocytes are only partially functional. However, in the setting of CMV retinitis, our experience has been that the use of protease inhibitors has resulted in significant increase in time to progression of CMV retinitis in a group of patients who have responded with elevation of CD4+ T lymphocyte counts.

The posterior segment inflammatory responses seen in our series could therefore be attributed to increased immune function as a result of HAART including protease inhibitors in eyes with CMV retinitis.

It is unclear precisely what factors are necessary to cause the inflammatory response in these patients. It is associated with the presence of inactive CMV retinitis, combination antiretroviral therapy with protease inhibitors, and evidence of at least partial immune reconstitution suggested by elevated CD4+ cell counts. However,
there are many patients with all 3 factors who do not develop inflammation. The reason is not known. The inflammatory reaction could be in response to CMV antigens expressed on cells that have been latently infected, near the areas of previously active CMV retinitis. Indeed, the immune response to CMV antigens varies in patients with AIDS. Schier et al. previously have shown that this may be related to the predisposition to CMV retinitis. The heterogeneity of the T-lymphocyte response has already been documented. Further studies are necessary to evaluate the incidence and pathogenesis of this newly described syndrome.

Immune recovery vitritis emerges as a new syndrome and a cause of visual morbidity in patients with AIDS. It is associated with improving immune status as a result of HAART and may be reversible with corticosteroid treatment without reactivation of CMV retinitis.

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19. 4th Conference on retroviruses and opportunistic infections; January 24, 1997; Washington, DC.


ARCHIVES OF OTOLARYNGOLOGY—HEAD & NECK SURGERY

Optic Nerve Decompression for Compressive Neuropathy Secondary to Neoplasia
Kasey K. Li, MD, DDS; Mark J. Lucarelli, MD; Jurij R. Bilyk, MD; Michael P. Joseph, MD

Objective: To evaluate the outcome of extracranial optic nerve decompression in patients with compressive optic neuropathy secondary to intracranial, para nasal sinus, or orbital neoplastic processes.

Design: A retrospective chart review and clinical follow-up of patients who underwent optic nerve decompression.

Setting: Tertiary care referral center.

Patients: During an 8-year period, 95 extracranial optic nerve decompressions were performed by one of us (M.P.J.) for optic neuropathy resulting from traumatic, inflammatory, infectious, iatrogenic, neoplastic, and idiopathic processes. Thirty patients with compressive optic neuropathy secondary to histopathologically confirmed tumors were identified.

Intervention: Optic nerve decompression via external ethmoidectomy approach.

Result: Twenty (67%) of 30 patients showed improvement in vision. Improvement in 17 of the 20 patients has been stable. Seven patients (23%) showed no improvement but there was no further worsening of vision after surgery. In 3 patients (10%) vision deteriorated following surgery.

Conclusion: Extracranial optic nerve decompression may be considered for the preservation or improvement of vision in selected patients with compressive optic neuropathy from neoplasms. Arch Otolaryngol Head Neck Surg. 1997;123:425-429

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