Clinical characteristics of acute HSV-2 retinal necrosis

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Abstract

Purpose

To report the clinical features and evaluate the visual outcome of eleven cases of herpes simplex virus-2 (HSV-2) related acute retinal necrosis syndrome (ARN).

Design

Retrospective interventional case series.

Methods

Twelve eyes of eleven patients from two European centers, diagnosed with HSV-2 related acute retinal necrosis syndrome were retrospectively reviewed. Herpes simplex virus-2 DNA was detected.
by polymerase chain reaction in intraocular fluids (aqueous and/or vitreous). Findings at initial examination, clinical evolution with antiviral therapy, complications and final visual acuity were evaluated.

Results

Herpes simplex virus-2 DNA was detected in all cases. No sample was positive for more than one virus. The mean age of disease in the first eye was 36 years (ranged from 10 to 57 years). Five patients were women and six were men. All patients were immunocompetent. Previous medical history included neonatal herpes \( (n = 1) \), previous ARN \( (n = 3) \), trauma \( (n = 1) \) and systemic corticosteroid administration before occurrence of ARN \( (n = 3) \). Preexisting pigmented chorioretinal scars were found in three cases. Patients were treated with high dose intravenous acyclovir or foscarinat and intravitreal ganciclovir and interferon. The mean follow-up was 14.5 months (from 5 to 22 months). At the end of the follow-up period, five eyes \( (41.7\%) \) showed improvement of visual acuity of two or more lines. Final visual acuity was 20/60 or better in four eyes \( (33.3\%) \), 20/400 or better in four eyes \( (33.3\%) \) and less than 20/400 in four eyes.

Conclusion

History of neonatal herpes, triggering events such as neurosurgery, periorcular trauma, high-dose corticosteroids, and chorioretinal scars suggest that HSV-2 retinitis reflects reactivation of HSV-2 infection.

Article Outline

Patients and methods
Results
Discussion
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Vita

Acute retinal necrosis syndrome (ARN) is, according to the Executive Committee of the American Uveitis Society, characterized by peripheral necrotizing retinitis, retinal arthritis, and a prominent inflammatory reaction in the vitreous and anterior chamber.1

Various studies, including examination of retinal biopsy specimens,2 viral culture,3 immunocytochemical study,4 intraocular antibody production, and polymerase chain reaction (PCR),5, 6, 7, 8, 6, 10, 11 and 12 have implicated varicella-zoster virus (VZV), herpes simplex virus-1 (HSV-1) and -2 (HSV-2), cytomegalovirus (CMV), and, rarely, Epstein-Barr virus (EBV)13 and 14 as causative agents of ARN syndrome.

Polymerase chain reaction is a sensitive and specific method, which has been performed successfully to detect viral DNA in ocular samples from immunocompetent and immunocompromised patients presenting with viral retinitis.5, 10, 12, 13, 15, 16 and 17 It has been used as a molecular tool for the diagnosis and the determination of causal agents in ARN syndrome.

The association of herpetic encephalitis12, 18, 19, 20, 21, 22, 23, 24 and 25 or herpes neonate with ARN syndrome12, 18 and 26 has been reported previously. Herpes simplex virus-2 is one of the causative agents of the ARN syndrome, particularly in Japan.11 It has been suggested that in patients less than 25 years old, ARN syndrome is more likely to be caused by HSV-2.12 In this study, we describe the clinical features and
visual outcome of the HSV-2-related ARN syndrome proven by PCR.

Patients and methods

Patients diagnosed with HSV-2-related ARN syndrome recruited in adult referral centers in Brussels (patients 1, 3, and 4) and in Paris (patients 2, and 5–11) between April 1999 and February 2002 were retrospectively selected. All patients fulfilled the clinical diagnostic criteria of ARN syndrome according to the Executive Committee of the American Uveitis Society. Patients with necrotizing retinopathy caused by other agents were excluded. Implication of HSV-2 was confirmed by PCR in all patients in the aqueous humor or vitreous, with negative results for HSV-1, VZV, EBV, and CMV.

Intraocular fluid (aqueous or vitreous) samples were obtained in nine cases from anterior chamber paracentesis or at the time of posterior segment surgery (retinal detachment, epiretinal membrane, severe vitreous haze). None of the patients had bilateral paracentesis. Ten to 20 μl of aqueous humor were used for each PCR reaction. Sensitivity and specificity of assays performed in patients from Paris for detection of HSV-1, HSV-2, EBV, CMV, and VZV have been described previously.[13], [27] and [28] Detection of HSV-2 in patients from Brussels was performed by nested PCR.[29] and [30]

In addition to PCR analysis of intraocular fluids, nonspecific HSV antibodies in sera and screening for acquired immunodeficiency syndrome were performed in all patients.

In each center, all patients were followed by the same physician. Therapeutic management, complications, and final visual acuity at the last examination are reported.

Results

Diagnosis of an ARN syndrome was made in 24 patients at the Pitié-Salpêtrière Hospital and in 15 patients at the Saint Pierre Hospital between April 1999 and February 2002. Demographic characteristics and initial clinical presentation of the HSV-2-related ARN syndrome are shown in TABLE 1 and TABLE 2. Six males and five females with a mean age of 40 years (ranging from 10 to 57 years) were included. One patient (patient 9) presented with bilateral disease (BARN), and 10 with a unilateral form, yielding a total of 12 involved eyes.

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Eye</th>
<th>Duration of Symptom Before Examination</th>
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<tr>
<td>1</td>
<td>57</td>
<td>F</td>
<td>OS</td>
<td>2 wk</td>
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<tr>
<td>2</td>
<td>51</td>
<td>F</td>
<td>OD</td>
<td>3 wk</td>
</tr>
<tr>
<td>3</td>
<td>26</td>
<td>F</td>
<td>OS</td>
<td>2 wk</td>
</tr>
<tr>
<td>4</td>
<td>47</td>
<td>M</td>
<td>OD</td>
<td>3 wk</td>
</tr>
<tr>
<td>5</td>
<td>10</td>
<td>M</td>
<td>OS</td>
<td>4 d</td>
</tr>
<tr>
<td>6</td>
<td>51</td>
<td>M</td>
<td>OS</td>
<td>3 d</td>
</tr>
</tbody>
</table>

Visual loss because of ARN of OD 12 years before
Globe atrophy of OS of undetermined origin. Deta
Herpetic keratitis of OD. Ocular toxoplasmosis of t
None
Visual loss of OD because of traumatic RD at 9 ye
ARN of OD 10 years before. Preexisting inactive c
<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Initial VA</th>
<th>Final VA</th>
<th>IOP</th>
<th>Extent of the Retinitis, Clock Hours</th>
<th>Samples</th>
<th>Antiviral Therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20/200</td>
<td>LP</td>
<td>36</td>
<td>Peripheral, 12</td>
<td>V</td>
<td>Acyclovir</td>
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<tr>
<td>2</td>
<td>20/30</td>
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<td>26</td>
<td>Peripheral, 12</td>
<td>AH</td>
<td>Acyclovir then foscanet</td>
</tr>
<tr>
<td>3</td>
<td>20/50</td>
<td>20/60</td>
<td>19</td>
<td>Peripheral, 4</td>
<td>V and AH</td>
<td>Acyclovir</td>
</tr>
<tr>
<td>4</td>
<td>20/100</td>
<td>CF</td>
<td>14</td>
<td>Peripheral, 12</td>
<td>AH</td>
<td>Acyclovir</td>
</tr>
<tr>
<td>5</td>
<td>CF</td>
<td>20/60</td>
<td>6</td>
<td>Peripheral, 6</td>
<td>AH</td>
<td>Foscarnet + ganciclovir IVT + IFN-γ</td>
</tr>
<tr>
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<td>20/40</td>
<td>20/100</td>
<td>30</td>
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<td>AH</td>
<td>Foscarnet + ganciclovir IVT</td>
</tr>
<tr>
<td>7</td>
<td>20/25</td>
<td>20/20</td>
<td>13</td>
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<td>AH</td>
<td>Foscarnet</td>
</tr>
<tr>
<td>8</td>
<td>20/200</td>
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<td>35</td>
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<td>AH</td>
<td>Foscarnet + ganciclovir IVT then IFI</td>
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<tr>
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<td>Peripheral, 5</td>
<td>V and AH</td>
<td>Foscarnet + ganciclovir IVT</td>
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</tbody>
</table>

**Notes:**

AH = aqueous humor; ARN = acute retinal necrosis; CF = counting fingers; CME = cystoid macular edema; ERM = epiretinal membrane; HSV-2 = herpes simplex virus type 2; IFN = interferon; IOP = intraocular pressure; IVT = intravitreal injection; LP = light perception; MH = macular hole; PPVT = pars plana vitrectomy; RD = retinal detachment; V = vitreous; VA = visual acuity.

All patients were immunocompetent. Three patients with unilateral disease had a history of ARN syndrome (patients 1, 6, and 7). Of these, two patients were referred for ARN syndrome of the second eye (patients 1 and 6) with a bilateralization after a delay ranging from 10 to 12 years. History of patient 7 was remarkable for neonatal herpes and vision loss in the right eye at birth. He presented with two previous episodes of ARN syndrome in the left eye and was referred for management of a third relapse. Patient 2 had a history of retinal
detachment in the same eye 15 years earlier and atrophy of the left eye for unknown causes. She had no pathologic myopia or major traumatism, and there was no history of uveitis. Patient 3 had a history of PCR-positive ocular toxoplasmosis of the nasal quadrant of the same eye years before the ARN syndrome, which had regressed upon antiparasite treatment. Funduscopic revealed two retinal scars in the temporal and nasal quadrants and peripheral retinal necrosis, which ultimately progressed despite antitoxoplasmic treatment. The new aqueous tap performed in the same eye was positive for HSV-2 and negative for toxoplasmosis. From analysis of medical histories, the corrected mean age of first eye involvement was age 36 years, and bilateral disease occurred in four of 11 (27.2%) cases (patients 1, 5, 7, and 9).

Acute retinal necrosis syndrome appeared within 2 weeks after systemic corticosteroid administration in three patients. One of them (patient 8) had been treated with oral prednisone for anterior uveitis before being referred. Two other patients received intravenous methylprednisolone after a negative large exploration for chronic panuveitis. Patient 2 presented with a history of blurred vision for 3 weeks in his left eye. Slit-lamp examination disclosed a panuveitis with moderate inflammation and retinal vasculitis without retinitis. Systemic investigation (blood cell count, antinuclear antibodies, fluorescent treponemal antibody-absorption test and Venereal Diseases Research Laboratory tests, rheumatoid factor, angiotensin-converting enzyme, cerebral magnetic resonance imaging, bronchoscopy and bronchial biopsies, audiogram, and lumbar puncture) was performed because the diagnosis of viral retinitis remained uncertain. Patient 9 presented with chronic panuveitis, retinal vasculitis, and peripheral scars for 5 years. Corticosteroids were immediately stopped after confirmation of ARN syndrome.

A history of periorcular trauma was found in patient 5. He suffered a head injury 1 week before the ARN syndrome appeared, but his eye had not been injured.

The duration of symptoms before examination ranged from 2 days to 1 year. Most of the patients (10/11) were seen within 1 month after the beginning of symptoms. Initial visual acuity ranged from 20/25 to counting fingers. Visual acuity was 20/60 or better in four eyes (33.3%), from 20/70 to 20/400 in four eyes (33.3%), and less than 20/400 in four eyes (33.3%).

All patients had active necrotizing retinitis. Healed areas demonstrated retinal pigment epithelial abnormalities and neuroretinal atrophy. Three patients (patients 3, 6, and 9) presented with inactive chorioretinal scars in the eye with ARN syndrome. The clock-hour numbers of peripheral involvement sites for each patient are given in Table 2. Nine of 12 eyes (75%) presented the involvement of at least two quadrants.

Herpes simplex virus-2 DNA was detected in two vitreous and nine aqueous samples obtained from 11 patients. Antibody simple virus antibodies (anti-HSV-1 + 2 IgG) were detected in the sera of all patients. Results of syphilis and human immunodeficiency syndrome serologies were negative in all patients.

Antiviral therapy was started at admission and continued for at least 3 weeks. Four patients (1–4) were treated with acyclovir (acyclovir 10 mg/kg/8 h). Of these cases, retinitis progressed in two patients (patients 2 and 3) despite acyclovir. Foscarnet was added to control retinitis in patient 2, and vitrectomy was performed in patient 3 because of severe vitreous haze. Two patients (patients 7 and 9) were treated with intravenous foscarnet (180 mg/kg/day). Five patients were treated with a combination of intravenous foscarnet and intravitreal ganciclovir (2,000 μg/0.5 ml) for rapid regression of retinitis. In nine patients, retinitis resolved completely within an average of 1 month after initiation of an antiviral regimen. In two other patients (patients 5 and 8), relapses occurred each time foscarnet was switched to oral valacyclovir. In these cases, foscarnet had to be continued for several weeks, and immunomodulators, such as interferon, were associated to stop major antiviral regimens without further relapse. High-dose corticosteroids were administered to reduce inflammation (patients 1, 5–8, and 10).
only after efficient control of retinitis under antiviral therapy. Patients were placed on oral antivirals for at least 6 months following the intravenous course of administration.

Retinal detachment occurred in five eyes (41.7%) (patients 1, 4, 6, 9, and 11) and was successfully treated with a single surgical procedure, including pars plana vitrectomy, endolaser photoagulation, and silicone oil injection with or without scleral buckling. Despite anatomical success, only two eyes (patients 6 and 9) achieved a final visual acuity greater than 20/200 at the last examination. Patient 10 presented with a retinal tear, which was treated with laser photoagulation. Scleral buckling was performed secondarily. Other complications included: epiretinal membrane (four eyes, 33.3%), cataract (five eyes, 41.7%), optic nerve atrophy (two eyes, 16.7%), cystoid macular edema (one eye, 8.3%). Of 11 originally phakic patients, five underwent secondary cataract surgery after retinal surgery.

The mean follow-up was 14.5 months, ranging from 5 to 22 months. At the last examination, five eyes (41.7%) showed improvement of visual acuity of two or more lines of the Snellen chart. Final visual acuity was 20/60 or better in four eyes (33.3%), 20/200 or better in four eyes (33.3%), and less than 20/400 in four eyes (33.3%).

**Discussion**

The results of this study suggest distinct clinical features of HSV-2-related ARN syndrome: young age of the patient, history of herpes at birth, preexisting chorioretinal scar in the eye presenting the ARN syndrome, triggering events such as trauma or systemic corticosteroids. Patients with HSV-2 may show a relatively aggressive clinical course and a variable response to treatment.

Clinical criteria of ARN syndrome have been described previously. The disease can progress rapidly, with vision loss due to macular involvement, retinal detachment, or optic neuropathy. Previous studies demonstrated that ARN syndrome is caused by herpes viruses: VZV, HSV, occasionally CMV, or EBV.[6], [12], [13] and [14] Polymerase chain reaction analysis and intraocular antibody testing are the two laboratory techniques that are used to determine the causative agent of ARN syndrome.

Series reporting HSV-2 as the causative agent of ARN syndrome are rare, and most reports are of sporadic cases. Margolis and associates first suggested HSV-2 as a causative agent in the ARN syndrome using serologic analysis. The prevalence of HSV-2-related ARN syndrome may be underrepresented because several studies used intraocular antibody testing with antibodies reactive to both HSV-1 and HSV-2. The use of PCR analysis[11], [12], [13], [18], [24], [32], [33], [34], [35] and [36] or specific antibody testing[37] may explain increased reporting of HSV-2-related ARN syndrome.

Combining data from previous and present studies, 38 cases of HSV-2-related ARN syndrome have been reported up to October 2002. These data are summarized in Table 3. The mean age of HSV-2-related ARN syndrome at the first eye is 22.6 years, ranging from 25-day-old neonates to 56-year-old patients. Of these, at least 12 cases (31.6%) presented with bilateral involvement. All patients were immunocompetent. Many authors found that HSV-2 is an important cause in younger patients, whereas VZV is predominant in older patients. It has been suggested that ARN syndrome in patients under 25 years old is likely due to HSV-2.[12], [13] and [37]

**TABLE 3.**

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Report</th>
<th>Age</th>
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</thead>
</table>

Review of HSV-2 Related ARN in the Literature
<table>
<thead>
<tr>
<th>Study:</th>
<th>Year</th>
<th>Type</th>
<th>Age</th>
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<tbody>
<tr>
<td>Margolis and associates</td>
<td>1988</td>
<td>Case report</td>
<td>48</td>
<td>Parase</td>
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<td>Thompson and associates</td>
<td>1994</td>
<td>3 patients</td>
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</tr>
<tr>
<td></td>
<td></td>
<td>1</td>
<td>30</td>
<td>Premat</td>
</tr>
<tr>
<td></td>
<td></td>
<td>2</td>
<td>4</td>
<td>Preexis</td>
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<td></td>
<td>3</td>
<td>10</td>
<td>Premat</td>
</tr>
<tr>
<td>Rahhal and associates</td>
<td>1996</td>
<td>Case report</td>
<td>44</td>
<td>Craniot</td>
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<td>Schlingemann and associates</td>
<td>1996</td>
<td>Case report</td>
<td>28</td>
<td>ARN of</td>
</tr>
<tr>
<td>Rappaport and Tang</td>
<td>2000</td>
<td>Case report</td>
<td>29</td>
<td>System</td>
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<td>Ganatra and associates</td>
<td>2000</td>
<td>Case series, 6 patients</td>
<td>Mean age = 26.8</td>
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<tr>
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<td>Van Gelder and associates</td>
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<td>Varicella</td>
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<td></td>
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<td>patient 3</td>
<td>10</td>
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<tr>
<td>Tran</td>
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<td>Case series, 11 patients</td>
<td>36</td>
<td>Neonatal</td>
</tr>
</tbody>
</table>

ARN = acute retinal necrosis; ERM = epiretinal membrane; HSV = herpes simplex virus; OD = right eye; OS = left eye; PCR = polymerase chain reaction; RD = retinal detachment.

Medical histories of patients in our study are remarkable for herpes neonate in one patient (patient 7), high-dose corticosteroids treatment within 2 weeks before the ARN syndrome appeared in three patients (patients 2, 8, and 9), and trauma 1 week before the ARN syndrome in one patient (patient 9). Kychenthal and associates reported the case of a 25-day-old infant who suffered from neonatal herpes infection with encephalitis and retinitis. Herpes simplex virus-2 was confirmed in cerebrospinal fluid and maternal blood. Thompson and associates described three cases of ARN syndrome caused by apparent reactivation of a congenital HSV-2 infection after pericocular trauma. In each case, the ARN syndrome was preceded by trauma. Margolis and associates reported a case of ARN syndrome with serologically confirmed HSV-2 following meningioma excision and high-dose systemic corticosteroids. Rahhal and associates presented a patient with bilateral, biopsy-proven HSV-2-related-ARN syndrome who had undergone craniotomy for removal of parasellar tumor and received dexamethasone. Other findings in medical histories that may be associated with an HSV-2-related ARN syndrome include encephalitis,[12], [24] and [35] cranial surgery,[7] and [35] and ocular trauma.35 Ganatra and associates found that HSV-1-related ARN is likely to be associated with a past or concomitant history of...
encephalitis and HSV-2-related ARN with a past or concomitant history of meningitis.12

Corticosteroids are known to affect cellular immunity, and there is experimental evidence suggesting that corticosteroids alone can induce HSV reactivation in the cornea.36 Cases of ARN syndrome that occurred after systemic corticosteroids39 have been reported, although the role of these medications in HSV reactivation in the retina has not been clearly demonstrated. In two patients (patients 2 and 9), typical ARN syndrome occurred after methylprednisolone administration for chronic panuveitis without retinitis after a broad negative exploration. In these cases, high-dose corticosteroids might play a triggering role in the HSV-2-related ARN syndrome. Another hypothesis is that HSV-2 might be responsible for chronic panuveitis. This has to be confirmed by a prospective study.

Chorioretinal scars have been reported in others cases of HSV-2-related ARN syndrome.[24], [34] and [35] Thompson and associates35 considered it a striking feature of this disease. Because these patients had a history of maternal or congenital herpetic infection, it has been suggested that their disease occurred as recrudescence of congenital herpetic retinitis.35

In our study, three patients (patients 3, 6, and 9) had preexisting, inactive chorioretinal scars upon initial examination. In these patients, PCR analysis was negative for toxoplasmosis, and retinitis resolved under antiviral treatment. In patient 3, the scar might have been due to previous ocular toxoplasmosis. The second patient (patient 6) has a history of neonatal herpes. The third patient (patient 9) had no previous history of herpetic infection. It was noted that bilateral hyperpigmented equatorial lesions might correspond to earlier HSV-2 infection of the retina.40 Although pigmented chorioretinal scars might be a marker of previous herpetic infection, alternative diagnoses have to be ruled out.

Although we had no case of exudative retinal detachment, this feature has been described in HSV-1 or -2-related ARN syndrome.[33], [36] and [41] It has been suggested that exudative retinal detachment early in the course of disease may be helpful in distinguishing HSV from other infectious etiologies.42

In this study and previous reports, history of neonatal herpes, triggering events such as neurosurgery, periocular trauma, high-dose corticosteroids, and chorioretinal scars suggest that HSV-2 retinitis reflects reactivation of HSV-2 infection in the pathogenesis of ARN syndrome.

These observations must be viewed with caution because of a selection bias in a study of cases selected from multiple institutions. The results of our study reveal the need for further investigation about the relationship between age, history, findings of funduscopy, and outcome of the ARN syndrome caused by HSV-2.

References


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