ACUTE RETINAL NECROSIS SYNDROME FOLLOWING CHICKENPOX

SÍNDROME DE NECROSIS RETINIANA AGUDA TRAS PRIMOINFECCIÓN POR VARICELA

BOLÍVAR G\(^1\), GORROÑO MB\(^2\), PAZ J\(^1\), PAREJA J\(^1\)

ABSTRACT

Case report: A 34-year-old male patient developed acute retinal necrosis in his left eye about three weeks after the onset of chickenpox. Systemic anti-viral treatment with intravenous acyclovir (10 mg/kg/8 hours) and systemic corticosteroids (1 mg/kg/day) controlled the retinitis and the patient suffered no loss of visual acuity.

Discussion: Acute retinal necrosis is an unusual complication of chickenpox. A mild form of this entity has been described during the course of primary varicella-zoster infection. Adequate and early therapy during the acute phase of the disease with intravenous acyclovir and systemic corticosteroids is recommended to achieve a satisfactory visual acuity and prevent complications (Arch Soc Esp Oftalmol 2007; 82: 579-582).

Key words: Acute retinal necrosis, chickenpox, treatment.

RESUMEN

Caso clínico: Varón de 34 años que desarrolló una necrosis retiniana aguda en su ojo izquierdo tres semanas después de padecer varicela. Con tratamiento sistémico precoz con aciclovir intravenoso (10 mg/kg/8 horas) y corticoides sistémicos (1 mg/kg/día) la retinitis fue controlada y el paciente no sufrió pérdida de agudeza visual.

Discusión: La necrosis retiniana aguda es una complicación inusual de la varicela que necesita atención. Se ha descrito una forma leve durante el curso de la primoinfección por varicela zoster. Se recomienda un tratamiento adecuado y precoz durante la fase aguda de la enfermedad con aciclovir intravenoso para conseguir una buena agudeza visual y prevenir complicaciones.

Palabras clave: Necrosis retiniana aguda, varicela, tratamiento.

INTRODUCTION

Acute retinal necrosis syndrome (ARN) is a rare entity characterized by necrotizing vaso-occlusive retinitis, retinal arteritis, vitritis, and it often evolves into regmatogenous retinal detachment. It tends to result from the reactivation of viruses belonging to the herpes group and affecting healthy and immunocompromised patients. It rarely occurs during a primo-infection caused by the Varicella-zoster virus, while the present cases were slighter and had a favorable visual prognosis (1-3).
CASE REPORT

A 34-year-old male diagnosed with varicella three weeks earlier, already cured without complications. He checks in the ER reporting a reduction in visual acuity in the left eye.

The ophthalmologic exploration revealed a corrected visual acuity of 1 in the right eye (RE) and 0.6 in the left eye (LE). The slit lamp study showed a 4+ cellular tyndall in the LE, lower keratic precipitates (3+) and no corneal staining with fluorescein, while the RE was normal. Intraocular pressure was 16 mmHg for both eyes.

The LE’s initial funduscopic exploration revealed a slight vitritis (1+) without retinitis spots.

The topical treatment prescribed consisted of corticoids and miotics. Two days later, there was a slight reduction in the cellular tyndall (3+) in the anterior chamber, whereas the eye fundus revealed a peripheral source of necrotizing retinitis in the temporal region associated with a retinal vasculitis.

The patient was admitted and treatment was prescribed including intravenous aciclovir (10 mgr/kg/8 hours), antiaggregation therapy (150 mgr of acetylsalicylic acid every 24 hours) while maintaining the topical treatment. Additionally, prophylactic photocoagulation with argon laser was applied around the retinal necrosis area (figs. 1 and 2).

Forty-eight hours later and after checking the patient’s good response with a reduction in the retinitis spot, 1 mg/kg/d of systemic corticoids was administered, with rapid improvement in visual acuity, decreased vitritis and small-sized retinitis spot. Two weeks later, oral antiviral treatment was prescribed (famciclovir 500 mgr/12 hours), gradually decreasing corticoids by 10 mgr per week. The antiviral treatment was kept at maintenance doses during six weeks, performing regular hemogram and biochemistry checkups in order to detect renal involvement.

Six months after the onset of his condition, the patient remains non-symptomatic, with a visual acuity of 1 in the LE, scarring of retinal lesions and full absence of ocular inflammatory activity in both eyes.

During the patient’s stay in the hospital, a recent infection induced by the Varicella-zoster virus seroconversion was appreciated.

DISCUSSION

Acute retinal necrosis was first defined as a clinical entity in 1971 by Urayama et al. It is characterized by a necrotizing vaso-occlusive retinitis, with a significant inflammatory reaction in the vitreous and anterior chamber, retinal vasculitis and frequent regmatogenous retinal detachments (up to 75 percent), with rapid progression in the absence of treatment. It may affect both healthy and immunocompromised patients. Approximately one third of patients develop bilateral involvement, generally within 6 weeks and 4 months after the emergence of symptoms in the first eye. It is caused by the reactivation of a virus belonging to the herpes group,
mainly the Varicella-zoster virus, simple herpes types 1 and 2 and, rarely by the cytomegalovirus. The treatment consists of the systemic administration of antiviral drugs, corticoids and antiaggregants (4,5).

There is a slighter and less frequent form of acute retinal necrosis associated to the primo-infection caused by the Varicella-zoster virus, usually affecting adult patients, both healthy and immunocompromised patients (1-3). Some cases involving children have been described (2). The symptoms may appear days after diagnosing varicella, generally when the disease has already remitted. The condition appears as a peripheral retinitis progressing at a slow pace, with a moderate inflammatory reaction in the vitreous and anterior chamber, good visual acuity and no retinal detachment. Bilateral involvement is not frequent (it has only been described in one immunocompromised patient) (3).

Some authors state the uncertain role of treatments including aciclovir and corticoids based on the more moderate and, possibly limited, progression of this disease without treatment (1). However, some untreated cases with bad progression have also been reported (3), subsequently its administration is advised.

The case described herein represents a typical acute retinal necrosis after primo-infection induced by the varicella virus, presenting a favorable progression not only because it was probably a slighter form of herpetic infection but also thanks to the early administration of the appropriate systemic treatment.

A more indolent progression may be due to the patient’s good cellular and humoral response to varicella at the time of onset of the retinitis (1), that is, when the skin disorder had already remitted.

Since varicella’s ocular complications are rare, it is important to keep in mind that this clinical entity could be a complication of the disease, since early diagnosis and treatment are crucial for an optimal handling of these patients.

REFERENCES