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Scientific letters

Keratitis due to *Colletotrichum gloeosporioides* and *Herpesvirus* reactivation

Queratiris por Colletotrichum gloeosporioides *y reactivación de* Herpesvirus

Colletotrichum spp. are common plant pathogens worldwide. Fruit rots (anthracnose) are often attributed to *C. gloeosporioides*.[1]

Colletotrichum spp. have been reported as unusual cause of ophthalmic infections in several countries as United States,[2] Japan[3] and India.[4] Recently, two cases have been reported in our country (Spain).[5,6]

We present a case of keratitis due to *C. gloeosporioides* aggravated with herpes virus reactivation and antifungal therapy failure.

A 75-year-old man attended Ophthalmic Outpatient Department (OPD) with a sudden loss of visual acuity after trauma with an orange tree branch (genus *Citrus*). Slit lamp examination revealed an old walleye secondary herpes infection, corneal oedema and high intraocular pressure (IOP 50). Treatment was initiated with antiglaucoma agents, prophylactic antibiotic (tobramycin) and dexamethasone (1 mg/mL plus 3 mg/mL, one drop every 4 h). Ten days later, the patient presented with aggravation. Biomicroscopic examination showed corneal ulcer and associated hypopyon. Corneal scraping of the ulcer was sent for bacterial culture that was negative. The patient was treated with topical application of antiglaucoma agents, moxifloxacin (5 mg/mL, four times a day) and ciprofloxacin ophthalmic ointment (3 mg/g, at night) for 4 weeks. Two weeks later there was no enhancement and descemetocele appeared. Given the history of Herpesvirus infection, real-time PCR detection of *Herpesvirus* DNA was requested (positive to Herpes simplex-1). Topical acyclovir (30 mg/g, five times daily for 1 month) and valacyclovir (1 g every 8 h during 2 months) were added to treatment. Nevertheless, the patient did not respond to therapeutic changes. Corneal scrapings were sent to bacterial and fungal culture and inoculated into usual culture media. Hyphal elements were detected on Gram stain. Thus, the patient received oral (400 mg/12 h loading dose and 200 mg/12 h maintenance dose), topical (10 mg/mL daily for 7 weeks), intravitreal (100 mcg/0.1 mL), and intrastromal (1%) voriconazole treatment.

A rapidly growing fungus produced colonies on Sabouraud-chloramphenicol culture media. Lactophenol-cotton blue (Merck Millipore®, Madrid, Spain) mount of the smear, showed unbranched hyphae with cylindrical conidia typical of the genus *Colletotrichum*. Molecular studies were utilized to species identification.[7] The isolate was identified as *C. gloeosporioides* using the Basic Local Alignment Search Tool (BLAST) program. The nucleotide sequence showed 98% homology match with accession number KC341915.

The patient did not respond to the 7 weeks voriconazole treatment and suffered corneal melting (Fig. 1). Therapeutic penetrating keratoplasty was performed. New fungal recurrence was seen after surgery with melting of donor corneal button. Due to poor prognosis and lack of response to therapy, eye evisceration was performed.

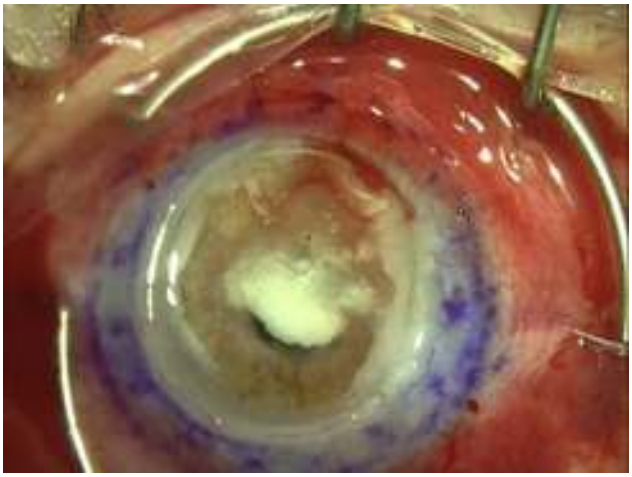

Fig. 1. Image taken during surgery prior to the completion of penetrating keratoplasty. Corneal melting is observed with full involvement of all corneal layers and in all quadrants of the ocular fundus, including severe mixed conjunctival hyperaemia.

Colletotrichum spp. are an uncommon cause of keratitis. The species implicated in human infections include *C. dematium*, *C. coccodes*, *C. graminicola*, *C. gloeosporioides*, *C. crassipes* and *C. truncatum*.[8] They have mainly been involved in keratitis, although subcutaneous and systemic infections among immunosuppressed patients have also been reported.[9,10] A Pubmed search using "Keratitis" and "*Colletotrichum*" as keywords revealed 18 reports, including 64 patients with ophthalmic infections. *Colletotrichum* spp. eye infections are increasingly reported, even though the rate of isolation is still low (from 1.9% to 2.8%).[2,4]

Fernandez et al. described principal risk factors associated to *Colletotrichum* keratitis; they reported that ocular trauma followed by diabetes mellitus and corticosteroid use were the principal characteristics of a series of patients with *Colletotrichum* keratitis.[2] Our patient fulfilled these main risk factors. The route of entry was through trauma with an orange tree branch, similar to that of other cases reported in our country.[5,6]

Due to the difficulty of morphological identification, molecular techniques are the most suitable method for isolates identification to the species level.[8]

There is a lack of consensus in the literature regarding the optimal therapy against *Colletotrichum* spp. ophthalmic infection. Most articles reported successful therapy with topical natamycin.[2,4] Combination therapy with natamycin plus other antifungal agents have shown resolve on infection in several cases too.[3,11] Our patient

received oral, topical, intravitreal and intrastromal voriconazole showing no improvement of eye lesion, as well as Navalpotro et al. reported recently.[5] The unsuccessful treatment and fatal outcome of our patient may possibly be due to diagnostic and surgical delay procedures along with Herpes simplex-1 virus reactivation in an already compromised cornea. Combination therapy may be more effective than monotherapy. Moreover, natamycin and voriconazole could be a good treatment as suggested by Shiraishi et al.[3]

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Pileflebitis por *Aeromonas caviae* secundaria a colecistitis aguda

Pylephlebitis due to Aeromonas caviae *secondary to acute cholecystitis*

Sr. Editor:

El género *Aeromonas* está formado por bacilos gramnegativos móviles, ubicuos, de reservorio acuático, heterótrofos, oxidasa e indol positivos, anaerobios facultativos y productores de diversas β-lactamasas (cefalosporinasas de clase C, penicilinasas de clase D y metalo-β-lactamasas) y factores de virulencia (enterotoxinas, hemolisinas, adhesinas y verotoxinas, entre otros)[1]. La infección causada por estos microorganismos es más frecuente en áreas de clima templado y subtropical, en especial tras el contacto con agua dulce o salobre, mordedura por animales acuáticos o ingesta de alimentos contaminados[1,2]. Tres de las 21 especies clasificadas en este género son responsables del 85% de los casos de infección: *A. hydrophila*, *A. caviae* y *A. veronii* biovar *sobria*[1-3]. El espectro clínico es muy variable, abarcando desde gastroenteritis, infección de partes blandas o neumonía aspirativa en ahogados, hasta ejemplos ocasionales de empiema, artritis séptica, endocarditis, meningitis o infección del tracto urinario[1-3]. La inmunosupresión y ciertas comorbilidades (neoplasia hepatobiliar o hepatopatía crónica) han sido identificadas como factores de riesgo para el desarrollo de bacteriemia por *Aeromonas*[2,3]. La tromboflebitis séptica de la vena porta, también denominada pileflebitis, es una complicación asociada a procesos de localización intraabdominal y pélvica en el territorio de drenaje del sistema venoso portal[4,5]. Presentamos a continuación un caso de pileflebitis por *A. caviae* secundaria a colecistitis aguda.

Se trata de un varón de 85 años con diagnósticos previos de hipertensión arterial, deterioro cognitivo leve y neoplasia primaria múltiple (adenocarcinoma de próstata bajo bloqueo androgénico, adenocarcinoma gástrico tratado mediante gastrectomía total, y adenocarcinoma multifocal de colon sometido a hemicolectomía derecha) en remisión completa y sin datos de recidiva. Se encontraba en tratamiento con sertralina, lormetazepam y suplementos de vitamina B_{12}. Consultó por un cuadro de 24 h de evolución consistente en sensación distérmica no termometrada, postración y sensación de mareo. La exploración física mostró una temperatura axilar de 38,3 °C, presión arterial de 162/58 mmHg, frecuencia cardíaca de 105 lpm, desorientación, tinte ictérico y una masa dolorosa de consistencia elástica en el hipocondrio derecho, sin datos de reacción peritoneal y dudosa positividad del signo de Murphy (1 punto en el *score* de bacteriemia de Pitt). Analíticamente destacaba la presencia de leucocitosis ($14,5 \times 10^3$ células/μl con 92% de neutrófilos) asociada a elevación de reactantes de fase aguda (proteína C reactiva 7,5 mg/dl [rango normal: 0,1-0,5]) y alteración de las pruebas de función hepática (glutamato-piruvato transaminasa [GPT] 531 UI/l, glutamato-oxalacetato transaminasa [GOT] 565 UI/l, gamma-glutamil transpeptidasa [GGT] 269 UI/l, fosfatasa alcalina 284 UI/l, lactato deshidrogenasa [LDH] 475 UI/l, bilirrubina total 6,1 mg/dl y actividad de protrombina 66%). Ante la sospecha de un foco de infección en la vía biliar fue solicitada una ecografía abdominal urgente, que reveló una marcada dilatación de la vesícula biliar (13,5 × 6,5 cm) con paredes engrosadas, colelitiasis y barro biliar en su interior, signos compatibles con colecistitis aguda, y una trombosis de la rama portal izquierda. El estudio fue completado mediante una tomografía computarizada (TC) abdominal, que demostró la existencia de una trombosis de las ramas principales de la vena mesentérica superior con extensión a la vena porta principal y sus ramas, así como múltiples lesiones hipodensas en el parénquima hepático sugerentes de microabscesos y una marcada distensión de la vesícula biliar) y del conducto cístico con engrosamiento difuso de su pared (fig. 1). El diagnóstico de pileflebitis en el contexto de colecistitis aguda con hidrops vesicular fue confirmado mediante una resonancia magnética nuclear hepática. Tras la obtención de 2 sets de hemocultivos