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Oropouche virus meningoencephalitis. Case presentation

Meningoencefalitis por virus Oropouche. Presentación de caso

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RESUMEN

Introducción: La fiebre de Oropouche, causada por el *orthobunyavirus* Oropouche, es una arbovirosis transmitida por insectos hematófagos. Aunque suele ser leve, se han descrito manifestaciones neurológicas graves. En 2024, Cuba reportó transmisión activa en todas sus provincias. **Objetivo:** Describir las características clínicas, diagnósticas y evolutivas de un caso de meningoencefalitis por virus Oropouche con desenlace fatal. **Presentación del caso:** Varón de 36 años, procedente de zona rural, con antecedente de hipospadias corregido, amaurosis del ojo derecho de 10 días de evolución y crianza de palomas. Ingresó por cefalea occipital intensa, vómitos y mareos. Presentó síndrome meníngeo, fiebre, deterioro neurológico progresivo, shock, oliguria, hipopotasemia y paro cardiorrespiratorio. La PCR confirmó Oropouche, y la autopsia reveló edema cerebral severo secundario a meningoencefalitis. **Conclusiones:** El virus Oropouche puede causar meningoencefalitis grave y mortal en pacientes jóvenes sin comorbilidades. Este caso resalta la necesidad de incluir esta arbovirosis en el diagnóstico diferencial de síndromes meníngeos en áreas endémicas y de realizar confirmación virológica precoz.

ABSTRACT

Introduction: Oropouche fever, caused by the Oropouche orthobunyavirus, is an arboviral disease transmitted by blood-sucking insects. Although usually mild, severe neurological manifestations have been described. In 2024, Cuba reported active transmission in all its provinces. **Objective:** To describe the clinical, diagnostic, and evolutionary characteristics of a case of Oropouche virus meningoencephalitis with a fatal outcome. **Case presentation:** A 36-year-old male from a rural area with a history of surgically corrected hypospadias, right eye amaurosis of 10 days' duration, and pigeon rearing. He presented with severe occipital headache, vomiting, and dizziness. He developed meningeal signs, fever, progressive neurological deterioration, shock, oliguria, hypokalemia, and cardiorespiratory arrest. PCR confirmed Oropouche, and autopsy revealed severe cerebral edema secondary to meningoencephalitis. **Conclusions:** Oropouche virus can cause severe and fatal meningoencephalitis in young patients without comorbidities. This case highlights the need to include this arbovirosis in the differential diagnosis of meningeal syndromes in endemic areas and to perform early virological confirmation.

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INTRODUCTION

Oropouche fever is an emerging arbovirosis caused by the Oropouche orthobunyavirus (OROV), transmitted primarily by biting midges of the genus *Culicoides* and, in Cuba, also by *Culex quinquefasciatus*. Although it is classically considered a self-limited febrile illness, in recent years an increase in severe neurological forms, such as meningitis, encephalitis, or meningoencephalitis, has been documented, which can occur in up to 4 % of patients after the initial febrile phase [(1)]. Since its identification in Trinidad and Tobago in 1955, OROV has circulated in Central and South America. In 2024, Cuba reported active transmission in all 15 provinces, with more than 110 municipalities affected [(2)].

The classic clinical picture includes fever, intense occipital headache, myalgias, arthralgias, dizziness, nausea, and vomiting. However, presentation as severe meningoencephalitis with multisystem involvement and fatal outcome is exceptional and has been scarcely documented [(3)]. The present case provides a valuable clinical lesson, as it describes a young patient without known immunosuppression who progressed from an initial meningeal syndrome to shock, multiple organ dysfunction, and death, despite management in intermediate care.

The objective of this report is to describe the clinical, diagnostic, and evolutionary characteristics of a case of Oropouche virus meningoencephalitis with a fatal outcome, in order to alert clinicians to the possible severity of this emerging arbovirosis.

CASE PRESENTATION

A 36-year-old male patient, from a rural area, with a personal history of surgically corrected hypospadias in childhood, amaurosis of the right eye of 10 days' evolution, and pigeon rearing. He reported no other pathological history or immunosuppression.

He presented to the emergency department accompanied by family members due to a 10-day history of headache, located in the occipital region, of high intensity, with frontal irradiation, without relief with analgesics. It was accompanied by two episodes of vomiting with food remnants, frequent dizziness, and elevated blood pressure values (150/90 mmHg) documented at the health area. He was admitted to the Internal Medicine service of the "Dr. Agostinho Neto" General Teaching Hospital with the diagnostic impression of meningeal syndrome and suspected leptospirosis. Due to lack of improvement, he was transferred to the Intermediate Care Unit (UCIM), where he

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remained until his death. Data were obtained through review of the medical record.

Physical examination on admission:

Vital signs: blood pressure 120/70 mmHg, heart rate 70 bpm, respiratory rate 18 rpm, temperature 36,8 °C.

Nervous system: conscious, oriented, with positive meningeal signs (Kernig and Brudzinski). Cranial nerves without alterations.
Rest of the examination: no relevant findings.

Hospital evolution

Day 1: Lumbar puncture was performed: cloudy, colorless cerebrospinal fluid (CSF), with undetectable glucose, 296 cells × 10^6/L (lymphocytic predominance), positive Pandy test, and crenated red blood cells. Viral meningoencephalitis was diagnosed.

Days 2–3: Initial improvement of headache, no fever, Glasgow 15. On the third day, recurrence of headache requiring metamizole (dipyrone).

Day 4: Deterioration: disorientation, psychomotor agitation, Glasgow 13. Abdominal ultrasound showed moderate intra-abdominal free fluid. Urethrovesical catheterization was requested due to meatal stenosis.

Day 5: Transfer to UCIM due to fever of 40 °C, tachycardia (105 bpm), tachypnea (30 rpm), Glasgow 9, and persistence of meningeal signs.

Day 6: Hypotension (BP 110/70 mmHg), tachypnea (36 rpm), oliguria (0,7 ml/h), hypokalemia (3,2 mmol/L), and respiratory alkalosis.

Day 7: Febrile (38 °C), Glasgow 13, neck stiffness, without improvement.

Day 8: Afebrile, but with signs of dehydration, Glasgow 13. He suffered cardiorespiratory arrest that did not respond to resuscitation maneuvers.

Complementary studies (performed between days 1–3):

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Hemogram: leukocytes 17,0 × 10^9/L (neutrophils 82 %, lymphocytes 13 %), hematocrit 0,45 L/L.

Blood gas: respiratory alkalosis.

Peripheral blood smear: Döhle bodies, band cells 7 %, metamyelocytes 5 %, myelocytes 2 %.

Polymerase chain reaction (PCR): Oropouche virus RNA in clinical sample; negative for dengue, Zika, and chikungunya.

Electrocardiogram, chest X-ray, and computed axial tomography of the skull: normal.

Abdominal ultrasound: moderate intra-abdominal free fluid.

Treatment

Intravenous hydration, acyclovir (given suspicion of herpetic encephalitis), hydrocortisone, paracetamol, fraxiparin, mannitol, and ceftriaxone (doses and regimens not detailed due to record limitations).

Autopsy results:

- Direct cause of death: severe cerebral edema.
- Intermediate cause: intracranial hypertension.
- Basic cause: Oropouche virus meningoencephalitis.

Informed consent: Written informed consent was obtained from the closest family member for the publication of this case, guaranteeing the anonymization of all identifying data. The document is in the possession of the authors and available to the editorial committee.

DISCUSSION

The presented case documents Oropouche virus meningoencephalitis with a fatal outcome, an exceptionally severe clinical form. The definitive anatomopathological diagnosis established severe cerebral edema secondary to intracranial hypertension as the direct cause of death, and Oropouche meningoencephalitis as the basic cause. In the patient's epidemiological context (rural origin, pigeon rearing) and the active outbreak in the province between April and June 2024, OROV infection constitutes the most plausible etiology, confirmed by specific PCR.

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Oropouche meningoencephalitis is rare, but possible. Wesselmann and collaborators point out that the disease classically presents with fever, headache, arthralgias, and myalgias, with spontaneous recovery in most cases; however, up to 4 % develop neurological manifestations [1]. Vernal and collaborators described patients with OROV meningoencephalitis in Brazil, all with favorable evolution [4]. Unlike those reports, our case presented shock, multiple organ dysfunction, and death, highlighting the potential severity of this arbovirosis.

Differential diagnosis with other arboviroses is complex in areas of co-circulation. Myalgias and arthralgias are less frequent in Oropouche than in dengue, while retro-orbital pain, typical of dengue, is rare in Oropouche [5]. Zika presents with low-grade fever, rash, and conjunctivitis, absent in our patient. PCR allowed ruling out dengue, Zika, and chikungunya, confirming OROV as the causative agent.

The presence of intra-abdominal free fluid, hypotension, and oliguria suggests a component of septic shock or multiple organ dysfunction, possibly related to the systemic inflammatory response secondary to intracranial hypertension. Da Silva Menegatto and collaborators point out that abdominal pain is frequent in Oropouche fever, due to greater abdominal inflammation [6]. This finding was corroborated in our patient.
Given the history of pigeon rearing, meningeal cryptococcosis was considered as a differential diagnosis; however, the absence of known immunosuppression and the acute evolution make this etiology less likely [7]. Virological confirmation by PCR also ruled out leptospirosis, initially suspected due to the rural setting.

The amaurosis of the right eye, present 10 days before admission, could represent an early neurological manifestation of OROV (involvement of the optic nerve or central visual pathway) or an independent finding. No previous reports of amaurosis associated with Oropouche were found, so this case suggests the need to investigate possible ocular complications in future outbreaks.

From a pathophysiological standpoint, the Oropouche virus interferes with ionic transport and neuronal synaptic regulation, and induces downregulation of type 1 interferon signaling, which could explain its neuroinvasive capacity [6]. Furthermore, OROV has been shown to induce oxidative stress in organs such as the liver and spleen, which could contribute to the multisystem involvement observed [8].

Limitations of the case: This is a single report, so the findings are not generalizable. A complete autopsy of the peripheral nervous

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system and histopathological study of the eyeball were not performed to confirm the cause of amaurosis. Inflammatory cytokines were also not measured to characterize the immune response.

Lessons learned: Oropouche meningoencephalitis can present with rapidly progressive evolution toward shock and death, even in young patients without comorbidities. In the presence of a meningeal syndrome with neurological deterioration and multisystem involvement in endemic areas, OROV should be included in the differential diagnosis, beyond the classic arboviroses. Early diagnostic confirmation by PCR is essential to guide management and avoid unnecessary treatments.

CONCLUSIONS

Oropouche virus meningoencephalitis can present with rapidly progressive neurological deterioration, shock, and death, even in young patients without comorbidities. This case highlights the need to include OROV in the differential diagnosis of severe meningeal syndromes in endemic areas, and to implement early virological confirmation to optimize clinical management.

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AUTHORSHIP STATEMENT

RHHK: Conceptualization, investigation, methodology, validation, writing the original draft, review, editing.

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CONFLICT OF INTEREST

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