

In summary, this case highlights diagnostic challenges in endemic fungal infections and rare drug-associated hyperpigmentation. Diagnostic stewardship, particularly in endemic regions, is critical to reduce delays in appropriate antifungal therapy and minimize unnecessary antimicrobial exposure.

The patient provided written informed consent for the publication of her photograph.

About the Author

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Jorge Lobo's Disease in Child with Tick Exposure, Brazil

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Jorge Lobo's disease (JLD), caused by *Paracoccidioides lobogeorgii*, primarily affects inhabitants of the Amazon Forest. We report a 9-year-old boy in Brazil who had JLD diagnosed after a tick bite. The rarity of pediatric cases likely reflects surveillance gaps. Increased clinical awareness is crucial for early JLD detection and intervention, especially in endemic regions.

Jorge Lobo's disease (JLD) is a chronic infection caused by the noncultivable fungus *Paracoccidioides lobogeorgii* (previously *Lacazia loboi*) (1). JLD primarily affects persons in the Amazon Basin and parts of Central America, and most reported cases originate from Acre state in Brazil (2). Infection is thought to occur when skin trauma involving contaminated vegetation or arthropods permits direct fungal inoculation or enables pathogen implantation from secondary environmental sources (3). Initial lesions can appear as subtle papules or nodules, often on the ears, arms, or legs, and might evolve into extensive verrucous plaques (3,4). The hypothesis of environmental transmission is supported by the cessation of new cases after relocation of the Caiabi Indigenous population from endemic to nonendemic areas.

JLD disproportionately affects men involved in rural occupations and forest extractivism. Left undiagnosed, JLD can cause visible disfigurement, functional impairment, and psychosocial stigma, which can undermine the ability to work and can contribute to social isolation. Although few pediatric cases have been documented, the long disease course, which can persist for decades, supports the hypothesis of early-life acquisition and delayed clinical recognition (4–6). We describe a pediatric male patient with JLD, providing insight into early acquisition and clinical appearance.

In February 2018, a child from a forested area in Brasília, Acre state, Brazil, was seen at the Serviço Estadual de Dermatologia do Acre (Rio Branco, Brazil) for a single lesion on his right ear. His mother, who frequently brought him to forest work areas, said that a tick had been attached at the site for 2 days in 2017. Initial treatment with topical dexamethasone (1 mg/g) was ineffective. Over 6 months, the lesion evolved from mild inflammation to a nodular lesion. One year later, a dermatologist suspected JLD on the basis of a solitary indurated lesion on the ear (Figure 1, panel A). Histopathology confirmed the JLD diagnosis, revealing granulomatous inflammation with spherical, double-walled yeast cells in branching chains (Figure 1, panel B) and prominent histiocytes and

multinucleated giant cells (Figure 1, panel C).

Treatment began with pediatric multidrug therapy for multibacillary leprosy, as previously described (7): monthly doses of rifampin (300 mg), clofazimine (150 mg), and dapsone (50 mg), combined with daily dapsone (50 mg) and clofazimine (50 mg on alternate days) for 2 years, and quarterly follow-up visits (7). After 4 months, lesion atrophy made surgical excision possible. By November 2020, the patient was lesion-free but was lost to follow-up. In August 2022, during a health outreach mission for leprosy surveillance in the Amazon, the patient was relocated, and a small recurrent lesion on the pinna of the same ear was observed and excised. Itraconazole (200 mg/d) was prescribed for 3 months, but adherence to follow-up consultations was irregular. By June 2024, a 5-cm lesion recurred at the same site. A novel biopsy confirmed JLD recurrence showing chronic granulomatous dermatitis with multinucleated giant cells, fibrotic stroma, and yeast-like cells (Figure 1, panel D). Treatment with itraconazole continued, along with regular medical monitoring.

JLD in children is underreported, although evidence suggests many cases originate in childhood (8,9). JLD progression, often spanning decades, underscores the need for early detection in endemic

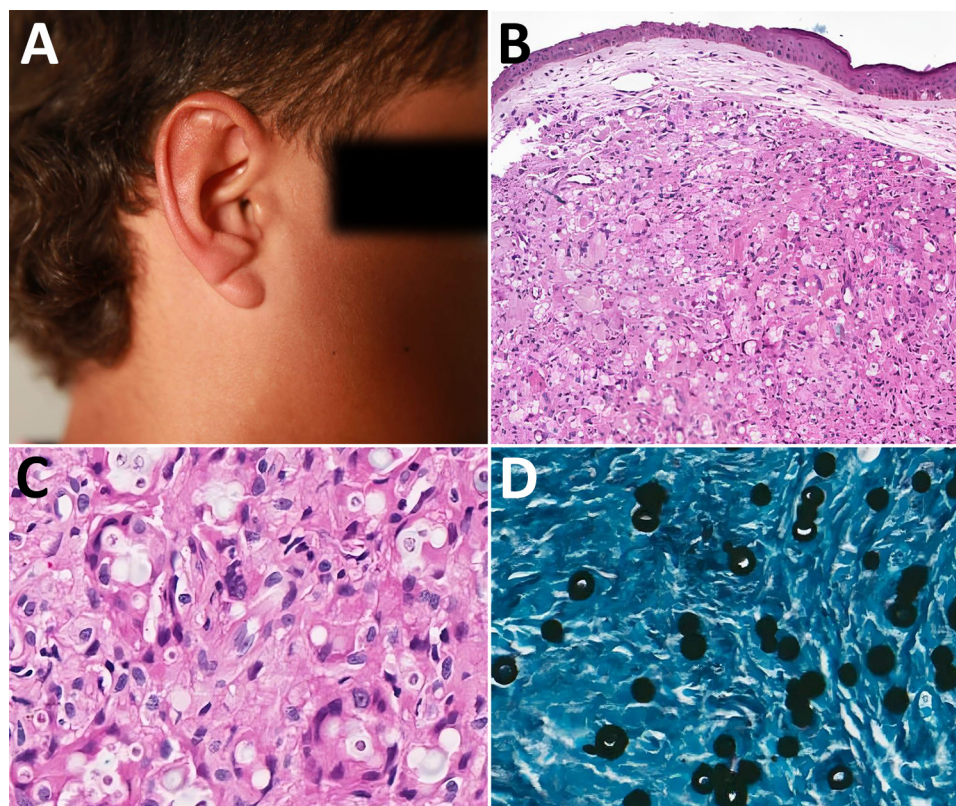


Figure 1. Nodule and histopathology in a case of Jorge Lobo's disease in child with tick exposure, Brazil. A) Single granulomatous lesion on the right ear, which was surgically excised and histologically examined in 2018. B, C) Hematoxylin and eosin stain of nodule showing cells of the mononuclear phagocyte system, including monocytes, macrophages, epithelioid cells, and multinucleated giant cells, revealing granulomatous inflammation with spherical, double-walled yeast cells in branching chains (B) and prominent histiocytes and multinucleated giant cells (C), which are predominantly dense with areas of mild neovascularization, stromal hyalinization, and a slight infiltrate of lymphocytes and neutrophils. D) Grocott–Gomori methenamine silver stain of recurrent nodule from 2024 in which yeast-like fungal structures are diffusely distributed throughout the stroma and in the cytoplasm of phagocytic cells.



Figure 2. Location of a case of Jorge Lobo's disease in child with tick exposure, Brazil. The patient resided near the Brazil–Bolivia border and was diagnosed in Brasiléia, Acre state, Brazil. Brasiléia is situated between the municipalities of Xapuri and Epitaciolândia in Acre, also known as Brasiléia–Epitaciolândia–Xapuri immediate region. This zone borders Bolivia within a dense rainforest region of the western Amazon and is marked by rubber plantations and extractive activities.

regions. Long latency periods support the hypothesis of early-life acquisition (3,4). The patient's slow lesion development, despite a year without treatment, further suggests a gradual pediatric course. Timely diagnosis combined with surgical and antifungal therapy can prevent complications (5,10). JLD elicits a robust inflammatory response with histiocytic infiltrates, multinucleated giant cells, and fungal elements, progressing to fibrosis, chronicity, and immune containment. In this case, a tick bite might have led to fungal entry, as observed in other reports linking JLD onset to arthropod trauma (3). Despite several attempts, molecular confirmation (rDNA sequencing) failed because of poorly preserved paraffin-embedded material. Histopathological examination confirmed the diagnosis.

Brasiléia is a municipality in western Acre state, Brazil, bordering Bolivia. It falls under the Brasiléia–Epitaciolândia–Xapuri immediate region (Figure 2), an area marked by rubber plantations and extractive activities, which might increase environmental exposure. Because river transport is seasonal, remote communities face year-round delays in healthcare; floods during the wet season wash out roads and make navigation hazardous, whereas low water in the dry season strands boats and slows travel.

In summary, treatment for JLD remains empirical, and no standardized protocols are available (4,5). This case illustrates a 9-year disease course marked by relapses and partial responses and echoes adult reports of forest exposure during childhood, suggesting underdiagnosed childhood infections. Polychemotherapy regimens used for multibacillary leprosy have shown efficacy in localized JLD cases, although prolonged treatment is often necessary (7). In this case, therapy lasted 2 years and was complicated by irregular follow-up. Itraconazole regimens in adults typically span 12–24 months (5), but data on pediatric treatments are scarce. The patient's rediscovery during a targeted field mission highlights the need to strengthen active surveillance and outreach in endemic forested areas to enable early detection, timely treatment, and improved management and to mitigate the long-term effect of JLD on at-risk populations.

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Neonatal Gonococcal Conjunctivitis Caused by Fluoroquinolone-Resistant *Neisseria gonorrhoeae*

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Prophylaxis for ophthalmia neonatorum remains in use despite decreased incidence of the condition. We report a breakthrough case of neonatal conjunctivitis in Japan caused by a levofloxacin-resistant *Neisseria gonorrhoeae* bacteria strain, co-infected with *Chlamydia trachomatis* bacteria. This case highlights failures in screening, prophylaxis, and treatment, underscoring the need to reassess prevention strategies.

Since the introduction of prophylactic ophthalmic solutions by Carl Credé in 1881, the incidence of neonatal gonococcal conjunctivitis has declined markedly (1,2). Over time, the agents used for ocular prophylaxis have shifted from silver nitrate to erythromycin or tetracycline ophthalmic ointments and povidone/iodine (3).

In Japan, erythromycin/colistin ophthalmic formulations had been widely used since 1970. However, production of erythromycin/colistin preparations was discontinued in 2015, prompting some institutions to adopt fluoroquinolone-based ophthalmic agents for neonatal prophylaxis. Although fluoroquinolone-resistant *Neisseria gonorrhoeae* bacteria strains have been reported in adults, neonatal infections with such strains remain rare (2). We describe a breakthrough case of neonatal gonococcal conjunctivitis caused by a fluoroquinolone-resistant *N. gonorrhoeae* strain and further complicated by concurrent *Chlamydia trachomatis* bacteria infection. This case highlights the need to reevaluate current strategies for preventing and managing neonatal conjunctivitis.

In 2023, a 12-day-old female infant was brought to a hospital in Shizuoka, Japan, where she was observed to have purulent ocular discharge and peri-orbital swelling. She was born at full-term through spontaneous vaginal delivery. The mother had a negative *C. trachomatis* nucleic acid amplification test at 12 weeks' gestation; however, no screening for *N. gonorrhoeae* was performed. The infant received prophylactic levofloxacin ophthalmic solution immediately after birth. From day 4 of life, purulent ocular