

Cat Scratch Disease Presenting With Concurrent Pityriasis Rosea in a 10-Year-Old Girl

Shane M. Swink, DO, MS; Luther Pat Rhodes, MD; Joshua Levin, MD

PRACTICE POINTS

- Dermatologists should familiarize themselves with the physical examination findings of cat scratch disease.
- There are numerous clinical variants and triggers of pityriasis rosea (PR).
- There may be a new infectious trigger for PR, and exposure to cats prior to a classic PR eruption should raise one's suspicion as a possible cause.

To the Editor:

Cat scratch disease (CSD) is caused by *Bartonella henselae* and *Bartonella clarridgeiae* bacteria transferred from cats to humans that results in an inflamed inoculation site and tender lymphadenopathy. Pityriasis rosea (PR) and PR-like eruptions are self-limited, acute exanthems that have been associated with infections, vaccinations, and medications. We report a case of PR occurring in a 10-year-old girl with CSD, which may suggest an association between the 2 diseases.

A 10-year-old girl who was otherwise healthy presented in the winter with a rash of 5 days' duration. Fourteen days prior to the rash, the patient reported being scratched by a new kitten and noted a pinpoint "puncture" on the left forearm that developed into a red papule over the following week. Seven days after the cat scratch, the patient experienced pain and swelling in the left axilla. Approximately

1 week after the onset of lymphadenopathy, the patient developed an asymptomatic rash that started with a large spot on the left chest, followed by smaller spots appearing over the next 2 days and spreading to the rest of the trunk. Four days after the rash onset, the patient experienced a mild headache, low-grade subjective fever, and chills. She denied any recent travel, bug bites, sore throat, and diarrhea. She was up-to-date on all vaccinations and had not received any vaccines preceding the symptoms. Physical examination revealed a 2-cm pink, scaly, thin plaque with a collarette of scale on the left upper chest (herald patch), along with multiple thin pink papules and small plaques with central scale on the trunk (Figure 1). A pustule with adjacent linear erosion was present on the left ventral forearm (Figure 2). The patient had a tender subcutaneous nodule in the left axilla as well as bilateral anterior and posterior cervical-chain subcutaneous tender nodules. There was no involvement of the palms, soles, or mucosae.

The patient was empirically treated for CSD with azithromycin (200 mg/5 mL), 404 mg on day 1 followed by 202 mg daily for 4 days. The rash was treated with hydrocortisone cream 2.5% twice daily for 2 weeks. A wound culture of the pustule on the left forearm was negative for neutrophils and organisms. Antibody serologies obtained 4 weeks after presentation were notable for an elevated *B henselae* IgG titer of 1:640, confirming the diagnosis of CSD. Following treatment with azithromycin and hydrocortisone, all of the patient's symptoms resolved after 1 to 2 weeks.

From Lehigh Valley Health Network, Allentown, Pennsylvania. Drs. Swink and Levin also are from Advanced Dermatology Associates, Ltd, Allentown. The authors report no conflict of interest.

Correspondence: Shane M. Swink, DO, MS, Division of Dermatology, Lehigh Valley Health Network, 1259 S Cedar Crest Blvd, Ste 100, Allentown, PA 18103 (shanesw@pcom.edu).

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FIGURE 1. A, A 2-cm pink, scaly, thin plaque with collarette of scale on the left upper chest as well as multiple pink papules with central scale on the chest. B, Multiple pink papules with central scale were present on the upper back.

Cat scratch disease is a zoonotic infection caused by the bacteria *B henselae* and the more recently described pathogen *B clarridgeiae*. Cat fleas spread these bacteria among cats, which subsequently inoculate the bacteria into humans through bites and scratches. The incidence of CSD in the United States is estimated to be 4.5 to 9.3 per 100,000 individuals in the outpatient setting and 0.19 to 0.86 per 100,000 individuals in the inpatient setting.¹ Geographic variance can occur based on flea populations, resulting in higher incidence in warm humid climates and lower incidence in mountainous arid climates. The incidence of CSD in the pediatric population is highest in children aged 5 to 9 years. A national representative survey (N=3011) from 2017 revealed that 37.2% of primary care providers had diagnosed CSD in the prior year.¹

Classic CSD presents as an erythematous papule at the inoculation site lasting days to weeks, with progression to tender lymphadenopathy lasting weeks to months. Fever, malaise, and chills also can be seen. Atypical CSD occurs in up to 24% of cases in immunocompetent patients.¹



FIGURE 2. A singular pustule with an adjacent linear erosion on the left ventral forearm.

Atypical and systemic presentations are varied and can include fever of unknown origin, neuroretinitis, uveitis, retinal vessel occlusion, encephalitis, hepatosplenic lesions, Parinaud oculoglandular syndrome, osteomyelitis, and endocarditis.^{1,2} Atypical dermatologic presentations of CSD include maculopapular rash in 7% of cases and erythema nodosum in 2.5% of cases, as well as rare reports of cutaneous vasculitis, urticaria, immune thrombocytopenic purpura, and papuloedematous eruption.³ Treatment guidelines for CSD vary widely depending on the clinical presentation as well as the immunocompetence of the infected individual. Our patient had limited regional lymphadenopathy with no signs of dissemination or neurologic involvement and was successfully treated with a 5-day course of oral azithromycin (weight based, 10 mg/kg). More extensive disease such as hepatosplenic or neurologic CSD may require multiple antibiotics for up to 6 weeks. Alternative or additional antibiotics used for CSD include rifampin, trimethoprim-sulfamethoxazole, ciprofloxacin, doxycycline, gentamicin, and clarithromycin. Opinions vary as to whether all patients or just those with complicated infections warrant antibiotic therapy.⁴⁻⁶

Pityriasis rosea is a self-limited acute exanthematous disease that is classically associated with a systemic reactivation of human herpesvirus (HHV) 6 and/or HHV-7. The incidence of PR is estimated to be 480 per 100,000 dermatologic patients. It is slightly more common in females and occurs most often in patients aged 10 to 35 years.⁷ Clinically, PR appears with the abrupt onset of a single erythematous scaly patch (termed *the herald patch*), followed by a secondary eruption of smaller erythematous scaly macules and patches along the trunk's cleavage lines. The secondary eruption on the back is sometimes termed a *Christmas or fir tree pattern*.^{7,8}

In addition to the classic presentation of PR, there have been reports of numerous atypical clinical presentations. The herald patch, which classically presents on the trunk, also has been reported to present on the extremities; PR of the extremities is defined by lesions that appear as large scaly plaques on the extremities only. Inverse PR presents with lesions occurring in flexural areas and acral surfaces but not on the trunk. There also is an acral PR variant in which lesions appear only on the palms, wrists, and soles. Purpuric or hemorrhagic PR has been described and presents with purpura and petechiae with or without collarettes of scale in diffuse locations, including the palate. Oral PR presents more commonly in patients of color as erosions, ulcers, hemorrhagic lesions, bullae, or geographic tongue. Erythema multiforme-like PR appears with targetoid lesions on the trunk, face, neck, and arms without a history of herpes simplex virus infection. A large pear-shaped herald patch has been reported and characterizes the gigantea PR of Darier variant. Irritated PR occurs with typical PR findings, but afflicted patients report severe pain and burning with diaphoresis. Relapsing PR can occur within 1 year of a prior episode of PR and presents without a herald patch. Persistent PR is defined by PR lasting more than 3 months, and most reported cases have included oral lesions. Finally, other PR variants that have been described include urticarial, papular, follicular, vesicular, and hypopigmented types.⁷⁻⁹

Furthermore, there have been reports of multiple atypical presentations occurring simultaneously in the same patient.¹⁰ Although PR classically has been associated with HHV-6 and/or HHV-7 reactivation, it has been reported with a few other clinical situations and conditions. Pityriasislike eruption specifically refers to an exanthem secondary to drugs or vaccination that resembles PR but shows clinical differences, including diffuse and confluent dusky-red macules and/or plaques with or without desquamation on the trunk, extremities, and face. Drugs that have been implicated as triggers include ACE inhibitors, gold, isotretinoin, nonsteroidal anti-inflammatory agents, omeprazole, terbinafine, and tyrosine kinase inhibitors. Smallpox, tuberculosis, poliomyelitis, influenza, diphtheria, tetanus, hepatitis B virus, pneumococcus, papillomavirus, yellow fever, and pertussis vaccinations also have been associated with PR.^{7,11,12} Additionally, PR has been reported to occur with active systemic infections, specifically H1N1 influenza, though it is rare.¹³ Because of its self-limited course, treatment of PR most often involves only reassurance. Topical corticosteroids may be appropriate for pruritus.^{7,8}

Pediatric health care providers including dermatologists should be familiar with both CSD and PR because they are common diseases that more often are encountered in the pediatric population. We present a unique case of CSD presenting with concurrent PR, which highlights a potential new etiology for PR and a rare cutaneous manifestation of CSD. Further investigation into a possible relationship between CSD and PR may be warranted. Patients with any signs and symptoms of fever, tender lymphadenopathy, worsening rash, or exposure to cats warrant a thorough history and physical examination to ensure that neither entity is overlooked.

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