Chest-Wall Abscess Due to Cat-Scratch Disease (CSD) in an Adult with Antibodies to Bartonella claridgeiae: Case Report and Review of the Thoracopulmonary Manifestations of CSD

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We describe a patient who presented with a massive chest-wall abscess after a severe debilitating illness that lasted 3 months. Steroid therapy, administered for 4 weeks, masked the slow development of an extensive axillary and chest-wall abscess. After multiple negative tests, the patient’s prolonged illness was diagnosed as cat-scratch disease (CSD). An indirect fluorescent antibody test revealed that two convalescent serum samples were positive for IgG to Bartonella claridgeiae, but no other Bartonella species. We also review 12 cases of severe chest and pulmonary disease due to CSD that were reported in the English-language literature. Thoracopulmonary findings associated with CSD, pathogenic mechanisms of bartonella infections, diagnostic criteria, and management of CSD are presented.

Cat-scratch disease (CSD) is a relatively common, self-limited, benign infectious disease reported in persons of all ages. It occurs worldwide, and in the United States >80% of patients with this disease are <21 years of age. It is the most common cause of chronic (>3 weeks) infectious lymphadenopathy that develops after contact with and/or scratches by cats in children and young adults [1]. A primary dermal inoculation papule or pustule or an ocular granuloma is found in 60%–90% of patients [2]. Epidemiological, serological, and PCR studies have revealed that Bartonella henselae is the etiologic agent in the majority of cases [1, 3]. Recently, the first case of typical CSD due to Bartonella claridgeiae was reported in a veterinarian [4]. In 1988 Aafia felis was isolated and confirmed, on the basis of Koch’s postulates, to be the cause of CSD in 10 patients [3]. Subsequent studies have shown that A. felis is rarely the causative agent of CSD [1]. However, a dual role for A. felis and B. henselae has been reported [3, 5].

Although many systemic manifestations of CSD have been described, only 12 cases of pulmonary involvement have been reported [2, 6–16]. During the last 40 years (1957–1997), one of the authors (A. M. M.) has observed 2,065 patients with CSD. Two adults and one adolescent had pulmonary disease [2, 10]. Our purpose herein is to report the unique clinical course of a patient who had a massive chest-wall abscess and axillary abscess due to CSD and to review the pulmonary findings in 12 published cases. Although several patients with intrathoracic bacillary angiomatosis due to Bartonella have been described recently, our discussion will be limited to the clinical context of CSD [1, 3, 17, 18].

Case Report

A previously healthy, 35-year-old male physician was seen by several colleagues for left shoulder-girdle pain that had persisted for 6 weeks. There was no history of trauma. Initially, he had had fever, chills, sweating, malaise, severe headaches with cognitive impairment, and anorexia for 2 weeks. During the last 4 weeks, he had noted a slowly enlarging anterior chest mass. Severe fatigue, anorexia, and shoulder pain with stiffness continued, and he had lost 20 lb. Findings on roentgenograms of the shoulder and an MRI of the neck were normal. The presumed diagnosis was brachialpseudopoly.

Treatment included analgesics, massage, and dexamethasone, 2 mg daily for 7 days, with minimal response. The results of a complete blood count and a metabolic panel (chemistry 18) were normal. By day 15 a tender mass had developed over the left pectoral region. Findings on chest and cervical spine roentgenograms were normal. Prednisone was administered at a dosage of 20 mg daily for 4 days; the dosage was increased to 60 mg daily for 10 days, then tapered over another 5–7 days. The results of an electromyogram were consistent with myositis. The creatine kinase level and an erythrocyte sedimentation rate were normal. On day 18, a chest MRI revealed a noncystic mass between the pectoralis major and minor, suggesting the presence of focal myositis.

By day 29 the mass was quite tender and measured 12 cm. By day 44 the mass involved the upper chest and left anterior axilla. The overlying skin was tender, red, and hot. A contrast-enhanced CT of the neck and thorax showed a large loculated abscess in the left upper chest wall, extending into the axilla. Spontaneous rupture occurred a few hours later, releasing...
### Table 1. Summary of cases of cat-scratch disease (CSD) in 13 patients.

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)/gender</th>
<th>Diagnostic criteria</th>
<th>Major signs and symptoms; lymphadenopathy location</th>
<th>Systemic involvement (diagnostic test)</th>
<th>Therapy (result)</th>
<th>Course, outcome (time)</th>
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<tbody>
<tr>
<td></td>
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<td>Fever (temperature, 39.3°C), pleuritic pain, cough, dyspnea, L axilla, L epityrochlear</td>
<td>Pneumonia, pleural reaction</td>
<td>Pen, Tet, Sm (not effective)</td>
<td>Hospital, 7 w; pneumonia resolved; recovered, 2 mo</td>
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<td>Dry cough, fever (temperature, 39°C), malaise; R axilla, R epityrochlear</td>
<td>Patchy RUL infiltrate</td>
<td>Symptomatic</td>
<td>Pneumonitis resolved, 10 d; no adenopathy after 1 mo</td>
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<td>Cough, breast pain, headaches, mental confusion, lethargy, fever (temperature, 39.5°C); L axilla, L epityrochlear</td>
<td>Pleuritic chest pain, pleural effusion, hepatitis, tinnitus, dysartria</td>
<td>Clex, Dox, Oxa (not effective)</td>
<td>Effusion cleared, 3 w; recovered, 2 mo</td>
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<td>Headaches, dry cough, fever (temperature, 40°C), arthralgias, seizure; L axilla, L epityrochlear</td>
<td>Bilateral infiltrates, widened mediastinum, hypotension, encephalopathy</td>
<td>TMP-SMZ, Tm, Em, Prd, Aza (effective)</td>
<td>Rapid recovery, 72 h; hospital, 2 w; recovered, 1 mo; adenopathy resolved</td>
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<td>Fever (temperature, 38.3°C), malaise, fatigue, 8-lb weight loss, back pain; L axilla, mediastinum</td>
<td>Pleuritic chest pain, apical pleural thickening, 4-cm postmediastinal mass</td>
<td>Pen, Oxa, Cph (not effective)</td>
<td>Adenopathy resolved, 4 mo; mediastinal mass, 7 mo, recovered, 4 mo</td>
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<td>Malaise, fatigue, anorexia, splenomegaly; L axilla, L epityrochlear</td>
<td>7-cm central mediastinal mass (CT)¹</td>
<td>Ctn, Amp, Gm (not effective)</td>
<td>Adenopathies resolved, 8 mo; recovered, 4 mo</td>
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<td>Fever (temperature, 39°C), diarrhea, 20-lb weight loss, CD4/CD8 ratio, &lt;0.14; HIV positive; L axillary, L epityrochlear</td>
<td>Bilateral disk papilledema, exudative retinitis, pleural effusion, liver abscess (CT)³</td>
<td>Kaposi’s sarcoma</td>
<td>Rapid response, 2 w, relapsed; cytomegalovirus, hepatitis, pneumonitis, renal failure, and death, 3 mo</td>
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<td>Fever (temperature, 39°C), coma; R submaxillary</td>
<td>Bilateral pneumonia, 450-ml pleural effusion, encephalopathy, seizures, recurrent hypotension</td>
<td>Thoracotomy tube, extracorporeal support for 89 h, Em, Cm, Ctr (not effective)</td>
<td>Effusions cleared, 5 d; recovered, 2.5 w</td>
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<td>Abdominal pain, emesis, fever (temperature, 39.7°C); WBC count, 45,400/mm³; L axilla</td>
<td>Pneumonia, 500-ml pleural effusion, T-10 vertebra osteomyelitis, paraspinal mass</td>
<td>Thoracotomy tube (effective), Rif, TMP-SMZ, Gm (effective)</td>
<td>Effusion cleared, 2 w; recovered, 3 mo</td>
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<td></td>
<td>Fever (temperature, 38.5°C), tachypnea, dyspnea; L axilla</td>
<td>Bilateral pneumonia, pleural effusions, seizures, encephalopathy</td>
<td>Acy, Ctr, Dox, oxygen (effective!)</td>
<td>Effusions and encephalopathy cleared, 1 w; recovered, 2 w</td>
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<td>Fever (temperature, 41°C), dry cough, emesis, diarrhea; retroperitoneal nodes (CT)³</td>
<td>Multiple bilateral pulmonary nodules, immunocompromised, four renal transplantsations</td>
<td>Clex (ineffective), Dox, 8 w (effective)</td>
<td>Pulmonary nodules and retroperitoneal nodes decreased, 6 w; recovered, 2 mo</td>
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<td>Fever (temperature, 40°C), coma, posthermorrhaphy; inguinal nodes</td>
<td>Bilateral pleural effusions, seizures, severe encephalopathy</td>
<td>Ctx, Gm (not effective)</td>
<td>Surgical excision LN; recovered, 3 w</td>
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</table>
~500 mL of thin, yellow, odorless pus. A culture of the drainage yielded a few colonies of *Streptococcus pneumoniae* that was susceptible to all antibiotics. A smear for acid-fast bacilli and routine cultures were negative. A specific culture for *Bartonella* species was not performed. However, the diagnosis of bartonella infection is usually made serologically, since culture of the bacteria is difficult. Two Penrose drains were placed, effecting complete drainage. After 2 days in the hospital, the patient was discharged, and his condition was much improved. He received cefadroxil for 7 days. His recovery was rapid, with removal of the clavicular drain in 7 days and the axillary drain in 14 days. Physical therapy resulted in full range of motion and good shoulder strength in a few weeks.

The patient’s medical and family histories were unremarkable. No prior lymphadenopathy, history of abscesses, or serious infections had occurred. Three domestic, originally stray, pet cats had scratched the patient often. Findings on physical examination during the 7th week of illness were normal, except for extensive pustular folliculitis over the anterior chest and two healing incisions with serous drainage in the left subclavicular and left anterior axillary areas.

Three skin tests were done. Indurations at 72 hours and 5 days after testing with PPD tuberculin, PPD-Battey (for *Mycobacterium avium complex*), and cat-scratch antigen (CS Ag) were 5 × 5 mm, 17 × 15 mm, and 17 × 18 mm, respectively. The CS Ag was positive for *B. henselae* DNA by PCR [3]. An indirect fluorescent antibody (IFA) test and EIA for *B. henselae* and *Bartonella quintana*, performed on serum of the patient and his wife at the Centers for Disease Control and Prevention (Atlanta) and Specialty Laboratories (Santa Monica, CA), were negative. Four months later repeated IFA tests for *B. henselae* and *B. quintana* were negative. The results of a follow-up examination and a complete blood count were normal 2 years later. Two HIV tests were negative.

In 1997 Kordick et al. [4] reported a case of typical CSD in a veterinarian, which was caused by a newly recognized zoonotic pathogen, *B. clarridgeiae*; this report prompted us to have our patient’s serum reanalyzed for IgG antibodies to other *Bartonella* species [4, 18, 19]. IFA tests for IgG antibodies to seven *Bartonella* strains, including *B. henselae* (Houston-1, NCSU strain 93-F006/type II, and NCSU strain 93-F012/type I), *B. clarridgeiae*, *B. quintana*, *B. elizabethae*, and *B. vinsonii berkhoffii*, were performed. Seroreactivity was detected only to *B. clarridgeiae*, at a titer of 1:128. Although some concern exists regarding cross-reactivity and the interpretation of serology for *B. clarridgeiae* (D. L. Kordick, personal communication), three control sera tested in the same assay were negative. Five months later a fourth convalescent serum sample was tested against the same panel of *Bartonella* strains, and seroreactivity was again detected only to *B. clarridgeiae* (titer, 1:256). At that time, blood samples from the patient, his dog, and three cats were cultured. *B. clarridgeiae* was isolated from one cat, whereas all other cultures remained negative after 8 weeks of incubation.

### Discussion

Atypical CSD occurs in 10%–14% of patients with CSD [2, 10]. The most common unusual manifestation is the oculoglandular syndrome, followed by encephalopathy, systemic disease, neuroretinitis, erythema nodosum, and granulomatous hepatosplenia [1–3, 18]. Of the 13 patients described herein, 10 were immunocompetent patients, and three were immunocompromised adults (table 1). Six cases of CSD with pneumonia and eight cases associated with pleural thickening and/or effusion have been reported [6–16]. The pulmonary features usually developed 1–5 weeks after the onset of lymphadenopathy. A 19-year-old with depressed cell-mediated immunity due to immunosuppressive therapy after renal transplantation who had

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### Table 1. (Continued)

<table>
<thead>
<tr>
<th>Case no.</th>
<th>Age (y)</th>
<th>Major signs and symptoms; lymphadenopathy location</th>
<th>Systemic involvement (diagnostic test)</th>
<th>Therapy (result)</th>
<th>Course, outcome (time)</th>
</tr>
</thead>
<tbody>
<tr>
<td>13 [PR]</td>
<td>35/M</td>
<td>Shoulder girdle pain (6 w), 20-lb weight loss, fatigue, chills, sweats, headaches, malarase, L axilla and chest, 12-cm abscess</td>
<td>Noncystic 12-cm mass, L upper chest, between inflamed pectoralis major and minor (MRI)</td>
<td>Dex, 1 w; Pred. spontaneous abscess rupture, Penrose drains, 2 w (effective)</td>
<td>Abscess ruptured after 3 mo; rapid recovery, 2 w</td>
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</tbody>
</table>

**NOTE.** Acy = acyclovir; Amp = ampicillin; Aza = azathioprine; Clex = cephalaxin; Cm = clindamycin; Cph = cephradine; CS Ag = cat-scratch antigen; Ctx = cephalexin; Cln = cephalothin; Ctri = ceftriaxone; Cm = clindamycin; Dox = doxycycline; Em = erythromycin; Gm = gentamicin; HP = histopathology; IFA = indirect fluorescent antibody; L = left; LN = lymph node; Oxa = oxacillin; Pen = penicillin; Pred = prednisone; Rif = rifampin; RUL = right upper lobe; Sm = streptomycin; Tet = tetracycline; Tm = tobramycin; TMP-SMZ = trimethoprim-sulfamethoxazole; WSS = Warthin-Starry stain; + = positive; = negative.

* Renal allograft recipient.
1 Patent had AIDS.
2 IgG titer, 1:1,025.
3 Detected on a CT.
4 Dosage: 40–60 mg over 21 days.
pulmonary nodules due to *B. henselae* was described recently [15]. Of the 13 patients described herein, 11 (85%) had severe systemic signs of infection, including fever (temperature, \(>38.3^\circ\text{C}\)) and/or multisystem involvement. Although afebrile, our patient had a massive chest-wall abscess that persisted for 2 months before spontaneous rupture.

Diagnoses in the 12 reported cases and in our case were confirmed by a history of cat contact and/or animal scratches in all cases (history of contact with a dog was reported in only one case), by positive CS Ag skin tests in seven cases, exclusion of other causes for lymphadenopathy in 12 cases, and a positive Warthin-Starry stain in five cases. *B. henselae* DNA was detected by PCR analysis of the CS Ag skin test used in our patient [3]. Titer of antibody to *B. henselae*, detected by an IFA test, were positive in three cases but negative for our patient. However, two convalescent serum samples from our patient, obtained 5 months apart 2 years after recovery, reacted only to *B. clarridgeiae*. Since cats can remain bacteremic with *Bartonella* species for years [4, 19] and *B. clarridgeiae* was cultured from one of our patient’s cats 2 years after his illness, it is conceivable that *B. clarridgeiae* could have been the cause of his CSD. Alternatively, because cats can be coinfected with *B. henselae* and *B. clarridgeiae* (D. L. Kordick, personal communication), the patient may have had CSD due to *B. henselae* and antibodies to *B. clarridgeiae* from recent exposure.

The histopathology of lymph nodes was compatible with CSD in eight cases. For one child, a 6.5-year-old with encephalitis and massive pleural effusions, the 16S rDNA target sequence of *A. felis* was detected in an extract from a submandibular lymph node biopsy specimen [12]. For an adult with pulmonary nodules, a lung homogenate was positive for *B. henselae* 16S rDNA sequence, amplified by PCR [15]. The CSD bacillus (not identified to the species level) was cultured from pleural fluid, a lymph node, and a liver abscess in a 27-year-old patient with AIDS [11] (case 7, table 1).

The patients’ ages ranged from 4 years to 46 years (mean age, 21 years); five were children, and eight were adults. The outcome was excellent in 12 cases, with complete resolution of CSD after 1–8 months (mean time, 2.0 months). The antibiotic therapy used was effective in only four of 12 patients. Spontaneous recovery occurred in eight patients. Trimethoprim-sulfamethoxazole was effective in two immunodeficient patients [1, 9, 11]. The 19-year-old immunosuppressed patient with pulmonary nodules responded to treatment with doxycycline, given for 2 months [1, 15].

Several findings in our patient were unique. During 2 weeks of high-dose prednisone therapy for a presumed diagnosis of focal myositis of the chest, he developed a massive chest-wall abscess, severe debilitating malaise, fatigue, and anorexia with a 20-lb weight loss. Lymph node abscess occurs in 12% of patients with CSD and in 50% of patients with severe CSD [2, 10]. Although our patient was seen by several physicians, none inquired about cat contact or scratches until the abscess ruptured. Furthermore, there was no comment about the folliculitis on his chest, which was likely the inoculation site for *B. henselae* or *B. clarridgeiae* after a cat scratch. The positive PPD-Battey skin test was interpreted as a normal reaction noted in most healthy individuals residing in the southeastern United States. Recently, bacillary angiomatosis associated with myositis was reported in an HIV-positive patient [20].

There have been case reports in which serological results predicted infection with one *Bartonella* species, while blood cultures yielded a different species. IFA IgG serology is not a species-specific diagnostic test. It is still possible that *B. henselae* was responsible for our patient’s disease, especially since high-dose steroid therapy may have caused temporary immunosuppression. In addition, the pitfalls and fallacies of IgG and IgM assays by IFA or EIA resulted in low sensitivities (41% sensitivity for the IFA IgG assay vs. 71% sensitivity for the EIA IgM assay for patients who fulfilled two or more criteria for CSD [21]).

Recent studies in healthy cats with bartonella bacteremia have demonstrated *B. henselae* in the cats’ erythrocytes by transmission electron microscopy [22]. These and prior studies by Hadfield, which showed the causative bacilli in the endothelium of blood vessels of lymph nodes by use of electron microscopy, may explain the pathogenesis of multisystem organ involvement noted in the patients described herein and also the lack of therapeutic response to routinely prescribed antibiotics in immunocompetent patients [1, 22]. Studies by Kordick et al. and Fumarola et al. suggest that the prolonged survival, intracellular growth, and multiplication of *B. henselae* within phagocytic cells may explain the variable clinical severity of CSD and the granulomatous, nongranulomatous, and/or angio-proliferative host-tissue pathology seen in human and feline infections [19, 23]. The genetic differences between *B. henselae* and *B. quintana* might account for the different clinical features seen in patients with CSD and the occurrence of bacillary angiomatosis [18, 23, 24].

Treatment of the healthy patient with typical CSD is supportive, since this disease is self-limited. In patients with thoracic and/or pulmonary disease, especially in association with prolonged fever, systemic symptoms, and/or severe multisystem disease, a trial of oral trimethoprim-sulfamethoxazole, ciprofloxacin, or azithromycin, two-to-three times daily for 7–21 days, is recommended [1, 25]. Alternatively, rifampin, two or three doses daily, may be effective. In the rare severely ill patient, im gentamicin sulfate, 5 mg/(kg · d), may be effective, usually within 72 hours. In immunodeficient patients, therapy with erythromycin, clarithromycin, azithromycin, doxycycline, or tetracycline alone was found to be effective [18]. Rifampin with erythromycin was more effective [1]. Treatment in these patients should continue for at least 6–8 weeks. Patients who relapse should be retreated for 4–6 months.

In conclusion, physicians should always inquire about recent cat or cat-flea contact and/or animal scratches when patients present with chronic and/or recurrent pulmonary complaints and associated adenopathy [24]. Performing IFA or EIA...
and/or a PCR hybridization assay on blood or aspirated or biopsied material to detect *Bartonella* species may provide a prompt diagnosis of CSD, obviate the need for multiple nondiagnostic studies, and direct appropriate therapy [1, 3, 4, 18, 21, 23–26].

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**References**