# Human Lymphadenitis Due to *Corynebacterium pseudotuberculosis:* Report of Ten Cases from Australia and Review

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***Corynebacterium pseudotuberculosis*** **commonly causes caseous lymphadenitis in Australian sheep. We describe 10 cases of human lymphadenitis due to *C. pseudotuberculosis;* in all cases, isolates were submitted to a reference laboratory in Victoria, Australia. Most of the patients were occupationally exposed to sheep. We also review the 12 previously published cases of this infection, most of which were reported from Australia. No patient had any underlying disease or predisposing condition. Surgical excision of the affected lymph glands is the mainstay of management, and antibiotic therapy is supplementary treatment. Diagnosis was delayed for some patients, and some patients had a protracted or recurrent clinical course and/or a slow recovery. These 10 additional cases from one Australian state indicate that human lymphadenitis caused by *C. pseudotuberculosis* has not been as rare as the number of published reports indicates, at least in Australia. However, the increasing use of a vaccine against caseous lymphadenitis in sheep in Australia should result in the decreasing human incidence of this zoonosis.**

*Corynebacterium pseudotuberculosis* (formerly *Corynebacterium ovis*) causes caseous lymphadenitis in sheep and in feral goats in Australia. Human lymphadenitis due to *C. pseudotuberculosis* was first described in 1966 in a Panamanian man whose source of infection is unclear [1]. Reports of human cases from Australia in persons who were occupationally exposed to sheep soon followed [2–4]. Australian reports have continued to dominate the world literature and account for nine of the 12 published human cases. In this report, we describe 10 more cases of human lymphadenitis due to *C. pseudotuberculosis* and review the previously published reports.

## Methods

*Laboratory procedures.* Excised glands were submitted for histopathologic examination and microbiological culture. Special staining procedures (e.g., Warthin-Starry silver stain and gram stain for bacteria, Ziehl-Neelsen acid-fast stain for *Mycobacterium* species, Grocott-Gomori methenamine–silver nitrate stain and periodic acid–Schiff stain for fungi) were usually carried out on the tissue sections in laboratories that performed histopathologic examinations. Pus discharged or drained from the affected lymph node or a sample of the excised node was cultured for bacteria in microbiology laboratories. Bacterial isolates were either identified in the laboratory and then sent to the Microbiological Diagnostic Unit in Parkville (Victoria, Australia) for confirmation of identity or were referred directly for identification.

On receipt of an isolate at the Microbiological Diagnostic Unit, subcultures were made to conventional culture media for the determination of growth and morphologic characteristics and biochemical activities. Isolates were also characterized biochemically by the API Coryne identification system (bioMérieux, Marcy l'Etoile, France). Production of phospholipase D exotoxin was tested on sheep blood agar plates against standard strains of $\beta$-hemolysin-producing *Staphylococcus aureus* NCTC (National Collection of Type Cultures) 7428 and *Rhodococcus equi* NCTC 1621. Isolates were tested for the production of diphtheria toxin by the Elek agar immunodiffusion plate technique and by PCR.

*Literature review.* We reviewed the world literature from the date of publication of the first case of human lymphadenitis due to *C. pseudotuberculosis* (1966) through 1995.

## Selected Case Reports

The following cases are illustrative of the delay in diagnosis and the recurrent or protracted course of disease that typically characterize human lymphadenitis caused by *C. pseudotuberculosis.*

*Case 7.* A 40-year-old abattoir worker presented to his physician with a 10-day history of an enlarged left epitrochlear lymph node. The patient did not have a recent history of sore throat, night sweats, rigors, or lacerations at his workplace and was well except that he had had increasing lethargy over the preceding week. The enlarged lymph node was excised. Histopathologic examination of the excised tissue showed a partly fibrotic node (dimension, 10 × 15 × 25 mm) containing germi-

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nal centers and seven large, irregular granulomas, each consisting of necrotic, basophilic material surrounded by palisaded fibroblasts. Numerous gram-positive coryneform bacteria were seen within the central necrotic area of one of the granulomas in sections stained by gram stain and Warthin-Starry silver stain. The results of culture for *Mycobacterium* species were negative, but no other bacterial cultures were requested or performed. No antibiotic treatment was given.

Twenty-two months later, the patient presented to his physician again, this time with a 12-month history of symptoms of left axillary lymphadenopathy, generalized malaise, and muscular pain. He had no fever, gastrointestinal symptoms, or weight loss. Fibrofatty tissue containing nodes of 20 and 25 mm in diameter was excised. The nodes contained multiple, suppurative foci up to 10 mm in diameter. Histopathologic examination revealed the almost total replacement of lymph node structure by a chronic granulomatous inflammatory reaction with central foci of suppurative necrosis.

A piece of the excised node was referred to the microbiology laboratory, where it was coarsely pulped by slicing. Smears of this material were examined after gram staining and Ziehl-Neelsen staining and were found to be negative for bacteria and acid-fast bacilli. However, culture of the material yielded light pure growth of a gram-positive coryneform bacillus. The isolate was identified as probable *C. pseudotuberculosis* and referred to the Microbiological Diagnostic Unit, where the identification was confirmed. The patient was prescribed a course of erythromycin (250 mg q.i.d. for 2 months), but no report on patient compliance was available. As of this writing, the patient had not presented with any further lymphadenopathy.

*Case 8.* A 27-year-old male employee of a sheep export saleyard presented to a physician with pain in the left axilla. No laboratory investigations were undertaken, and no treatment was given at that time. He presented again to a different physician 4 months later with a palpable lump in the left axilla. A course of flucloxacillin was prescribed, and the size of the lump decreased significantly. He next presented 3 years and 5 months later, when a swelling had developed in his left axilla. Differential diagnoses considered at that presentation were lymphoma and infection. A CT scan and a roentgenogram of the chest were normal. His WBC count ($12.2 \times 10^9$/L with 77% neutrophils) revealed mild neutrophilia. A course of amoxicillin/clavulanate therapy was prescribed, and the patient was instructed to return if his axillary swelling persisted.

The patient did not present again until 7 months later, when the mass in his left axilla had reached the size of a small orange. His WBC count ($12.5 \times 10^9$/L with 74% neutrophils) again revealed mild neutrophilia. Pus (70 mL) was drained and forwarded to the microbiology laboratory for gram staining and culture. A course of flucloxacillin therapy was prescribed. Microscopic examination of a gram-stained smear of the pus showed numerous gram-positive coccobacilli. Culture of the pus revealed pure growth of a gram-positive, rod-shaped bacterium that was identified as *C. pseudotuberculosis.* The identity of the bacterium was confirmed by the Microbiological Diagnostic Unit.

Six weeks later, the axillary lymph nodes and surrounding soft tissue were excised. Enlarged nodules (up to $45 \times 30 \times 10$ mm in dimension) were present within the excised tissue. The lymph nodes and soft tissue showed a necrotizing granulomatous and inflammatory process with sinus tract formation. No bacteria were seen in tissue sections or in imprints on staining, and cultures of the excised tissue were negative. Altogether, this patient's course of disease spanned a period of about 4.5 years.

## Results

Patient characteristics, clinical presentation, exposure history, histopathologic findings, and management for the 10 cases of lymphadenitis due to *C. pseudotuberculosis* that occurred in Victoria, Australia, are summarized in table 1. Table 2 summarizes these data for the 12 previously reported cases of this infection [1–10].

*Patient characteristics.* The mean age of the 10 patients described in table 1 and the 12 previously reported cases (table 2) is 31 years (range, 18–53 years), and only three of the 22 patients are female. All but five of the 22 patients were occupationally exposed to live or dead sheep. Two of the five reported previous contact with sheep. One patient had had contact with sheep more than a year before his presentation, at which time he had a 40-day history of axillary lymphadenopathy, and a second patient had worked in an abattoir 2 years before his presentation [6].

One of the female patients, an immigrant from Vietnam, denied both contact with sheep and consumption of lamb or mutton. With respect to sheep contact, a notable exception was the first published case of human lymphadenitis due to *C. pseudotuberculosis;* this infection occurred in a 37-year-old man who lived on the outskirts of Panama City, worked as a grass cutter, and frequently hunted edible iguanas in the surrounding jungle. He denied any contact with farm animals over the 3-year period before his presentation [1].

Another exception with respect to sheep contact was the first (and only) reported case of *C. pseudotuberculosis* lymphadenitis from the United States. This patient was an unemployed urban dweller who presented with cervical lymphadenopathy. He denied travel outside the western region of Washington State or significant occupational exposure to livestock. However, he regularly drank unpasteurized cow's and goat's milk, which may have been the source of his infection [8].

*Clinical characteristics.* Of the 22 patients, 13 male patients had axillary lymphadenitis, and one had epitrochlear involvement as well. One of the female patients had inguinal lymphadenitis, and a second had supraclavicular lymphadenitis; the third female patient had involvement of the cervical gland. In three cases there was primary involvement of the inguinal glands. The remaining three cases involved multiple lymph nodes, including one each of axillary, inguinal, and cervical lymph nodes. None of the 22 patients was immunosuppressed or had a known underlying disease.

**Table 1.** Summary of data from 10 cases of human lymphadenitis due to *Corynebacterium pseudotuberculosis* in Victoria, Australia.

| Case no. | Age (y)/sex | Year diagnosed | Lymph nodes involved (duration)* | Other symptoms | Occupation (exposure) | Histopathologic findings | Treatment and outcome |
|---|---|---|---|---|---|---|---|
| 1 | 41/M | 1985 | Right axillary | NA | Farm worker (sheep contact) | Granulomatous nodes | Excision of nodes |
| 2 | 29/F | 1985 | Left inguinal | Malaise, myalgia; cellulitis after surgery | Farm worker (sheep contact) | Chronic granulomatous reaction | Excision of node<br>Penicillin and flucloxacillin |
| 3 | 29/M | 1986 | Left axillary | Painless abscess in left arm initially | Meat inspector (sheep contact); cuts on his hands | Suppurative granulomatous lymphadenitis | Drainage of abscess<br>Excision of abscess and axillary node (1 mo after drainage) |
| 4 | 22/M | 1988 | Right axillary (3 mo) | None | Butcher (handled sheep) | Caseous necrosis; coryneform bacteria | Excision of nodes<br>Erythromycin |
| 5 | 20/M | 1988 | Right axillary | Discharging sinus | Slaughterman | Acute necrotizing granulomatous lymphadenitis | Drainage initially<br>Sinus formation<br>Excision of node (9 mo after drainage)<br>Erythromycin† |
| 6 | 53/F | 1988 | Left supraclavicular (1 mo) | Intermittent fever, malaise, nausea | Unknown exposure; urban dweller with no sheep contact | Inflammation; fibrosis | Excision of node |
| 7 | 40/M | 1989 | Left epitrochlear (10 d) | Lethargy | Abattoir worker | Granulomas with central necrosis; palisaded fibroblasts; bacteria | Excision of node<br>Relapsed |
| | | | Left axillary (1 y)‡ | Malaise, myalgia | | Chronic granulomatous inflammatory reaction; suppurative necrosis | Excision of nodes<br>Erythromycin |
| 8 | 27/M | 1992 | Left axillary | Pain (initially) | Worker in sheep saleyard | Necrotizing granulomatous inflammatory process | No antibiotics initially<br>Flucloxacillin, Amox/clav<br>Drainage, flucloxacillin therapy, excision |
| 9 | 26/M | 1992 | Right axillary | Malaise, weight loss | Abattoir worker | Granulomas with palisaded histiocytes, necrotic centers; numerous bacilli | Excision of node<br>Penicillin |
| 10 | 40/M | 1992 | Right axillary (40 d) | None | Contact with sheep 1 y before presentation; cuts on his hands | Giant cell reaction; abscess formation | Excision of node<br>Erythromycin and flucloxacillin<br>Recovery over 3 mo |

NOTE. Amox/clav = amoxicillin/clavulanate; NA = not available.

* Duration of history of enlarged lymph node at presentation.

† The patient was allergic to penicillin.

‡ Presentation 22 months after the first presentation.

Most patients with lymphadenitis due to *C. pseudotuberculosis* received antibiotic therapy. *C. pseudotuberculosis* is susceptible to penicillin and to other antibiotics that are active against gram-positive bacteria or have broad-spectrum activity. All patients eventually recovered, but usually not until the affected lymph nodes had been surgically removed.

*Bacteriologic and histopathologic findings.* The morphologic and biochemical characteristics of *C. pseudotuberculosis* are given in table 3. The API Coryne numerical profile was consistently 0111324, which means that the strains tested also gave positive results for alkaline phosphatase, $\alpha$-glucosidase, and acid from ribose in this system. The isolates did not produce diphtheria toxin with use of the Elek test or by PCR.

Histopathologic findings indicate that the histopathologic features of human lymph nodes infected by *C. pseudotuberculosis* are variable and fall in the broad category of necrotizing and suppurative granulomatous lymphadenitis.

## Discussion

*C. pseudotuberculosis* is a nonmotile, gram-positive coccobacillus and small bacillus that produces catalase, grows

**Table 2.** Summary of data from 12 previously reported cases of human lymphadenitis due to *Corynebacterium pseudotuberculosis*.

| Reference/ year | Age (y)/ sex | Location of case | Lymph nodes involved | Other symptoms and signs | Occupation (exposure) | Histopathologic findings | Treatment and outcome |
|---|---|---|---|---|---|---|---|
| [1]/1966 | 37/M | Panama Canal Zone | Right inguinal | Fatigue, myalgia, painful right leg, hepatomegaly | Grass cutter, hunter (no farm animal contact for 3 y before presentation) | Follicular hyperplasia with epithelioid phagocytic reaction; granulation tissue; chronic inflammatory reaction | Tetracycline (some response)<br>Excision of node |
| [2]/1967 | 28/M | New South Wales, Australia | Right inguinal, leg and thigh | Painful right calf lesion | Manager of a large sheep and cattle farm | Reported as "similar to that of cat-scratch fever or lymphogranuloma inguinale" | Drainage of inguinal node; sinuses formed<br>Excision of leg nodes, then inguinal node (2 mo later) then thigh node (4 mo later)<br>Relapsed (4 mo after excision of thigh node)<br>Drainage of inguinal node<br>Penicillin (49 d)<br>Inguinal sinus reopened<br>Recovery over 1 y |
| | | | Right inguinal and iliac (relapse) | | | | |
| [3]/1968 | 24/M | Victoria, Australia | Right axillary | None | Sheep shearer | Patchy degeneration with foci of caseous necrosis | Tetracycline (no response)<br>Excision of nodes (7 mo later)<br>Antituberculous therapy<br>Wound breakdown<br>Penicillin and tetracycline<br>Recovery over 1 y |
| [4]/1968 | 23/M | Queensland, Australia | Left axillary | Malaise, left finger wound, fever, palpable spleen and liver | Butcher (handled sheep) | Foci of eosinophilic necrosis with palisaded epithelioid cell margins | Tetracycline (no response)<br>Excision of nodes<br>Wound discharge for 2 mo |
| [5]/1974 | 20/M | Western Australia | Right axillary, then left axillary | None | Stationhand on sheep property | Round or oval foci of necrosis (20/M, 40/M) or suppuration (50/F), usually surrounded by a zone of palisaded histiocytes, beyond which is often a prominent fibrous collar | Excision of right node<br>Drainage, then excision of left node (6 mo later) |
| | 40/M | Western Australia | Left inguinal | None | Rural worker (sheep contact) | | Tetracycline and co-trimoxazole (no response)<br>Erythromycin and potassium iodide<br>Excision of nodes (6 mo later) |
| | 50/F | Western Australia | Cervical (2 mo)* | None | Homemaker (sheep contact) | | Excision of two nodes |
| [6]/1979 | 21/M | Western Australia | Left axillary | Pain | Abattoir worker 2 y before presentation | Central necrosis; palisaded epithelioid cells; perinodal inflammation; numerous bacilli | Excision of node<br>Sinus formation<br>Cloxacillin (3 mo) |
| [7]/1980 | 27/M | Hautes Alpes, France | Left axillary | Debilitation, fever, splenomegaly | Shepherd | Suppuration with follicular hyperplasia; necrosis; palisaded histiocytes; fibrosis; rare gram-positive bacilli | Antituberculous therapy<br>Excision of nodes<br>Tetracycline and chloramphenicol<br>Recovery over 4 mo |
| [8]/1981 | 30/M | United States | Left cervical | Malaise, myalgia, arthralgia | Urban dweller (drank raw cow's and goat's milk) | Granulomas; central suppuration and necrosis; palisaded epithelioid histiocytes; fibrosis; gram-positive bacilli | Penicillin (some response)<br>Excision of node<br>Penicillin<br>Drainage (sinus)<br>Relapsed (21 d later) |
| | | | Left lateral neck | | | | Drainage, then excision<br>Erythromycin |
| [9]/1985 | 18/M | Victoria, Australia | Right epitrochlear, supraclavicular, and axillary | Fever, sweating, headache, anorexia, weight loss | Butcher (handled offal with cut hands) | Vascular fibrosis and granulation tissue in wall of abscess | Tetracycline, then penicillin and flucloxacillin<br>Excision of epitrochlear node (2 mo later)<br>Tetracycline and erythromycin<br>Drainage of supraclavicular node, then drainage of axillary node |

**Table 2.** *(Continued)*

| Reference/year | Age (y)/sex | Location of case | Lymph nodes involved | Other symptoms and signs | Occupation (exposure) | Histopathologic findings | Treatment and outcome |
|---|---|---|---|---|---|---|---|
| [10]/1986 | 29/M | North Canterbury, New Zealand | Right inguinal (6 mo)* | Fever, malaise, anorexia | Farm manager (squeezed superficial abscesses on sheep with bare hands) | Necrotizing granulomatous inflammation with caseous necrosis; palisaded epithelioid histiocytes; perinodal fibrosis | Exicision of two nodes<br>Penicillin and cloxacillin<br>Penicillin and flucloxacillin<br>Wound debridement and skin grafting (2 mo later)<br>Erythromycin |

* Duration of history of enlarged lymph node at presentation.

aerobically and anaerobically, and metabolizes and ferments carbohydrates. *C. pseudotuberculosis* is closely related to two other species of *Corynebacterium, C. diphtheriae* and *C. ulcerans,* and shares two distinctive characteristics with these species: negative pyrazinamidase activity [11] and positive cystinase activity, as determined by the production of brown halos around colonies on modified Tinsdale medium [12].

*C. pseudotuberculosis* is differentiated from *C. diphtheriae* by its positive urease activity and by the negative reaction for nitrate reduction that is given by the biovar infecting sheep and goats. *C. pseudotuberculosis* differs from *C. ulcerans* in that it is unable to ferment glycogen and trehalose or to hydrolyze gelatin [13]. In contrast with the reaction listed for *C. pseudotuberculosis* in some texts [14], none of our isolates fermented sucrose. Isolates of *C. pseudotuberculosis* were correctly identified in the API Coryne identification system.

Phospholipase D produced by *C. pseudotuberculosis* hydrolyzes sphingomyelin in mammalian cell membranes, such as the endothelial cells of blood vessels, thereby facilitating the establishment and spread of these bacteria in the host. The inhibition of staphylococcal $\beta$-hemolysin on sheep blood agar plates, or reverse CAMP reaction, is indicative of phospholipase D [14, 15]. Another in vitro correlate of phospholipase D is the enhancement of the hemolysis of sheep RBCs by synergistic interaction with the hemolysin of *R. equi* [16].

Both *C. pseudotuberculosis* and *C. ulcerans* can be lysogenized by the bacteriophage of *C. diphtheriae* that confers the ability to produce diphtheria toxin [17]. Hence, although no clinical cases of diphtheria have been attributed to *C. pseudotuberculosis*, isolates of this species need to be tested for toxigenicity by the Elek immunodiffusion test [13] and/or by PCR [18].

*C. pseudotuberculosis* is primarily a pathogen of ungulates. Sheep, then goats, are the most commonly affected animal hosts, but horses, cattle, and deer may also be infected [19]. *C. pseudotuberculosis* causes caseous lymphadenitis in sheep and goats, a disease characterized by the development of chronic abscesses containing caseous pus in superficial lymph nodes and in visceral nodes and organs, particularly the lungs [20]. The prevalence of caseous lymphadenitis in ewes that were >3 years of age and that were slaughtered in a Western Australian abattoir in early 1984 was found to be 54% [21]. It has been postulated that the etiologic agent of caseous lymphadenitis was introduced into Australia with Merino sheep, as the disease does not occur naturally in sheep in the United Kingdom [3]. The prevalence of caseous lymphadenitis in feral goats presented for slaughter at a Western Australian abattoir was reported to be 7.8% [22].

A nitrate-positive biovar of *C. pseudotuberculosis* causes two different forms of major disease in horses. One is characterized by extensive abscesses, especially in the pectoral muscles and ventral abdominal wall, but also in the internal organs; the other is characterized by ulcerative lymphangitis, with suppurative inflammation of the lymphatics. This biovar has also rarely been isolated in cases of equine abortion and mastitis [23]. The equine biovar of *C. pseudotuberculosis* apparently does not occur in Australia [20], and equine disease has never been reported in Australian horses. *C. pseudotuberculosis* infection is uncommon in cattle worldwide, although single, sporadic cases and occasional outbreaks among cattle herds have been reported [23]. Bovine disease includes mastitis, with excretion of the bacteria in milk.

**Table 3.** Identifying characteristics of *Corynebacterium pseudotuberculosis.*

| Positive characteristics | Negative characteristics |
|---|---|
| Aerobic growth | Motility |
| Anaerobic growth | Gelatin hydrolysis |
| Gram stain reaction (coccobacilli) | Nitrate reduction |
| Hemolysis (horse blood) | Fermentation of: |
| Fermentation of: | Glycogen, mannitol, sucrose, trehalose, and xylose |
| Glucose, maltose | Pyrazinamidase activity |
| Production of: | |
| Catalase | |
| Cystinase | |
| Phospholipase D | |
| Polyphosphate granules | |
| Urease (Christensen's medium) | |

NOTE. Carbohydrate fermentation was tested in a peptone-based medium containing 5% heat-inactivated newborn calf serum and bromcresol purple indicator.

It is generally believed that *C. pseudotuberculosis* infection in sheep and goats is initiated through superficial wounds to the skin or mucous membranes followed by extension to the regional lymph nodes with, perhaps, further dissemination [20]. Evidence that shearing wounds are the primary route for infection in sheep has been provided by the demonstration of a specific serological response to *C. pseudotuberculosis* toxin in sheep 3 months after shearing [24]. Infected animals are not the only reservoir of infection, as *C. pseudotuberculosis* survives well in the environment [25].

Most of the cases of human lymphadenitis due to *C. pseudotuberculosis* have been reported from Australia, usually in those who have been occupationally exposed to sheep. Axillary lymphadenitis predominates, presumably because the hands and arms are frequently the site of primary infection. Two of the 10 patients whose cases are reported herein (cases 3 and 10) and one of the patients whose case was previously reported [9] admitted to inflicting cuts on their hands, which would provide an obvious portal of entry for the bacterium. Axillary lymphadenitis was prominent in those who worked in occupations such as butcher, slaughterman, abattoir worker, and sheep shearer; in contrast, the occupations of those with inguinal lymphadenitis were given as hunter and grass cutter [1], manager of a large sheep and cattle farm [2], rural worker [5], farm manager [10], and farm worker (case 2 of current series).

It is likely that sheep are the source of *C. pseudotuberculosis* infection and that the hands and arms are the primary sites of infection in patients with axillary lymphadenitis; in contrast, environmental contamination may well be the source in patients with inguinal lymphadenitis, with the feet and legs as the primary sites of infection.

Involvement of the cervical lymph nodes was considered compatible with *C. pseudotuberculosis* infection due to ingestion in the case of a North American man who denied significant animal contact but consumed raw goat's and cow's milk [8]. Dissemination of infection to visceral lymph nodes and internal organs, as may occur in animals, has not been reported in human infections.

However, in the only other case of human disease caused by *C. pseudotuberculosis* to be reported from the United States, the patient had unusual eosinophilic pneumonia [26]. The patient, a 28-year-old male student of veterinary medicine, had been exposed to "sick" horses. He developed a spiking fever, chills, and a cough with peripheral blood eosinophilia and an eosinophilic pulmonary infiltrate. *C. pseudotuberculosis* was isolated from the transtracheal aspirate and bronchoscopy washings. In addition, there was a specific antibody response to his isolate. The patient responded to antibiotic treatment with erythromycin. No other case of pulmonary disease in humans has been attributed to *C. pseudotuberculosis*.

As has been noted by other investigators [5], human illness caused by *C. pseudotuberculosis* is characterized by its chronicity rather than its severity. Furthermore, constitutional signs and symptoms other than lymphadenopathy are usually relatively mild and may be absent. As is illustrated by the more-detailed descriptions of the clinical course of lymphadenitis in cases 7 and 8 of the current series, patients may present with a history of lymphadenopathy of many months' duration and then experience a chronic, protracted, or recurrent course of disease until the affected lymph nodes are surgically removed.

The present series of 10 cases of human lymphadenitis caused by *C. pseudotuberculosis* from the state of Victoria in Australia almost doubles the number of cases reported worldwide. It indicates that the disease has been more common, at least in Australia, than published accounts suggest. Cases of suppurative lymphadenitis might be treated by incision and drainage plus antibiotic therapy, without a concerted effort being made to determine the microbial cause. Cases might also be misdiagnosed clinically or in the laboratory. The histopathologic appearance of the lesions may be mistaken as being caused by other infectious agents. Furthermore, if a *Corynebacterium* species is cultured in a microbiology laboratory when the bacteria have not been detected in stained preparations of the sample, the isolate may be regarded as nonsignificant and wrongly dismissed as a "skin diphtheroid."

A commercial toxoid vaccine against caseous lymphadenitis (*C. pseudotuberculosis*) was released in Australia in 1983 as Glanvac vaccine [20]. The Glanvac 3 and Glanvac 6 vaccines that are currently available (CSL Limited; Parkville, Victoria, Australia) are multicomponent, adjuvanted vaccines intended for administration to sheep and goats and designed for the control of caseous lymphadenitis, as well as other important diseases affecting these animals. It may be expected that, as vaccination against caseous lymphadenitis is more widely used in Australian sheep flocks, the incidence of the human form of this disease in those occupationally exposed to sheep or their environment will also decline.

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