gressive loss of strength, and trouble with swallowing and opening his eyes. These complaints had started after the first dosing of clarithromycin and progressed rapidly with each new dose. There were no signs of meningism, and the temperature was normal. His blood pressure was 120/80 mm Hg and his pulse 84/min. A neurological examination revealed flaccid tetraplegia and generalized hypotonia with only minimal traces of contraction, an impaired swallowing reflex, and lagging of the eyelids. There were no sensorial disturbances, and the reflexes were symmetrically lowered. Laboratory investigations showed no abnormalities except for a leukocyte count of 1.7 × 10^9/L. The clinical picture resembled myasthenia gravis. The patient was given pyridostigmine (30 mg), and clarithromycin was withdrawn. All symptoms disappeared within the next 6 hours. Electromyography was performed 3 days after withdrawal of clarithromycin and showed no abnormalities (e.g., no sign of myasthenia gravis). An assay for antibodies to acetylcholinesterase receptors was negative. For ethical reasons we did not perform a rechallenge.

Estimates of the incidence of clinical and subclinical neuromuscular disease in HIV-infected patients vary from 15% to almost 50% [1]. To our knowledge, myasthenia gravis in HIV-infected patients has been described two times before [2, 3]. In both cases myasthenia gravis was thought to derive from an alteration of the immune system at the level of the thymic gland, resulting in antibody production to the acetylcholine receptor. In one of these patients, a tumor in the anterior mediastinum was found and antibodies to acetylcholine receptors were detected. After surgery the patient became asymptomatic, following initiation of treatment with low-dose pyridostigmine bromide [2]. An ocular pseudo-mycophenic reaction to interferon therapy in a patient with AIDS has been reported as well [4].

Drug-induced myasthenic syndromes and aggravation of myasthenia gravis with other macrolides—in particular, erythromycin—have been described [5, 6, 7, 8]. Erythromycin is thought to lead to neuromuscular blockade and exacerbation of myasthenia gravis through inhibition of the presynaptic release of acetylcholine. In patients without neuromuscular disease who were taking erythromycin, electromyographic findings demonstrated myasthenia-like changes without clinical weakness. The loss of motor unit contractions improved with intravenous administration of edrophonium or neostigmine [9]. In view of the clinical picture of our patient and his reaction to withdrawal of clarithromycin, we assumed that he must have had a clarithromycin-induced myasthenic syndrome.

Addendum. A review of data on file at Abbott Laboratories (Amstelveen, the Netherlands) revealed that no cases concerning the association of clarithromycin with myasthenia gravis or a myasthenic syndrome have been reported.

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References

Pyomyositis Due to *Eubacterium lentum* and *Streptococcus constellatus* from a Periodontal Source

*Eubacterium lentum* has never been reported as an etiologic agent of pyomyositis, and *Streptococcus intermedius* group microorganisms have hardly ever been implicated as pathogens that cause this condition [1, 2].

We report herein a case of pyomyositis caused by a mixed bacterial infection due to *E. lentum* and *Streptococcus constellatus* that was probably secondary to seeding from periodontal disease. To our knowledge, this mixed bacterial infection has never been reported as a cause of pyomyositis.

Pyomyositis in a 34-year-old man was admitted to our hospital because of a 4-week history of pain and gradual enlargement of a mass on the right thigh; he had had a fever during the week prior to admission. He denied recent trauma, travel to tropical countries, homosexuality, and intravenous drug use. He drank 75–100 g of alcohol per day. On admission, he was in good condition and his temperature was 39.0°C. He had various dental caries and signs of gingivitis and periodontitis. Tenderness on palpation, swelling, and a 10 × 6-cm mass in the lateral aspect were noted on the right thigh. The remainder of the physical examination was normal.

The WBC count was 19.3 × 10^9/L, with a differential cell count of 89% neutrophils, 8% lymphocytes, and 3% monocytes. Routine laboratory tests revealed values within the normal ranges, except for the serum levels of aldolase (9.1 IU/L) and gamma-glutamyltransferase (96 IU/L). A test for antibodies to HIV was negative. A CT scan (figure 1) showed a multilobed hypodense fluid collection within the body of the quadriceps femoris muscle.

An incision was performed, and foul-smelling purulent material was drained from an abscess involving the quadriceps femoris muscle. Gram staining revealed gram-positive cocci and

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gram-positive bacilli, and empirical treatment with intravenous cloxacillin, tobramycin, and ornidazole was begun. Cultures of blood and urine disclosed no pathogens. Culture of pus yielded *E. lentum* and *S. constellatus* (API 20 A and API 20 Strep systems; bioMérieux, Marcy l’Etoile, France), and then the treatment was changed to intravenous penicillin G (2 × 10⁶ U q4h) on the basis of the results of antimicrobial susceptibility testing.

Panoramic radiography disclosed no periapical or periodontal abscesses. Chest radiographic findings were normal, ultrasonography of the abdomen revealed no abnormalities, and a barium enema ruled out lesions in the large intestine. The patient’s condition improved, and a 19-day course of intravenous penicillin G was prescribed; the symptoms gradually disappeared. The patient was discharged from the hospital in good health. He was asymptomatic at follow-up examinations performed during months 1 and 4 after discharge.

*Eubacterium* species are anaerobic nonsporulating bacilli that usually reside in the mouth and bowel [3] and have been associated with periodontitis and gingivitis [4]. Furthermore, *Eubacterium* species have been implicated in head and neck, abdominal, and gynecologic pyogenic infections; periodontitis is a known predisposing factor [5]. Nevertheless, this microorganism has not yet been reported as an etiologic agent in pyomyositis. To date, only rare cases of anaerobic pyomyositis have been described. The organisms involved in these cases have included *Fusobacterium* species plus *Peptostreptococcus* species [6], *Clostridium septicum* [7], *Fusobacterium* species [8], and *Fusobacterium nucleatum* plus *Peptococcus* species [9].

*S. intermedius*, *S. constellatus*, and *Streptococcus anginosus* (collectively referred to as the *S. intermedius* group or the *S. milleri* group) also form part of the normal flora of the mouth and gastrointestinal tract, and they are often associated with various purulent infections [10]. In relation to odontogenic infections, *S. intermedius* has been implicated in refractory periodontal disease, and *S. anginosus* in chronic gingivitis [4]. *S. intermedius* group isolates have rarely been reported as causative pathogens in pyomyositis. To date, only two cases of *S. anginosus* pyomyositis have been described [1], but no case of *S. constellatus* pyomyositis has been documented.

Pyomyositis remains a poorly understood disease. Bacteremia and seeding of a previously damaged skeletal muscle could be the most likely etiologic mechanism. However, other predisposing factors such as diabetes mellitus, alcoholic liver disease, thiamine deficiency, parasitic myositis (in the tropics), corticosteroid therapy, and HIV infection may contribute to suppuration in the muscle [1]. In the present case, the clinical evidence of periodontal infection, the bacteriology of the pus from the muscular abscess, and the absence of any other infectious focus all suggest periodontal disease as the source of the pyomyositis. Bacteremia could not be demonstrated, nor was muscle trauma evident. The only predisposing factor involved was alcoholism, which could explain the thiamine deficiency.

In summary, this unique case of *E. lentum* and *S. constellatus* pyomyositis serves to expand the spectrum of bacterial infections causing this condition; it also suggests that these organisms may cause pyomyositis through a transient bacteremia and seeding of the skeletal muscle from a periodontal source.

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**Figure 1.** CT scan obtained for a patient with pyomyositis caused by a mixed bacterial infection; the scan, which was obtained on the first day of hospitalization, shows low-density areas in the right musculus vastus lateralis.

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**References**

Isolated Tricuspid Valve Endocarditis Due to \textit{Streptococcus bovis}

Involvement of the tricuspid valve occurs in 10\%-15\% of patients with infective endocarditis. \textit{Staphylococcus aureus} is the most common cause of tricuspid valve endocarditis (TVE), particularly in drug addicts and patients with central venous catheters. Various species of streptococci cause TVE, which most often occurs in association with left-sided endocarditis. \textit{Streptococcus bovis} is a rare cause of TVE. We report what we believe to be the second documented case of isolated TVE secondary to \textit{S. bovis}. In the first reported case, the patient passed away, and the organism was isolated from the bloodstream only postmortem. We present the findings of a patient with an isolated TVE due to \textit{S. bovis}, which was isolated from the bloodstream and the valve tissue.

A 34-year-old male with a history of mild asthma and hepatitis B, but no high-risk behavior, was admitted to the hospital because of dyspnea, persistent dry cough, and high fever. These symptoms had gradually worsened during the 2 weeks before admission. The patient did not have a history of heart murmur and engaged in daily vigorous aerobic exercise. On physical examination, he appeared restless; his oral temperature was 104°F, and his respiratory rate was 40. He appeared to have mild jaundice. A systolic murmur could be heard over the tricuspid area. Auscultation of the lungs revealed bilaterally decreased respiratory sounds, with a few rales in both lung bases. No other abnormalities were detected.

The WBC count was 9.9 \times 10^9/L with 87\% segmented neutrophils, 6\% band forms, 5\% lymphocytes, and 1\% monocytes. Laboratory studies revealed the following values: hemoglobin, 74 g/L; hematocrit, 21.4\%; and platelet count, 124 X 10^9/L. Liver function tests revealed the following abnormal values: alkaline phosphatase, 194 U/L; alanine aminotransferase, 152 U/L; aspartate aminotransferase, 117 U/L; \gamma-glutamyl transpeptidase, 103 U/L; and lactate dehydrogenase, 843 U/L. The prothrombin time was 13.4 seconds, and the partial thromboplastin time was 25.5 seconds. Levels of electrolytes, blood urea nitrogen, creatinine, and bilirubin were within normal limits. Analysis of arterial blood gases while the patient breathed room air showed a pH of 7.5, a P\textsubscript{CO}_2 of 27 mm Hg, a P\textsubscript{O}_2 of 74 mm Hg, and an O\textsubscript{2} saturation of 96.1\%. Chest radiographs obtained on admission revealed diffuse patchy infiltrates in both lung fields. An electrocardiogram did not reveal any abnormalities. A transesophageal echocardiogram obtained on the second hospital day showed a 1.4 X 1.1-cm vegetation on the septal leaflet of the tricuspid valve, as well as significant tricuspid regurgitation.

Two cultures of blood drawn on admission were positive for a \textit{Streptococcus} species. The organism was identified by the Mini ID Screen (Carr-Scarborough Microbiological, Decatur, GA) as \textit{S. bovis}; it was susceptible to ampicillin, cephalothin, clindamycin, erythromycin, tetracycline, and vancomycin. Susceptibility to gentamicin was not determined, as \textit{S. bovis} does not fit the screen for testing with this method.

The patient was initially treated with ampicillin and gentamicin, followed by vancomycin and gentamicin. Despite aggressive antibiotic treatment, he continued to have high spiking fevers and respiratory compromise, and bilateral infiltrates persisted on the chest radiographs, indicating pulmonary septic embolization from a tricuspid valve vegetation. Repeated blood cultures remained negative. Two weeks after admission, transesophageal echocardiography was repeated and showed a new 2 X 1-cm vegetation on the anterior leaflet of the tricuspid valve, in addition to the septal leaflet vegetation present on the initial echocardiogram. He therefore underwent excision of the tricuspid valve, which was replaced with a porcine valve (#29). Pathological examination of the excised valve tissue revealed two tricuspid valve vegetations with multiple ruptured chordae. Cultures of the tissues subsequently yielded \textit{S. bovis}. The postoperative medical regimen included anticoagulative therapy with intravenous heparin and antibacterial therapy with vancomycin and gentamicin.

In the postoperative period, the patient developed several complications including acute renal failure secondary to gentamicin toxicity, metabolic encephalopathy, and mechanical ventilation-associated pneumonia and bacteroides empyema that was treated by adding clindamycin to his regimen. Because he had difficulty being weaned off mechanical ventilation, he required tracheostomy. Two weeks after surgery, when he had been successfully weaned off the ventilator and the complications had resolved, he underwent pancolonoscopy for a possible source of \textit{S. bovis} because there is a recognized association between colon cancer or polyps and \textit{S. bovis} endocarditis. This procedure did not reveal any abnormalities. The patient’s liver function abnormalities gradually abated, and 8 weeks after admission he was discharged to his home.