**Mycoplasma hominis-Associated Parapharyngeal Abscess following Acute Epstein-Barr Virus Infection in a Previously Immunocompetent Adult**

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**CASE REPORT**

A previously healthy 20-year-old man was referred to a tertiary-care hospital for management of a parapharyngeal abscess complicating acute Epstein-Barr virus (EBV) infection. Two weeks previously, he was admitted to a rural hospital with dehydration associated with a 5-day history of sore throat, fever, and progressive dysphagia. He was treated with an intravenous fluids and benzylpenicillin at 1.2 g every 6 h. Acute EBV infection was diagnosed on the basis of a positive monospot test, positive EBV capsid antigen immunoglobulin G (IgG) and IgM results, and negative EBV nuclear antigen IgG results. Treatment with intravenous dexamethasone at 8 mg daily was commenced, with improvement of the symptoms. The man was discharged from the hospital after 7 days. On the day of discharge, he noted a painful papular rash, which evolved rapidly over the subsequent 3 days into pustules and vesicles covering the forehead, nose, and left cheek. He was readmitted to the hospital and treated for 3 days with intravenous acyclovir at 600 mg three times daily and cefazolin at 1 g twice daily. Acute EBV infection was diagnosed on the basis of a positive monospot test, positive EBV capsid antigen immunoglobulin G (IgG) and IgM results, and negative EBV nuclear antigen IgG results. Treatment with intravenous dexamethasone at 8 mg daily was commenced, with improvement of the symptoms. The man was discharged from the hospital after 7 days. On the day of discharge, he noted a painful papular rash, which evolved rapidly over the subsequent 3 days into pustules and vesicles covering the forehead, nose, and left cheek. He was readmitted to the hospital and treated for 3 days with intravenous acyclovir at 600 mg three times daily and cefazolin at 1 g twice daily. Direct immunofluorescence analysis of a skin scraping confirmed infection with herpes simplex virus type 1. During this time, he complained of persistent dysphagia. A computed tomography (CT) scan of the neck demonstrated a 3-cm hypodense mass in the poststyloid parapharyngeal space with mild peripheral contrast enhancement and fat stranding consistent with a parapharyngeal abscess. He was transferred to a tertiary-care hospital and treated with intravenous floxacillin (flucloxacillin) at 1 g four times daily, ceftriaxone at 1 g twice daily, and metronidazole at 500 mg twice daily. Serology for human immunodeficiency virus was negative. When a CT scan performed 1 week later (Fig. 1) failed to demonstrate improvement, the collection was surgically drained. The operative findings were of a 2-ml collection with a large amount of associated soft-tissue swelling. Gram staining of the abscess fluid showed moderate numbers of polymorphic cells but no bacteria. After 72 h of incubation, there was heavy growth of small colonies on horse blood agar incubated aerobically in 5% CO₂ and on anaerobic agar. Gram staining of the colonies failed to distinguish any organisms, and a provisional identification of Mycoplasma sp. was made. This identification was supported by electron microscopy (Fig. 2). A 1,282-bp fragment from the organism was amplified by PCR using eubacterial domain-specific broad-range PCR primers for the 16S rRNA gene. The sequence of the PCR product showed 100% homology to the 16S rRNA gene of Mycoplasma hominis (GenBank accession number AF443616.3) in a BLAST search (www.ncbi.nlm.nih.gov/BLAST). Susceptibility testing was performed by using Etest (AB Biodisk, Solna, Sweden) and an inoculum with a 0.5 McFarland standard on Mueller-Hinton agar with sheep blood, incubated anaerobically at 35°C, and results were read at 48 h. The isolate was regarded to be sensitive to clindamycin (MIC < 0.016 μg/ml) and ciprofloxacin (MIC 0.125 μg/ml) but resistant to tetracycline (MIC > 256 μg/ml) and erythromycin (MIC > 256 μg/ml). The man was discharged 2 days postsurgery on a regimen of oral Amoxicillin (amoxicilline)-clavulanic acid at 875 mg/125 mg twice daily. This treatment was changed to clindamycin at 450 mg three times daily 2 days later, once the presence of Mycoplasma sp. was suspected. The dysphagia completely resolved within 2 weeks, and he remained asymptomatic 4 months later.

* M. hominis is a common commensal of the genitourinary tract (13). Colonization of the oral or respiratory tracts by M. hominis occurs in only 3% of healthy adults (11, 15), although it was found in 20% of children undergoing tonsillectomy for recurrent adenotonsillitis (6). The first reported case of M. hominis infection was associated with Bartholin’s abscess in 1937 (2). M. hominis has subsequently been implicated in pyleonephritis, pelvic inflammatory disease, postpartum fever, caesarean wound infection, premature birth, and respiratory illness in newborns (15, 19). Less commonly, M. hominis causes extragenital infections. There have been 25 reported cases of M. hominis infection involving the lower respiratory tract (3–5, 7, 9, 10, 13, 14, 18) but none involving the upper respiratory tract or oral cavity. In all but two cases, there was a history of...
organ transplantation, trauma, or tracheal intubation. Pharyngitis due to *M. hominis* has been induced experimentally following oropharyngeal and nasopharyngeal inoculation (16); however, natural infection has not been reported.

The isolation of a *Mycoplasma* sp. in the laboratory may be difficult, as the organism is slow growing, produces small, translucent colonies that may be missed unless examined under a stereomicroscope, produces little turbidity in broth culture, and may be overgrown by other bacteria. *Mycoplasma* spp. are also inhibited by sodium polyanethol sulfonate in blood culture medium. Although this difficulty can be overcome by the addition of gelatin, automated detection systems fail to detect the growth of *Mycoplasma* spp. (8, 21). In addition, most *Mycoplasma* spp. do not grow on standard bacteriologic medium, requiring specialized medium (e.g., SP4 or Shepard’s 10B broth), which may not be available in all laboratories or used routinely for extragenital specimens. It is therefore likely that *Mycoplasma* sp. infections, particularly extragenital infections, are underdiagnosed. *M. hominis* is one species which may grow on routine bacteriologic media, such as blood agar (22); however, this method of isolation is not always reliable. *Mycoplasma* spp. are facultative anaerobes, growing best either anaerobically or in room air supplemented with 5 to 10% carbon dioxide. *M. hominis* growth appears usually within 2 to 4 days, typically as pinpoint translucent colonies with a fried-egg appearance (22). The inability of *Mycoplasma* spp. to take up the Gram stain allows for a presumptive identification of *Mycoplasma/Ureaplasma* sp. Species identification may be suggested by the colony morphology, rate of growth, site of origin, and biochemical properties. *M. hominis* is unable to produce acid from glucose but hydrolyzes arginine. Molecular techniques, however, such as 16S rRNA gene sequencing, may be more accurate and practical methods of species identification. There are no universally accepted standards for *Mycoplasma* sp. susceptibility testing or specific MIC breakpoints. *Mycoplasma* spp. are innately resistant to penicillins, cephalosporins, rifampin (rifampicin), sulfonamides, and trimethoprim (12, 20). *M. hominis* is also resistant to macrolides but sensitive to clindamycin. Tetracycline susceptibility is variable, with the frequency of resistance associated with the *tet*(M) gene increasing in certain locations (1, 17, 20). The drainage of collections and the debridement of tissue, in the absence of specific antimycoplasma antibiotics, have been associated with cure (10).

This is the first reported case of upper respiratory tract infection with *M. hominis* and is particularly notable in that it occurred in a young adult with no prior history of immunosuppression, urinary tract manipulation, surgery, or trauma. The *M. hominis*-associated abscess complicated acute EBV pharyngitis, suggesting that the localized inflammation induced by the EBV infection may have facilitated secondary infection by *M. hominis*. The use of antibiotics active against cell walls, inhibiting normal flora, and the immunosuppressive effect of dexamethasone may have also contributed to the infection. The reactivation of herpes simplex virus infection is consistent with a transient deficiency in cell-mediated immunity. We cannot entirely dismiss the possibility that other oral-type bacteria were contributing to the infection, being inhibited in culture by the antibiotics. The early institution of intravenous benzylpenicillin treatment following the onset of symptoms of EBV infection and the absence of visible bacteria upon Gram staining of the abscess fluid, despite the presence of moderate numbers of polymorphic cells, are evidence against the involvement of other bacteria. It is also difficult to determine whether the clinical response was due
providing the electron micrograph image.

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We declare that we have no conflicts of interest.

REFERENCES


