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Isolation of *Mycoplasma hominis* from a Brain Abscess

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***Mycoplasma hominis* is a commensal in the genital tract of women and has been associated with urogenital and extragenital infections. However, central nervous system infections with this organism in adults are very rare. Here we describe the recovery of *M. hominis* from a brain abscess associated with a postpartum infection. Seroconversion to the isolated strain was detected by both a metabolic inhibition test and an immunoblotting assay. This case demonstrates the pathogenic potential of *M. hominis* and the need for rapid recognition of the organism so that appropriate chemotherapeutic intervention can occur.**

Mycoplasma hominis colonizes the oral cavity and respiratory tract but is more frequently (20 to 50%) found as a commensal in the lower genitourinary tract of women. Prevalence of this organism is linked to young populations, low socioeconomic status, sexual activity with multiple partners, and oral contraceptive use (3, 16, 18). *M. hominis* has been associated with pyelonephritis in males and pelvic inflammatory disease, spontaneous abortion, postpartum endometritis, and bacteremia in women. The organism causes pneumonia and meningitis in neonates, especially those born prematurely, who acquire the organism by passage through an infected birth canal (3, 10, 20).

Although the organism is less fastidious than other mycoplasmas and frequently will grow on conventional blood agar medium, specific laboratory methods are required for the identification of *M. hominis*. In addition, the virulence of this organism is minimal in most normal hosts; however, immunosuppressed patients and individuals with other risk factors may experience clinically apparent infection with *M. hominis*. We describe herein the recovery of *M. hominis* from a postpartum brain abscess of a patient during the postpartum period.

Case report. A 22-year-old female patient underwent normal spontaneous vaginal delivery. Over a period of 3 days, she developed left-sided weakness and numbness. A computerized tomography (CT) scan of the head revealed a right frontal intracranial hematoma. Magnetic resonance imaging, pre- and postcontrast, confirmed this finding and also demonstrated a thrombosed central vein. There was no evidence of arterial-venous malformation (Fig. 1A). Her body temperature was 38.2°C. Her leukocyte count was 13,100/mm^3 with 85% neutrophils. The patient underwent craniotomy and drainage of a hematoma. Ceftriaxone and nafcillin were given intravenously. Eight days following the initial operation, the patient developed increased left-sided weakness, with fever (temperature, 38.6°C) and leukocytosis (20,800 leukocytes/mm^3, 90% neutrophils). Increased cerebral edema with a midline shift was found by CT scan; an enhancing lesion was apparent with CT scan with contrast. The second craniotomy revealed purulent material. A few polymorphonuclear neutrophils, but no microorganisms, were detected by Gram stain. The specimen was processed for culture. The infection site was debrided and irrigated with bacitracin. Hemovac drains were placed to allow infusion of antibiotics (2 g of cefazolin per liter at an infusion rate of 50 ml per h). Ceftriaxone and metronidazole were given intravenously. The patient continued to have fever (temperature, 38.6 to 40.3°C) and leukocytosis (15,600 to 21,200/mm^3). The patient's symptoms and neurodeficit continued to worsen. A third craniotomy was performed and the bone flap was removed. An estimated 70 ml of straw-colored fluid was present. The dura was released, opened, and reinforced. The galea was loosely sutured and the skin was stapled. The patient recovered slowly and was dismissed for rehabilitation.

Significant laboratory findings included a low protein C activity of 36% (reference range, 72 to 136%). Histopathology findings of the brain tissue obtained during the first operation demonstrated a moderate, acute perivascular inflammatory infiltrate involving the small vascular channels of the meninges. There was associated hemorrhaging and small amounts of necrotic tissue in these areas of neutrophilic inflammation (Fig. 1B). Multiple blood cultures for bacteria and fungi were all negative. The brain abscess fluid obtained during the second and third operations was cultured. At 4 days, tiny pinpoint colonies were observed on Centers for Disease Control anaerobic agar incubated under anaerobic conditions. The organism also grew in thioglycollate broth at 5 days. The organism was subcultured to SP-4 broth for 24 h and then plated on SP-4 agar plates, which were incubated under anaerobic conditions (GasPak) at 37°C for 24 h. *M. hominis* was identified with conjugated antiserum specific to this organism in an epi-immunofluorescence test (5). The organism (from specimens obtained during two operations) was also recognized by monoclonal antibody 8B1.2 to *M. hominis* as demonstrated by an immunoblotting assay (4). To detect the host antibody reaction to this organism, three plasma specimens from the patient, obtained 13, 29, and 60 days after onset of the disease, were analyzed by the metabolic inhibition test and an immunoblotting test. The patient produced a strong (≥256-fold) specific antibody response to *M. hominis* as measured by the metabolic inhibition test (Table 1). Similarly, all three plasma specimens contained antibodies (29- and 60-day specimens showed identical results) to both the *M. hominis* reference strain PG21 and the isolate from the patient, as demonstrated by immunoblotting (Fig. 2). Previous analyses of acute- and convalescent-

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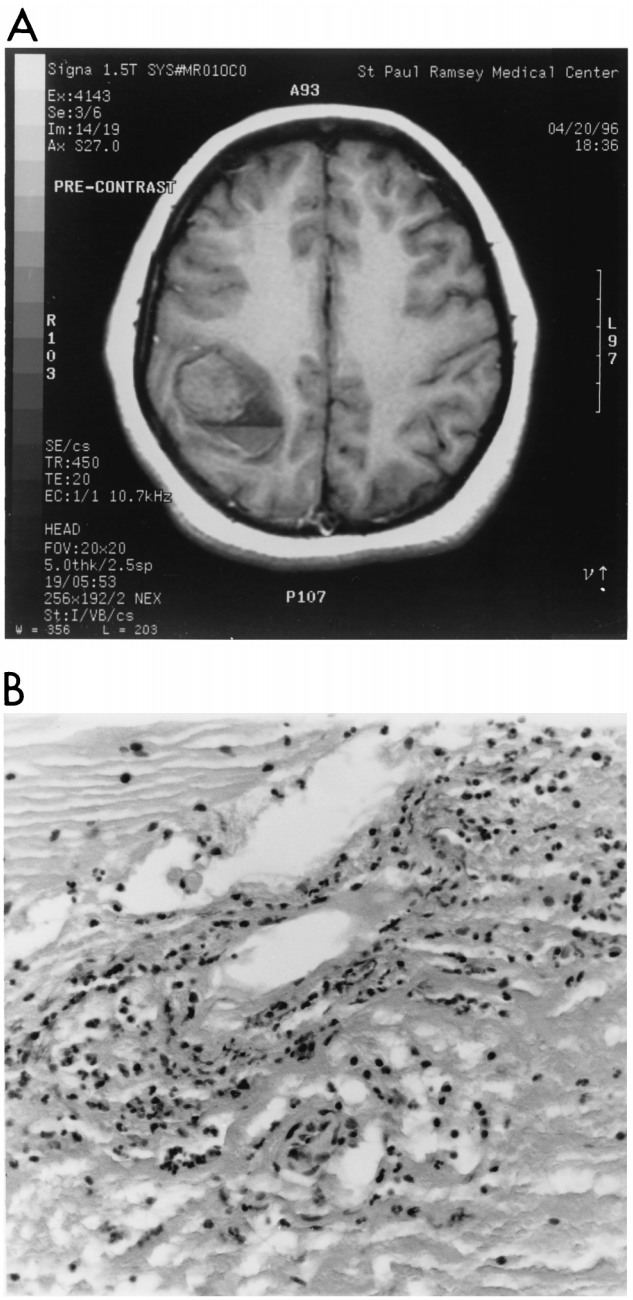

FIG. 1. (A) Results of precontrast magnetic resonance imaging, demonstrating an extensive right frontal-parietal hematoma. (B) Hematoxylin-and-eosin-stained brain tissue. Shown are vascular channels adjacent to right frontal-parietal hematoma with neutrophilic inflammatory response (magnification, ×400).

phase sera from patients with culturally proven invasive *M. hominis* infections demonstrated that four specific *M. hominis* antigens were recognized by at least 80% of the patient's antibodies (4). Of these four antigens, the one migrating at 102 kDa was the most frequently recognized (95%) and is the best indicator of *M. hominis* infection.

Discussion. *M. hominis* has been associated with both urogenital and extragenital infections (9, 11), including postpartum fever (14, 19), septicemia (15, 17), neonatal central nervous system infections (21), sternal and wound infections (13), and abscesses (1, 12). Recently, experiments using chimpanzees demonstrated the pathogenicity of *M. hominis* (2). Host predisposing factors such as immunosuppression, malignancy, trauma, and manipulation or surgery of the genitourinary tract are considered to be associated with extragenital infections.

TABLE 1. Serologic response of patient to *M. hominis*

Specimen	Time of specimen collection (days)	Titer with test antigen:	
		Type strain PG21	Patient isolate
Patient plasma	13	<1:16[a]	<1:16
	29	1:512	1:1,024
	60	1:128	1:256
Anti-PG21 control serum	NA[b]	>1:4,096	>1:4,096

[a] Dilution of plasma.
[b] NA, not applicable.

To date, there has been only one report documenting a case of brain abscess caused by *M. hominis* (12). The patient was infected by the organism after a surgical procedure to manage a subdural hematoma caused by trauma. In our case, *M. hominis* was isolated as a pure culture twice (from four specimens) and identified specifically by an immunofluorescence test and an immunoblot assay with monoclonal antibodies.

The actual mechanism of the infection was not clear. It is possible that the protein C deficiency caused a hypercoagulable state and, subsequently, the formation of a hematoma, where transient bacteremic seeding occurred. It is recognized that protein C deficiency is an inherited characteristic (6). Interestingly, the protein C activity of the patient's mother was 44%.

The initial source of the organism was most likely the genital tract. Although a genital specimen for culture was not obtained at that time, this assumption is supported by the evidence that the infection was postpartum and that the histopathology of the brain tissue from the first operation demonstrated an acute inflammatory infiltrate. Furthermore, detection of specific an-

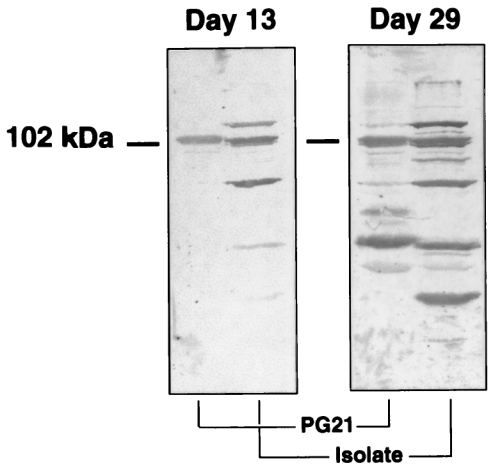

FIG. 2. Immunoblot showing the reaction of patient plasma specimens with *M. hominis* antigens. Ten micrograms of organism protein per well was separated by sodium dodecyl sulfate-polyacrylamide gel electrophoresis and transferred to nitrocellulose, and immunological reactions were performed and visualized as described previously (4). The plasma specimens used were obtained from the patient 13 or 29 days after onset of disease and were diluted 1:100 for the immunoblotting procedure. Lanes: 1, *M. hominis* type strain PG21; 2, isolate from the patient. A protein migrating at 102 kDa is indicated in the figure and is the antigen most commonly recognized by *M. hominis*-infected individuals (4).

tibodies to *M. hominis* in the patient's plasma also supports acute infection with this bacterium.

Because of the length of time required to identify the organism, the antibiotics used in this case were not those considered most effective against *M. hominis* (8). Clinical experience has indicated that many infections with *M. hominis* resolve either spontaneously or by drainage and debridement of the infected focus without specific antimicrobial therapy. However, appropriate antimicrobial therapy is recommended for invasive infection of the blood and the central nervous system by *M. hominis* (7). Mycoplasmas do not possess a cell wall and do not synthesize folic acid. Therefore, they are resistant to antibiotics that inhibit cell wall synthesis or interfere with folic acid synthesis, such as sulfonamides (8). Although mycoplasmas are susceptible to protein synthesis inhibitors and DNA gyrase-inhibiting agents, antibiotics are usually not considered to be mycoplasmacidal. *M. hominis* is usually resistant to erythromycin, so tetracyclines are considered to be the drugs of choice for treatment of these infections. However, the increasing prevalence of tetracycline-resistant strains of *M. hominis* in humans has suggested that clindamycin might be an alternative choice for treatment.

This case again emphasizes the pathogenic potential of *M. hominis*, the need for the prompt clinical recognition of such infections, and the institution of rapid and appropriate chemotherapy in the early stages of the infection.

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