formed and revealed a large adenomatous polyp in the sigmoid that was removed endoscopically.

Because we were interested in the biotype of the strain, it was sent to a reference laboratory, which typed it as *Streptococcus galloyticus* subsp. *galloyticus*. A quick search of literature revealed that *Streptococcus galloyticus* is the new name for *S. bovis* biotype 1 [2].

Shortly after the first patient was admitted to the hospital, another patient was admitted with signs of aortic valve endocarditis. Streptococci were cultured from the blood, and this time the strain was directly identified as *S. galloyticus* (with use of a Vitek 2 system; bioMérieux) and was later differentiated as *S. galloyticus subsp. galloyticus* by the same reference laboratory. Further analysis of the patient showed adenocarcinoma of the ascending colon.

The new nomenclature for the so-called *Streptococcus equinus*/*S. bovis* complex is based on DNA-DNA reassociation experiments by which 4 different DNA clusters are identified. Group II contains 3 *S. galloyticus* subspecies [3], including the one isolated from our patients. We were not aware of this new nomenclature, nor were several of our colleagues (both microbiologists and infectious diseases specialists). Lack of awareness of this change can lead to underdiagnosis of serious underlying conditions, including colon carcinoma. If it had not been for the first patient, we would almost certainly not have performed a colonoscopy for the second patient.

We therefore propose that isolation of *S. galloyticus* subsp. *galloyticus* be reported to clinicians along with the old name of the organism, *S. bovis*, to prevent life-threatening omissions in the diagnostic work-up of patients. Because the other newly proposed *S. galloyticus* subspecies, *S. galloyticus subsp. pasteurianus* and *S. galloyticus subsp. macedonius*, also include some of the “old” *S. bovis* strains, the same advice can be applied for those subspecies.

In other words, it is all right to call Romeo by another name, as long as we all know that we are referring to the young nobleman that is associated with Juliet.

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**Cytomegalovirus Hepatitis Associated with Use of Anti-Tumor Necrosis Factor—α Antibody**

Sir—We read with interest the report by Haerter et al. [1] of cytomegalovirus (CMV) retinitis in a patient receiving infliximab for treatment of rheumatoid arthritis. Here, we present what is, to our knowledge, the first case of CMV hepatitis associated with this agent.

A 45-year-old woman with a history of Crohn disease had been treated with 6-mercaptopurine at a dosage of 50 mg/day for the past 8 years until the fall of 2002, when intravenous infliximab (5 mg/kg body weight), administered at 4–8 week intervals, was added to the regimen for a better control of the disease. She had also been receiving long-term treatment with prednisone (10 mg daily) and levophenoxine replacement therapy for Sheehan syndrome. In October 2003, the patient was admitted to our hospital with a 4-week history of daily fever and chills unresponsive to several courses of empirical antimicrobial therapy. She denied gastrointestinal symptoms.

At admission, the patient’s temperature was 39.7°C, her blood pressure was 100/66 mm Hg, her pulse was 112 beats/min, and her respiratory rate was 18 breaths/min. She appeared fatigued; however, physical examination failed to detect focal abnormalities. Laboratory tests yielded the following values: WBC count, 2500 cells/mm³, with 89% neutrophils and 10% lymphocytes; CD4⁺ cell count, 170 cells/mm³; hemoglobin, 10.2 g/dL; platelet count, 149,000 platelets/mm³; aspartate aminotransferase, 468 U/L; alanine aminotransferase, 282 U/L; alkaline phosphatase, 845 U/L; and total bilirubin, 1.1 mg/dL. CT showed mild hepatomegaly without abnormalities of the biliary tract. The results of the following studies were unremarkable: blood culture, peripheral blood smear, tests for antinuclear antibodies, and tests for antibodies against hepatitis viruses, human immunodeficiency virus, *Rickettsia rickettsii*, and *Anaplasma phagocytophilum*. Finally, histological examination of a liver biopsy specimen revealed intranuclear inclusion bodies, confirmed as CMV by paraffin immunoperoxidase staining with antibody against this virus. Levels of anti-CMV IgM and IgG were elevated. After treatment with intravenous ganciclovir, 5 mg/kg twice daily, the patient’s symptoms and laboratory values improved within a few days.

To date, CMV hepatitis after the administration of anti-TNF-α antibodies has not been reported. As the indications for use of these antibodies increase, clinicians need to be aware of CMV infection as one of the potential complications of treatment with anti-TNF-α antibodies.
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References


