**M. tuberculosis** belonging to SIT 52 that caused tuberculosis meningitis was reported in a human in Thailand (10), but that case was not related to the case reported here. Our finding of a relatively novel spoligotype of *M. tuberculosis* in an animal destined for the pet trade underscores the need for intensive testing of and extended quarantine for all imported nonhuman primates to prevent the spread of newly isolated *M. tuberculosis* (4,7,8).

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**Treatment of Mycobacterium abscessus subsp. massiliense Tricuspid Valve Endocarditis**

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**To the Editor:** *Mycobacterium abscessus* is a ubiquitous, rapidly growing mycobacteria (RGM) found in water supplies, soil, and dust. *M. abscessus* is considered the most pathogenic and difficult to treat of the RGM and is most often associated with pulmonary, skin, and soft tissue infections; it has also been reported to cause ocular infections, otitis, lymphadenitis, arthritis, osteomyelitis, disseminated disease, and prosthetic valve endocarditis (1,2). Most prosthetic valve endocarditis cases have been fatal.

*M. abscessus* subsp. *massiliense* is 1 of 3 subspecies of *M. abscessus*. *M. abscessus* subsp. *massiliense* has an identical 16S rRNA gene sequence to the other 2 subspecies, *Mycobacterium abscessus* subsp. *bolletii* and *Mycobacterium abscessus* subsp. *abscessus*, but can be differentiated by rpoB and erm41 gene sequencing (3,4). *M. abscessus* subsp. *massiliense* grows readily in blood culture media and on sheep’s blood agar within 2–4 days. Care should be taken in interpreting Gram staining of isolates because RGM is not identifiable by this method and could be mistaken for corynebacteria or diphtheroids (5,6). Such isolates could be further tested by acid-fast staining and, if positive, sent to a reference laboratory for definitive identification and susceptibility testing.

Five cases of *M. abscessus* native valve endocarditis have been reported; 4 were fatal and 1 was lost to follow-up (1,5–9). One of the 4 fatal cases also involved the tricuspid valve and was associated with intravenous heroin abuse.
and lingular nodules/infiltrates. Tigecycline, amikacin, showed nearly complete resolution of the RUL cavitary treated isolates are susceptible to the macrolides (truncated) macrolide-inactivating gene (erm41)

**(10)** and 14-day susceptibility to clarithromycin quencing (erm by hsp65 gene se-

A 52-year-old man who used intravenous drugs was admitted to our hospital describing a 25-pound weight loss, fever, and night sweats. He reported injecting crushed opioid tablets mixed with tap water. He had tachycardia and pitting edema of the legs and feet. Laboratory data revealed elevated aminotransferase levels, thrombocytopenia, and opiates in the urine. Computerized chest tomographic scan showed cavitary right upper lobe and lingular nodules. Routine blood cultures (BacT/ALERT3D; bioMérieux, Marcy l’Etoile, France) of samples drawn at admission and on hospital day 3, before the initiation of antimicrobial drug therapy, grew acid-fast bacilli (AFB) in broth medium on days 3 and 4 of incubation. A transthoracic echocardiogram on hospital day 5 revealed a 1-cm vegetation on the tricuspid valve. An empiric regimen for RGM consisting of intravenous cefoxitin and amikacin and oral clarithromycin and moxifloxacin were administered. Based on preliminary (3-day) susceptibility test results showing susceptibility to amikacin, resistance to the quinolones, and intermediate susceptibility to cefoxitin, linezolid, and imipenem, the regimen was changed to tigecycline, linezolid, clarithromycin, and amikacin (10). Routine blood cultures on hospital days 11 and 17 were negative.

On hospital day 19, linezolid was stopped, and imipenem was added. A transthoracic echocardiogram on hospital day 31 showed the vegetation had enlarged to 1.5 \( \times \) 0.5 cm. We concluded that antibiotics alone were unlikely to be curative; cardiac catheterization was performed on hospital day 38. On the basis of hemodynamic findings, the cardiologist inferred that valve replacement would be of no value and recommended valvectomy alone.

Surgery on hospital day 41 revealed a 2-cm nodule on each anterior and posterior leaflet and a 2–3 mm nodule on the septal leaflet of the tricuspid valve. The anterior and posterior leaflets were removed, and the septal leaflet was segmentally resected. Routine cultures of valve tissues, in which *M. abscessus* would have grown, were negative. Pathologic examination confirmed suppurative vegetations with numerous bacterial colonies consistent with AFB; AFB staining disclosed numerous mycobacteria (Figure, http://wwwnc.cdc.gov/EID/article/21/3/14-0577-F1.htm).

Identification and final susceptibilities of the RGM from the original blood culture isolate revealed *M. abscessus* subsp. *massiliense* by hsp65 PCR and *erm* gene sequencing (4) and 14-day susceptibility to clarithromycin (10). *M. abscessus* subsp. *massiliense* has a nonfunctional (truncated) macrolide-inactivating gene (*erm41*), and untreated isolates are susceptible to the macrolides (4).

Repeat chest tomographic scan on hospital day 69 showed nearly complete resolution of the RUL cavitary and lingular nodules/infiltrates. Tigecycline, amikacin, imipenem, and clarithromycin were continued until hospital day 77, when amikacin was stopped because of moderate hearing loss. The patient was discharged without antibiotics after 2 months of postoperative antibiotic therapy. At follow-up visits 2 and 8 weeks later he was doing well except for peripheral edema. AFB and routine blood cultures drawn at both visits were negative. He is periodically seen in the cardiology clinic; his edema has resolved with diuretic therapy.

Cure of *M. abscessus* native valve endocarditis has not been previously reported. A case of *M. chelonae* native tricuspid valve endocarditis associated with a pacemaker lead was successfully treated with wire removal, valve debridement, and antimicrobial therapy (11). The patient in the current study likely acquired his infection from the tap water diluent he injected. Clinicians should consider the possibility of mycobacterial endocarditis when evaluating a septic patient with intravenous drug use history or cardiac prosthetic devices.

We successfully treated mycobacterial native tricuspid valve endocarditis with combination antimicrobial therapy and surgical debridement. The location of the infection in the tricuspid valve and favorable hemodynamics enabled debridement without implantation and the subsequent possibility of intraoperative infection of a prosthetic valve.

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To the Editor: Rickettsia rickettsii is the etiologic agent of Rocky Mountain spotted fever (RMSF), a highly lethal tick-borne rickettsiosis restricted to the Western Hemisphere (1,2). In Colombia, R. rickettsii was first reported during the 1930s, when 62 (95%) of 65 affected persons died of RMSF in Tobia town (Cundinamarca Department) (3), from where highly virulent strains of R. rickettsii were isolated through the inoculation of patient blood or of Amblyomma cajennense sensu lato (s.l.) extracts into guinea pigs (4). Thereafter, RMSF remained unnoticed in Colombia until the 21st century, when new outbreaks with high case-fatality rates were reported in different regions, including Villete, a nearby locality of Tobia (1).

Recent studies have shown that A. cajennense s.l., widely distributed from the southern United States to Argentina, is actually a complex of 6 different species: A. cajennense sensu stricto (Amazonian region), A. mixtum (from Texas, USA, to western Ecuador), A. sculptum (northern Argentina, Bolivia, Paraguay, Brazil), A. interandinum (inter-Andean valley of Peru), A. tonelliae (dry areas of northern Argentina, Bolivia, and Paraguay), and A. patinoi (eastern cordillera of Colombia) (5). With this new classification, A. patinoi, originally described from Villete, is the only species of this complex known to occur in the RMSF-endemic area of Cundinamarca, Colombia (5).

In August 2013, we collected 15 A. patinoi adult ticks from cattle in Naranjal village (5°33′31.52″N, 74°26′50.24″W), Villete town, an area of Cundinamarca, Colombia, to which RMSF is endemic. Ticks were taken alive to the laboratory, where they were frozen at –80°C for further analysis. The 15 ticks were defrosted, surface sterilized with iodine alcohol, and processed individually by the shell vial technique for isolation of rickettsiae in Vero cells, as described (6). Infected cells were always incubated at 28°C. Rickettsiae were observed by Gimenez staining within cells (online Technical Appendix Figure 1, http://wwwnc.cdc.gov/EID/article/21/3/14-0721-Techapp1.pdf) from only 1 (inoculated from a female tick) of the 15 inoculated shell vials. This isolate was subjected to at least 7 Vero cell passages, each achieving >90% infected cells.

DNA was extracted from an aliquot of first passage–infected cells and tested by a battery of PCR protocols targeting fragments of the rickettsial genes gltA, ompA, and ompB and the intergenic regions RR0155-rpmbR, RR1240-tlc5′, and cspA-ksgA (Table). We sequenced 1,106 bp, 512 bp, and 799 bp of the gltA, ompA, and ompB genes, respectively. By BLAST analyses (http://www.ncbi.nlm.nih.gov/blast), these sequences were 100% identical to corresponding sequences of R. rickettsii from Colombia and Brazil (GenBank accession nos. CP003306, CP003305). Generated sequences for 2 intergenic regions, RR0155-rpmbR (228 bp) and RR1240-tlc5′ (306 bp), were 100% identical to corresponding sequences of the same 2 R. rickettsii isolates from Colombia and Brazil. A 337-bp sequence of the cspA-ksgA intergenic region was 100% (337/337 nt) identical to R. rickettsii from Brazil (CP003305) and 99.7% (336/337) to R. rickettsii from Colombia (CP003306). Partial sequences from R. rickettsii generated in this study were deposited into GenBank and assigned nucleotide accession nos. KJ735644–KJ735649.

Whole-body remnants of the 15 ticks used to inoculate shell vials were also subjected to DNA extraction and processed by PCR for the rickettsial gltA gene (Table); only 1 tick (the one that provided the rickettsial isolate) contained rickettsial DNA, indicating a 6.6% (1/15) infection rate. We confirmed the taxonomic identification of this