

strains but was loosely related to genogroup 1, the basis of the vaccine used for inoculation against Korean PEDV strains. This isolate may need further evaluation as a candidate for a vaccine to prevent reemerging PEDVs in South Korea.

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Tickborne Lymphadenopathy Complicated by Acute Myopericarditis, Spain

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To the Editor: *Dermacentor*-borne necrosis erythema lymphadenopathy/tickborne lymphadenopathy (DEBONEL/TIBOLA) is an apparently benign, self-limiting rickettsial disease transmitted by *Dermacentor* ticks (1,2). *Rickettsia slovaca* was the first etiologic agent isolated, but other species, such as *R. raoultii* and *Candidatus R. rioja*, also might be involved (3–6). If the scalp is affected, a larger number of agents (including *Francisella tularensis*, *Bartonella henselae*, *R. massiliae*, *R. sibirica mongolitimonae*, and *Borrelia burgdorferi*) should be considered within the differential diagnosis of a similar syndrome recently named scalp eschar associated with neck lymphadenopathy after a tick bite (SENLAT) (7). Nevertheless, in Spain, only *R. slovaca*, *Candidatus R. rioja*, and *F. tularensis* are known to cause DEBONEL/TIBOLA/SENLAT (4,6). This entity is considered an emerging rickettsiosis in Europe; cases have been reported from Italy, France, Hungary, Germany, and Portugal (8).

We recently saw a patient in whom acute myopericarditis developed after he was bitten by a large tick on the scalp and showed clinical signs of DEBONEL/TIBOLA/SENLAT, most likely attributable to *R. slovaca* or *Candidatus R. rioja* infection. The patient, a previously healthy 28-year-old man, went on a day-long hiking trip to the northern mountains of Madrid (central Spain; mean altitude 1,300 m) on November 2, 2014. Three days later, he noticed a mild ache on the occipital area of his scalp and found an attached tick that he removed with his fingers. A week later, he sought care from an infectious disease specialist because of itchy discomfort at the area of the tick bite.

Examination revealed an erythematous and elevated punctiform lesion with mild fluctuation in the occipital region accompanied by tender, small lymph node enlargement of both occipital lymphatic chains (Figure). No widespread rash was present. DEBONEL/TIBOLA/SENLAT was diagnosed, and doxycycline (100 mg every 12 hours) was initiated. IgG titer against spotted fever group *Rickettsia*



Figure. *Dermacentor*-borne necrosis erythema lymphadenopathy/tickborne lymphadenopathy/scalp eschar associated with neck lymphadenopathy after a tick bite. Shown is an erythematous, punctiform lesion in the scalp (arrow), accompanied by enlarged occipital lymph nodes. A color version of this figure is available online (<http://wwwnc.cdc.gov/EID/article/21/12/15-0672-F.htm>).

(SFGR) was 1:128. Four days later, the patient sought care at an emergency department, reporting retrosternal chest pain. Electrocardiogram revealed a diffuse ST-segment elevation with PR-segment depression; serum creatine phosphokinase and troponin T levels were 327 IU/L (reference range 10–190 IU/L) and 420 ng/mL (reference <14 ng/mL), respectively. Myopericarditis was diagnosed. A transthoracic echocardiogram ruled out pericardial effusion, valve vegetations, and left ventricular dysfunction; cardiovascular magnetic resonance imaging performed 4 days later showed myocardial inflammation. Blood cultures were sterile, pneumococcal urinary antigen test result was negative, and IgM against coxsackievirus and *Mycoplasma pneumoniae* were not detected. Nonsteroidal antiinflammatory drugs were prescribed. The patient improved clinically, and electrocardiogram findings resolved. The patient received doxycycline for 4 weeks.

On a convalescent-phase serum specimen collected after 8 weeks, indirect immunofluorescence assays (IFA) for IgG against SFGR were performed in Spain's national reference center for rickettsioses (Hospital San Pedro—Centro de Investigación Biomédica de La Rioja [CIBIR], Logroño, Spain). Commercial (Focus Diagnostics, Cypress, CA, USA) and in-house *R. conorii*, *R. slovaca*, and

R. raoultii antibody testing showed an IgG titer of 1:512 against the 3 species. A subsequent cross-adsorption assay using *R. slovaca*, *R. raoultii*, and *R. conorii* antigens prepared on the basis of strains from the collection at Hospital San Pedro-CIBIR showed a decrease in IgG titers against *R. conorii* and *R. raoultii* to 1:64 and 1:256, respectively, whereas titer against *R. slovaca* remained at 512. IFA against *Bartonella* spp. and *C. burnetii* (Focus Diagnostics), chemiluminescence immunoassay for *B. burgdorferi* (Liason, DiaSorin, Spain), and in-house microagglutination assay for *F. tularensis* were not reactive. The patient recovered, with only a residual scarring alopecia on the occipital region of the scalp and without cardiac dysfunction after 9-month follow-up.

Myopericarditis is a rare complication of rickettsiosis, usually associated with *R. rickettsii* and *R. conorii* (9). Although tetracycline-induced cardiac adverse reactions have been described (10) and the patient reported here had signs of myopericarditis shortly after the initiation of doxycycline, he completed a 4-week treatment without recurrence. Therefore, the clinical picture seems unlikely to be attributable to doxycycline-induced toxicity. Because the patient was bitten in November (when only *Dermacentor* spp. ticks are active in central Spain), we have further epidemiologic evidence for attributing the infection to SFGR causing DEBONEL/TIBOLA/SENLAT. After serum adsorption, IFA titer against *R. slovaca* was 3-fold higher than that against *R. conorii*. *R. slovaca* and *Candidatus R. rioja* are the species most commonly found in *D. marginatus* ticks and in cases of DEBONEL/TIBOLA/SENLAT in Spain (8).

In view of the seroconversion to *Rickettsia* spp. with negative test results for other possible causative agents and the clinical response to doxycycline, rickettsiosis caused by *R. slovaca* or *Candidatus R. rioja* remains the most probable diagnosis. Because DEBONEL/TIBOLA/SENLAT is an emerging disease, physicians should consider that this entity may be associated with systemic complications similar to those of other tickborne rickettsioses.

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Parainfluenza Virus 5 as Possible Cause of Severe Respiratory Disease in Calves, China

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To the Editor: Parainfluenza virus 5 (PIV5), family *Paramyxoviridae*, genus *Rubulavirus*, was previously known as simian virus 5 because of its discovery in primary

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monkey kidney cells in 1954 (1). PIV5 was later isolated from various hosts, including humans, dogs, pigs, cats, and rodents. The neutralizing antibody for PIV5 is detectable in symptomatic and asymptomatic humans; thus, its association with human disease remains controversial (2). In addition, previous studies have not documented illness in infected animals, except kennel cough in dogs (1,3). Isolation of PIV5 from cattle has not previously been reported.

Since 2012, an infectious respiratory disease has been prevalent in weaning calves (≈10 d to 2 mo of age) in Baicheng City, Jilin Province, China. Initial clinical signs included secretion of clear nasal mucus, anorexia, sluggishness, and loss of body weight. After 10–20 d, ≈10% of the sick calves died of dyspnea and interstitial pneumonia. Farmers observed that 80%–90% of calves in the affected farms demonstrated clinical signs, but most recovered. All attempts of local veterinarians to treat the animals with various chemical compounds and antimicrobial drugs failed. The disease persists throughout the year but occurs mainly during spring (from February through March), which has resulted in substantial economic losses in the cattle industry.

To identify the causative agent of the disease, we tested 15 lung samples from calves that had died and 10 lung samples from healthy calves that were slaughtered for serum products (all from 1 farm). The samples were homogenized in phosphate-buffered saline and analyzed by using electron microscopy. Paramyxovirus-like particles were identified in the tissues of sick calves. Reverse transcription PCR with the generic primers for the paramyxovirus polymerase gene was performed (4).

Of the 25 calf specimens, the 15 samples from the sick calves were positive by reverse transcription PCR, and amplicons of the expected size were obtained and sequenced. The generated sequences were closely related to the PIV5 sequences available from GenBank, particularly to sequences of the recently identified KNU-11 and SER viruses in pigs (5,6).

The suspensions of lung tissue from sick calves were purified by centrifugation at 12,000 × *g* for 5 min, and 0.2 mL of the supernatant was added to a Vero cell monolayer in a 25-cm² cell culture flask (EasyFlasks; Thermo Fisher Scientific, Odense, Denmark). After virus adsorption for 1 h at 37°C, the cell monolayer was rinsed with phosphate-buffered saline (pH 7.4) and then incubated in Dulbecco minimal essential medium/2% newborn calf serum at 37°C in a 5% CO₂-humidified incubator. The infected cells were serially passaged every 3 days at 37°C and detected by using monoclonal antibody against SV5 (AbD Serotec; Bio-Rad, Kidlington, UK) by indirect fluorescent antibody test (7). A PIV5 strain was isolated in the cell culture and designated PIV5-BC14 (BC14 stands for Baicheng City 2014).

For amplification and analysis of the full-length viral genome, 13 pairs of primers covering overlapping