

Three of the four cases of congenital TB appeared in mothers who had remained undiagnosed during pregnancy and one in a patient with miliary and meningeal TB with late gestational infection. The risk of transmission is higher in mothers with undiagnosed extrapulmonary TB (especially genital TB) or diagnosed in the last month of pregnancy or in the puerperium. Neonatal TB has high morbidity and mortality rates and requires a high degree of suspicion, as the clinical presentation tends to be atypical. A maternal history of immunosuppression, latent tuberculosis infection or TB before or during pregnancy, constitutional symptoms, productive cough, recurrent miscarriages or tubal infertility are crucial as support for the diagnostic process. Various studies show a better prognosis for newborns when treatment is started aggressively and at an early stage.^{9,10}

Our aim is for this letter to serve as a reminder that gestational and congenital TB are still present in our environment and of the importance of considering these conditions in the differential diagnosis in our day-to-day.

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Paula Rodríguez Molino^{a,*}, María de la Calle^b, Teresa del Rosal^a, Fernando Baquero-Artigao^a

^a Servicio de Pediatría Hospitalaria, Enfermedades Infecciosas y Tropicales, Hospital Universitario La Paz, Madrid, Spain

^b Servicio de Obstetricia y Ginecología, Hospital Universitario La Paz, Madrid, Spain

* Corresponding author.

E-mail address: paularmolino@gmail.com (P. Rodríguez Molino).

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Tubular acute lymphangitis caused by *Rickettsia sibirica mongolitimonae*[☆]



Linfangitis aguda tubular por *Rickettsia sibirica mongolitimonae*

Acute lymphangitis is an acute inflammatory process in healthy or anatomically altered lymphatic vessels in either immunocompetent or immunocompromised patients. It generally develops after inoculation of microorganisms into the lymphatic vessels through a skin defect or as a complication of a distal infection.¹ It is commonly caused by *Streptococcus pyogenes* and *Staphylococcus aureus*; in immunocompromised patients, Gram-negative bacteria; *Pasteurella multocida* or *Spirillum minus* after dog, cat or rat bites. Less common culprits are mycobacteria, viruses, fungi, parasites, *Rickettsias* or non-infectious causes.¹ Clinically speaking, acute lymphangitis may be: reticular, tubular, nodular, phlyctenular or necrotising.

We report here a case of tubular acute lymphangitis caused by *Rickettsia sibirica mongolitimonae* in Valencia, Spain.

This was a 59-year-old man with no relevant medical history who consulted in April 2019 with a four-day history of pre-syncope, general malaise, headache, arthralgia, myalgia and fever of 38.5 °C. Three days before the onset of his symptoms, he developed an erythematous papule on his right leg. In the previous 48 h it had become enlarged so, suspecting cellulitis, he was started on amoxicillin/clavulanic acid 500 mg/8 h. Despite the antibiotic, the lesion

had worsened, spreading to the knee, and he had developed painful enlarged right inguinal lymph nodes.

He stated that he had not travelled outside Valencia. He had been in the countryside in an area with pigeons, rabbits, cats and dogs.

On physical examination, he was found to have a temperature of 37.6 °C, the lesions shown in Fig. 1 and left inguinal lymphadenitis. He had no peripheral lesions or rashes. The only blood test finding was CRP 35 mg/l. Urinalysis and chest X-ray were normal.

The appearance of the eschar raised the suspicion of rickettsiosis and he was started on treatment with oral doxycycline 100 mg/12 h for five days. Within 48 h his temperature had returned to normal and the rest of the symptoms and lesions gradually disappeared.

For the aetiological diagnosis, serological chemiluminescence immunoassay (CLIA) (Viracell, Granada, Spain) was performed for *Rickettsia conorii*, *C. burnetti* and *B. burgdorferi*, which were negative. A sample of exudate was obtained from the eschar, and Real-Time PCR (Fast Track Diagnostic, Luxembourg) detected *Rickettsia* spp DNA. Definitive species identification was obtained by molecular sequencing of a 525bp fragment of the *ompA* gene, which confirmed the presence of *Rickettsia sibirica mongolitimonae* (99.7% similarity with sequences deposited in GenBank®).² The serology was repeated at six weeks using CLIA and indirect immunofluorescence (IIF) (Viracell, Granada, Spain): IgG and IgM were positive against *Rickettsia conorii*, with evidence of cross reactions between *R. sibirica* and *R. conorii*.³

Acute tubular, trunk- or branch-like lymphangitis affects the main superficial branches of the lymphatic vessels. An erythematous plaque appears and an indurated, erythematous, warm, palpable and painful sinuous tubular cord develops (Fig. 1), extending proximally to the regional lymph nodes (lymphadenitis). It is usually accompanied by systemic symptoms: chills, fever, general malaise, headache, arthralgia, myalgia and nausea and/or vomiting. Laboratory tests may show leucocytosis, increased inflammatory

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Fig. 1. Eschar with surrounding erythema on the right leg. Erythematous cord of trunk-like lymphangitis from the eschar to the knee.

parameters, and positive blood cultures.¹ It generally responds to empirical oral antibiotics. Infants and small children may require hospital admission in the case of significant systemic involvement or antibiotic resistance. Further exploratory tests are not usually necessary, unless there is a lack of response or atypical aetiology is suspected.¹

The presence of lymphangitis, fever and an inoculation eschar should make us suspect lymphangitis-associated rickettsiosis (LAR)⁴ and start treatment with doxycycline. LAR do not have a specific clinical-aetiological correlation, and can be caused by different species of *Rickettsiae*.^{5,6} One of these is *R. sibirica mongolitimonae*, which is involved in less than half of the published cases of LAR.^{6,7} This is a rare type of rickettsiosis,⁸ which causes lymphangitis in approximately 35% of cases.⁹ It tends to be transmitted in spring and summer by tick bites of the genera *Rhipicephalus* and *Hyalomma*.⁵ We confirmed a case of lymphangitis due to *R. sibirica mongolitimonae* by PCR analysis of the eschar sample. This technique allows the definitive diagnosis of rickettsiosis with precision,

speed and high sensitivity and specificity.¹⁰ Serology requires 2–3 weeks for seroconversion and shows cross-reactions, as in our case. The existence of cross reactions means that *R. sibirica mongolitimonae* is probably underdiagnosed, not only as a cause of LAR, but also of other types of rickettsiosis, as the microbiological diagnosis is based mainly on serology, and in many cases it is only performed against *R. conorii*.

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Sonia Miguélez Ferreiro^{a,*}, David Navalpotro Rodríguez^b

^a Servicio de Urgencias de Atención Primaria, Hospital General de Valencia, Valencia, Spain

^b Servicio de Microbiología, Hospital General de Valencia, Valencia, Spain

* Corresponding author.

E-mail address: tormentabranca@hotmail.com (S. Miguélez Ferreiro).

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Recovery of *Chlamydia trachomatis* and other pathogens in urine from men with sterile pyuria attended in an emergency department[☆]



Recuperación de *Chlamydia trachomatis* y otros patógenos en la orina de varones con piuria estéril atendidos en urgencias

Urinary tract infection is a rare condition in young men with no predisposing factors. In contrast, sexually transmitted infections (STI) are becoming more common and, with many having an acute onset (haematuria, dysuria, haemospermia, testicular inflamma-

tion and/or epididymitis, etc), these patients often seek help from emergency departments. Various studies suggest that STI play a central role in the origin of sterile pyuria in young men^{1,2}, and our aim in this study was to find out if this was true in our environment.

From May 2014 to July 2017, we looked for microorganisms related to STI in samples sent from the Accident and Emergency department (A&E) from men aged 15–59 with sterile pyuria of unknown cause. We used urine samples with >14 leukocytes/μl (Sysmex UF-1000i analyser) which had been sent for urine culture and been negative, and the detection of STI-causative pathogens was performed by PCR (STI-7, Seegene, Seoul, South Korea). In cases with severe pyuria, the urine was also spread on chocolate agar (to rule out gonococcus and other bacteria which do not grow in the media usually used for urine culture) and in others, studies were

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