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Gestational and congenital tuberculosis: An ongoing problem☆

Tuberculosis gestacional y congénita: un problema aún vigente

Although the incidence of tuberculosis (TB) has declined considerably in recent years, a significant proportion of those affected are young adults,[1] including women of childbearing potential. In Spain an estimated 39% of cases affect this population group.[2] However, gestational TB is very rare in our setting. In a recent study, only 2/2320 TB cases were diagnosed during pregnancy.[3]

The foetal transmission rate ranges from 0 to 16%, but transmission is very unusual in pregnant women with pulmonary TB who are properly treated before delivery.[4,5] Congenital TB is rare, but very serious, so adequate treatment in the pregnant woman and study and follow-up of the newborn are essential.[6,7]

We carried out a review of gestational and congenital TB in our centre over 12 years (2007–18). We included women with a pre- or post-conception diagnosis of TB who received treatment during pregnancy. We analysed cases of congenital TB according to the Cantwell et al. criteria[8]: Confirmed TB in the infant and at least one of the following: symptoms in the first week of life; demonstration of primary complex or caseating hepatic granulomas; infection of the placenta or maternal genital tract; and exhaustive exclusion of postnatal transmission.

In the end we included 12 women who had been given anti-tuberculosis therapy during pregnancy: five had started treatment before becoming pregnant and the other seven, during their pregnancy (median gestational age at baseline 16 weeks, interquartile range [IQR]: 11.8–20). They were all immigrants, predominantly from Morocco (4) and Latin America (4). Eight (67%) were diagnosed with pulmonary TB, two with lymph node TB and two with miliary and meningeal TB. No resistant isolates were identified.

They were started on induction treatment for two months (90% including pyrazinamide) and then maintenance treatment to complete 6–12 months, with good tolerance and recovery, and without sequelae.

The newborns were asymptomatic, with three being premature and one having low weight. The tuberculin test (n = 11) and the QuantiFERON-TB Gold® test (n = 9) were negative. Imaging tests were normal. Two preterm infants received isoniazid prophylaxis for three months. One child of a mother with tuberculous meningitis diagnosed two weeks before delivery had positive PCR and culture in gastric juice and received anti-tuberculosis therapy, but without ever developing symptoms. The rest of the neonates had negative microbiological studies. All completed follow-up for one year, with negative tuberculin and QuantiFERON-TB Gold® checks at 3, 6 and 12 months.

During the study period, three infants with congenital TB were identified in mothers not diagnosed with TB during pregnancy, two with pulmonary involvement and one with miliary dissemination. One had Spanish parents and the mothers of the other two were Moroccan immigrants. All three were premature and had PCR, smear microscopy and positive culture in gastric juice (2) or bronchial aspirate (1). The mothers were diagnosed by PCR on biopsies of the endometrium in two and the placenta in the other. Two of the infants had been conceived by *in vitro* fertilisation due to tubal infertility. TB was not suspected as the initial diagnosis in any of the three infants (1 respiratory infection, 2 sepsis). All three made good progress with anti-tuberculosis therapy.

Gestational and congenital TB in our environment primarily affects the immigrant population. The possibility of TB should therefore be considered in any immigrant pregnant woman with suggestive symptoms. The classic treatment seems to be safe for the foetus, and makes vertical transmission to the newborn very unlikely. We therefore need to stress the importance of screening programmes in at-risk populations in order to provide early treatment for TB infection and disease in pregnant women.

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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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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) (Vircell, 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) (Vircell, 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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