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https://doi.org/10.1016/j.eimc.2018.08.001
0213-005X/
Published by Elsevier España, S.L.U.

Tuberculosis of the elbow: A rare form of presentation of extrapulmonary tuberculosis

Osteoartritis tuberculosa del codo: forma poco frecuente de presentación de tuberculosis extrapulmonar

Osteoarticular tuberculosis (OTB) remains a major diagnostic and management challenge. Globally, OTB accounts for 2.2–4.7% of all tuberculosis cases in Europe and the USA.[1] The most frequent form of OTB is tuberculous spondylitis or Pott's disease, followed by osteomyelitis.[2] Tuberculous (TB) arthritis, less frequent, mainly affects large weight bearing joints, such as the hip and knee, being the elbow exceptionally affected.

We present a case of a 53-year-old Spanish man who was admitted to the emergency department with a 12 years history of pain and swelling of his right elbow which had worsened over the last six months. He denied previous injury and had no underlying disease. He had received corticosteroid infiltrations, last time two months before consulting. Physical examination revealed inflammation of the posterolateral region of the right elbow with associated distal paraesthesia, wrist drop and complete inability to extend the wrist and move the fingers.

On admission, he was afebrile and acute-phase reactants were slightly elevated. The chest X-ray was completely normal. The ultrasound image of the elbow showed discrete articular effusion and thickening of the articular capsule with hairy and nodular projections. Magnetic resonance imaging showed bone destruction with severe subchondral erosions. Under suspicion of pigmented villonodular synovitis, a biopsy was performed for anatomopathological study, which reported chronic synovitis with necrotizing granulomas and no hemosiderin or malignancy features. *Mycobacterium tuberculosis* (MTB) DNA was detected without mutations in the genes associated with resistance to first and second line antituberculous drugs (ANYPLEX™ II MTB Detection/MDR/XDR, Seegene). Ziehl–Neelsen stain of the biopsy was positive. Latent tuberculous infection was confirmed with both the tuberculin skin test and the IFN-γ release assay (Quantiferon-TB Gold®). Treatment with isoniazid (INH), rifampicin (RIF), ethambutol (EMB) and pyrazinamide (PZA) was started for four months and reduced to INH and RIF for 14 months. After 10 days of treatment, the patient reported a transitory increase of the pain and swelling followed by drainage of semi-liquid caseous material, concordant with fistulization, through the puncture site of the elbow. By then, the liquid medium culture (BD MGIT 960) of the biopsy was positive (10 days). Growth on the solid medium (Löwestein–Jensen) was observed after 14 days of incubation. Isolated non-chromogenic mycobacteria were identified as *M. tuberculosis* by PCR and reverse hybridization (GenoType MTBC, HAIN Lifescience). Susceptibility to first line anti-TB drugs was confirmed by culture in Middlebrook (BD MGIT 960). Whole Genome Sequencing (WGS) of the *M. tuberculosis* strain (Nextera XT protocol and Illumina MiSeq platform) showed no resistance-related mutations and was identified as Lineage 4.10/PGG3, one of the most recently described sublineages within the globally distributed Lineage 4.[3]

The patient progresses favorably a year after treatment completion, with decreased inflammation, resolution of the fistula and recovery of the elbow motility with complete flexion and extension of the right wrist.

OTB follows a progressive course of several months, developing fistulous paths from the cartilage to the skin surface when the infection is advanced. Fever or systemic symptoms are infrequent. Acute-phase reactants are often raised, but levels are lower than those seen in pyogenic vertebral infections.[1] Differential diagnosis includes other subacute or chronic infections such as those caused by *Brucella* spp., *Burkholderia pseudomalle* or *Candida* spp.,[4] together with local or adjacent tissue tumors. Imaging tests are nonspecific but can help to delimit the extent of soft-tissue and bone affection. Anatomopathological examination of the biopsy of the injury might reveal the presence of granulomas. Given the lack of specificity of the manifestations, clinical suspicion is difficult, and the diagnosis is often delayed with subsequent prolonged therapy needed in some cases.[5] After suspicion, MTB growth in conventional culture media takes at least one week in liquid broth and more than two weeks in solid medium.[6] Recently developed molecular techniques might accelerate the detection of MTB, with sensitivity in extrapulmonary samples over 75%.[7]

In summary, we describe a very rare case of TB arthritis in a Spanish patient with no documented previous history of tuberculosis. Since delay in both early diagnosis and adequate treatment of this condition is associated with high morbidity and risk of severe complications, TB must be considered as part of the differential diagnosis of osteoarticular illness in non-endemic regions.

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https://doi.org/10.1016/j.eimc.2018.08.002
0213-005X/

Methicillin-resistant *Staphylococcus aureus* community-acquired pneumonia in infants☆

Neumonías adquiridas en la comunidad por Staphylococcus aureus *resistente a meticilina en lactantes*

Pneumonia is a major cause of morbidity and mortality in children. Over the past 2 decades, a slight increase in the prevalence of *Staphylococcus aureus* as the causative agent has been observed.[1] Among children hospitalised with community-acquired pneumonia (CAP), *S. aureus* is currently the causative agent in 1% of cases, and 15% of cases of typical CAP are of bacterial origin.[2]

A gradual increase in the rate of community-acquired methicillin-resistant *S. aureus* (MRSA) strains has also been observed in some countries.[1,3] These isolates are characterised as being more virulent since Panton-Valentine leukocidin (PVL) is produced more readily,[4] with MRSA pneumonia being more common in younger children, especially infants.[1]

A retrospective review of children under the age of 2 years hospitalised at our hospital with CA-MRSA pneumonia over the past 5 years (2013–2017) has been performed in order to describe their clinical and epidemiological characteristics.

Three cases were identified, all involving children under the age of 6 months born in Spain to immigrant families (Table 1). The 3 patients developed severe pneumonia with pleural effusion and parenchymal necrosis (X-ray images included in Appendix. Supplementary material) and clinical deterioration was observed with the usual antibiotic therapy. All isolates were susceptible *in vitro* to clindamycin and trimethoprim-sulfamethoxazole. After getting the culture results back (3–4 days after hospitalisation), the antibiotic regimen was changed to linezolid in 2 cases while the third patient continued to receive vancomycin. However, this had to be changed to linezolid after 72 h due to no signs of clinical improvement. The outcome was favourable in all cases, with complete clinical resolution at the time of discharge.

Methicillin resistance in community-acquired *S. aureus* in children was first described in Spain in 2006. These strains currently account for 5–10% of all community-acquired *S. aureus* isolates in children.[5] Most of the cases reported are skin and soft tissue infections.[5]

Community-acquired *S. aureus* pneumonia is associated with high mortality rates (1–5%)[6] and a higher risk of lung necrosis and abscesses.[7] In a recent European study on invasive

Table 1
Characteristics of infants admitted with community-acquired methicillin-resistant *Staphylococcus aureus* pneumonia.

	Case 1	Case 2	Case 3
Age	One month	Three months	Five months
Gender	Male	Male	Male
Family origin/MRSA isolate from family	Paraguayan mother/MRSA nasal colonisation	Filipino mother/MRSA pneumonia	Dominican family/grandmother and brother/MRSA nasal colonisation
Clinical manifestations	Fever without a focus	Fever, tachypnoea and grunting	Fever, grunting and difficulty breathing
Empirical antibiotic therapy	Ampicillin and cefotaxime	Cefotaxime and vancomycin	Cefotaxime and clindamycin
Chest X-ray	Necrotising pneumonia in the right upper lobe with pleural effusion	Necrotising pneumonia in the right lower lobe with pleural effusion	Necrotising pneumonia in the left lower lobe with pleural effusion
Clinical deterioration	Day 4 of hospitalisation (tachypnoea and hypoventilation of the right hemithorax)	Day 3 of hospitalisation (increased pleural effusion)	Day 4 of hospitalisation (increased difficulty breathing)
Admission to the PICU	Admission upon deterioration and need for chest drain, non-invasive ventilation required	Not required	Not required
Microbiology	MRSA in pleural fluid	MRSA in pleural fluid	MRSA in pleural fluid
Panton-Valentine leukocidin	Positive	Positive	Positive
Targeted therapy	Linezolid	1. Vancomycin 2. Linezolid	Linezolid
Clinical outcome	Favourable	Favourable	Favourable

MRSA: methicillin-resistant *Staphylococcus aureus*; PICU: paediatric intensive care unit.

☆ Please cite this article as: Martín Espín I, Aguilera-Alonso D, García-Perea A, Baquero-Artigao F. Neumonías adquiridas en la comunidad por *Staphylococcus aureus* resistente a meticilina en lactantes. Enferm Infecc Microbiol Clin. 2019;37:551–552.