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Orange sputum in a kidney transplant patient with *Legionella micdadei* pneumonia☆

Esputo anaranjado en el contexto de neumonía por *Legionella micdadei* en un paciente trasplantado renal

Dear Sir,

We present the case of a 54-year-old male with a history of IgA nephropathy that has progressed to stage 5 chronic kidney disease, for which he received a cadveric kidney transplant in 2011. His immunosuppression treatment included: tacrolimus, mycophenolate mofetil (MMF) and prednisone, and he had a baseline creatinine level of 2.11 mg/dl. Two months prior to admission, he was started on treatment with corticosteroids at a dose of 1 mg/kg, subsequently in tapering regimen, due to a recent diagnosis of borderline rejection.

The patient was admitted to our hospital with respiratory failure in the context of bilobar pneumonia. Table 1 shows some of the patient's analytical parameters on admission.

The chest X-ray showed infiltrates in the right and left lower lobes. It was initially treated empirically with ceftriaxone and levofloxacin. Due to the poor clinical progress, the ceftriaxone was changed to meropenem, and the levofloxacin was withdrawn as the urinary antigen test for *Legionella pneumophila* (*L. pneumophila*) was negative. He was then also started on co-trimoxazole. The serial blood and sputum cultures obtained on admission were negative, as were the selective cultures for *Legionella*, *Nocardia* and fungi. The test for influenza A and B in nasopharyngeal aspirate was negative. Acid-fast bacillus staining and fluid and solid cultures also resulted negative. Quantitative PCR for the detection of cytomegalovirus in blood only revealed 93 CMV copies/ml.

Despite broad-spectrum antibiotic treatment, the patient remained febrile and had high oxygen requirements at day five after admission; thus, the treatment of immunosuppression with tacrolimus and MMF was discontinued. A bronchoscopy was performed with non-invasive ventilation, bronchoalveolar lavage (BAL) and tracheal aspirate were collected for cultures of conventional bacteria, *Pneumocystis jirovecii*, acid-fast bacilli and selective cultures for *Legionella*, *Nocardia*, fungi and mycobacteria.

On day 10, the patient's general condition deteriorated, with high fever (39.3 °C), coinciding with expectoration of a large amount of orange colour sputum (Fig. 1). We decided to add vancomycin and clindamycin to the treatment. Finally, on day 12 in hospital, colonies of *Legionella micdadei* (*L. micdadei*) were isolated in the BAL and tracheal aspirate cultures. All

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Table 1 – Patient's analytical parameters on admission.

Leucocytes (μl)	5200
Haemoglobin (g/dl)	10.9
Platelets (l)	103×10^9
Creatinine (mg/dl)	3.89
Urea (mg/dl)	176
C-reactive protein (mg/l)	378.2
pH	7.38
PaO_2 (kPa)/FiO_2 50%	10.21
$PaCO_2$ (kPa)	2.58
HCO_3 (mmol/l)	12

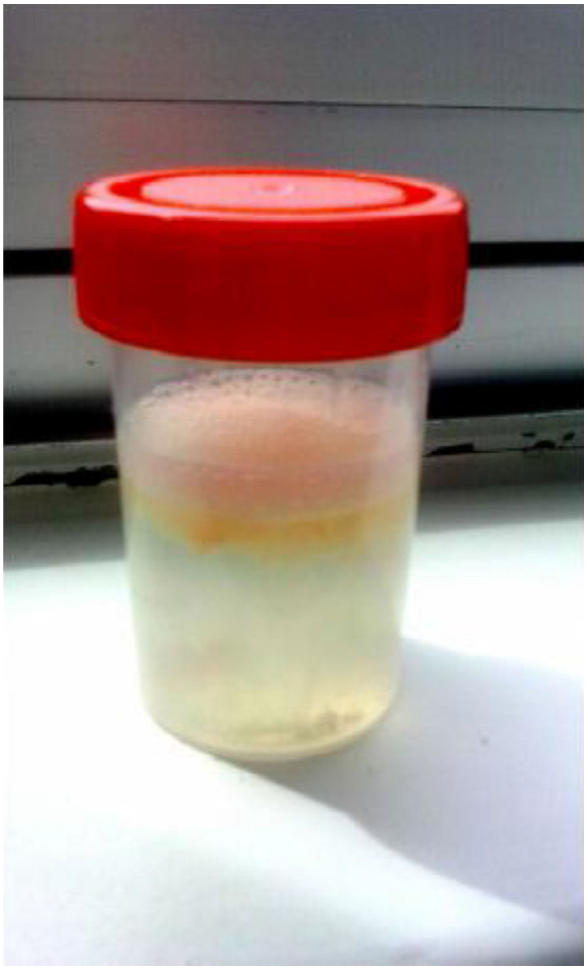

Fig. 1 – Orange sputum.

antibiotics were discontinued and treatment with levofloxacin was re-introduced for an additional 10 day period. The patient continued to have intermittent episodes of expectoration of large amounts of orange like sputum for a few more days but, ultimately, he made good clinical progress. It was possible to re-introduce the immunosuppressant treatment and his kidney function returned to previous levels.

Discussion

One of the most common causes of undiagnosed community-acquired pneumonia is infection by *Legionella* spp.; *L. pneumophila* is responsible for around 85% of the cases of *Legionella* infection, with *L. micdadei* the second most common cause, responsible for 60% of infections by *Legionella* species other than *L. pneumophila*.[1]

Outbreaks of community-acquired pneumonia due to *Legionella* species other than *L. pneumophila* are uncommon,[1,2] although the fact that culture and isolation of *Legionella* is so difficult means that the diagnosis is omitted in some cases. Infection by *L. micdadei* is more likely than *L. pneumophila* to be found in immunocompromised patients, and lower mortality rates have been reported.[3]

No differences in clinical presentation have been described between *L. pneumophila* and *L. micdadei* pneumonia. The gold standard test for diagnosis is the culture of respiratory secretions using Buffered Charcoal Yeast Extract (BCYE) agar. However, due to its viability and speed, detection of *L. pneumophila* serogroup 1 antigen in urine has become one of the methods most commonly used. The tests used, are able to detect the urinary antigen for all serogroups of *L. pneumophila* and some for other species, but the highest sensitivity is achieved with the detection of *L. pneumophila* serogroup 1 antigen.[4]

It has been reported that orange-like sputum in a patient with pneumonia is suggestive of infection by *L. pneumophila*. Fujita et al.[5] put forward that the orange-coloured sputum is caused by factors released by *L. pneumophila* that interact with the tyrosine in the fluids in the epithelial lining.

In our case, we were dealing with an isolated case of community-acquired *L. micdadei* pneumonia in an immunosuppressed patient. Although the infection was originally being empirically covered with a quinolone, the negative urine test for *Legionella* led to us withdraw that treatment. However, that test has very low sensitivity for the diagnosis of *Legionella* infections other than *L. pneumophila*. For that reason, in the particular case of immunocompromised patients with pneumonia without alternative aetiology, we suggest that empirical treatment against *Legionella* spp. should not be discontinued until infection by species other than *L. pneumophilia* has been ruled out, or until the patient shows good progress with the conventional treatment.

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Conflicts of interest

The authors have no conflicts of interest.

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Rare brown tumour location in chronic renal failure☆

Rara localización de tumour pardo en paciente pediátrico

Dear Editor,

Brown tumours (BT) in chronic kidney disease (CKD) are the result of high bone turnover, one type of renal osteodystrophy (ROD), which is caused by secondary hyperparathyroidism (HPT). BT lesions are characterised by the presence of multinucleated cells and brown haemosiderin deposits; this differentiate BT from osteitis fibrosa cystica (OFC) .BT has specific clinical presentation, with imaging and histopathology feature that are different from other disorders caused by HPT or renal osteodystrophy.[1]

This is a case of a 13-year-old male patient from a rural area of Loja in Ecuador. He had a history of CKD and symmetrical growth retardation. His symptoms began with chronic nocturnal pain in his left knee that was exacerbated by trauma. Anthropometric measurements showed that, a short stature, low weight and severe thinness (below Z score −3 of the median on the weight/age, height/age and BMI/age curves). Sexual infantilism was observed, in addition to little muscle mass in limbs and genu-valgus angular deformity (Fig. 1).

Further tests showed altered phosphorus/calcium metabolism (Table 1). Imaging and biopsy obtained with a Jamshidi® needle identified the BT (Fig. 1).

Treatment included intravenous calcitriol (0.5–1 μg) and dialysis. Once the parathyroid hormone (PTH) was reduced the patient had orthopaedic surgery.

The pseudoneoplasm is caused by proliferation of multinucleated giant cells and the mineral bone tissue is being replaced by fibrosis.[2]

The criteria used in diagnosis were: presence of hypocalcaemia and hyperphosphataemia and deficiency of 1,25(OH)3D related to secondary HPT (SHPT)[3,4]; multinucleated giant cells and haemosiderin coexistent with OFC[5]; thyroid and parathyroid gland with no structural alterations[6]; and no history of prolonged steroid therapy. His PTH values were greater than 120 pg/l and he had no

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