Acute post infectious glomerulonephritis by *Streptococcus equi* in a pediatric patient☆

Glomerulonefritis aguda postinfecciosa por Streptococcus equi *en paciente pediátrico*

Streptococcus equi (*S. equi*), subspecies *zooepidemicus*, is a micro-organism belonging to Lancefield group C beta-haemolytic *streptococci*. It is an opportunistic bacteria, particularly in horses, although it has also been isolated in cattle, pigs, rabbits and dogs.[1] It can cause serious bacterial infections in humans such as septic arthritis, meningitis, pneumonia and endocarditis following contact with colonised or sick animals or due to consumption of their derivative products.[2] We report the case of a previously healthy 12-year-old girl who presented with palpebral oedema and oedema in her legs for 48 h plus gross haematuria. Two weeks earlier, she had presented with a throat infection, which was treated with oral amoxycillin for 10 days. She lives in a rural location and looks after horses; she also rides horses as a regular sporting activity. Physical examination revealed peripheral oedema, ascites, bilateral crackles and blood pressure 159/100 mmHg (95th percentile for the patient's age, gender and height 121/78 mmHg). Blood testing yielded values consistent with acute renal failure (Table 1). A lung ultrasound confirmed bilateral pleural effusion. *Streptococcus equi* was isolated in a throat culture. She was treated with salt and water restriction, intravenous furosemide 2 mg/kg/day/for 3 days and oral amlodipine 0.1 mg/kg/day. Her condition followed a favourable course with improved kidney function (Table 1) and normal blood pressure (115/63 mmHg) at discharge after 7 days of hospitalisation. She is currently continuing follow-up with outpatient appointments at the Paediatric Nephrology department with good kidney function, no haematuria or proteinuria and no anti-hypertensive treatment (amlodipine was stopped after two months had elapsed since she was admitted). *S. equi* is a nephritogenic toxin-producing bacteria, although it does not always produce haemolysin of the streptolysin O type, meaning that anti-streptolysin O (ASO) antibodies may not be elevated; in this way, it differs from other members of the genus *Streptococcus*. However, it does usually cause an increase in anti-DNase levels, which can be useful in its diagnosis. Microscopically, glomerular deposition of plasminogen receptor has been seen, which explains secondary nephritis.[1] Most reported cases of acute glomerulonephritis (AGN) caused by *S. equi* have been in outbreaks in an adult population secondary to consumption of unpasteurised dairy products; there are few data on the link to horses seen in the case of our patient. Between December 1997 and July 1998, 253 cases (134 of which were microbiologically confirmed) of AGN caused by *S. equi* were reported in Nova Serrana (Brazil) following consumption of unpasteurised dairy products. In 94% of all the cases, the patients were over the age of 15. Regarding kidney function, 5 cases required dialysis during the acute phase. In the 5-year follow-up, 30% had persistent hypertension and 15% had some degree of chronic kidney disease (defined as a glomerular filtration rate below 60 ml/min/1.73 m^2).[3] Between December 2012 and February 2013, there was another similar outbreak in Monte Santo de Minas (Brazil) among 175 patients, 4 of whom required acute haemodialysis.[4] To date, multiple cases of *S. equi* infection have been reported, primarily in the adult population; the infection is considered rare in the paediatric population. In the literature, we found a single isolated published case, in addition to the paediatric patients included in the above-mentioned outbreaks.[5] Therefore, we find it interesting that our case is described as the first paediatric case reported in Spain, and that it also does not form part of any outbreak associated with consumption of unpasteurised products. Unlike *S. pyogenes*, AGN caused by *S. equi* is more commonly associated with hypertension and chronic kidney disease. Even so, with early detection and treatment, the prognosis is usually favourable as with our patient, although it should be highlighted that it started with a hypertensive crisis, which is very rare in AGN caused by other members of the *Streptococcus* genus. Therefore, we must highlight the importance of taking a medical history that covers consumption of unpasteurised products and contact with animals, particularly horses, which may be carriers of *S. equi*, in cases of acute post-infectious glomerulonephritis, since this will allow for suitable treatment with particular attention to the possible acute complications and long-term sequelae that may occur in these patients.

Table 1
Changes in our patient's laboratory results.

Date	Blood testing	Urinalysis
Admission	Urea 84 mg/dl; creatinine 1 mg/dl; eGFR 63 ml/min/1.73 m^2; ions normal; CRP 4 mg/l; ASO 311 U/mL (NVs <200 U/mL); C3 11 mg/dl (NVs 90–180 mg/dl); C4 21 mg/dl (NVs 10–40 mg/dl)	Proteins 70 mg/dl; erythrocytes 300 mg/dl; FENa 2.8%; ACR 551 mg/g
7 days	Urea 91 mg/dl; creatinine 0.9 mg/dl; eGFR 70 ml/min/1.73 m^2	Proteins 0 mg/dl; erythrocytes 66 mg/dl; ACR 13.2 mg/g
14 days	Urea 26 mg/dl; creatinine 0.6 mg/dl; eGFR 105 ml/min/1.73 m^2	Proteins 0 mg/dl; erythrocytes 0 mg/dl
3 months	Urea 34 mg/dl; creatinine 0.71 mg/dl; eGFR 90 ml/min/1.73 m^2; ASO 381 U/l; C3 141 mg/dl; C4 25 mg/dl	Proteins 0 mg/dl; erythrocytes 0 mg/dl; ACR 7.3 mg/g

ACR: albumin-to-creatinine ratio; ASO: a n ti -streptol ysin O; FENa: fractional excretion of sodium; eGFR: estimated glomerular filtration rate according to Schwartz's formula (2009 update); NVs: normal values.

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Systemic nocardiosis in acquired aplastic anaemia: Report of 2 cases☆

Nocardiosis sistémica en pacientes con aplasia medular adquirida: descripción de 2 casos

Dear Editor,

Nocardia spp. is a filamentous, branching Gram-positive bacteria that can cause serious infections in immunocompromised patients.[1] Since the 1970s, cases of nocardiosis have been reported in solid-organ transplant recipients.[2] In oncohaematological patients, infections caused by *Nocardia* have been documented in haematopoietic stem cell transplant recipients.[3] Acquired bone marrow aplasia is an immunomodulatory disease that presents with bone marrow failure. Treatment for this disease essentially consists of immunosuppression.[4] We report 2 cases of systemic nocardiosis in patients with bone marrow aplasia who did not undergo a bone marrow transplant.

Case 1

An 83-year-old woman diagnosed with bone marrow aplasia was not offered a bone marrow transplant due to her age.

The patient was hospitalised with signs and symptoms consisting of fever for 15 days, asthenia and lumbar pain. Her fever persisted despite treatment with amoxycillin, and a previously absent palpable mass was detected in her lumbar spine. A com-

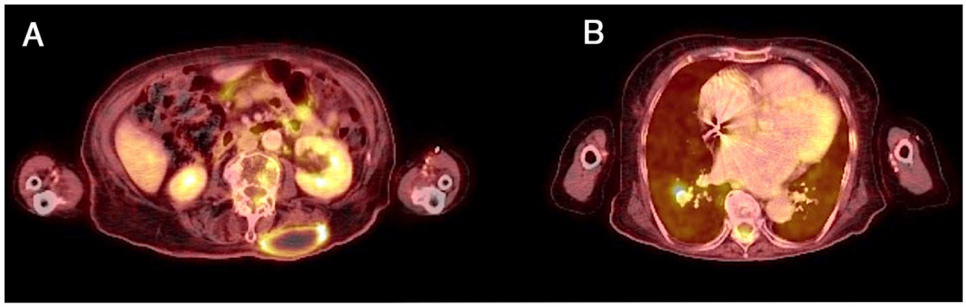

Figura 1. A) Colección paravertebral izquierda con realce periférico. B) Nódulo pulmonar hipercaptante peribronquial derecho.

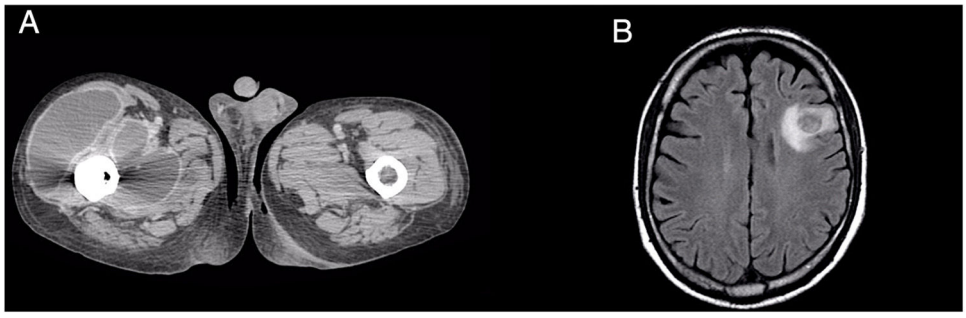

Figura 2. A) Extensa colección periprotésica en miembro inferior derecho que se extiende a cuádriceps y músculos aductores. B) Lesión con realce periférico y edema vasogénico en lóbulo cerebral frontal izquierdo.

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