

Infection caused by *Aeromonas sobria*, complicated by lower extremity paraplegia and the cauda equine syndrome in a patient with well-controlled type 2 diabetes

Małgorzata Szafrąńska¹, Jacek Szafrąński², Agata Bronisz¹, Roman Junik¹

¹Department of Endocrinology and Diabetology, Nicolaus Copernicus University, Ludwik Rydygier *Collegium Medicum*, Bydgoszcz, Poland

²Second Department of Cardiology, Dr Jan Bizioł University Hospital No. 2, Bydgoszcz, Poland

Submitted: 5 July 2011

Accepted: 25 August 2011

Arch Med Sci 2013; 9, 2: 384-385

DOI: 10.5114/aoms.2012.30832

Copyright © 2013 Termedia & Banach

Corresponding author:

Małgorzata Szafrąńska MD
Department of Endocrinology
and Diabetology
Nicolaus Copernicus
University
Ludwik Rydygier
Collegium Medicum
9 Skłodowskiej-Curie St
85-094 Bydgoszcz, Poland
Phone: +48 52 585 40 20
Fax: +48 52 585 40 41
E-mail: zakma@wp.pl

Aeromonas species are Gram-negative anaerobes with the natural habitat of fresh and seawater watercourses and have also been found in stagnant and flowing water reservoirs, water tanks as well as moist soil [1]. The literature primarily describes cases of alimentary tract infections [2] and occasionally sepsis or inflammation of the fascia and muscles [3].

A 75-year-old woman was admitted to the Neurology Department of the University Hospital in Bydgoszcz because of lower extremity muscle weakness developing for 2 weeks and was accompanied by bilateral foot drop and lumbosacral pain irradiating along the posterior surface of both thighs. Approximately 7 days before admission urinary and faecal incontinence as well as an increase of body temperature up to 38.5°C were observed. Type 2 diabetes was diagnosed a year ago, treated with a diet, and with a well-controlled glycaemia. The patient has a negative family history of neoplastic and haematological diseases without addictions.

A neurological examination at admission revealed that the patient suffered from cauda equine syndrome accompanied by lower extremity flaccid paresis and multiple neuritis syndrome accompanied by reduced sensation of gloves-socks type. Aberrations discovered during additional examinations were as follows: C-reactive protein (CRP) 56 mg/l, white blood cells (WBC) $12.25 \times 10^3/\mu\text{l}$, hyperglycaemia 19.8 mmol/l, hypoalbuminaemia 19.2 g/l. A general urine analysis revealed presence of acetone, protein, glucose and microscopic haematuria. Whereas the concentration of total bilirubin in the blood serum was increased to 124 $\mu\text{mol/l}$, aspartate transaminase, alanine transaminase and alkaline phosphatase were normal, γ -glutamyl transpeptidase level was slightly increased (97 U/l).

Spine magnetic resonance imaging with contrast showed as follows: presence of fluid collections resembling abscesses on various levels of the vertebral canal, in the right iliopsoas muscle, where the suspicion of abscesses was additionally intensified by the presence of gas bubbles. Gas bubbles were also visible in the left iliopsoas muscle, vertebral canal as well as L4 and L5 vertebral bodies. A small amount of pathological tissue not enhanced by contrast was visible instead of fat in the sacral canal, on the S1 level with sacral bone defects in front of it.

Empirical antibiotic therapy was introduced through intravenous administration of amoxicillin with clavulanic acid (2×1.2 g). Gradual normali-

zation of the WBC and the total bilirubin level were observed. In spite of the antibiotic therapy the patient suffered from fever of over 38°C. *Aeromonas sobria* was cultured in one of three blood samples. The antibiotic therapy that had been used hitherto was changed according to the antibiogram and ceftazidime was administered intravenously (3 × 750 mg). Human immunodeficiency virus (HIV) infection, hepatitis B, hepatitis C, tuberculosis, syphilis, toxoplasmosis and Lyme disease were all excluded.

A fragment of the L4 vertebral body was collected and histological examination revealed small fragments of bone tissue without any pathological changes. A microbiological examination of a bone fragment and swabs from surrounding tissue indicated *A. sobria* presence in both cases. Due to occasional hyperglycaemia exceeding 11.1 mmol/l human insulin (4-6 units) was introduced. The 24-hour glycaemic profile gradually normalized up to the level where subcutaneous injections of insulin were no longer necessary.

After 12 days of treatment a control X-ray computed tomography scan showed that the size and the number of hypodense areas that could stand for abscesses with gas bubbles in iliopsoas muscles and vertebral bodies decreased and all the changes in the vertebral canal were absorbed. During the patient's hospitalization lasting over a month, improvement of general feeling and condition, normalization of body temperature, lowering of the inflammation index (CRP 68 mg/l), increase of albumin concentration, slight return of lower extremity motor activity, and return of normal urethral and alimentary system sphincter activity were achieved. The patient was advised to continue antibiotic therapy commenced during hospitalization with oral administration of ciprofloxacin (2 × 750 mg) and a check-up visit at the Neurosurgery and Neurology Department after 8 weeks of treatment.

Aeromonas sobria is a rare human pathogen. The most frequently described cases of bacterial infections concerning humans referred to diarrhoea [4] and were usually in a good clinical condition. The bacteria were also found in preparations from appendectomy [2, 5] and it was suggested that they are an aetiological factor of acute cholangitis [6, 7]. There were no descriptions of the cauda equine syndrome or paraplegia caused by the said bacteria. The publications in English only describe a single case of inflammation of the nervous system in the form of purulent meningitis secondary to bacteraemia [8].

A severe course of *A. sobria* was connected with a weakened immune response such as in cases of HIV, acute leukaemia [9], neutropenia during the course of a neoplastic disease [7, 10] and liver cirrhosis [7]. There were descriptions of two cases of

acute necrotic skin and fascia inflammation that ended with the patients' death [3]. In both described cases the infection was preceded by an injury.

It seems that mild gastrointestinal infection (reported as a diarrhoea a few days before admission) was the primary infection site. Jaundice as well as a fever present on admission implies that *A. sobria* bacteraemia was secondary to acute cholangitis. Biliary tract infection is the most common primary infection site of *A. sobria* secondary bacteraemia [7]. We cannot explain why the course was so severe because the patient did not have any diseases predisposing to *A. sobria* inflammation [9, 10]. The only disease was type 2 diabetes diagnosed a year ago, based on periodic health examinations without hyperglycaemia symptoms. The patient had been treated only with a diet. Glycated haemoglobin (HbA_{1c}) level amounting to 5.3% indicated well-controlled glycaemia.

We would like to draw attention to the possibility of *A. sobria* being an etiological factor not only with regard to persons with weakened immunity, chronically ill or wasted, but also to persons with well-controlled type 2 diabetes.

References

1. Janda JM, Abbott SL. Evolving concepts regarding the genus *Aeromonas*: an expanding Panorama of species, disease presentations, and unanswered questions. *Clin Infect Dis* 1998; 27: 332-44.
2. Lim PL. Appendicitis associated with travelers' diarrhea caused by *Aeromonas sobria*. *J Travel Med* 2009; 16: 132-3.
3. Tsai YH, Huang KC, Huang TJ, Hsu RW. Case reports: fatal necrotizing fasciitis caused by *Aeromonas sobria* in two diabetic patients. *Clin Orthop Relat Res* 2009; 467: 846-9.
4. Gröbner S, Bissinger AL, Raible A, Heeg P, Autenrieth IB, Schmidt SM. Severe diarrhoea caused by *Aeromonas veronii* biovar *sobria* in a patient with metastasised GIST. *Pol J Microbiol* 2007; 56: 277-9.
5. Van Noyen R, Selderslaghs R, Bekaert J, Wauters G, Vandepitte J. Causative role of *Yersinia* and other enteric pathogens in the appendicular syndrome. *Eur J Clin Microbiol Infect Dis* 1991; 10: 735-41.
6. Sánchez-Céspedes J, Figueras MJ, Aspiroz C, et al. Development of imipenem resistance in an *Aeromonas veronii* biovar *sobria* clinical isolate recovered from a patient with cholangitis. *J Med Microbiol* 2009; 58: 451-5.
7. Wang JH, Wang CY, Chi CY, Ho MW, Ho CM, Lin PC. Clinical presentations, prognostic factors, and mortality in patients with *Aeromonas sobria* complex bacteremia in a teaching hospital: a 5-year experience. *J Microbiol Immunol Infect* 2009; 42: 510-5.
8. Jacob L, Carron DB, Haji TC, Roberts DW. An unusual case of pyogenic meningitis due to *Aeromonas sobria*. *Br J Hosp Med* 1988; 39: 449.
9. Martino R, Santamaría A, Pericas R, Sureda A, Brunet S. Acute rhabdomyolysis and myonecrosis complicating *aeromonas* bacteremia in neutropenic patients with hematologic malignancies: report of two cases. *Haematologica* 1997; 82: 692-4.
10. Harris RL, Fainstein V, Elting L, Hopfer RL, Bodey GP. Bacteremia caused by *Aeromonas* species in hospitalized cancer patients. *Rev Infect Dis* 1985; 7: 314-20.