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A case of community-acquired Acinetobacter Junii-Johnsonii cellulitis

Celulitis por *Acinetobacter Junii-Johnsonii* adquirida en la comunidad: una presentación de caso

Community-acquired Acinetobacter cellulitis

Andrés F. Henao-Martínez<sup>1</sup>, Guido R. González-Fontal<sup>2</sup>, Steven Johnson<sup>1</sup>

<sup>1</sup> Division of Infectious Diseases, University of Colorado, Denver, USA

<sup>2</sup> División de Hematología-Oncología, Hospital Militar Central, Bogotá, D.C., Colombia

# **Corresponding author:**

Andres F. Henao-Martinez, University of Colorado Denver, 12700 E, 19th Avenue,

Denver, USA. Mail Stop B168, Aurora, CO 80045.

Telephone: (303)-724-6451; fax :(303)-724-6462

andres.henaomartinez@ucdenver.edu

All authors contributed with the preparation and edition of the manuscript.

Acinetobacter skin and soft tissue infections outside of the traumatic wound setting are a rare occurrence. The majority of cases reported occur in the presence of significant comorbilities and by *Acinetobacter baumanii*. We describe a case of community-onset, health-care-associated, non-traumatic cellulitis caused by *Acinetobacter*, species *junii-johnsonii* with bacteremia. To our knowledge, this is the first reported case of *Acinetobacter junii-johnsonii* skin and soft tissue infections. Hemorrhagic bullae might be one of the clinical features of *Acinetobacter* cellulitis.

**Key words:** *Acinetobacter*, cellulitis, community-acquired infections, bacteremia, blister, therapy.

Infección de piel y tejidos blandos por *Acinetobacter* no relacionadas con trauma es una presentación inusual. La mayoría de los casos descritos son en presencia de comorbilidades y causados por *Acinetobacter baumanii*. Describimos aquí un caso de celulitis no traumático por *Acinetobacter junii-johnsonii* con bacteremia de inicio en la comunidad, y asociado a la salud. De acuerdo a nuestro conocimiento este sería el primer caso reportado de infección de tejidos blandos y piel por *A. junii-johnsonii*. Vesícula hemorrágica podría ser una característica clínica de celulitis por *Acinetobacter*.

**Palabras clave:** *Acinetobacter*, celulitis, infecciones comunitarias adquiridas, bacteriemia, vesícula, terapia.

Acinetobacter is a widely recognized pathogen in the hospital setting. On the other hand, cases in the community are uncommon. Among community-acquired cases the most frequent presentation is pneumonia. Non-traumatic skin and soft tissue infection remains an uncommon described presentation in any setting, although necrotizing fasciitis by *Acinetobacter baumanii* is well characterized.

## Case report

In August 2010, a 48 year old man with a history of metastatic, stage IV, prostate cancer, complicated by bladder obstruction, T8 fracture, spinal cord involvement treated with androgen deprivation therapy and local radiation therapy was admitted to our institution after being transferred from an outside hospital. The patient had been admitted one month prior to this presentation with a pulmonary embolus, methicillinsensitive Staphylococcus aureus (MSSA) bacteremia of unclear origin and also with a multidrug resistant (MDR) Serratia marcescens urinary tract infection. The patient recovered from that episode and was discharged home on intravenous (IV) ertapenem 1gram every 24 hours. Few weeks later patient was readmitted to an outside hospital after he developed a rapid progression of symptoms characterized by marked fatigue, lightheadness, fever, chills and right lower extremity swelling, erythema, tenderness and warmness. On admission the patient was found to be in septic shock requiring intensive care unit care. He received aggressive fluid resuscitation therapy, pressor support and he was placed empirically on IV vancomycin, piperacillin-tazobactam and levofloxacin. His hemodynamic parameters improved over the next two days and he was transferred to our hospital with the documentation of a non fermenting gram negative rod (GNR) in blood culture (1 of 2 bottles). CT (computerised tomography) scan of his leg showed a

marked subcutaneous edema (figure1). The positive culture was from a peripheral site. Physical exam was remarkable for marked right lower extremity swelling, with erythema involving the distal 2/3 of the leg along with local tenderness, warmness and the presence of a purpuric rash and hemorrhagic bullae. The PICC line was present and was without local signs of inflammation. His labs were significant for leukopenia, anemia and thrombocytopenia. The gram negative rod was a nonmotile, catalase-positive, oxidase-negative bacteria; finally identified using the Vitek 2 system (an automated system for bacterial identification using a phenotypic approach) as *Acinetobacter junii-johnsonii* sensitive to quinolones and B-lactams and intermediate to Aztreonam. His antibiotic therapy was narrowed to Levofloxacin only and he was discharged 4 days later. He was seen again in the clinic 4 weeks later and complete resolution of his local right leg inflammatory changes were noted.

## **Discussion**

Acinetobacter is a ubiquitous, encapsulated, nonmotile, aerobic gram negative cocobacillus. It is widely recognized as an opportunistic pathogen and well known to cause hospital acquired infections ranging from pneumonia to bacteremia, UTI and soft tissue infections (1,2). It is also recognized for its ability to develop multi-drug resistant mechanisms, making the treatment often times difficult and challenging.

Although less common, community acquired Acinetobacter (CA-AB) infections have also been described (3). A prior systematic review of 123 patients with CA-AB infections showed that the most prevalent type of infection was pneumonia followed by bacteremia. In that review only one case of SSTI (skin and soft tissue infections) was

reported (4). Among the descriptions of cases of ocular infections are conjunctivitis, preseptal cellulitis, endophtalmitis and corneal perforation.

Acinetobacter SSTI frequently complicates traumatic injuries as a consequence of war or natural disasters, especially in areas like Afghanistan, Iraq or tropical such as Southeast Asia (5-8). Non traumatic Acinetobacter SSTI has been rarely reported. However, when it is reported, it's by Acinetobacter baumanii and associated with more extensive infections such as necrotizing fasciitis and increase mortality (1,9,10). In the best of our knowledge there are not other cases of reported SSTI caused by A. junii-johnsoni.

Based on prior descriptions; it seems that older age, prior antibiotic use and comorbilities such as cirrhosis, nursing home residency, dementia and diabetes may play a role as potential risk factors for this type of infection. In our case, the patient had metastatic cancer and was already on IV antibiotics at the time of presentation.

Interestingly enough, he acquired this infection while on broad spectrum antibiotic: ertapenem is well known to be less effective against non fermenting GNR such as 

Acinetobacter. Another important consideration is that from the described cases, the development of necrotizing fasciitis from a multi-drug resistant A. baumanii is translated in high mortality (1,9). It also recognized a trend to a MDR pattern in the antibiotic susceptibility profiles of the community Acinetobacter strains.

As described previously with an *Acinetobacter* infection traumatic wound injury cases (5) and in a necrotizing fasciitis case (9), hemorrhagic bullae is a possible finding in this type of skin and soft tissue infection, as featured in our case. Therefore, although infrequent, it is critical to diagnose *Acinetobacter* cellulitis in patients that otherwise are

bullae. There are no clinical data to favor combination therapy vs. monotherapy for this type of infection. However depending of the severity of the infections like in this case, we consider reasonable to have double antibiotic coverage initially while awaiting final susceptibilities. Once they are available, we advocate simplifying to monotherapy. Finally it is important to recognize not only *A. baumanii* as the possible cause of SSTI but also other species such as *A. junii-johnsonii*. The severity described with the former might be due to the MDR profile or virulent factors. *A. baumanii* has been isolated from multiple body sites. However its main impact is as a cause of ventilator associated pneumonia. In contrast *A. junii-johnsonii* has been described as a skin colonizer and as a cause of catheter related bacteremias in the setting of prior antibiotic use, malignancy or invasive procedures (11,12).

not responding to the initial antibiotic therapy, especially in the presence of hemorrhagic

## **Conflicts of interest**

No conflicts of interest were reported by Drs. Andres F. Henao-Martinez, Guido R. Gonzalez-Fontal and Steven Johnson.

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**Figure 1.** CT scan of the right lower extremity demonstrating marked subcutaneous edema throughout the entire leg with skin thickening without hematoma or abscess formation.

