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Ecthyma Gangrenosum Caused by Klebsiella Pneumoniae: A Rare Entity with Devastating Consequences

[1]Department of Dermatology, Barros Luco Trudeau Hospital, Santiago, Chile.
[2]Resident Department of Dermatology, Faculty of Medicine, University of Chile, Santiago, Chile.

Andrea Elgueta[1], Claudia Schroder[2].*

Ectima gangrenosa causado por Klebsiella Pneumoniae: Una entidad poco común con consecuencias devastadoras

ABSTRACT
This report describes the case of a 64-year-old female patient, with multiple comorbidities which presented an ecthyma gangrenosum due to Klebsiella pneumoniae confirmed by blood and tissue cultures. It finally ended in the amputation of the limb. The case is presented given the low prevalence of positive cultures for this microorganism in this pathology associated to devastating consequences for the patient.
Keywords: *Antibiotic Resistance; Ecthyma; Infections, Klebsiella pneumoniae.*

**Corresponding author: Claudia Schroder / clau.schroder@gmail.com*
Independencia 1027, Santiago, Chile.

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RESUMEN
Este reporte describe el caso de una paciente de sexo femenino de 64 años, con múltiples comorbilidades, que presentó una ectima gangrenosa por Klebsiella pneumoniae confirmado por hemocultivos y cultivo de tejidos. Finalmente terminó con la amputación de la extremidad. Se presenta el caso dada la baja prevalencia de cultivos positivos para este microorganismo en esta patología, asociado a consecuencias devastadoras en el caso de nuestra paciente.
Palabras clave: *Ectima; Infecciones; Klebsiella pneumoniae; Resistencia a los Antibióticos.*

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Pseudomonal ecthyma gangrenosum (EG) is a well-known condition in immunosuppressed patients[1]. EG was first described in 1897 by Canadian pathologist Dr. Lewellys Barker as a manifestation of *Pseudomonas aeruginosa*[2]. However, ecthyma gangrenosum associated with *Klebsiella pneumoniae* is a rare entity that requires early recognition and optimal antibiotic and surgical management[3]. Only five cases with *Klebsiella pneumoniae* have previously been reported to be a cause of EG[3].

Case Report

Female patient, 64 years old, with a history of type 2 diabetes mellitus, chronic arterial hypertension, and stage V chronic kidney disease on hemodialysis. She was admitted to the surgery service for management of a diabetic foot ulcer. Given the poor response to treatment and healing, the patient underwent trough a supra-patellar amputation of the left leg. A couple days after medical discharge, the patient started with purulent drainage from the amputation stump, dehiscence of sutures, and appearance of hemorrhagic blisters and bullae on the right leg in less than 24 hours, and she was admitted again. Blood cultures informed *Klebsiella pneumoniae* extended-spectrum beta-lactamases (ESBL) and started treatment with Piperacillin/Tazobactam. The lesions on the right leg increased in size, including even the dorsal aspect of the right foot, associated with intolerable pain. Finally, the blisters and bullae unroofed spontaneously, forming a thick black eschar, and small ulcers with punched-out edges were evident (Figure 1). A tissue sample was taken from the edge of the ulcer and sent for regular culture, fungi and mycobacteria, and histological study as well. It resulted positive for *Klebsiella pneumoniae* ESBL, only sensitive to Amikacin, Imipenem, Meropenem and Ertapenem (Figure 2), so the antibiotic regimen was adjusted. She underwent surgical debridement of the right leg, however due to the null response of the lesions after approximately one month, it had to be amputated.

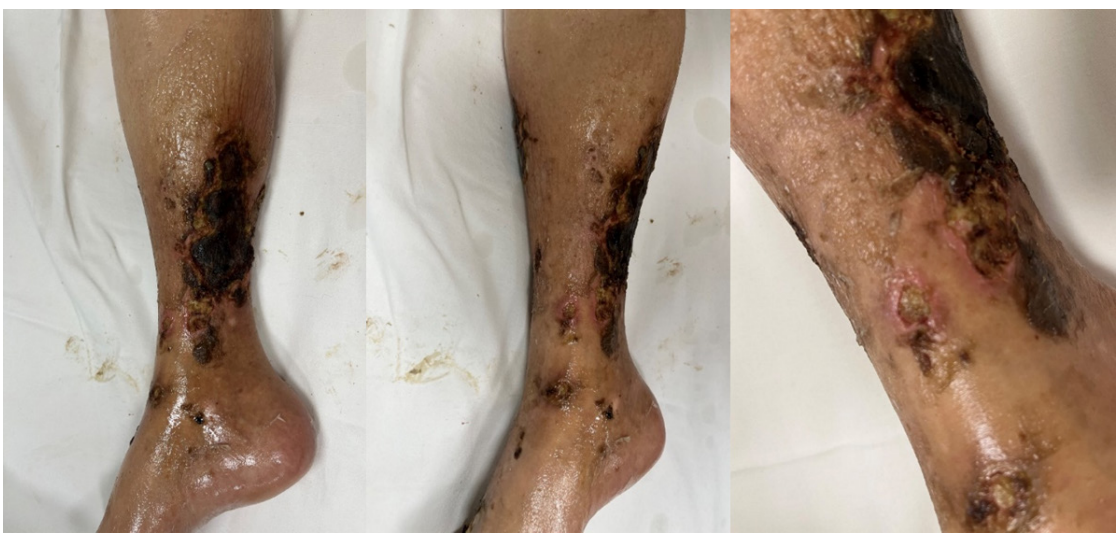

Figure 1: *Thick black eschar and small ulcers with punched-out edges on the right leg.*

	Results
Bacterial culture	Positive
Fungal culture	In process

Identified microorganism	Commentary
1 *Klebsiella pneumoniae* (KLEPEP)	Small quantity, positive ESBL

Antibiotic	1 CMI	KLEPNEP SIR	2 CMI	. SIR	3 CMI	. SIR
Amikacina	<=8	S				
Cefazolina	>8	R				
Cefepima	>16	R				
Ceftriaxona	>4	R				
Ciprofloxacino	>2	R				
Ertapenem	0,5	S				
Gentamicina	>8	R				
Imipenem	<=0,25	S				
Meropenem	<=0,5	S				
Piperacilina - Tazobactam	64/4	N				
Trimetoprim - Sufametoxazol	>2/38	R				

Figure 2: *Tissue culture positive for Klebsiella pneumoniae ESBL.*

Discussion

Ecthyma gangrenosum is a relatively uncommon condition, previously thought to be pathognomonic for Pseudomona aeruginosa's sepsis[5]. Since the 1980s, more and more data have been accumulated that various bacterium like *Escherichia coli*, *Citrobacter freundii*, *Klebsiella pneumonia*, other Pseudomonas species, and *Morganella morganii* can be etiologic agents for EG, as well as some fungi (Candida albicans, Fusarium, and others)[5].

Klebsiella pneumoniae (KP), a Gram-negative bacteria is an uncommon agent for EG, with only five cases reported so far[4].

EG affects all age groups and genders. Immunocompromised individuals are particularly susceptible to developing this condition, with up to 62% to 75% of affected individuals having an underlying immunodeficiency. It is also known to occur in otherwise healthy immunocompetent individuals. Common predisposing conditions include neutropenia, leukemia, multiple myeloma, diabetes mellitus, malnutrition, and extensive burn wounds[2].

It may develop both through hematogenic seeding or direct skin inoculation. The bacteremic form of EG is more common than the non-bacteremic form. It initially appears as a single erythematous papule or a hemorrhagic blister that evolves rapidly into a necrotic ulcer with a black eschar and a red halo[3]. The lesions can be localized and solitary, or widespread. The gluteal/perineal area is most common (57%), followed by the extremities (30%), the trunk (6%), and the face (6%)[2]. Perivascular invasion and resultant ischemic necrosis of the associated skin result in the classic macroscopic appearance[2].

Excisional biopsy or punch biopsy with Gram stain, culture, and histopathologic examination

can be performed to confirm diagnosis[2]. Microscopic examination reveals an ulcerated zone of necrotic epidermis and dermis with neutrophilic predominance. The epidermis adjacent to the necrotic region reveals reactive hyperkeratosis. The underlying subcutaneous tissue may also be involved[2].

Other conditions with similar macroscopic features include autoimmune vasculitides, vasopressor-induced necrosis, calciphylaxis, warfarin-induced necrosis, and disseminated intravascular coagulation, pyoderma gangrenosum, necrolytic migratory erythema, and livedoid vasculopathy[2].

Because significant morbidity is associated with EG, broad spectrum antimicrobial coverage should be initiated until a pathogen is identified[4]. Neutropenia at the time of diagnosis is the most important prognostic factor[2].

Initial empiric therapy includes antipseudomonal beta-lactams (piperacillin/tazobactam), cephalosporins (cefepime), fluoroquinolones (levofloxacin), and carbapenems (imipenem). Combined therapy is recommended in high-risk individuals, such as those with neutropenia and septic shock. Once the causative organism and antimicrobial susceptibilities are identified, antimicrobial coverage should be narrowed[2].

Timely diagnosis, appropriate antibiotics, and surgical debridement remain the only potential cure[3]. Management of ecthyma gangrenosum requires an interprofessional team approach, including providers from infectious disease, surgery, dermatology, hematology, and critical care[2]. EG is a life-threatening infection often complicated by septic shock. Prompt clinician recognition and appropriate antimicrobial therapy (always thinking about the possibility of atypical pathogens like KP), are necessary to achieve optimal outcomes and avoid results as the ones with our patient.

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