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Community-Associated Methicillin-Resistant *Staphylococcus Aureus* (CA-MRSA) Impetigo Successfully Treated with Oral Rifampicin in a Rural Kenyan Hospital

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Abstract: **Methicillin-resistant *Staphylococcus aureus* (MRSA) is resistant to beta-lactam antibiotics (and other drug classes) through altered penicillin-binding proteins that maintain cell wall synthesis despite antimicrobial pressure. MRSA can be hospital-associated (HA-MRSA) or community-associated (CA-MRSA). The major risk factors for MRSA infection in Africa include hospitalization, prior antibiotic use, diabetes, invasive procedures, intravenous drug use, and HIV infection. The prevalence of CA-MRSA is widely variable and stands at about 26.7% in the US, predominantly among younger patients with skin infections. In Kenya, the prevalence is 1.8% according to limited data. The Infectious Diseases Society of America (IDSA) recommends using clindamycin, trimethoprim-sulfamethoxazole, tetracycline, and linezolid for skin and soft tissue infections caused by CA-MRSA and discourages the use of rifampicin in this context. However, we report on the case of a young Kenyan woman in a rural setting whom we successfully treated for CA-MRSA impetigo with oral rifampicin monotherapy after ruling out active tuberculosis infection prior to treatment. She had failed treatment with all the other IDSA-recommended antibiotics.**

Keywords: **Methicillin-resistant *Staphylococcus aureus*, MRSA, community-associated MRSA, CA-MRSA, hospital-associated (HA-MRSA), Kenya.**

1. INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA) is resistant to beta-lactam antibiotics through altered penicillin-binding proteins that maintain cell wall synthesis despite antimicrobial pressure (1). MRSA possesses the *mecA* and *mecC* genes, which confer methicillin resistance by encoding a different penicillin-binding protein (PBP) that allows cell wall synthesis in the presence of beta-lactams due to low binding affinity (1). MRSA can be hospital-associated (HA-MRSA) or community-associated (CA-MRSA). In a systematic review and meta-analysis, the prevalence of MRSA was at about 15%, with higher prevalence observed in Africa (22.5%) compared to Asia (13.1%) and South America (5.4%). In Kenya, the prevalence of MRSA varies between 3.7% in the private hospitals and 27.8% in the public hospitals (2). Njenga J collected

228 wound swab samples from 5 health centers and noted a CA-MRSA prevalence of 1.8% (3). Increased infection prevention measures and antimicrobial stewardship programs have led to a decline in HA-MRSA by as much as 17.1% in the US (4). MRSA infection is one of the leading causes of nosocomial infections, with significant morbidity, mortality, and increased hospital-related costs (5). The risk factors for MRSA infection in Africa include hospitalization, prior antibiotic use, diabetes, invasive procedures, intravenous drug use, and HIV infection (6). Host susceptibility and virulence factors like capsular polysaccharides, staphylococcal hemolysin, and Panton-Valentine leukocidin interact to cause MRSA infections (7). The spectrum of MRSA infections includes skin and soft tissue infections, bacteremia, including infective endocarditis; pneumonia; lung abscess and empyema; osteomyelitis, and meningitis (1). The Infectious Diseases Society of America (IDSA) has issued guidelines for the management of MRSA infections. These guidelines emphasize rapid diagnosis through appropriate cultures, effective source control measures, and tailored antibiotic therapy using drugs like vancomycin, daptomycin, or linezolid (8).

2. CASE SUMMARY

Clinical history, physical examination, and work-up

A 26-year-old married mother of 3, a shopkeeper from Lanet, Nakuru County, Kenya, came to us in September 2025 with a 3-month history of recurrent pustular lesions on both legs, hands, and ears. She had no underlying medical conditions. These lesions started following a scratch on both legs while working in her garden. A few days later, the scratched areas developed hyperpigmented, painful nodular swellings, which soon became pustular, with similar eruptions on the palms of her hands and external ears. She had been seen in various outpatient clinics and thought to have maggot infestations, atopic dermatitis, contact dermatitis, and impetigo. She had been treated with oral flucloxacillin 500 mg QID for 1 week on 3 different occasions in 3 separate health centers, oral clindamycin 300 mg TDS for 1 week on 2 different occasions, oral azithromycin 500 mg OD for 3 days on 1 occasion, oral steroids on 3 different occasions, and topical steroids on 2 occasions and was presently on another course combining oral clindamycin, metronidazole, and clotrimazole-beclomethasone ointment.

On examination, she was a healthy young woman with normal vital signs and no personal or family history of atopy. She had pustular nodules with crusting on the left external ear and on the palms of both hands and the lateral aspect of both mid legs extending to the gastrocnemius region of the left leg. The lesions on the legs were coalescing and looked like infected plaques. See figures 1 and 2 below. She had no local or regional lymphadenopathy. She had no clinical features of lupus or rheumatoid arthritis that would make us consider erythema nodosum, which causes nodular lesions on the legs. She had a normal random blood sugar of 110 mg/dL, an ESR of 10 mm/hr, a normal complete blood count, and she was HIV negative.

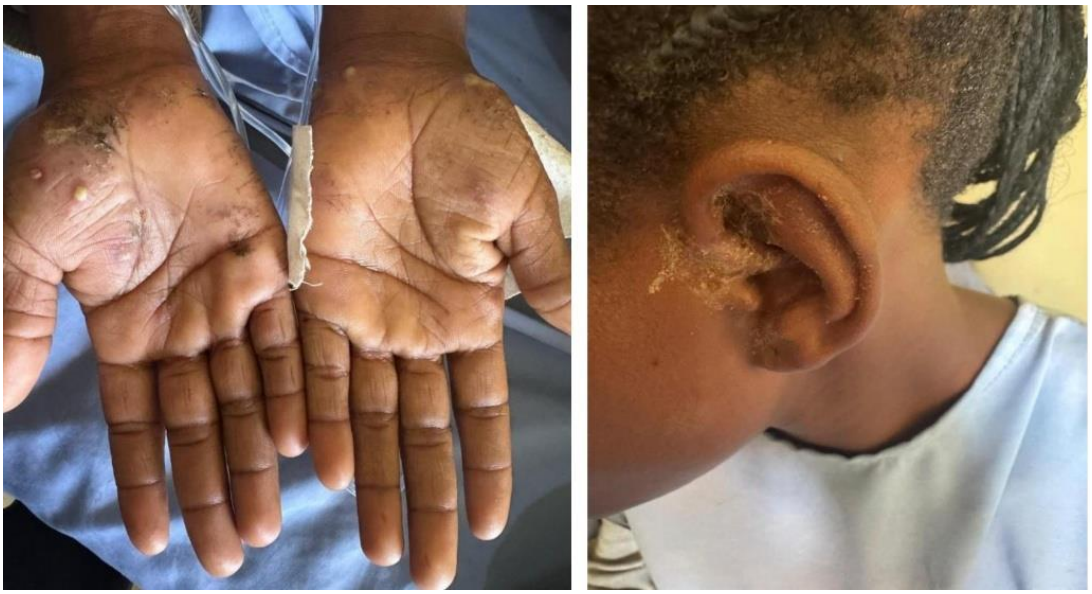

Figure 1: Pustular nodules on the palms of both hands with crusting on the right proximal palm and the left external ear.

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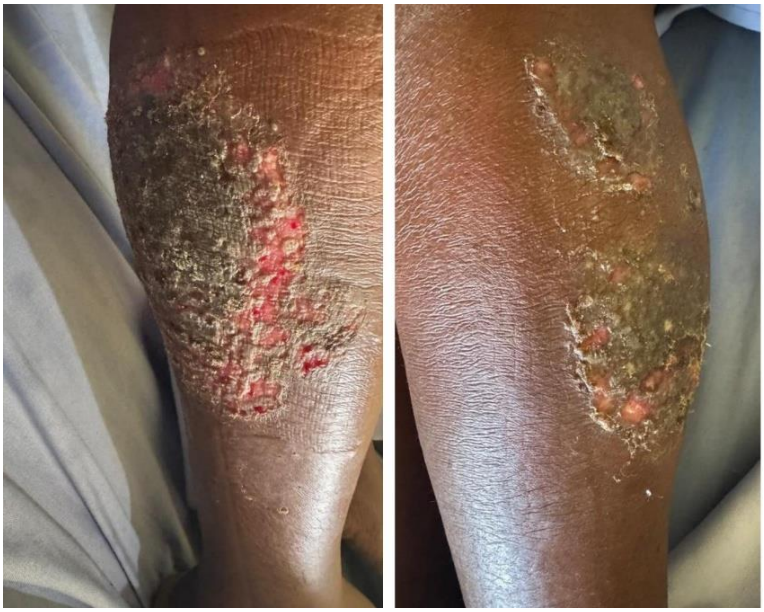

Figure 2: Pustular nodules on both legs with crusting. The nodules are coalescing to form plaque-like septic lesions bilaterally.

Diagnosis, management, and follow-up

We made a clinical diagnosis of impetigo, probably caused by resistant bacteria in view of the multiple courses of antibiotics she had already used. We put her on a course of oral co-amoxyclav 1 gm BD combined with Bactrim (sulfamethoxazole-trimethoprim) 960 mg BD for 1 week, together with daily skin cleansing using chlorhexidine 200 ml in her bath water for 2 weeks. When she came back 2 weeks later, the lesions had persisted. We then took pus swabs for microscopy, culture, and sensitivity, which were done in a major quality-assured private laboratory in town, and whose report was as follows:

DRUGS	**MICROORGANISM AND SENSITIVITY**	
	Klebsiella pneumoniae	Methicillin-resistant *Staphylococcus aureus* (MRSA)
AMOXYCILLIN-CLAVULANATE	**Sensitive**	Resistant
CEFTRIAXONE/CEFOTAXIME	**Sensitive**	Resistant
CEFUROXIME/CEFPROZIL	**Sensitive**	Resistant
CLINDAMYCIN	Resistant	Resistant
CLOXACILLIN	Resistant	Resistant
ERYTHROMYCIN	Resistant	Resistant
FUCIDIN	Resistant	Resistant
LEVOFLOXACIN	**Sensitive**	Resistant
AMPICILLIN	Resistant	Resistant
RIFAMPICIN	-	**Sensitive**

NOTES:

MICROSCOPY-GRAM STAIN:

- Pus cells: moderate
- Gram-positive cocci: moderate
- Gram-negative bacilli: moderate

ORGANISM CULTURE:

Organism 1: *Klebsiella pneumoniae*

- Growth: moderate

Organism 2: Methicillin-resistant *Staphylococcus aureus (MRSA)*

- Growth: moderate

In light of these findings, we believed that the main cause of the recurrent and persistent infection was MRSA and elected to treat her with oral rifampicin as per the antibiogram. We ruled out active tuberculosis (TB) first clinically (she had no known contact with an active TB case, no clinical features of TB, and had a normal chest X-ray and repeat ESR.) She also had a normal plain x-ray of both legs that ruled out osteomyelitis. An ultrasound of the infected areas ruled out abscesses and collections that would have warranted surgical drainage. A normal cardiac evaluation and a normal point-of-care cardiac ultrasound ruled out infective endocarditis. Accordingly, we put her on oral rifampicin (600 mg daily) for 2 weeks with analgesics and ongoing daily chlorhexidine baths. We advised her on practical steps to prevent the infected skin coming in contact with her family members and household items. Two weeks later, the lesions on the ears and both hands had completely cleared while the ones on both legs had cleared about 80%. We added another week of rifampicin at the same dosage. During follow-up two weeks after this, the lesions on both legs had completely cleared. She has had no further recurrence of the lesions on subsequent reviews and has since been discharged from the clinic.

3. DISCUSSION

Our patient was a young woman who had a CA-MRSA skin infection (impetigo). S. L. Davies et al. reported the prevalence of CA-MRSA of 26.7% in the midwestern US, predominantly among younger patients (median age of 46 years). The risk factors for CA-MRSA were a younger age, sports team participation, and incarceration (9). The skin was the most commonly infected site (80%) in the study (9). Her history of scratching her legs while working in the garden served as a gateway to the infection. Breakages in the skin are a common route by which staphylococcal organisms cause human infections. Beyond resistance to the beta-lactams, the MRSA in our patient was also resistant to fluoroquinolones (levofloxacin), lincosamides (clindamycin), and macrolides (erythromycin). This is not unusual, for various strains of MRSA have widely been reported to develop resistance to multiple antibiotic drug classes. This is due to such mechanisms as single nucleotide polymorphisms (SNPs) that confer daptomycin resistance (10); heterogeneity of the *SCCmec* gene that confers resistance to lincosamides, macrolides, and tetracyclines (11); and an acquired resistance to linezolid caused by, e.g., mutations of the ribosomal proteins near the binding pocket of linezolid in the central core of ribosomal peptidyl transferases (*rplV*, *rplD*, and *rplC*) (12, 13). In Kenya, Njenga J et al. isolated two strains of CA-MRSA from wound swabs from two health facilities. They sequenced resistance genes, i.e., sequence types (ST) 7460 and ST 7635, that were sensitive to vancomycin, rifampicin, linezolid, and tigecycline. They were resistant to cefoxitin, penicillin, erythromycin, clindamycin, ciprofloxacin, tetracycline, and sulfamethoxazole-trimethoprim (14). As seen in the antibiogram, the CA-MRSA was sensitive to rifampicin, which we successfully used as a single agent to treat her impetigo. We clinically excluded active TB first before using rifampicin given the high burden of TB in Kenya with a prevalence of 558 per 100,000. Only 46% of TB cases are diagnosed and treated in Kenya, leaving 54% undiagnosed and at risk of spreading the disease (15). Rifampicin is the backbone of TB treatment, and its use as a monotherapy or adjunctive therapy for skin and soft tissue infections caused by MRSA is discouraged by the IDSA in the CA-MRSA treatment guidelines (class A-III recommendation) (8). This is designed to prevent the selection of multidrug-resistant TB. Instead, the IDSA recommends the use of clindamycin, trimethoprim-sulfamethoxazole, a tetracycline, and linezolid (8). We had unsuccessfully tried these options for our patient. We opted against using oral linezolid as a first line because it was expensive and was not on the antibiogram and preferred to preserve it as a potential reserve drug for future use should she fail to respond to the rifampicin. Luckily, the impetigo completely resolved on an oral course of rifampicin.

4. CONCLUSION

Multi-drug resistant CA-MRSA should be suspected in patients with skin and soft tissue infections that are not responding to the usual beta-lactams, beta-lactamase inhibitors, and clindamycin. In this case, a rapid diagnosis should be obtained by tissue culture and sensitivity studies and treatment tailored to achieve effective source control and the judicious use of

antibiotics. Institutional infection prevention protocols and antimicrobial stewardship programs will curtail the emergence and spread of multidrug-resistant infections, including MRSA.

Ethical Consideration

Informed consent was obtained from the patient to publish this case.

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