MRSA association with Tinea corporis in Al-Anbar governorate/ west of Iraq

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Abstract
Backgraund: Staphylococcus aureus, a significant human pathogen throughout history, is now the most common cause of bacterial infection worldwide. S. aureus has the remarkable ability to cause a spectrum of illnesses, from non-fatal necrotizing pneumonia to mild skin infections. Additionally, a significant therapeutic worry is the spread of extremely virulent, drug-resistant bacteria like methicillin-resistant S. aureus in clinical and community settings. Dermatophytes are responsible for the superficial fungal skin ailment known as tinea corporis. Tinea corporis is widespread across the world. The position of the lesions, which may affect the trunk, neck, arms, and legs, is carefully specified. Aims of study: To study the relationship between Bacterial infection with the Tinea corporis. Isolation and Identification of fungal species in clinical isolates MRSA by Classical methods. To show the frequency of bacterial (MRSA) associated with mycotic dermatitis infections and in the patients from Al-Anbar governorate. Methods: Swab for bacteria and scrapping for fungi isolation from skin infection culture in classical method in) blood agar and then. MSAfor MRSA **diagnosisi** and Loctophynol cotton blue orf ,SDA dermatophyte(Tinea corporis). Result: There was a significante correlation between MRSA and Tina corporis at the site of the infaction. Conclusion: the skin infected with tinea corporis destroy the skin defence wich lead to secondary bacterial infection MRSA wich transmission by contact and itching hand to stie infection led to contamination.
Keywords: Dermatophytes,Bacteria, MRSA, Tinea corporis ،skin ،methillicine.

1. Introduction

Tinea corporis is a very common form of superficial dermatophytosis infection of the skin in patients seeking treatment at our hospital, a tertiary care center. Dermatophytes are fungi capable of causing skin changes of the type known as ringworm or dermatophytosis. The ringworm species are all moulds belonging to three asexual genera: Microsporum, Trichophyton and Epidermophyton. With increasing number of people travelling worldwide, mycoses that were previously restricted to certain geographic areas can now be seen in other areas as well. In recent years, the number of fungi recognized as human pathogens has risen, partly in debilitated and immunocompromised patients (1). According to a World Health Organization study on the prevalence of dermatophytic infection, 20% of people needing medical help have cutaneous fungal infections. worldwidepatients(1). Any tinea infection on the body other than the scalp, beard, feet, or hands is referred to as tinea corporis. Ringworm is the common name for this lesion, which appears as an annular plaque with a slightly elevated, frequently scaly, advancing border. One, two, or more concentric rings with red papules or plaques in the middle may be present on each lesion. The middle of the lesion may resolve as it develops, leaving post-inflammatory hypo- or hyperpigmentation. Dermatophyte infections can be treated with topical and systemic treatments. For simple tinea corporis of tiny regions and of short duration, topical treatment is typically helpful.(2).

Tinea corporis is a skin disease that affects the body's surface that is brought on by dermatophytes. All around the world, tinea corporis exists. It is specifically described by the sites of the lesions, which can affect the arms, legs, neck, and body's trunk. There are several names for dermatophyte infections that affect different body parts. The scalp (tinea capitis), face (tinea faciei), hands (tinea manuum), groin (tinea cruris), and feet are among them (tinea pedis).(3).

Trichophyton rubrum, T. tonsurans, and Microsporum canis are the most common causes of tinea corporis(3-9). In North America and around the world, T. rubrum is by far the most frequent cause of tinea corporis and dermatophytosis..(10-12) T. tonsurans frequently causes tinea corporis, which is a subsequent illness to tinea capitis..(13). Tinea corporis, also known as ringworm, often manifests on the trunk, limbs, or face as a single or several annular, scaly lesions with core clearing, a slightly raised, reddish edge, and acute margination (abrupt transition from abnormal to normal skin). Pustules or follicular papules might be seen along the lesion's border. The itch might change. Clinical features and KOH examination of skin scrapings from the active edge are used to make the diagnosis of tinea corporis (14).

Transmission oftinea corporis

Dermatophytes infect both people and animals when they come into contact with spores (conidia). In a vertebrate host, dermatophytes typically exclusively

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produce arthrospores (arthroconidia), asexual spores that originate inside the hyphae. They can also create microconidia and macroconidia in the environment (for example, in laboratory culture), which are asexual spores that form outside the hyphae. The dermatophyte first infects a hair in growth or the stratum corneum of the skin. Due to the lack of or restricted availability of the vital nutrients these organisms require for development, they typically do not infect dormant hairs. Hyphae finally created contagious arthrospores as it traveled through the hair and keratinized skin. both anthropophilic and zoophilic the primary means of dermatophyte transmission between hosts is by arthrospores in skin scales or hair. The environmental phases may also produce other contagious asexual or sexual spores. Fomites like brushes and clippers play a crucial role in transmission. It has been claimed that certain spores can survive in saline water for at least a year. Spores may survive in proper settings for up to 12 to 20 months. Athrospores may be used to distribute specific spore types (such microconidia) through the air. both anthropo- and zoophilic(15).

Pathogenesis

Some dermatophytes, including T. rubrum, have immune-inhibitory mannans in their cell walls..(16). As a result, the fungus can infect the skin without being shed before it can take hold. The causative fungus can produce proteases, which break down keratin, serine-subtilisins, which break down protein by starting a nucleophilic attack on the peptide bond through a serine residue at the active site, and keratinases, which eat through keratinized tissue. These enzymes allow the fungus to enter the horny layer of the skin and spread outward..(17). In healthy immunocompetent hosts, host defensive mechanisms such as the activation of serum inhibitory factor, polymorphonuclear leukocytes, and complements prevent the fungus from entering deeper tissues. (17).

Staphylococcus aureus

One of the main microorganisms responsible for human infections ranging from moderate, non-life-threatening illnesses to severe, life-threatening illnesses is Staphylococcus aureus. A key risk factor for Staphylococcal infections in carriers is endogenous source, which is a common commensal bacteria that often colonizes the skin and mucosal membranes..(18).

Staphylococcus aureus is a common cause of bacterial skin infections and soft tissue diseases globally. In the USA, S. aureus is in fact the most common cause of skin and soft tissue infections.(19) . The majority of skin and soft tissue infections (SSTIs) in people are caused Staphylococcus aureus (S. aureus). Since S. aureus is becoming much more resistant to antibiotics, new approaches to treatment S. aureus infections are urgently needed. (20). Antibiotics like B-lactams, daptomycin, and glycopeptides cause S. aureus to acquire resistance to them. Additionally, Methicillin-Resistant Staphylococcus aureus (MRSA) increases the production of B-lactamases, which decreases the effectiveness of antibiotics..(21). Skin and soft tissue infections (SSTIs) are caused by the bacteria Staphylococcus aureus, with methicillin-resistant S. aureus (MRSA) emerging as the primary factor and causing a major strain on the healthcare system. Regarding the management of MRSA infections and SSTIs, the Infectious Diseases Society of America (IDSA) last revised its recommendations in 2011 and 2014, respectively. Published in 2021 were the revised UK recommendations for treating MRSA infections. Older therapy methods could be harmful and need regular doses. The arsenal of novel agents for treating MRSA SSTIs has received little recent evaluations. (22). There are several virulence factors that contribute to S. aureus's pathogenicity, including enterotoxins, exfoliative toxins, and Panton-Valentine leukocidin (PVL) (23, 24). Due to the production of these virulence factors, it can therefore infect healthy people with illnesses. (24, 25). Additionally, it may result in the development of biofilms on tissues and medically implanted equipment. (23). S. aureus may spread across the body and damage tissues, causing systemic illnesses. Different strains of S. aureus have developed that exhibit multi-drug or methicillin resistance (MDR) (26). Methicillin resistance is often caused by mecA on the staphylococcal chromosomal cassette mec (SCCmec), which also causes broad-spectrum resistance to all _-lactam antibiotics. S. aureus strains exhibit resistance to all commonly used antibiotics. MDR, in contrast, is characterized as an acquired resistance to three or more classes of antibiotics. (27) Methicillin-Resistant S. aureus (MRSA) Infection S. aureus shows a marked resistance to common antibiotic therapies. The first resistant S. aureus strains were found a few years after the introduction of penicillin; in this case, S. aureus produced the Antibiotics B-lactamase enzyme by evading drug therapy. In 1959, methicillin was introduced; as a consequence, in 1960 (28) , the first strains resistant to this antibiotic (MRSA) were highlighted. The ability to escape treatment by methicillin results from three different mechanisms The first mechanism produces a penicillin-binding protein called PBP2a, encoded by the mecA gene, causing a decrease in the antimicrobial activity of B - lactams (29). Recent research has shown that the new mecA gene homologues, such as mecB, mecc, and mecd, are generally undetected by present research methods. [47–49]. Borderline oxacillin-resistant S. aureus (BORSA)] is the second method of antibiotic evasion in which the amount of B-lactamase, the enzyme that causes oxacillin resistance, increases. escalation of MRSA in the community the majority of MRSA infections have historically been hospital-related, affecting immunocompromised people or patients with risk factors such as surgery, surgical incisions, the presence of indwelling medical devices, or pre-existing infections. [12, 29]. In addition to being

highly virulent, CA-MRSA spread fast among several groups of healthy people by showing an elevated ability for transmission and/or colonization. [11, 18, 30–41CAMRSA infections have been reported throughout the world, including in South America, Asia, Australia, Canada, Europe, and the USA. Pandemic levels have been achieved very quickly. [42–51]. The epidemic spread of CA-MRSA in the USA has increased the overall burden of MRSA. [52]. Since the introduction of CAMRSA, concurrent increases in staphylococcal load have been reported globally. [51, 53–55Approximately 90% of CA-MRSA infections manifest as skin and soft tissue infections, and the majority are abscesses or cellulitis with purulent discharge. [52, 53, 55–57]. The most common CA-MRSA strains can, however, also be known to cause serious invasive illnesses including necrotizing fasciitis and necrotizing pneumonia, which were previously uncommon until the emergence of CAMRSA [17, 29, 58–63]. Although CA-MRSA invasive infections are infrequent, they were responsible for 14% of all invasive MRSA-associated fatalities in the USA in 2005. [5].

2. Methods

70 of Sample collection will be collected from patients suspected by dermatologist at Ramadi Teaching General Hospital, Ramadi – Al-Anbar, Weat of Iraq. Each sample will be divided into two parts one for direct microscopic examination by KOH 10 – 20% and other for culturing on (sabouraud Dextrose Agar) with cyclohexamide and chloramphenicol (SDACC), then will be incubated at 29C for 3 – 7 days. macroscopical and Microscopical examination will be studied colonies, (Macro and Micro) conidia. The growth of fungus using lacto phenol cotton blue dye is examined microscopically. Nature assisted in the formation of the fungus and conidia (large and small conidia). Distinguish between these groups.
All clinical samples of suspected were cultured on blood agar incubated at 37°C for 24 h
Subsequently. Growth appears as beta – hemolysis. After that ticking sample from the growth of blood agar and cultured on monnitol salt agar Incubated at 37°C for 24h subsequently. Growth appears yellow colony. To confirmatory methicillin resistance staphylococcus aureus (MRSA) cultured on Mueller Hinton agar with methicillin discincubated at 37°C for 24h . and dignosied by vitek2 (bioMérieux-France).

3. Result and Discussion

Patients with tinea pedis, tinea manuum, tinea cruris, tinea corporis, or tinea capitis may suffer dermatophytid complications. (30, 31) .
Tinea corporis is an usual mycotic infection. Contacts with humans, animals, and soil all contribute to the spread of the disease. The species most usually implicated include Microsporum canis, Trichophyton rubrum, T. mentagrophytes, and M. gypseum. One to three weeks of latency are typical. Typically, the face and upper limbs are affected. A single round or oval erythematous lesion with scales in the center and well defined, vesicular margins characterizes the tinea corporis presentation. The majority of the time, pruritus is mild to moderate. (32).
No of the patient's age, the weather, or the location, Staphylococcus aureus is the most prevalent bacteria associated with skin infections globally. A few of the bacteria's toxins, which result in a wide range of clinical symptoms, are responsible for the majority of cutaneous clinical manifestations. The primary toxins involved in the majority of dermatological symptoms linked to S. aureus are Panton Valentine leucocidin, exfoliatins, enterotoxins, and toxic shock syndrome toxin 1(33).
this study disagrees with our study. From 2012 to 2014, we observed 108 children, aged between 4 and 11 years, with tinea corporis confirmed by mycological examinations. Clinical and mycological recovery was observed in 93 of them (86.1%). Only 4 of these children (3.7%) developed S. aureus superinfections., that staphylococcal superinfections of tinea corporis are due to scratching, Tinea corporis is a common mycotic infection in children. Staphylococcus aureus superinfections may be observed in atopic children with tinea corporis suffering from severe pruritus and consequent scratching (34).
In a healthy state, the human body contains a diverse range of microorganisms, including bacteria and fungi. The interactions between these taxonomically diverse microorganisms are highly dynamic and dependent on a multitude of microorganisms and host factors. Human disease can develop from an imbalance between commensal bacteria and fungi or from invasion of particular host niches by opportunistic bacterial and fungal pathogens. This Review describes the clinical and molecular characteristics of bacterial–fungal interactions that are relevant to human disease.(35).
Infections caused by dermatophytes may also be associated by secondary bacterial infections, especially in moist or occluded skin regions (eg, the feet)(36). A Gram stain and culture should be performed on patients who have erosions, ulceration, discomfort, or bad odor in the afflicted area to check for subsequent bacterial infection.
We explanation that tinea corporis infect the skin skin and make itching the skin and disrupted skin immune defense and the infected human itching site of infection help to contamination and easily transported bacteria which make that MRSA infected the skin. Staphyloccocci that under normal circumstances do not lead to cutaneous infections. However, when the skin barrier function is disrupted by a chronic skin disease the microorganisms at the skin surface have direct access into the skin. Massive microbial colonization may lead to subsequent clinically apparent cutaneous infection(37)..S. aureus may both amplify the skin inflammation in dermatitis and may lead to worsening of the barrier defect of

the skin because the exotoxins secreted by S. aureus act as superantigens that may penetrate the skin barrier (38).table (4-8).

Table (4 - 8): Relationship between Tinea and MRSA(N=100)							
Cross tabulation			MRSA				X2
			MSSA	negative	Positive	st.epid	
Tinea	Non	Count	0	30	0	0	Value (100) Df(3) p-value (0.001)
		% within Tinea	0.0%	100.0%	0.0%	0.0%	
	Positive	Count	8	0	54	8	
		% within Tinea	11.4%	0.0%	77.1%	11.4%	
Total		Count	8	30	54	8	
		% within Tinea	8.0%	30.0%	54.0%	8.0%	
X2= Chi-Square Tests							

The table (4 - 8): demonstration that relationship between Tinea and MRSA. (P-value = 0.001) at the ($p \leq 0.05$) level of significance.

Transmission of the fungus is facilitated by a moist, warm environment, sharing of towels and clothing, and wearing of occlusive clothing. Predisposing factors include personal history of dermatophytosis (e.g. Tinea pedis, tinea capitis, tinea cruris, and tinea unguium), concurrently afflicted family members, domestic pets, and Puducherry's hot and humid climate all lead to the dermatophytes' prolific growth. According to Jain N. et alstudy .'s on dermatophytoses in Jaipur, the city has hot, dry summers, while the monsoon season is marked by excessive humidity. Fungal infections are more likely to occur as a result of several climate factors. was of the idea that Assam has significant rainfall during the monsoon season and that the humidity is high for nearly the entire year. As a result of the high humidity, dermatophytes can develop on the skin easily, increasing their prevalence. There were many incidences of tinea corporis during the summer and before the monsoon season. (39). From the present study it is evident that dermatophytosis pose a significant health problem in this locality due to its hot and humid climate for a major part of the year. Hence, there is need for frequent monitoring and to create awareness regarding the adequate preventive measures in this locality. But in our study show that most of the patient 50 living in rural and 20 living in urban, as Table (4 - 3)

Table (5 - 3): distribution of patient according type of living and gender (N=70)			
	Rating	Frequency	Percent
Living	urban	20	28.57
	Rural	50	71.43
	Total	100	100.0
Gender	Female	55	55.0
	Male	45	45.0
	Total	100	100.0

The table (4 - 3): presentation that 60 % of patient living in rural and 40% living in urban. Also the results of study show that 55% of sample was female.

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