



ISSN: 0975-766X  
CODEN: IJPTFI  
Review Article

Available through Online  
www.ijptonline.com

### SUPERBUG SKIN INFECTION-AN REVIEW ARTICLE

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Received on 02-09-2012

Accepted on 20-09-2012

#### Abstract

Superbug caused by multi resistant Staphylococcus aureus and belonging to the family Staphylococcaceae a common cause of infection in hospitals and the community, it is becoming increasingly virulent a resistant to antibiotics. It spread quickly through patient contact, respiratory droplets and food. The symptoms are rashes on the body, low blood pressure, joint pain, headache, chest pain and fatigue. MRSA isolates by pulsed-field gel electrophoresis (PFGE), spa typing, multilocus sequence typing, and SCCmec typing. The mechanism of antibiotic resistance can be an effected of horizontal gene transfer and unlinked point mutation in the pathogen genome. After the identification of MRSA infection it is treated with Vincomycin, Linezolid and Clindamycin.

**Keywords:** Staphylococcaceae, resistant, skin and soft tissue infection, meningitis and septicaemia.

#### History

As early as 1959, the doctors and researchers were aware to “staphylococcus” ability to involve resistant to antibiotic medication. Methicillin was developing specifically to treat penicillin resistant in 1961, the reports of a “methicillin – resistant staphylococcus aureus (MRSA) were appearing in Europe. It has also found in Australia, Japan, and United State (U.S.) [1]. It seems that MRSA is suddenly in the news today. In October 2007 the centre of drug control (CDC) report was published in an issue of the journal of the American Medical Association [2].

It has been suggested that it is just now being noticed that these infections are more prevalent than earlier thought. It means that MRSA is now being deliberate better than measured in the past. In 1999 there were only 127,000 and in 2005 there were more than 278,000 cases. During this stipulation the time period death also augmented from 11,000 to more than 17,000 deaths [3]. There was primarily a hospital tribulation that has at present become an

community problems. There is no information as to whether MRSA superbug has complicated within the community. One thing is persuaded that the community – based form of MRSA did not instigate from the hospital form of MRSA. It is far and wide believed that drug resistant – bacteria may residential from the overuse and mistreatment of antibiotic. 85 % of MRSA skin infections are still establish within hospitals and health care facility, instead of community. The community - associated MRSA skin infection found in athletes, military members, children, pacific, islanders, Alaskan, American and Indians [4]. The study published in Journal Lancet infections Disease, establish the being there of a new gene called NDM-1 that gives assured class of bacteria the capability to produce a chemical that designate many antibiotic worthless. The newly superbugs found in 180 patient samples from Pakistan, India and U.S. in samples from patients who had surgery In India.

The NDM-1 gene is creating in bacteria which live in gut, such as E. coli, which come from a different family than MRSA. The bacteria can deliver useless the entire family of antibiotic which include penicillin and other copied antibiotics.

## **Introduction**

### **What is superbug:**

The superbug is called as MRSA (Methicillin – resistant Staphylococcus aureus) it is a STAPH infection that opposed to a very strong antibiotic. It is a bacterial infection that comes from informal skin to skin contact. MRSA infections are the most commonly transmitted from person to person by touching condition [5]. It is moreover possible to obtain MRSA infection from dust containing contaminated skin particles or from objects in the environment or from surface that may be contaminated with the MRSA infection [5]. The latest superbug was naked in India and Pakistan, scientists identified it by the name Of New Delhi Metallo – beta – lactamase or NDM – 1. This NDM – 1 superbug makes bacteria resistant to virtually antibiotics, including the majority powerful group of antibiotics such as Carbapenams [6].

### **What is MRSA:**

Methicillin - resistant Staphylococcus aureus have been well known within a squat period of time after antibiotic type “methicillin”. It was initial time applied in handling of Staphylococcus aureus and dissimilar infections bacteria type. MRSA progresses substantially inside 24 - 48 hours of first topical symptoms. Later on by subsequently 72 hrs, MRSA may acquire delicate on human tissues and ultimately become resistant to medicine.

Some symptoms of these bacteria are small red bumps that resemble pimples, spider bites, or boils that may be followed by fever and time to line rashes. The bumps get larger within a couple of days, more painful and pus-filled boils. MRSA superbug is due to the following risk factors that are nearby within these groups of people:

- Close skin- to- skin contact
- Cuts or abrasions in the skin
- Contaminated items and surface
- Crowded living conditions
- Poor hygiene

**Types of MRSA**

MRSA has three categories for these bacteria consequent to where the bacteria acquired

Which are as following:-

**1. Community- Acquired MRSA (CA - MRSA) :-**

It occurs in individual commonly healthy and not receiving healthcare as ongoing basis for treatment for chronic symptoms and complications [7].

CA – MRSA can be defined according to genetic, phenotype, and clinical characteristics: PVL (Panton – Valentine Leukocidin); SCC mec, S aureus that produced the (Staphylococcus Cassette Carrying Methicillin); SSTI (Skin and Soft Tissue Infection ); Methicillin resistant in S. aureus is explained by a number of alteration of penicillin – binding Proteins. SCC is unique family of mobile genetic elements found on the chromosomes of Staphylococcus [8]. The mecA gene is mediator of penicillin – binding protein 2b that encodes resistant to oxacillin [7].

**Table 1. Defining Characteristics of MRSA Infection**

Definitions	Characteristics
Clinical	Absence of healthcare exposure or risk factors SSTI Symptoms for 48 hours
Phenotypic	Susceptible to non-β-lactams antibiotics (eg, clindamycin)
Genetic	<p>SCC mec IV gene</p> <p>PVL gene</p>

## 2. Healthcare-Acquired (HA-MRSA):-

It refers Hospital or Healthcare acquired and Corresponding to the U.S. Centers for Disease Control (CDC), supplementary than 85% of MRSA Infections take place in healthcare Facilities [4]. At the create of HA - MRSA, an individual will develop red, inflamed, swollen and painful areas of the skin. Bumps may form that are described as pimples or boils and similarly from the outer shell to spider bites. The wounds are those the individual may have been treated in the hospital initially [9]. MRSA is a danger health problem, increasing from 2% to 63% of total STAPH infections between 1974 and 2004 years. HA-MRSA infections may comprise surgical wound infections, urinary tract infections, blood stream infections, and pneumonia.

### What is superbug NDM-1

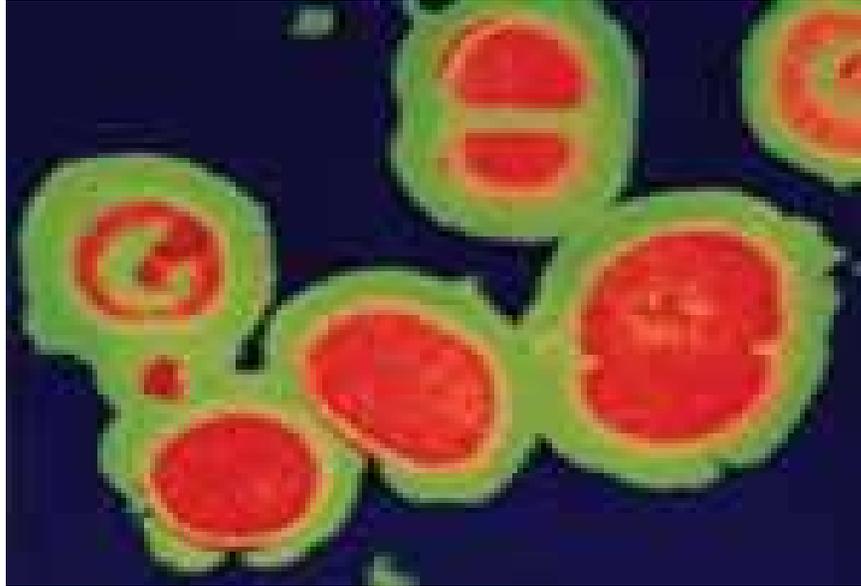
The New Delhi Metallo – beta - lactamase, or New Delhi Metallo  $\beta$  Lactamase, commonly referred to as Superbug NDM-1, is a set up enzyme which develops immunity in bacteria to beta-lactum antibiotics ( $\beta$  lactum). What evokes the further most anxiety is this resistance to beta-lactum antibiotics, a lot of the carbapenem family, that were hitherto successful in tackling otherwise antibiotic resistant bacteria. This killer gene in NDM-1 belongs to a larger gene family which is beta-lactamase enzymes called as Carbapenemases. Such killer genes are widely referred to as superbug as they have immense desolation credible due to their resistance to antibiotics. Consequently, the warning had come from the United Kingdom Health Protection Agency (UKHPA) on 3rd July 2009. Most isolates with NDM-1enzyme are resistant to all standard intravenous antibiotics for treatment of skin infections.

NDM-1 is a newly identified problem, only predictable since December 2009 in the medical literature. There have smaller amount than 100 cases identified external of the Indian subcontinent, so this is not a pandemic like bird flu or swine flu. On the other hand the number of cases is increasing and the apprehension is that these highly resistant bacteria could supplant additional antibiotic sensitive strains. If this happens, the antibiotic arsenal that has been built up in excess of the last 80 years will be critically compromised.

### Bacteria of superbug

Because of its colour on a laboratory plate, is generally harmless & sometimes cause minor infection in wounds or generate boils. Golden Staph is becoming resistant to the majority powerful of antibiotic and returning as a big

problem in most large Australian hospitals, attaching intravenous lines, catheters and wounds after operation. It spread quickly through patient contact, respiratory droplets and food. These resistant bacteria array from 20-40 percent of all Golden Staph infections in chief eastern Australian hospitals. About 5 percent of those multi resistant staphylococcus aureus (MRSA) can only treated with vancomycin [10].



Multi resistant Staphylococcus aureus(MRSA)

**Scientific classification:**

Kingdom: Bacteria

Phylum: Firmicutes

Class: Cocci

Order: Bacillales

Family: Staphylococcaceae

Genus: Staphylococcus

Species: S. aureus

VRE is another bacterium which hits the title from time to time. VRE or Vancomycin Resistant Enterococci was first acknowledged in Australia in 1994 [11]. The potent antibiotic Vancomycin was an in actuality last defense against cruel infections caused by staphylococcus and enterococcus bacteria [12]. Scientists now agonize that VRE not will persist to multiply but will allocate its genetic secrets for endurance with other bacteria.

## **Resistant Pathogens**

### **1. Escherichia coli:**

It is commonly known as E. coli, cause gastroenteritis, hemorrhage colitis, urinary and genital tract infection. Resistant of E. coli is dreadfully high and getting poorer. 50 % of Australian strains resist the most commonly used cure, take Amoxicillin. When the bacteria is spread severe health condition arise. Many people are Hospitalized every year becoming infected and some as a result.

### **2. Pseudomonas aeruginosa:**

It is exceedingly prevalent opportunistic pathogens. One of the most characteristic of P. aeruginosa consists in stumpy antibiotic susceptibility. This low confrontation is attributable to a concerted action of multidrug efflux pumps with chromosomally – encoded antibiotic genes and the low permeability of the bacterial cellular envelopes [13]. Development of multidrug resistance by P. aeruginosa isolates requires quite a lot of different genetic trial that contain attainment of different mutations and Horizontal gene transfer of Antibiotic resistance [14].

### **3. Clostridium difficile:**

It is a nosocomial pathogens that cause diarrheal disease in Hospital worldwide [15, 16]. Clindamycin - resistant C. difficile was reported as the causative agent of bulky out breaks of diarrheal disease in hospital in New York, Arizo, Florida and Massachusetts between 1989 –1992 [17].

### **4. Streptococcus pneumoniae:**

It causes nasty common problems like middle ear infection Sinusitis bronchitis and life aggressive pneumonia. S. pneumonia spreads through respiratory droplets and can also cause meningitis and septicaemia [17]. At least half of Australia's strains are resistant to one antibiotic and a third are resistant to 3 or 4 antibiotic. Given the great numbers of people who undergo infections caused by Streptococcus it, one sence good deal more important than VRE or Golden Staph.

### **5. Camphylobacter:**

On the international front wall, it cause gastroenteritis and is passed on through animals, is performance resistant to a class of antibiotics called quinolones. Like Salmonella, which is also showing resistant, it comes from food and water. Antibiotic resistant Camphylobacter is a giant issue in Europe and US especially in relation to the use of similar antibiotic in food producing animals.

## **Antibiotic misuse**

Antibiotic misuse refers to the antibiotic abuse or antibiotic overuse Which has serious effects on public health. This overuse constructs multi – antibiotic resistant life threatening infections by “superbug” sometimes out of relatively harmless bacteria [18]. The widespread use of antibiotics both inside and outside of medicine is playing an imperative role in the appearance of resistant bacteria [19]. Although the survival strategy of bacteria contribute to bacterial resistant, human bear mainly of the responsibility for the problem. Leading causes of bacterial resistant include:

### **1. Unnecessary antibiotic use in humans:**

MRSA is the result of decades of too much and unnecessary antibiotic use [20]. Antibiotic have been prescribed for colds, flu and other viral infections that don't respond to these drugs, as well for simple bacterial infections that normally clear on their own [21].

### **2. Antibiotic in food and water:**

Drugs are not the font of antibiotic. For example, in United State, antibiotic can be found in beef cattle, pigs and chickens. These drugs can affect the safety of the meat, milk and eggs produced from those animals and can be the source of superbug. For example, farm animals, particularly pigs, are supposed to be talented to contaminated people with MRSA [22]. Routine feeding of antibiotics to animals is banned in European Union and many other industrialized countries [23]. Antibiotic given in the suitable doses to animals that under the weather don't emerge to produce resistant bacteria.

### **3. Germ mutation:**

When antibiotic are used approximately, they supply to the rise of Bacterial resistance because they don't demolish each germ. Bacteria live on an evolutionary fast tracks, so that continue to exist treatment with one antimicrobial agent learn to resist other. Some germs end up resistant to just about everything. A handful of antimicrobial agents are now effectual against most forms of staph.

## **Mechanism**

Antibiotic resistance can be a effect of horizontal gene transfer and also of unlinked point mutations in the pathogen genome at a velocity of concerning 1 in  $10^8$  per chromosomal replication [24]. The antibiotic achievement against the pathogen can be seen as an environmental pressure, those Bacteria which have a alteration allowing them to

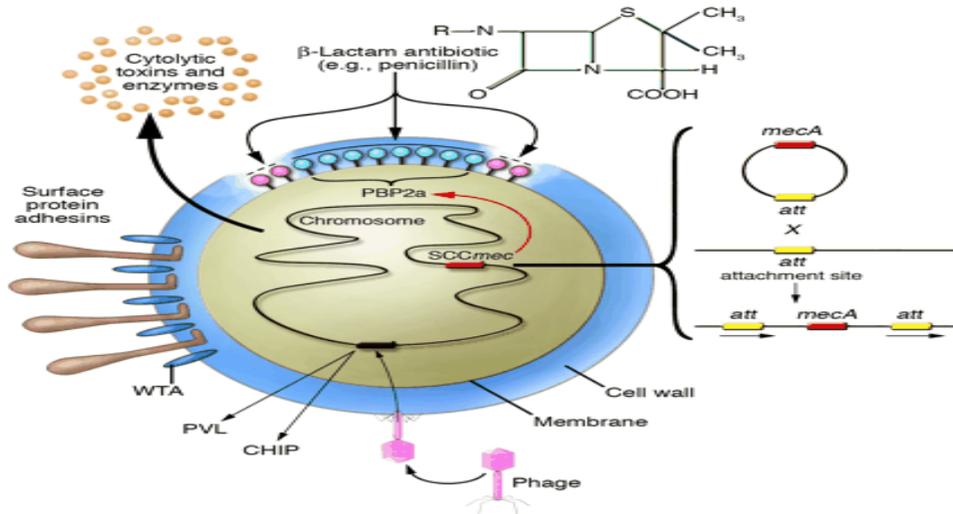
undergo will live on to replicate. They will pass this peculiarity to their offspring, which will result in a fully resistant colony.

The four chief mechanisms by which microorganisms exhibit resistance to antimicrobials are:

1. Drug inactivation or modification: e.g. enzymatic deactivation of Penicillin G in a quantity of penicillin-resistant bacteria through the production of  $\beta$ -lactamase.
2. Alteration of goal site: e.g. alteration of PBP - the binding target site of Penicillin's in MRSA and other penicillin-resistant bacteria.
3. Alteration of metabolic pathway: e.g. several sulfonamide-resistant bacteria do not require para-aminobenzoic acid (PABA), an important predecessor for the synthesis of acid and nucleic acids in bacteria reserved by sulfonamides.
4. Compact drug accretion: by falling drug permeability and ever-increasing active efflux (pumping out) of the drugs crosswise the cell surface [25].

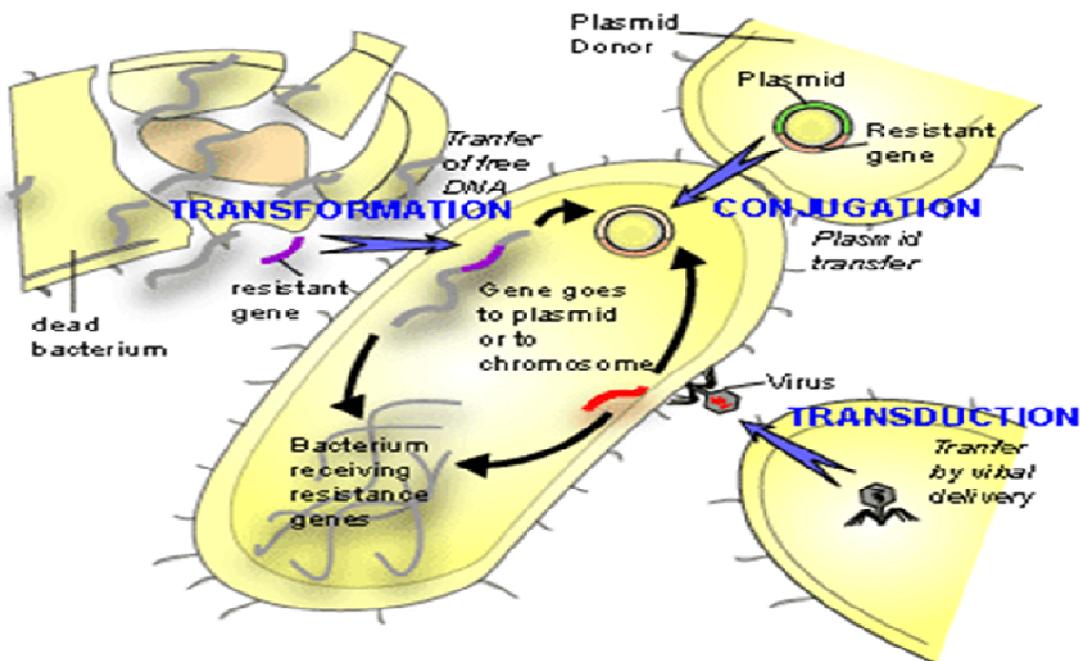
#### **Methicillin-resistant *S. aureus*:**

Healthy individuals have a minute but restricted risk of contracting an insidious infection caused by *S. aureus*, and this risk is enlarged among carriers. Hospital patients who are catheterized or who have been treated surgically have a significantly greater rate of infection. In some, but not all, developed countries, many nosocomial infections are caused by *S. aureus* strains that are multiply resistant to antibiotics known as methicillin-resistant *Staphylococcus aureus* (MRSA) [26, 27] although the acronym MRSA is somewhat ambiguous because the semi-synthetic  $\beta$ -lactam methicillin is no longer used to treat *S. aureus* infections. In MRSA, the straight acquired *mecA* gene encodes a penicillin-binding protein (PBP2a), which is basically insensate to methicillin and all  $\beta$ -lactamase that have been developed, including the isoxazolyl penicillin's (e.g., oxacillin) that superseded methicillin, in addition to the broad spectrum  $\beta$ -lactams (third-generation cephalosporins, cefamycins, and carbapenems) that were introduced chiefly to treat infections caused by Gram-negative bacteria [26]. In distinction to nosocomial MRSA strains, which are typically multidrug resistant, the freshly emerged community-acquired MRSA (CA-MRSA) strains are susceptible to drugs other than  $\beta$ -lactamase [28].



The *mecA* gene is the gene responsible for methicillin resistance and is fraction of a movable heritable aspect found in many MRSA strains called SCCmec. There are at least five different SCCmec elements. These elements incorporate at the same time site in the chromosome by a mechanism connecting site – specific recombination and removal from the chromosome at attBsc that is a part of an open analysis border of unknown function near the source of replication. The genetic mechanisms responsible for the transfer of these mobiles essential are uncertain. However, the MecA gene appearance alters PBP2A in *S. aureus* resulting in a loss of target resemblance. The *mecA* gene encodes a new b- lactase insensitive to penicillin [29].

**Horizontal Gene Transfer – another mechanism for Resistant**



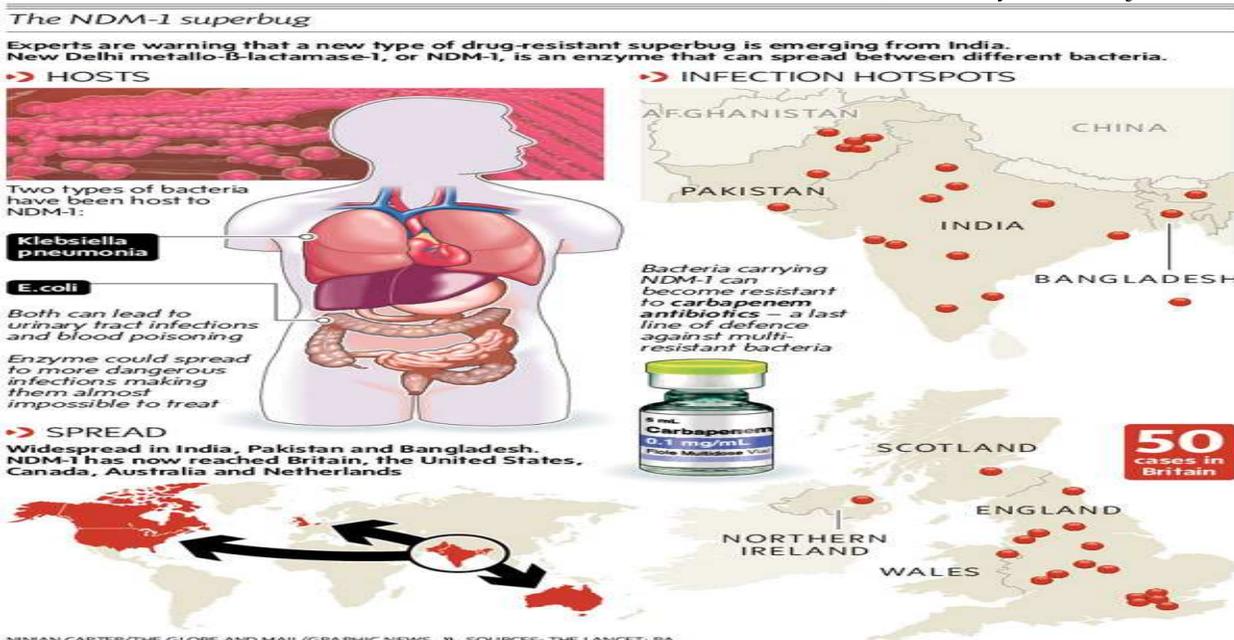
The expansion and achievement of resistance in MRSA also requires the presence of other mechanisms. Horizontal gene transfer is mechanism by which plasmids (resistant genes contained in small packets) establish within the cytoplasm of the bacteria have the ability to transfer resistance genes between the similar and dissimilar species.

The three different mechanisms involved in resistance are:

1. Conjugation is where there is cell-to-cell contact, this is bacterial sex. Some scientists consider that this is the main mechanism by which resistant gene transfer occurs.
2. Transformation is where bacteria from the outside environment are acquired.
3. Transduction involves bacteriophages transferring DNA between two personally Related Bacteria [30].

### **Signs and symptoms**

*S. aureus* most commonly colonizes the anterior nares (the nostrils), although the rest of the respiratory tract, opened wounds, intravenous catheters, and urinary tract are also probable sites for infection. Healthy individuals may carry MRSA asymptotically for periods ranging from a many years. Patients with compromised immune systems are at a barely some weeks to significantly better risk of symptomatic secondary infection. Combined with additional hygienic measures for those in contact with contaminated patients, screening patients admitted to hospitals has been establish to be effectual in minimizing widen of MRSA in hospitals in United States [31], Finland, and the Netherlands [32]. The most familiar way in which MRSA starts off is as a skin infection, in the form of either an abscess or a boil. This may glance like small red bumps resembling pimples or like a spider bite. However, these can rapidly become deep, painful abscesses that must be surgically drained [33]. Along with this skin concern flu - like symptoms, which include a high fever and sweating. If left entire MRSA can burrow deeper into your body and cause potentially life - threatening infections in your bones, joints, bloodstream, heart valves or lungs. MRSA can also illustrate up as a urinary tract infection. In the most horrible case, MRSA may actually pierce into the lungs and cause pneumonia, which includes a towering fever and difficulty with mouthful of air. Some of these symptoms are so subtle that they may truly be missed upon first glance However; the symptoms also depend upon where your infection is located [34].



**Fig: symptoms of superbug in NDM-1 [35]**

Other signs and symptoms of MRSA include:

1. A external skin infection that has a honey – colored scab and blisters.
2. A pussy infection in your hair follicles.
3. A compilation of pus under your skin that is red, hot to the contact, swollen and tender.
4. An infection that is bigger than an boil and has several openings.
5. An infection in your spongy tissue that may establish as what may look similar to a pimple or bug bite but then become hot to the touch, red, swollen and tender.
6. A pigpen which is basically an infection of the eyelids.

If it is a severe case of MRSA, then these signs and symptoms may be present:

- Low blood pressure.
- Joint pain.
- A severe headache.
- Shortness of breath.
- A rash over the majority of your body.
- Chest pain.
- Fatigue.

- Muscle aches.
- Generally feeling ill.

**Diagnosis:**

Diagnosis of MRSA is carried out culturing the bacteria isolated from suspected tissue samples. MRSA bacteria are created, further tests are carried out to settle on which antibiotics are most likely to treat the infection effectively. Many hospitals now test everyone who is admitted to see if they carry MRSA. Swabs from skin, the nose, and urine and blood samples will be testes and it can acquire three to five days for the results to come back [36]. A genetic test is also available. Real-time PCR and Quantitative PCR and are increasingly being employed in clinical laboratories for the rapid detection an identification of MRSA strains [37, 38].

A hospital patient may be tested for MRSA if:-

- Signs of infection are seen
- There is previous history of infection
- The patient is transferred from a hospital with MRSA infection to a new hospital

**Culture:** Once MRSA is suspected in a patient the following samples can be extracted from the patient and sent for culture to a microbiology lab:

- Blood sample
- skin tissue
- Pus from the wound, if any
- Urine
- Other biopsy material

Two First Steps to Identification

1. **Slide Agglutination:**

Determination of the “clumping factor” antigen, a cell wall-associated protein and adhesion for fibrinogen, differentiates *S. aureus* from many, but not all other staphylococcal species. A drop of plasma is added to a distilled water suspension (avoids auto-agglutination) of the organism. If positive, clumping occurs within 10 seconds. Latex agglutination kits can be used to detect clumping factor, protein A and other surface

[39].

## 2. Coagulase Test:

In humans, detection of the staphylocoagulase enzyme is considered definitive identification of *S. aureus*.

Mixing 0.1 ml suspension of an overnight culture with 0.5 ml of reconstituted plasma in a small test tube is followed by incubation from 4 hours to overnight at 37°C. Observation of a clot constitutes a positive test.

False positive and false negative results and Occur from impure plasma or a mixed culture [39].

### Genetic tests:

The Food and Drug Administration (FDA) of the USA approved a genetic test using a PCR (Polymerase Chain Reaction) which is done using a blood sample from the MRSA suspected individual. Although the genetic material of the MRSA bacteria can be detected in a span of two hours the test is recommended only for diagnosis and not for monitoring or in deciding the course of treatment [40].

### How accurate can the Diagnosis of MRSA:

**TABLE 1.** Comparison of the levels of accuracy of reactions of *S. aureus* isolates on CHROMagar Staph aureus, DNase, and MSA and of coagulase testing of CoNS after 18 to 24 h of incubation

Identification method	% of isolates showing positive reactions		Accuracy of medium in discriminating <i>S. aureus</i> and CoNS	
	<i>S. aureus</i> (n = 114)	CoNS (n = 22)	Sensitivity (%)	Specificity (%)
CHROMagar Staph aureus	100	0 <sup>a</sup>	100	100
DNase	98.0	4.6	98.0	95.4
MSA	98.0	36.5	98.0	63.5
Coagulase <sup>b</sup>	100	0	100	100

<sup>a</sup> *S. chromogenes* produced a natural carotenoid (orange or red) pigment and gave a slightly pink color. The isolate was identified by API20 Staph.

<sup>b</sup> All coagulase testing was confirmed by the standard tube method.

## **Prevention**

Hospitals are fighting back against MRSA infection by using surveillance system that track bacteria outbreaks and by investing in product such as silver coated catheters, silver impregnated gowns and gloves ionic silver complexes disinfectants [41]. There is Clinical evidence that topical dermatological preparations such as those containing tea tree oil and thyme oil may be effective in preventing transmittal of CA-MRSA [42]. In addition, other phytotherapeutic medicines too can reduce the use of antibiotics or eliminate their use entirely [43].

### **Top 10 Ways To Prevent MRSA:**

People infected with MRSA superbug have been growing around the United State in hospitals, homes, schools. People can prevent MRSA from harming others to help the Cause with Top 10 Ways to Prevent MRSA list [44, 45]. Which are as following -

1. Careful hand washing remains your best defense against germs. Scrub hand briskly for at least 15 seconds, then dry them with a disposable towel and use another towel to turn off the faucet.
2. Covered the open wound with sterile immediately and use dry bandages until they heal. The pus from infected sores may contain MRSA, and keeping wounds covered will help keep the bacteria from spreading.
3. Don't share personal items such as towels and razors, and just in case you have a scratch that would offer entry to MRSA, always keep your clothing or a towel between your skin and any shared surfaces. When using public toilets wipe the seat with toilet paper.
4. Shower immediately after each game or practice. Bath on regular bases by using soap and water and don't share towels.
5. Sanitize linens - If you have a cut or sore, wash towels and bed linens in a washing machine set to the "hot" water setting and dry them in a hot dryer.
6. Never swim in a swimming pool with an open wound.
7. When handling blood, wear gloves and a mask.
8. Avoid unnecessary surgeries.

9. Get tested - If you have a skin infection that requires treatment, ask your doctor if you should be tested for MRSA. Testing specifically for MRSA may get you the specific antibiotic you need to effectively treat your infection.
10. Use antibiotics appropriately and do not stop until your doctor tells to stop.

**Treatment:**

According to U.S.A’s Centers for Disease Control and Prevention (CDC) “The first line treatment for mild abscesses is incision and drainage. If antibiotic treatment is clinically indicated, it should be guided by the susceptibility profile of the organism.” [46, 47] Fortunately for mankind, most MRSA still can be managed by a few but specific antibiotics some of which are mention below-

**1. Treatment options for Mild-to-Moderate SSTI, [48, 49]:-**

S.No.	Drug	Oral Dose	Monitoring	Adverse Reactions	Drug Interactions
1.	<b>TMP-SMX</b>	1 DS table twice daily (Consider higher dosing with more serious infections.)	Routine lab tests are not indicated. In case of prolonged treatment or in complicated patients: Monitor CBC/platelets, and renal & hepatitis parameters.	Rash, erythema, multiforme, Stevens-Johnson syndrome, hemolysis w/ G-6-PD deficiency, hepatitis, pancreatitis, bone marrow suppression.	Dapsone, anticoagulants, phenytoin, cyclosporine, diuretics, MTX.
2.	<b>Clindamycin</b>	450 mg three times daily OR 300 mg four times daily	Routine lab test are not indicated.	GI upset and relatively high incidence of C. difficile- induced colitis as compared to other antibiotics.	

- For less serious infections, antibiotic treatment may be avoided by using conservative measures. When antibiotics are administered, do so conjunctions will conservative measures.
- Minocycline or doxycycline, 100 mg twice daily, may an alternative treatment option; however, laboratory susceptibility results must be carefully reviewed.[52]

- Do not use flouroquinolones to treat MRSA. MRSA isolates may be sensitive to quinolones in vitro; however, the potential for resistance limits the use of this class of antibiotic.
- Rifampin is not recommended for treatment of uncomplicated SSTI<sub>s</sub>. For treatment of recurrent of complicated SSTI<sub>s</sub>, rifampin can be considered on a case-by-case basis.
- When rifampin is administered in combination with TMP-SMX, the dose of TMP-SMX must be increased.  
Example: 2 tablets twice daily.

## 2. Treatment options for Serious MRSA Infections:-

S.No.	Drug	Dose	Monitoring	Adverse Effects	Drug Interactions
1.	<b>Vancomycin</b>	500 mg IV every 6 hours OR 1,000 mg IV every 12 hours Infuse of 1 hour.	Monitor through drug levels within 1 hour of the next dose: Target 1s 10-15 mcg/ml. Auditory function, Renal function/CBC.	Ototoxicity, nephrotoxicity, drug fever, rash, reversible neutropenia. If used with aminoglycosides, increased nephrotoxicity. Histamine reaction, flushing.	Anesthetics
2.	<b>Linezolid</b>	600 mg twice daily, orally or IV Can take with or without meals.	CBC with different/platelets count weakly, Monitor BP if hypertensive or talking a sympathomimetic.	Diarrhea, bone marrow suppression, nausea, headache. Peripheral and optic neuropathy have been reported in patients treated with linezolid.	Avoid adrenergic and serotonergic agents, including decongestants and SSTI antidepressants.

- Sepsis requires at least 2 weeks of IV antibiotics. Endovascular infections such as endovascular infections such as endocarditis, osteomyelitis and deep seated infections require 4-6 weeks of therapy and may require combination antibiotic therapy, consult with expert on treatment regimen and length of treatment.[50, 51]
- Linezolid is costly and has potential for serious toxicities. Linezolid should only be used after consultation with a physician expert to determine if alternative antimicrobials would be more appropriate.
- Avoid foods with very tyramine content such as packaged soups, pickled/smoked fish, orange pulp, fava beans, and aged cheeses.

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