A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA IN A TERTIARY CARE HOSPITAL

Dissertation submitted to

THE TAMILNADU DR.M.G.R.MEDICAL UNIVERSITY

In partial fulfillment of the regulations

for the award of the degree of

M.D. (MICROBIOLOGY)

BRANCH - IV

MADRAS MEDICAL COLLEGE

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU.

MAY 2019

CERTIFICATE

This is to certify that this dissertation titled "**A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA IN A TERTIARY CARE HOSPITAL**"is a bonafide record of work done by **DR.KOKILA S,** during the period of her Post Graduate study from 2016 to 2019 under guidance and supervision in the Institute of Microbiology, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai- 600003, in partial fulfillment of the requirement of **M.D MICROBIOLOGY** degree Examination of **The TamilnaduDr.M.G.R Medical University to be held in May 2019.**

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ACKNOWLEDGEMENTS

First and foremost, I would like to thank God Almighty for giving me the strength, knowledge, ability and opportunity to undertake this research study and to persevere and complete it satisfactorily. Without his blessings, this achievement would not have been possible.

I wish to express my sincere thanks to **Dr.R.Jayanthi, M.D.FRCP (Glas),** Dean, Rajiv Gandhi Government General Hospital & Madras Medical College, Chennai-3 for permitting me to use the resources of this institution for my study.

I express my thanks to **Dr.J.Euphrasia Latha, M.D.DGO,** Director, Institute of Microbiology for her guidance and support.

Sincere thanks to Former Professor **Dr.MangalaAdisesh M.D.,** Institute of Microbiology for her constant encouragement and support during this work.

I owe my heartfelt gratitude and sincere thanks to my guide **Dr.R.Vanaja, M.D.,**Professor, Institute of Microbiology for her valuable suggestions, guidance,constant support, motivation and encouragement throughout this study.

My Sincere thanks to Professor **Dr.V.R.Dhanalakshmi,MD,DD,Dip.NB,** Head of the Department,Department of Dermatologyfor her constant encouragement and support during my study.

I would like to thank all my Professors **Dr.S.ThasneemBanu M.D., Dr.U.Umadevi M.D., and Dr.C.P.Ramani M.D.,** for their support during this study.

I extend my gratitude to my co-guide**Dr.P.ShanmugapriyaM.D.,**Assistant Professor, Institute of Microbiology for his valuable guidance and constant support in this study.

I extend my gratitude to my former co-guide**. Dr. David Agatha M.D.,**Assistant Professor, Institute of Microbiology for her valuable guidance and constant support in this study.

I wish to extend my thanks to our Assistant Professors**Dr.Lakshmipriya.N M.D.DCH., Dr.K.G.VenkateshM.D., Dr.R.KesavanM.D.DCH.,Dr.C.Nithya M.D., Dr.J.PadmaKumariM.D., Dr.S.Vinotha M.D., Dr. B.GomathiManju M.D.,** for their support.

I wish to extend my thanks to our former Assistant Professors **Dr.Deepa.R M.D., Dr.N.RathnaPriya M.D, Dr.K.Ushakrishnan M.D., Dr.Sripriya.C.S M.D., Dr.B.Natesan M.D.DLO.,** for their support.

I would like to extend my thanks to all my postgraduate colleagues and technicians for their constant support and help in this study.

I am thankful to my dear parents, inlaws for their unconditioned love, sacrifice and constant emotional support. I owe my deepest gratitude towards my better half Dr.E.Mohanraj, for his eternal support and understanding of my goals and aspirations. His infallible love and support has always been my strength. His patience and sacrifice will remain my inspiration throughout my life. Without his help, I would not have been able to complete much of what I have done . I thank my my cute son **M.Pravasthik,**for his patience.

I would like to thank the **Institutional Ethics Committee ,**Madras Medical college for approving my study .

Last but not least, I would like to thank the patients participated in this study for their co-operation and support.

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Analysed Document:	A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA.docx (D42455016)
Submitted:	10/12/2018 7:32:00 AM
Submitted By:	varshinidr@yahoo.com
Significance:	1 %

Sources included in the report:

http://www.p-e-g.org/archiv_tmp/jahrestagung_22/work1/Pfeifer.pdf
https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3482783/
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LIST OF ABBREVIATIONS USED

AGN	-	Acute Glomerulonephritis
BHS	-	Beta Haemolytic Streptococci
CA-MRSA	-	Community Acquired Methicillin Resistant Staphylococcus aureus
HA-MRSA	-	Healthcare-associated Methicillin Resistant Staphylococcus aureus
LA-MRSA	-	Livestock-associated Methicillin Resistant Staphylococcus aureus
MRSA	-	Methicillin Resistant Staphylococcus aureus
MSSA	-	Methicillin Sensitive Staphylococcus aureus
MupRSA	-	Mupirocin Resistant Staphylococcus aureus
E-test	-	Epsilometer Test
ESBL	-	Extended Spectrum β-lactamases
CLSI	-	Clinical Laboratory Standards Institute
EUCAST	-	European Committee on Antimicrobial Susceptibility Testing
MIC	-	Minimum Inhibitory Concentration
SCC	-	Staphylococcal Cassette chromosome
PCR	-	Polymerase Chain Reaction
DNA	-	Deoxy ribonucleic acid
EB	-	EpidermolysisBullosa
CONS	-	Coagulase Negative Staphylococcus

ESBL	-	Extended Spectrum Beta Lactamase
GAS	-	Group AStreptococcus
WHO	-	World Health Organization
ECOFF	-	Epidemiological Cut-off value
PBP	-	Penicillin Binding Protein
GPC	-	Gram Positive Cocci
P.vulgaris	-	Proteus vulgaris
Ps. Aeruginosa	-	Pseudomonas aeruginosa
S.aureus	-	Staphylococcus aureus
S.epidermidis	-	Staphylococcus epidermidis
Spp	-	Species
S. pyogenes	-	Streptococcus pyogenes
E.coli	-	Escherichia coli

Introduction

INTRODUCTION

The skin is the body's largest and thinnest organ, with a surface area of 1.7 m^2 and accounts for 16% to 20% of the total body weight. It forms a self-repairing and protective boundary between the body's internal environment and the external environment. Skin plays an important role in the excretion of water and salts, control of body temperature, synthesis of important chemicals and hormones, and as a sensory organ. [1]

The skin continuously exposes to microbial pathogens, in order to prevent infection, cells within the epidermis and dermis have produced several innate strategies. The skin uses one of the Primary mechanisms in the early stages as immune defense by the synthesis, expression and release of antimicrobial peptides. The colonization by many pathogens were prevented by the skins normal microbial flora, pH, and chemical defenses (high salt and acidic environment) .[1]

Normal flora (also called "indigenous microbiota") refers to the diverse group of microbial population that every human being harbors on his/her skin and mucous membranes. Although there are many species of normal flora, these microbes typically fall into one of the two categories resident flora and transient flora. [4]

Resident Flora

These organisms are life-long members of the body's normal microbial community and are very closely associated with a particular area. When disturbed, they again re-establish themselves. For example, *Escherichia coli*is a resident flora of the intestine. They do not cause harm; rather they have beneficiary effect on the host. [4]

Transient Flora

The transient flora consists of microorganisms that inhabit the body surface or mucous membrane temporarily for a short interval. Many of the transient flora are potential pathogens which may cause disease under certain conditions, e.g. Pneumococcus and meningococcus in nasopharynx. In hospitals, patients may acquire many resistant organisms as transient flora from the healthcare workers and hospital environment.[4]

Skin microbiome

Microbiomes are thriving complex communities of bacteria, viruses and fungi, with approximately 1 million bacteria inhabiting each square centimeter of skin. That comprises mostly bacteria like *Actinobacteria, Firmicutes, Bacteroidetes and Proteobacteria*, with numerous subspecies thereof. *Actinobacteria* represents the largest phylum and includes *Propionibacteria*and *Corynebacteria*; *Firmicutes* includes *Clostridia and Bacilli*, the latter including the Class *Staphylococcus*. The composition of these organisms depends on

sebaceous gland concentration,temperature and moisture content, also on host genetics and exogenous environmental factors.

These organisms are not only just commensals but also play a significant role in immunemodulation and epithelial health than previously expected. By understanding microbe–hostinteractions and the factors that lead to microbial colonization will provide greater insight into thepathogenesis of skin diseases, such as the role of Staphylococci in atopic eczema, and the development of new antimicrobial and promicrobial agents. [5,6]

Bacterial skin infection is one of the most common clinical problem encountered in day to day clinical practice[10].Pyoderma is the generic term used to describe any variant of superficial bacterial skin infection. [8] Pyoderma constitutes a major portion among patients attending dermatological clinics in India. [9]

Pyodermas may present in two major forms.

- As a primary cutaneous infection
- As a superimposed conditionin the previously diseased skin

Immunosuppression, atopic dermatitis, pediculosis, scabies, pre-existing tissue injury and inflammation are the various predisposing factors. The source of infection may be family members, hostel inmates, school mates, military barracks, medical personnel, or inanimate objects like clothes, floor, walls and instruments used in hospitals. Overcrowded places and hospitalization of the sick provide increased opportunities for dissemination of the organism[2].

The two important pyogenic organisms *S. aureus* and *S. pyogenes* are the etiological agents most commonly isolated,the former being more common than the latter. Besides these, other organisms that are occasionally isolated from pyoderma are *Enterococci, Pseudomonas spp, E. coli, Proteus spp, Acinetobacterspp, and Klebsiella spp*.[8]

Most of the organisms isolated are found to be resistant to the antibiotics which were previously very effective so there is no response in many cases .The indiscreet and indiscriminate use of topical and systemic antibiotics leads to the antibiotic resistance. Multidrug resistant strains also possess the properties of transmissibility and virulence. As a result of introduction of newer antimicrobials and their extensive use, strains have been explodedthat they were resistant to greater number of antibiotics. Continuous use of antibiotics results in survival and spread of MRSA, ESBL producers and multidrug resistant Enterococci.[11] With the emergence of MRSA, vancomycin and linezolid were commonly used antibiotics for MRSA infections.

Mupirocin (MUP), a topical antibiotic was used for the treatment of skin- and soft-tissue infections as well as for decolonization of nasal carriers [104]. But the widespread use of mupirocin led to resistance among *S.aureus*, which was reported worldwide[105]

Ghadage D P&Sali Y A. (1999) et al states that most of the causative bacteria were found to be resistant to one or more antibiotics. They recommended

that while starting the antibiotic therapy, care should be taken to avoid unnecessary drug intake and so that in-vitro testing is essential for proper selection of antibiotics. Newer antibiotics must always kept in reserve for use only against resistant strains. Ideally, it would be better to carry out culture and sensitivity tests before prescribing antibiotics, but this is not always practical. [9]

Rapid emergence of multidrug resistance among most of the Gram positive bacterial isolates complicates the management of pyoderma and demonstrates the need for more judicious use of antibiotics. [12,13]Inspite of the fact that Pyoderma is easily treatable, it is known for their chronicity, recurrence, and other complications.Therefore timely recognition and prompt bacterial diagnosis with antimicrobial sensitivity is necessary for the effective management of pyoderma.[12]

By gaining knowledge about the pathogens and their antimicrobial susceptibility pattern, helps to prevent the emergence of resistant strains in future and guides the physicianin the management. Considering these aspects, the present study was conducted with the aimof isolation and identification of the causative organisms in different types of Primary and Secondary Pyodermas and detecting their latest antibiotic susceptibility pattern.

Aims & Objectives

AIM AND OBJECTIVES

- To isolate and identify the aerobic bacterial pathogens from skin lesions of patients with Primary and Secondary Pyodermas.

- To study the antimicrobial susceptibility pattern of all the isolates.

- To determine the antimicrobial resistance pattern of the most commonly isolated organism by phenotypic methods.

- To detect the Mupirocin Resistance among MRSA isolates by phenotypic and genotypic method.

Review of Literature

REVIEW OF LITERATURE

Historical aspects

Existence of microorganism was suspected by the roman philosopher Lucretius (about 98-55BC). Microorganism had been mentioned as a possible cause of disease by the roman Scholar, Marcus Varo in the first century BC. The physician Girolamo Fracrastro (1478-1553) suggested that the disease was caused by invisible living creatures. Fracrastro described "De contagione, contagiosismorbis et curatione (On Contagion, Contagious Diseases, and their Treatment)" in 1546. [14] Antony Von Leuwenhoek first observed the microorganisms accurately and reported it as bacteria. [15]

Pus forming skin infections caused by coccal bacteria was first clearly defined by Tilbury Fox and Sabouraud in the late 19th century [16]. In 1864, the contagious impetigo of children and Infants were described by Tilbury Fox. Sir Alexander Ogston gave the name Staphylococcus to the cocci due to the typical occurrence of grapelike clusters in pus culture. Von Recklinghausen first observed *Staphylococci* in human pyogenic lesions in 1871. [15]

In 1874, cocci in chains were first observed by Billroth in erysipelas and wound infection, he named it as *Streptococci.* In 1881, Ogston isolated *Streptococci* from acute abscess and distinguishes it from *Staphylococci.*In1884, Rosenbach isolated cocci from human suppurative lesion and named it as *Streptococcus pyogenes*. [15]

In 1887,Bockhart described superficial pustular folliculitis which is characterized by small pustules at the follicular openings, hence named Bockhart impetigo. In 1889, Folliculitis decalvans was first described by Quainquad. [1]

EPIDEMIOLOGY

In 1960, Desai observed that more than 30% of Dermatology Outpatient department consists of infections like Pyoderma, superficial fungal infections and reported them as disease of poor economy. [19]

In India, pattern of skin disease was due to various factors like poverty, malnutrition, overcrowding, poor hygiene, literacy and social backwardness. [20]

Incidence of Pyoderma was more common in summer and monsoon. When the skin is exposed, abrasions or insect bites are more likely to occur, thus predisposing the susceptible children to these infections. [21]

The relative incidence of clinical forms of pyoderma shows striking variation with age. Pyoderma occur most frequently among the economically poor children residing in tropical and subtropical climates[28].Impetigo occurs more frequently in early childhood, although all ages may be affected[29,31].

Bullous impetigo occurs characteristically in newborn and neonates. It was wide spread and contagious, although it can occur at any age. [29,30]Ecthyma occurs in children or neglected elderly patients or in patients with Diabetes[31].

Superficial folliculitis/Bockhart's impetigo occurs more often in children, on Scalp[30, 32] and on extremities, beard, buttock, axillary areas in adults[31]. Erysipelas was common in infants, young adults and older adults[28]. Staphylococcal scalded skin syndrome occurs largely in newborns and in children

younger than 5 years of age. It occurs rarely in older children and adults. Rarely distant focus consists of cutaneous infections or a septicemia[33].

Chronic folliculitis of legs occurs in young Indian males, predominantly affecting age group of 15-30 years[34]. Deep folliculitis occurs in males between 20-40 years. Hidradenitis suppurativa usually begins after puberty[30].The peak incidence of pyoderma was high in children up to 10 years of age[12].Peak incidence has also been reported in 2nd and 3rd decade followed by1st and 4thDecade. [36]

Pyogenic dermatoses occur usually due to secondary infection with Staphylococci and Streptococci. There is a higher incidence of pyoderma in the low socioeconomic strata[21, 22, 23, 24] whereas factors like poverty, malnutrition, overcrowding and poor hygiene plays an important role[22'23].

Impetigo was more likely to occur among children living in overcrowded homes and in poor hygienic situation. [25, 26] Often a history of similar lesion was present in other family members. [23, 24]

Common predisposing factors of pyoderma include skin diseases, skin damage due to insect bites, minor trauma, surgical wounds, burn, retained foreign body, injections in diabetics or injection drug use and poor personal hygiene. In patients with poorly controlled diabetes, renal insufficiency, hematological malignancies, nutritional deficiencies, alcoholism and in those receiving Corticosteroid or cytotoxic therapy, resistance to infection with Staphylococci was reduced.[22, 23, 26]

Classification of pyoderma

Pyoderma is classified into Primary and Secondary pyoderma.

Primary pyoderma is a Pyogenic infection of the normal skin and its appendages and is caused by direct invasion of normal skin and tend to have more characteristic course and morphology.

Secondary pyoderma arises in the previously diseased skin as superimposed condition and does not follow a characteristic course as it leads to either acute or chronic intermingling state of underlying skin disease.[1]

TYPES OY PYODERMA

Primary pyoderma	Secondary pyoderma
Impetigo	Eczema with secondary infection
Folliculitis	Infected pemphigus
Furunculosis	Infected contact dermatitis
Carbuncle	Infected psoriasis
Ecthyma	Trophic ulcer
Cellulitis	Infected scabies
Paronychia	Infected wound
	Hidradenitis suppurativa

IMPETIGO

Impetigo is a contagious superficial pyogenic infection of the skin, limited to the epidermis, forming pustules and crusty sores. Impetigo and folliculitis in the elderly is caused by Staphylococcus in contrast to impetigo in paediatric, which is usually caused by Streptococcus. Two clinical patterns of impetigo are recognized: bullous impetigo and non -bullous impetigo.

Bullous impetigo:

Bullous impetigo occurs most common in newborn and in older infants .It was mainly caused by phage group II *S.aureus* particularly strains 77 and 55, although *Streptococcal* bullous impetigo have also been reported[38.]It is characterized by rapid progression of vesicles to flaccid bullae around orifices.

Non bullous impetigo:

It accounts for >70% of cases of impetigo and occurs in children of all ages as well as in adults. Previously it was believed that *GAS* is the most common etiological agent causing non-bullous impetigo. Recent studies showed that *S. aureus* being the most common organism isolated, followed by *GAS*. However it may be a mixed infection caused by both *S.aureus* and *GAS*[38]. Theinitial lesion is a transient vesicle or pustule that quickly evolves into a honey coloured crusted plaque that enlarges to greater than 2 cm diameter.

FOLLICULITIS

Folliculitis is an inflammatory change confined to ostium or extends onlyslightly below it and heals without any scar formation[37]. It usually presents as crop of pustulesaffecting areas of skin with moist hair. *S.aureus* is the most common cause but can also caused by other organisms like *Pseudomonas aeruginosa* when associated with specific exposure like hot tubs and spar.

FURUNCLE AND CARBUNCLE

A furuncle or boil is defined as an acute usually necrotic, infection of a hair follicle with*S. aureus*. The term carbuncle was derived from a Latin word mean for a small, fiery coal. It is defined as deep infection of a group of contiguous follicles with *S. aureus*, which causes inflammatory changes in the surrounding

and underlying connective tissues, including the subcutaneous fat. It occurs as an extremely painful lesion at the nape of the neck, the back, or thighs. [37]

ECTHYMA

Ecthyma is a characterized by the formation of adherent crusts beneath which ulceration occurs. It is usually occurs as a consequence of neglected impetigo and classically evolves in impetigo occluded by footwear and clothing. *S. aureus* and *GAS* are the common causes. [37]

CELLULITIS

Cellulitis is an acute inflammatory condition of the skin that extends deeper into the dermis and subcutaneous tissue, characterized by localized pain, erythema and swelling. *GAS* and *S.aureus* are the most common etiological agents isolated MRSA is rapidly replacing MSSA as acause of cellulitis in both inpatient and outpatient settings.

HIDRADENITIS SUPPURATIVA

It is a chronic inflammatory, recurrent debilitating follicular disease usually begins after puberty. It involves apocrine bearing skin with a predilection for intertriginous areas, most commonly involves the genitofemoral area or axilla. *S.aureus* and *CONS* are most frequently isolated pathogen. [37]

PEMPHIGUS

The term Pemphigus was first described by Sauvages in 1760. Wichmann in 1971 described chronic bullous disease as Pemphigus. Pemphigus is a chronic autoimmune bullous dermatoses, characterized histologically by intraepidermal blister formation. [39]

Pemphigus is divided into two major subtypes depending on location of blisters in the epidermis.

Superficial	Deep
Pemphigus foliaceus	Pemphigus vulgaris
Pemphigus erythematosus	Pemphigus vegetans

It is mainly a disease of middle age group. Some Indian studies have found that men affected more frequently than women. In many studies, mean age of occurrence is 30-40 yrs, it was in accordance with Indian literature.

Pemphigus vulgaris presents as flaccid mucocutaneous blisters and have a tendency to rupture easily. Pemphigus was usually fatal prior to the advent of steroids and antibiotic therapy. [39]

Infection is the most important complication in these patients attributes to disruption of epidermal lesions because of the disease itself and immunosuppression induced by treatment.

Many reports states that predisposition to infection is due to immunosuppressive therapy and immunocompromised state of the Pemphigus patients.If left untreated progression of the disease may lead to death due to secondary bacterial infection and sepsis. *Staphylococcus aureus* is the most common cause of bacterial infection. [40]

BULLOUS PEMPHIGOID

It is a sub epidermalimmunobullous disorder and predominantly a disease

of elderly with age of onset between 69 and 83. It is an autoimmune blistering disease presents with large, tense, cutaneous blisters. Rupture of the bullae results in erosions, which is susceptible to bacterial infection. [39]

ECZEMA

Eczema is derived from Greek word meaning "to boil" [37]. The term eczema and dermatitis generally regarded as synonyms[39]. Clinically eczema ischaracterized by itching, redness, edema, papulovesicule in acute stage, edema and scaling in subacute and dry lichenfied skin in the chronic stage[37]. Most case of eczema in infants and young children are atopic and nummular dermatitis occurs particularly in elderly males. [39]

Skin of atopic patients of eczema carries high levels of *S.aureus* which correlate with severity of eczema. *S.aureus* releases a toxin with super antigenic actions and initiates a vicious circle in atopic eczema[41]. In most of the patients with atopic eczema even though there is an absence of skin lesion; colonization of *S.aureus* will be noticed due to altered immunological profile of atopic patients. [39]

Innate immunity is compromised in atopic eczema due to reduction in keratinocyte derived antimicrobial peptides (cathelicidin, betadefensin 2 & 3) and neutrophil chemo attractant. [37,41]

PSORIASIS

Psoriasis is chronic inflammatory and proliferative condition of the skin, associated with systemic manifestation in many organ systems.

Fatima Zahra Elfatokeret al. (2016) states that Psoriasis is a chronic inflammatory skin disease which has been found to affect up to 5% of the world's

population. The exact etiology is unknown concepts of pathogenesis indicate the genetic, immunologic and environmental factors. [42]

Gudionsson.E.J. (2003) and Malbris.L.et al (2005) states that the chronic plaque form psoriasis is the most common type.

Association between psoriasis and tonsillitis was noticed 100 years ago, now it is well recognized that Psoriasis is triggered by Streptococcal infection. [37]

TROPHIC ULCER

The term "trophic" is derived from Greek word Trophe means nutrition. The American heritage medical dictionary 2007 states that trophic ulcer is "an ulcer due to impaired nutrition of the part"[43]. Recent studies states that no correlation between nutritional indices and development of trophic ulcer. [37]

Now considerable evidence is there to suggest that this disease is due to infection. For successful management of chronic ulcer it is necessary to identify the etiology as well as local and systemic factors contributing to its non-healing nature. [41]

Etiology

Pyodermas are usually caused by Gram positive bacteria, which constitute majority of cases and less commonly by Gram negative organisms[23].Among the Gram positive organisms, *S.aureus* is the most common organism isolated followed by *CoNS*, *BHS* and E*nterococcus* has also been isolated from few cases.

Various Gram negative organisms isolated include *Pseudomonas spp, Klebsiella spp, Proteus spp, E.coli, and Acinetobacter spp*[23].In most of the cases infection is caused by a single Pathogen, although mixed infections may also

occur. Most of the mixed Infections are caused by*S. aureus* and Gram negative organism. [23, 24, 25]

STAPHYLOCOCCUS

Staphylococcus is the most common pathogen isolated from both primary as well as secondary pyoderma. [38]

The genus *staphylococcus* is classified under the family *Staphylococcaceae*, order *Bacillales* in the phylum firmicutes according to volume3 of the revised Bergy's manual of systematic microbiology.*S. aureus* is the most common human pathogen among the *Staphylococci*, although CONS has also been reported as the etiological agent by a few workers. [3]

Staphylococcal skin lesions are characterized by the formation of pus containing lesions which often begin in hair follicles and spread to adjoining tissues. [25]

Mode of infection

May be exogenous from direct contact,airborne or cross infection in hospitals or endogenous from colonization.

S.aureus is found in the external environment and in the anterior nares of 25-35% of healthy adults .Other sites of Colonization are intertriginous skin folds,the perineum, the axilla and vagina.

It was suggested that 10-20% of general population are persistent carriers of S.aureus with up to 50% of intermittent carrier and 20-30% non-carrier[37].Prevalence of nasal carriage in healthy adults is 27%[37]. A correlation has been noted between nasal flora and organisms causing pyoderma, high nasal

carriage contributes to recurrent pyoderma[44].The rate of colonization was higher among HIV infected patients, insulin dependent diabetics, haemodialysis patients and those with damaged skin. [25]

Factors which predispose to serious *S.aureus* infection includes defect in leucocyte chemotaxis, defects in opsonisation, skin injuries, presence of foreign bodies and chronic underlying diseases. [3]

Virulence factors of S.aureus[1, 2]

Polysaccharide capsule	Inhibits Phagocytosis
Peptidoglycan	Confers rigidity and resistance to cell wall. Induces inflammatory response Activates complement,IL1,chemototactic to PMNs
Teichoic acids	Protects from complement mediated opsonization Species specific,mediates binding to fibronectin
Protein A	Affinity for Fc receptor of IgG and complement
Adhesins	Clumping factor B associated with S.aureus nasal colonization.
Hemolysin and Leukocidin/ Panton valentine toxin	Hemolytic and leukocidal activity
Exfoliative toxin	Scalded skin syndrome
Enterotoxins: A-E, H& I	Food poisoning{ 2-6 hours}
Toxic Shock Syndrome Toxin I (TSST-1);	Toxic shock syndrome

Enzymes:	
Coagulase	Converts fibrinogen to fibrin
Clumping factor	Able to bind fibrinogen
Catalase	Inactivate hydrogen peroxide and free radicals
Hyaluronidase	Breaks down connective tissue network
Staphylokinase/ fibrinolysin	Breaks down fibrin clot
Lipases	Breaks down lipids
Nucleases	Hydrolyze DNA
Penicillinase	Hydrolyze Penicillin

Pathogenicity:

The pathogenicity of Staphylococcal infection was attributed to the surface antigens present in the *Staphylococcus aureus* and contributing to its antiphagocytic property. These include exotoxins like alpha toxin and leukocidin which are injurious to human leukocytes. [80, 81]

The presence of an extra cellular enzyme, coagulase plays a major role in Pathogenesis. The mechanisms with which it operates are[82]

- It promotes clot formation and then disturbs the functioning of phagocytic cell.
- It is responsible for the deposition of fibrin over the surface of Staphylococci giving it antiphagocytic envelope.
- Necrosis and abscess formation result from the formation of local thrombi.

The other enzymes like hyaluronidase and lipase, contribute a minor role in the pathogenicity of Staphylococcal infection.

The mechanism of cutaneous infections were mainly due to

a) Direct infections.
b) Toxin mediated disease.
c) Immunologically mediated disease.

Immunological response against *Staphylococci* :

Haemolysin and antileukocidin are the major antibodies against staphylococcal antigens. The passive transfer of these antibodies transplacentally provide protection against Staphylococcal infection during the first trimester of intrauterine life[80].

ANTIBIOTIC RESISTANCE AMONG *STAPHYLOCOCCUS AUREUS:*

In the early 1940's, Penicillin was introduced as drug of choice for treatment of serious *Staphylococcus aureus* infection. Resistant to penicillin was emerged in 1959 and it was due to acquisition of plasmid borne gene element blaZ encoding β -lactamase.

Penicillinase resistant penicillins (oxacillin, methicillin, nafcillin) were developed and introduced in1959 for clinical use. In 1960s ,with the emergence of methicillin-resistant strains of S. aureus (MRSA) in UK, the drug was rendered clinically ineffective. Resistant to methicillin was due to presence of PBP2a results from acquisition of chromosomal element known as SCC mec. *S.aureus* strains that contain SCC mec are termed MRSA and that lack this element MSSA.It was believed that *S.aureus* acquired the SCC mec transposon from a coagulase negative staphylococcal species *S.sciuri* which found on animals and in the environmemt.[2]

Methicillin resistant *Staphylococcusaureus* and *Coagulase-negative Staphylococci (CoNS),* were considered resistant to other β-lactam agents, ie, penicillin's, β-lactam/β-lactamase inhibitor combinations, cephems (with the exception of the cephalosporins with anti-MRSA activity), and carbapenems**.**

The Panton -Valentine leukocidin is an important virulence factor in MRSA. [58] MRSA can cause infection ranging from simple furuncles to life threatening necrotizing fasciitis and pyomyositis. [58] In 1968, the first case of MRSA was reported in the United States . MRSA have remarkably developed resistance against variety of antibiotics including penicillins, cephalosporins, aminoglycosides, macrolides and quinolones[57].MRSA was recognized initially in the health care setup, later MRSA spread to the community in 1980s. [55]

Thereafter community acquired MRSA has been increasingly reported in skin and soft tissue infections in India and globally[56]. In India, MRSA prevalence overall increased from 12% in 1992 to 80.83% in 1997. [54]

Types of MRSA

Healthcare-associated MRSA. Healthcare-associated MRSA (HA-MRSA) were isolated from the patients admitted to healthcare facilities such as nursing homes and long-term care facilities. HA-MRSA causes Healthcare-associated infections like bloodstream infections, urinary tract infections, respiratory tract infections, surgical-wounds and device-associated infections. [45,46,47]

Risk factors for acquiring HA-MRSA include previous admission to healthcare facilities, impaired immune system, use of multiple antibiotics, use of invasive medical devices and old age. [49] Genetically, the HA-MRSA carried SCC mec types I, II and III, which is usually multidrug resistant and tend to multiply slowly in culture. [49]

Community-associated MRSA

In late 1980s, Community-associated MRSA (CA-MRA) strains were initially reported among individuals with no previous history of hospitalization living in remote communities in Western Australia. [51]The initial report was followed by similar reports from USA, New Zealand, and later in Europe. Initially, CA-MRSA were mostly associated with skin and soft tissue infections such as impetigo, cellulitis, folliculitis and boils and the young patients are at risk. [48]

CA-MRSA are usually susceptible to non-beta lactam antibiotics which carry smaller-sized SCC mec types IV, V and VI. CA-MRSA strains often express lower levels of resistance to oxacillin (MIC; 8–32 mg/L) and also multiply faster than HA-MRSA strains with significant shorter doubling time, which may help CA-MRSA to achieve successful colonization by enabling it to compete out the normal bacterial flora. [48]

Livestock-associated MRSA

Staphylococcus aureus is also an important cause of infections in live stock resulting in economic losses in the food industry. Livestock-associated MRSA (LA-MRSA) strains were initially identified because they were non-typeable by pulsed-field gel electrophoresis following digestion with Sma I restriction enzyme. Further molecular typing revealed that LA-MRSA defined to a new lineage of MRSA that belonged to clonal complex 398 (CC398). [49]

Although LA-MRSA ST398 was initially reported among livestock, [49,50]it has also appeared in the community among human patients in contact with infected or colonized animals which was considered as a major risk factor for LA-MRSA colonization. [50]

Other LA-MRSA lineages reported in humans include ST9, ST97 and ST433[52]. LA-MRSA has also caused invasive infections including endocarditis, osteomyelitis, and ventilator-associated pneumonia in humans. [52,53]

Detection and identification of MRSA

MRSA can be detected by both phenotypic and genotypic methods. The ideal method of detection of MRSA is by detection of mecA gene or its product PBP2a.Because of high cost and need expertise it was not performed in most of the clinical laboratories.

Kacou- N doube et al (2011) states that pcr is the gold standard method to detect mec A gene in MRSA.

Methods of detection of MRSA

Screening methods

With cefoxitin/oxacillin disc by disc diffusion method.

Confirmatory methods

Oxacillin MIC detection (by broth dilution,agardilution,E-test method), oxacillin screen agar.

Molecular methods

Detection of mecAgene or PBP2a protein (its protein products)

Treatment options for MRSA infections

The emergence of methicillin resistance was accompanied by the development of resistance to most of the non-beta-lactam antibiotics and resulted in the reduction in options for treating infections caused by MRSA. [45]

In the 1980s, some MRSA strains were resistant to all available antibiotics except vancomycin. [59,60]The situation was compounded by the emergence of strains that expressed reduced susceptibility to vancomycin in the late 1990s, followed by vancomycin-resistant *S.aureus* strains (MIC: 32 mg/L) in the USA and other countries. [60]

The vancomycin-resistant *S. aureus* had apparently acquired vanA gene complex from vancomycin-resistant enterococci whereas vancomycin intermediate-resistant *S. aureus* strains have reduced susceptibility to vancomycin due to their thickened cell wall which is capable of binding vancomycin and reduce their diffusion into the cell. [61]

Newer antibioticslike daptomycin, linezolid, tedizolid, telavancin, oritavancin, dalbavancin, ceftaroline and ceftobiprole have been developed against MRSA strains. [45]

Control of MRSA infections

Globally, there is increase in number of multidrug resistant pathogens in healthcare facilities as well as in the community. The constant threat that resistance to even the newly developed antimicrobial agents may develop makes it necessary that other methods for limiting the spread of multidrug resistant organisms, to be developed and implemented. Some of these approaches include active surveillance of resistant pathogens, antibiotic stewardship, and for better implementation of infection control methods. [45]

The burden of multidrug resistant pathogens in healthcare settings will be reduced by Preventing infections. [62]

The main infection control interventions used against MRSA include screening, hand hygiene, contact isolation, cohosting and decolonization in addition to standard precautions. These procedures should be continued till patients become culture-negative for the target multidrug resistant pathogen. [45]

Topical agents to be used to reduce surface colonization of MRSA include the following:

- Hand wash with 70% alcohol
- Chlorhexidine gluconate, 4% (more active againstMRSA than MSSA)
- Triclosan (soap)
- Povidone iodine (equally active against MRSA and MSSA)[86]

Mupirocin

Mupirocin (pseudomonas acid A), a polyketide antibiotic was naturally produced by *Pseudomonas fluorescens* strain NCIMB 10586 [67,77]. It has antibacterial activity against a wide range of gram-positive and gram-negativebacteria, however many strains of Pseudomonas are resistant. Mupirocin is rapidly metabolized by the skin to an inactive, nontoxic substance, viz, monic acid, and thus appears to be ideal for use in patients with extensive areas of denuded skin. It is used topically for the treatment of skin infections, prevention of surgical site infections, and eradication of *Staphylococcus aureus* carrier state.[79]

Mupirocin was introduced into clinical practice in 1985, with mupirocin resistantS. aureus (MupRSA) was first reported in 1987. Resistance was classified into two categories:

Low-level resistance, with MICs ranges from 8 - 256 µg/ml, and high-level resistance, withMICs of >512 µg/ml. Isolates with MIC ≤4 µg/ml were considered mupirocin susceptible. In most cases, High-level resistance was conferred by acquisition of the plasmid-borne gene mupA, which is a phylogenetically distinct isoleucyltRNA synthetase gene, with no affinity for mupirocin. mupB, a related gene, has also been shown to confer high-level resistance.[76]

Low-level mupirocin resistance was caused by point mutations in the native isoleucyl- tRNA synthetase gene (ileS). It may be associated with higher rates of recolonization after measures to eradicate *S. aureus* carrier state. [76]

Many Studies on mupirocin resistance among *S. aureus* indicates that nearly all *S. aureus* isolates with high-level mupirocin resistance were mupA positive by polymerase chain reaction(PCR) .[69,70]

Isolates with low-level mupirocin resistance but positive for the mupA gene have been identified. In these isolates, the mupA gene was located on the chromosome and not on a plasmid[71]. Also, isolates that are mupirocin susceptible but mupA positive by PCR have been reported.

It was attributed due to a frameshift mutation in the mupA gene that inactivates the gene product[72].

Isolates with the frameshift mutation revert to wild-type sequence and develop high-level mupirocin resistance at a high frequency.

Few isolates have been identified that demonstrate high-level mupirocin resistance but are mupA negative by PCR despite the use of multiple primer sets. These isolates may carry a novel mechanism of mupirocin resistance.[75] The mupA

gene is typically located on mobile genetic elements, which likely facilitates the dissemination of this resistance mechanism. The mupA gene is typically plasmid mediated, and some of these plasmids are conjugative[73,74]

Insertion sequences have been identified flanking the mupAgene in plasmids, which might facilitate movement of the mupA gene between plasmids by recombination [74].

Retapamulin, a pleuromutilin antibacterial agent is a topical antibiotic effective against mupirocin resistant strains. Currently it was used against a variety of Gram positive pathogens associated with secondarily-infected dermatoses and secondarily-infected traumatic lesions. The pleuromutilins were potent inhibitors of protein synthesis in bacteria through the intervention of peptide bond formation by binding to the peptidyl transferase centre of the 50S ribosomal subunit. Retapamulin shows no target specific cross-resistance to other classes of antibiotics due to its unique mode of action .[102]

Detection of mupirocin resistance

Multiple laboratory testing methods have been described for determining the MIC of mupirocin, including agar dilution, broth microdilution and E-test. The Clinical and Laboratory Standards Institute (CLSI) recommends using broth microdilution or disc diffusion for screening of high-level mupirocin-resistant S. aureus, and it only differentiates between high-level resistance and the absence of high-level resistance. [65]

CLSI and the European Committee for Antimicrobial Susceptibility Testing (EUCAST) both recommends the use of 200 mcg discs for the detection of high-level mupirocin resistance by disc diffusion, whereas the British Society

for Antimicrobial Chemotherapy (BSAC) now recommended using 20 mg discs. EUCAST clinical thresholds for S. aureus are 1 mg/L for susceptible and >256 mg/L for resistant, placing the susceptible threshold at the epidemiological cut-off value (ECOFF). Isolates with MICs above the wild type (ECOFF 1 mg/L) but without a recognized resistance mechanism (MIC -4 mg/L) will thus be reported intermediate. Tilldate, no clinical data was found on the clinical relevance of S. aureus strains with these MIC levels (>1 and 4 mg/L) .[65]

MIC susceptibility thresholds of BSAC coincides with EUCAST thresholds, but disc diffusion cut-offs differ because of the 20 mg mupirocin discs used, rather than the 200 mcg discs recommended by EUCAST.

Genotypic techniques, i.e. mupA PCR, for identifying high level resistant isolates should be interpreted with utmost care, because genotypic and phenotypic results may vary. The mup-A positive isolates may be susceptible to mupirocin and high-level resistant may be mupA negative[66].

Organization	**Method**	**Thresholds and interpretation**		
		Susceptible	**Intermediate**	**Resistant**
CLSI	Disc diffusion Tablet: 200 mcg Broth microdilution Single well: 256 mg	Any zone - no high-level resistance No growth - no high-level resistance		No zone - high-level Resistance Growth - high-level Resistance
EUCAST	Disc diffusion Tablet: 200 µg MIC	30mm ≤1 mg/L	18-29mm 2-256 mg/L	<18mm >256 mg/L
BSAC	Disc diffusion Tablet: 20 mg MIC	≥27mm ≤1 mg/L	7-26mm 2-256 mg/L	≤6mm >256 mg/L

CoNS

Coagulase-negative staphylococci (CoNS) was considered to be one of the harmless skin commensal before 1970s; however, currently it was recognized as an importantcause of human infections. It has been recognized as major nosocomial pathogens in the context of prosthetic and indwelling device-related infections. CoNS are also isolated most frequently in clinical microbiology laboratories [63]. More importantly, CoNS often serve as reservoirs of antimicrobial resistance determinants since they usually have a high prevalence of multidrug resistance. Therefore, it is necessary to characterize and distinguish *S. aureus* strains and CoNS[64].

STREPTOCOCCUS

BHS was the second most common etiological agent to be isolated after Staphylococci from cases of pyoderma. *S. pyogenes* is the only species under Lancefield group A *Streptococcus* and is the commonest cause of Streptococcal pyoderma. It causes a variety of suppurative infections and it can also trigger post infectious non-suppurative complications such as acute rheumatic fever and acute glomerulonephritis.

Skin damage, although minor is necessary for the development of Streptococcal pyoderma.The organisms first colonize and multiply in the normal skin before invasion through minor breaks in the epithelium and the development of lesions.

GAS

GAS skin infections are often attributable to M types 49, 52, 57, and 59-61. They usually spread by transfer of organisms from an infected person or carrier

through close personal contact. Finger nails and the perianal region harbor GAS and play an important role in disseminating impetigo. However, in epidemics, fomites play animportant role in transmitting the disease.

Although serotypes causing impetigo may colonize the throat, spread is from skin to Skin and not via the respiratory tract.Streptococci of the same strain are recovered from the respiratory tract of approximately 30% of children with skin lesions but there is no clinical evidence of Streptococcal pharyngitis, and colonization occurs after the skin has become infected. The sequence of spread in a given patient was from normal skin to lesions and eventually to the respiratory tract.

The pathogenesis of S. pyogenes infection differs from *S. aureus* in many ways. *Streptococcus pyogenes* will not survive for prolonged period of time on intact normal skin.[78] The resident flora does not appear to be an important first line of defense, since disinfecting the skin surface does not increase the survival of *S. pyogenes* on intact skin.

Colonization of skin and subsequent infections develop quickly if the stratum corneum barrier was disrupted. [79]

Inoculation of S. pyogenes onto superficially scarificated skin results in an infection, but when the same inoculums was applied to intact skin, it dies off quickly. [78] It appears that serum provides enough nutrients for the growth of *S.pyogenes* and subsequent infection. Because *S. pyogenes* can penetrate the dermis and make its way into lymphatics and dermal vessels, systemic signs such as fever and regional lymphadenopathy will frequently develop early in the course of infection. [79]

Infections due to *S. pyogenes* most commonly occurs in the lower extremitiesand more common in children than in adults. This incidence probably reflects more frequenttrauma and minor cuts and abrasions, coupled with close contact from playing. Inpatients withEB, denuded skin and the abundance of serum facilitate infection due to *S. pyogenes*.[7]

Virulence factors	Biological functions
Cell wall associated polymers and proteins Capsule	Prevents phagocytosis
Teichoic acid	Binds to epithelial cells
M protein	Adhesin and antiphagocytic; inactivates C3b—an important complement factor responsible for phagocytosis. Strains that are rich in M protein are resistant to phagocytosis and intracellular killing by PMNs. Interferes with opsonization via the alternative complement pathway
F protein	Mediates attachment to epithelial cells
Enzymes	
Streptokinase	Breaks down the fibrin barrier around the infected site, thereby facilitating spread of the infection
Deoxyribonucleases Depolymerizes free DNA present in the pus	Depolymerizes free DNA present in the pus
Hyaluronidase	Hydrolyzes hyaluronic acids in the matrix of the connective tissues

Toxins	
Streptococcal pyrogenic exotoxins (SPEs)	Dissolves the clot, thrombi, and emboli; thereby facilitates spread of the bacteria in tissues
Streptolysin O and Streptolysin S	Lyse erythrocytes, leukocytes, and platelets; and stimulate production of lysosomal enzymes

ENTEROCOCCUS

Enterococcus faecalis is a rising cause for most of the nosocomial infections especially secondary skin and soft tissue infections (AgudeloHiguita and Huycke, 2014).

According to previous study, incidence of Enterococcal skin infections was 8.6 per 1000 admissions in New Delhi and among them *E. faecalis* was 3.4 per 1000 admissions and E. faecium was 4.8 per 1,000 admissions (Rajkumari et al., 2014). The increased incidence leads to increased antimicrobial resistance also. Enterococci has an intrinsic resistance to several antibiotics (Patel et al., 2013). Hence Vancomycin which is often given for severe infections also leads to rising vancomycin resistance (Brandl et al., 2008; Humphreys, 2014).

VRE

Vancomycin resistance among clinical Enterococcus strains developed in the past decade. Failure to adhere to strict infection control practices to prevent the spread of these pathogens, was responsible for the development of the resistance. It was suggested that the use of Extended-spectrum Cephalosporins

and drugs with potent activity against anaerobic bacteria plays a role in the spread of VRE, as they promote infection and colonization with these organisms.[91]

Treatment of Enterococcal infections usually requires a bactericidal combination of antibiotics which includes a cell wall-inhibitory agent to which the Enterococcus is susceptible and an aminoglycoside to which the Enterococcus does not exhibit high-level resistance. The combination commonly used includes PenicillinG/Ampicillin along with Gentamicin. Vancomycin was the recommended drug of Choice, only in cases of significant Penicillin allergy or in treatment of Ampicillin and Penicillin resistant strains. [92].Linezolid and Quinupristin/ Dalfopristin are approved therapeutic options for VRE on the basis of in vitro susceptibility and clinical efficacy from multicentre, pharmaceutical company sponsored clinical trials. [93]

MANAGEMENT OF PYODERMA

DIAGNOSIS

Diagnosis was usually based on clinical appearance and location of lesion. Patients history of travel, bite history, underlying disease status, and lifestyle also helpsin the diagnosis. Confirmation of diagnosis was obtained by laboratory investigations. [7]

TREATMENT

Primary pyodermas of mild to moderate severity can be treated with local measures, topical anti-infective therapy, oral antibiotics, or by a combination of these methods.[7,84]Topical therapy is generally the preferred mode of antibiotic administration in the management for reasons of convenience and ease of application.

Systemic therapy may be necessary only if the lesions are generalized, regional lymph nodes are involved, fever and / or if the lesions are deep asin the case of erysipelas, deep folliculitis, cellulitis and carbuncle. [84]

Extensive infections have to be vigorously treated with parenteral antibiotics in adequate dosage. Parenteral treatment is always recommended in the immunocompromised host.

TOPICAL THERAPY

Sisomicin and mupirocin[79] are used in the topical management of pyodermas.

Sisomicin cream 0.1% twice a day, or mupirocin ointment 2% thrice a day is the treatment of choice. Both are effective and safe, however sisomicin therapy results in faster and greater relief of signs and symptoms. [84]

Efficacy of sisomicin has been documented in the management of superficial infections of skin and skin structures. In vitro efficacy of sisomicin against a wide range of Gram positive and Gram negative clinical isolates, including Gentamicin resistant *Ps. aeruginosa* has been well established. [79,84]

Topical Sodium Fusidate cream also has shown excellent results in the group of impetigo, Bockhart impetigo, and furunculosis, better than topical Gentamicin and equal to systemic Erythromycin. . [85]

SYSTEMIC THERAPY

There is no hard and fast rule for use of systemic antibiotic, but it is based on few principles.

Systemic antibiotics are needed, if infection is wide spread or severe or accompanied by lymphadenopathy or if there is a reason to suscept a nephritogenic streptococcus or if other children are exposed to infection of if there is doubt whether topical medication is carried out properly, or if associated with systemic signs. (that is fever, malaise)[32]. Multiple lesions on the face and body are treated more aggressively with an oral antibiotic. [88]

For most uncomplicated skin and skin structure infection, empheric antibiotic therapy is directed against most likely pathogen, *Staphylococcus aureus* and *Streptococcus pyogenes*. Because of their broad-spectrum coverage, clinical efficacy, favorable tolerability, and safety profiles, oral β-lactam antibiotics (penicillin, cephalosporins) are one of the mostwidely used class of antibiotics for uncomplicated skin infection. Due to increasing *S. aureus* infections, penicillinase resistant penicillin's and β-lactam / β-lactamase inhibitors (e.g. Dicloxacillin and amoxicillin / clavulanate) are also appropriate options[89]. Cefdinir a third-generation cephalosporin is safe and effective for treating skin infection with *S. aureus, Streptococcus pyogenes* and gram-negative pathogen. In addition to traditional antibiotics like β-lactams macrolides and clindamycin, newer broad-spectrum antibiotics to treat resistant pathogens are available like streptogramins, oxazolidinediones and third generation fluroquinolones like moxifloxacin and gatifloxacin. [88]

Control measures

Pyoderma is best prevented by attention to adequate personal hygiene. [25] Isolation of patients with open draining Staphylococcal infections, strict hand washing procedures, good nursery techniques, and careful handling of patients are

important in the reduction of transmission of infection and thus serve as good control measures.[7]

A correct antimicrobial policy and the avoidance of inappropriate antimicrobial usage are mandatory to reduce the spread of MRSA in the community.[50]

Morbidity

Inspite of the fact that Pyoderma was easily treatable, it was known for its chronicity, recurrence and various other complications. [7]

S.aureus causing pyoderma, if left untreated can invade the blood stream,producing bacteremia, metastatic infections such as osteomyelitis, septic arthritis, brain abscess, pneumonitis and an acute infective endocarditis. [7] Staphylococci from boils and carbuncles in food handlers can be transmitted to food and can cause food poisoning. [90]

Scarlet fever, urticaria, and erythema multiforme may follow Streptococcal impetigo. Invasive complications of Streptococcal pyoderma include lymphangitis, lymphadenitis and bacteremia. The most important sequelae of Streptococcal skin infections were post streptococcal glomerulonephritis. The pyoderma associated nephritogenic strains belongs to the serotypes 2, 49, 42, 55, 56, 57 and 60. The frequency of AGN after infection with a known nephritogenic strain was 10% to 15%.[7]

Materials and Methods

MATERIALS AND METHODS

STUDY DESIGN:

Place of study:

The Study was conducted at theInstitute of Microbiology, Madras Medical College in association with the Department of Dermatology, Rajiv Gandhi Government General Hospital, Chennai**.**

Study period:

The study was conducted over a period of One year from March 2017 to February 2018.

Study type:

A hospital based prospective Cross-sectional study

Sample size:

The sample size of my study was 200 cases of pyoderma

Study population:

All out patients & in patients of Pyoderma attending the Departmentof Dermatology, Rajiv Gandhi General hospital were included in this study.

Ethical consideration

Ethical clearance was obtained from Institutional Ethics committee and Informed consent was obtained from all the patients who participated in this study.

Statistical analysis

Statistical analyses were carried out using Statistical Packages for Social Sciences (SPSS) software (Version 16.0; SPSS Inc., Chicago). The proportional data of this cross sectional study were analyzed using Pearson's Chi Square analysis test.

Inclusion Criteria:

- Patient aged more than 18 years.
- Inpatients/Outpatients with Primary and Secondary Pyoderma attended Dermatology Department of RGGGH, Chennai**.**
- Patients with Pustule, Papulovesicles, Bullae, Ulcer, Inflammatory plaques with oozing, crusting signifying bacterial skin infection.

Exclusion Criteria:

- Those who are on antibiotic therapy & denied consent will be excluded.

Sample collection:

Sample was taken before the start of treatment, samples(pus) for culture was collected from the base of the lesion using sterile swab.

The surrounding area of the lesion was cleaned with 70% alcohol followed by 10% povidone iodine solution before collecting the samples.Prior to the pus collection,iodine was removed from the area with alcohol

The intact pustule was ruptured with sterile needle and then the sample was taken with sterile cotton swab stick.

The debris was removed as far as possible in open wounds and then the lesion was rinsed thoroughly with sterile saline prior to sample collection.

In crusted lesions, the crusts were partly lifted and then the sample was taken from underneath.

All the samples were collected aseptically with two sterile cotton swabs for each sample from the lesion and kept in a sterile tube after proper labeling with name, age, gender, OP/IP number and date. Then the swabs were immediately transported to the microbiology laboratory without any delay.

Sample processing

In the laboratory the samples were processed immediately. Gram staining was done using one swab and aerobic culture using the other swab.

Gram staining

One swab is used for gram staining. A sterile glass slide was cleaned and exposed to Bunsen flame and allowed to cool and then a direct smear was made over the slide. The smear was allowed to air dry and then it was heat fixed by passing the under surface of the slide over the flame.

The fixed smear was covered with methyl violet stain for 60 seconds. Rapidly wash off the stain with clean water. After tip off all the water, cover the smear with Grams iodine for 60 seconds. Wash off the iodine with clean waterand decolorize rapidly (few seconds) with acetone. Wash immediately with clean

waterand Cover the smear with carbolfuchsin stain for 60 seconds. Wash off the stain with clean water, Wipe the back of the slide clean, and place it in a draining rack for the smear to air-dry. The smear was examined microscopically, first with the 40objectives to check the staining and to see the distribution of material, and then with the oil immersion objective to report the bacteria and cells.

CULTURE

The Second swab was inoculated onto the following media:

- Nutrient agar
- Blood agar
- MacConkey agar

The inoculated media were incubated aerobically at 37°C for 24 hours. In case ofno growth after 24 hours, the plates were further incubated for another 24 hours.

3) Identification of the isolates

Organisms grown were then identified on the basis of their colony characteristics and biochemical reactions as per the standard protocol.

BIOCHEMICAL TESTS

1. Catalase test
2. Coagulase test
3. Oxidase test
4. Bacitracin susceptibility test

5. Bile esculin agar
6. Growth at 10 &40^{o}C for enterococci
7. Hanging drop
8. Nitrate reduction test
9. Hugh and Leifson's oxidation fermentation test
10. Indole test
11. Methyl red and Voges-Proskauer test
12. Citrate utilization test
13. Urease test
14. Triple sugar iron agar test
15. Sugar fermentation test
16. Moeller's decarboxylase test
17. PYR test.

Catalase test

Principle

This test is used to differentiate the bacteria that produce the enzyme catalase, which acts as a catalyst in the breakdown of hydrogen peroxide to oxygen and water.

Procedure

With an inoculating needle or a wooden applicator stick, growth from the center of a Colony was transferred to the surface of a glass slide.One drop of 3% hydrogen peroxide is added and observed for bubble formation.

Interpretation

The rapid and sustained appearance of bubbles or effervescence constitutes a positive test. Because some bacteria possess enzymes other than catalase that can decompose hydrogen peroxide, a few tiny bubbles forming after 20–30 seconds is not considered a positive test. In addition, catalase is present in red blood cells; so care must be taken to avoid carryover of red blood cells.

Coagulase test

The coagulase test is used to identify *Staphylococcus aureus* and differentiate it from most other species of Staphylococci. Coagulase is present in two forms, bound and free, each having different properties that require the use of separate testing procedures.

Procedure

Slide test (bound coagulase):

Two drops of saline were placed in two circles drawn on a glass slide with a wax pencil. The colony material from the organism to be identified is gently emulsified in saline in each of the circles. A drop of plasma is placed in the suspension in one of the circles and mixed with a wooden applicator stick. Another drop of saline was placed in the other circle as a control, the slide was rocked back and forth and observed for agglutination of the test suspension.

Tube test (free coagulase)

A small amount of the colony growth of the organism was emulsified in a tube containing 0.5mL of plasma. The tube is incubated at 35°C for 4 hours and

observed for clot formation by gently tilting the tube. If no clot is observed at that time, the tube is reincubated at room temperature and read again after 18 hours.

Interpretation

Slide test:

A positive reaction was detected within 10–15 seconds of mixing the plasma with the suspension by the formation of a white precipitate and agglutination of the organisms in the suspension. The test was considered negative if no agglutination was observed after 2 minutes.

Tube test:

The tube coagulase test was considered positive if any degree of clotting is noted. The tube should be gently tilted and not agitated, because this may disrupt partially formed clotted material. Fibrinolysins produced by the organism may also dissolve the clot soon after formation. Tube tests that are negative after 4 hours should be incubated at room temperature overnight and read after 18 hours.

OXIDASE TEST

Principle:

The cytochrome oxidase test uses certain reagent dyes, such as p-phenylenediamine dihydrochloride, that substitute for oxygen as artificial electron acceptors. In the reduced state, the dye is colorless; however,in the presence of cytochrome oxidase and atmospheric oxygen, p-phenylenediamine is oxidized,forming indophenol blue.

Procedure:

Filter paper soaked with oxidase reagent (1% tetramethyl-P-Phenylene diamine dihydrochloride) was placed in a Petri dish and the colony to be tested was smeared on it using a sterile glass rod.

Interpretation

Bacterial colonies having cytochrome oxidase activity develop a deep blue color at the inoculation site within 10 seconds. Any organism producing a blue color in 10- to 60-secondperiod was considered negative, and it can be concluded that it does not belong to the familyEnterobacteriaceae.

NITRATE REDUCTION TEST

Principle:

This test demonstrates the presence of nitrate reductase enzyme which reduces nitrate to nitrite.

Reagent:

Reagent A

α-Naphthylamine	5 g
Acetic acid (5 N), 30%	1 L

Reagent B

Sulfanilic acid	8 g
Acetic acid (5 N), 30%	1 L

Procedure

With a loopful of the test organism isolated wasinoculated in the nitrate medium and incubated at 35°C for 18–24 hours. At the end of incubation, 1 mL each of reagents A and B was added to the test medium, in that order

Interpretation:

The development of a red color within 30 seconds after adding the test reagents indicates the presence of nitrites and represents a positive reaction for nitrate reduction.

INDOLE PRODUCTION TEST

Principle:

This test demonstrates the presence of enzyme tryptophanase, that degrades tryptophan to indole.

Procedure:

Kovacs reagent ingredients

P-Dimethylaminobenzaldehyde-10g

Isoamyl alcohol – 150ml

Concentrated hydrochloric acid-50ml

The test organism was inoculated into peptone water and incubated for 24 hours and then 15 drops of reagent was added along the inner wall of the tube.

Interpretation:

The development of a bright fuchsia red color at the interface of the reagent and the broth within seconds after adding the reagent was indicative of the presence of indole and interpreted as a positive test.

UREASE TEST

Principle:

This test demonstrates the presence of urease enzyme which splits urea to ammonia and CO2.

Procedure:

Test organism was inoculated on to slope of Christensen's urease medium and incubated at 37°C for 24 to 48 hours.

Interpretation:

Organisms that hydrolyze urea rapidly shows positive reactions within 1 or 2 hours; less active species may require 3 or more days. Development of pink color in the slope was interpreted as a positive test.

CITRATE TEST

Principle:

This test demonstrates the ability of an organism to use citrate as sole source of carbon.

Procedure:

The test organism was inoculated on to slope of Simmon's citrate medium and incubated at 37°C for 24 to 48 hour.

Interpretation:

Development of deep blue colour in the medium was interpreted as a positive test.If there is visible colony growth along the inoculation streak line it was considered as a positive test without development of blue color.

METHYL RED TEST

Principle:

This test detects the production of acid during the fermentation of glucose and maintenance of pH below 4.5.

Procedure:

The test organism was inoculated in to glucose phosphate broth and incubated at 37 °C for 48 to 72 hours. Few drops of 0.04% solution of methyl red was added to the broth.

Interpretation:

Development of red colour in the surface of the medium was interpreted as a positive test.

VOGES-PROSKAUER TEST

Principle:

This test detects the production of acetyl methyl carbinol from pyruvic acid as an intermediate stage in its conversion to 2:3 butylene glycol. In presence of alkali and atmospheric oxygen, small amount of acetyl methyl carbinol is oxidized to diacetyl which reacts with peptone in the broth.

Procedure:

The test organism was inoculated into glucose phosphate broth and incubated at 37°C for 48 to 72 hours. Aliquot 1 mL of broth to a clean test tube and add 0.6 mL of 5% α-naphthol followed by 0.2 ml of 40% KOH.

Interpretation:

The development of a red color 15 minutes or more after addition of the reagents was considered as a positive test.

TRIPLE SUGAR IRON AGAR TEST

It is a composite solid agar medium in tube having a butt and a slant. Its constituents include:

- Three sugars-glucose, sucrose and lactose in the ratio of 1:10:10 parts.
- Phenol red as an indicator of acid production.
- Ferric salt as an indicator of hydrogen sulfide (H2S) production.

Procedure

Medium was inoculated with a pure bacterial culture by a straight wire pierced deep in the butt (stab culture) and then doing a stroke culture on the slant area. the tube is incubated at37°C for 18- 24 hours. Under incubation or over incubation may lead to false interpretation of result.

Interpretation:

Alkaline slant / No change in butt (K / No change)	non fermenter
Alkaline slant / Acid butt (K/A)	glucose only fermenter
Acid slant / Acid butt (A/A)	glucose, sucrose and / lactose fermenter

A black precipitate in the butt indicates production of ferrous sulphide and H2S gas. Bubbles or cracks in the tube indicate the production of CO2 or H2.

SUGAR FERMENTATION TEST

It detects the ability of an organism to ferment a specific carbohydrate (sugar) incorporated in a medium producing acid with/without gas.

Procedure:

Sugar fermentation medium

Peptone	-	15g
Phenol red	-	10ml
Sugar	-	20g
Water	-	10ml

Each tube was inoculated with 1 drop of 18-24 hours broth culture and incubated at 35°-37°C for up to 7 days in ambient air. The tubes are examined for acid and gas production.

Interpretation:

Positive test is indicated by growth and change of colour to yellow. Gas production is indicated by the presence of bubbles in the inverted Durham tube.

OXIDATION –FERMENTATION TEST (Hugh-Leifson's method)

This test is used to differentiate microorganisms based on their ability to oxidize or ferment specific carbohydrates. Hugh-Leifson's basal medium is prepared and the carbohydrate to be added is sterilized separately and added to give a final concentration of 1 %.

Procedure

Two tubes are required for the OF test, each tube inoculated with the unknown organism, using a straight needle, stabbing the medium three to four times halfway to the bottom of the tube. One tube of each pair was covered with a 1-cm layer of sterile mineral oil or melted paraffin, leaving the other tube in air. Both tubes are incubated in ambient air for up to 7 days.

Interpretation:

Fermenting organisms produce an acid reaction throughout the medium in covered (anaerobic) as well as the open (aerobic) tube. Oxidizing organisms produce an acid reaction only in the open tube.

Organisms that cannot breakdown the carbohydrate aerobically or anaerobically produce an alkaline reaction in the open tube and no change in covered tube.

Moeller's Decarboxylase Tests

This test was used to differentiate decarboxylase producing Enterobacteriaceae from other gram negative rods.

Procedure

The tubes were inoculated with 1 drop of an 18- to 24-hour brain-heart infusion broth cultureand 4-mm layer of sterile mineral oil was added to each tube. Incubated at35°-37°C in ambient air.

Result :

Positive: Alkaline (purple) color change compared with the control tube

PYR Test

Principle

This test was now a standard assay for the presumptive identification of both group A β-hemolytic Streptococci and Enterococci.

Procedure

Two to three morphologically similar colonies are picked up with a sterile bacteriologic loop and emulsified in the small volume of PYR broth. The tube was incubated at 35-37°C for 4 hours. One drop of the PYR reagent was added and observed for color change. (The reaction should be read and recorded 1 minute after the addition of reagent.)

Interpretation

The development of a deep cherry red color within a minute of addition of the reagent is interpreted as Positive.A yellow or orange color was interpreted as Negative.

IDENTIFICATION TESTS:

Staphylococcus aureus- *[Mackey]*

- Morphology in culture Medias:

Nutrient agar- as smooth, low convex, glistening, densely opaque and of butyrous consistency[30]. The pigment formation in nutrient agar when kept in room temperature inaerobic atmosphere ranges from cream colour to golden yellow due topresence of carotenoids.

Blood agar- as white opaque colonies surrounded by a zone of β-hemolysis[31].On MacConkey agar(MAC)-Small opaque colonies and acquires the colour of the indicator and appear as pink.

- Gram staining showed Gram positive cocci in clusters.
- Colonies were subjected to the following biochemical tests.

S.NO	TESTS	RESULTS
1.	Catalase test	Positive
2.	Coagulase test(Slide and Tube method)	Positive
3.	Urease test	Urea hydrolysed
4.	Voges-Proskauer	Acetoin produced
5.	Hugh-Leifson's Oxidation Fermentation test	Fermentative pattern
6.	Mannitol fermentation test	Fermented with gas production

Enterococcus faecalis- *[Mackey]*

- Gram staining showed Gram positive oval cocci arranged in pairs and short chains
- Colony morphology

On 5% Blood Agar Plate(BAP) - Tiny Translucent non-haemolytic colonies

On MacConkey agar(MAC)-Small Magenta coloured colonies

- Colonies were subjected to the following biochemical tests.

S.NO	TESTS	RESULTS
1.	Catalase test	Negative
2.	Heat tolerance test at 45ºC	Positive
3.	Voges-Proskauer	Acetoin produced
4.	Bile esculin hydrolysis	Positive
5.	Sorbitol	Fermented
6.	Arabinose Fermentation Test	Not fermented

Klebsiella species-*[Mackey]*

- Colony morphology

  On MacConkey agar(MAC)-Large mucoid Lactose fermenting colonies

  On 5% Blood Agar Plate(BAP)-Large greyish-white mucoid colonies

- Gram staining showed Short plump Gram negative bacilli
- Motility test by Hanging Drop method- Non-Motile
- Colonies were subjected to the following biochemical tests.[Mackey

S.NO	TESTS	Klebsiella pneumonia	Klebsiellaoxytoca
1.	Catalase	Positive	Positive
2.	Oxidase	Negative	Negative
3	Nitrate Reduction test	Positive	Positive
4.	Hugh-Leifson's Oxidation Fermentation test	Fermentative	Fermentative
5.	Indole test	Indole not produced	Indole produced
6.	Methyl Red test(MR) Voges-Proskauer test(VP)	MR:Negative VP: acetoin produced(Positive)	MR: Negative VP: acetoin produced(Positive)

7.	Simmon'sCitrate Utilization test	Citrate utilized	Citrate utilized
8.	Christensen's Urease test	Urea hydrolyzed	Urea hydrolyzed
9.	Triple Sugar Iron agar test (TSI)	Acid butt/acid slant with gas production and no H_2S production	Acid butt/acid slant with gas production and no H_2S production
10.	Lysine Decarboxylation test	Decarboxylated	Decarboxylated

E.coli

- Colony morphology

  On MacConkey agar(MAC)- Smooth, glossy Lactose fermenting colonies

- Gram staining showed Gram negative bacilli
- Motility test by Hanging Drop method-Motile
- Colonies were subjected to the following biochemical tests.[Mackey

S.NO	TESTS	RESULTS
1.	Catalase	Positive
2.	Oxidase	Negative
3	Nitrate Reduction test	Positive
4.	Hugh-Leifson's Oxidation Fermentation test	Fermentative
5.	Indole test	Indole produced
6.	Methyl Red test(MR)	MR: Positive
	Voges-Proskauer test(VP)	VP: negative
7.	Simmon'sCitrate Utilization test	Citrate not utilized
8.	Christensen's Urease test	Urea not hydrolyzed
9.	Triple Sugar Iron agar test(TSI)	Acid butt/acid slant with gas production and no H_2S production
10.	Lysine Decarboxylation test	Decarboxylated
11.	Mannitol motility medium	Fermented and motile

Proteus spp

1.Colony morphology

On MacConkey agar(MAC)- Lactose non- fermenting colonies

On 5% Blood Agar Plate(BAP)-greyish white colonies with swarming

2.Gram staining showed Gram negative bacilli

3.Motility test by Hanging Drop method-Motile

4. Colonies were subjected to the following Identification tests. [Mackey

S.NO	TESTS	Proteus mirabilis	Proteus vulgaris
1.	Catalase	Positive	Positive
2.	Oxidase	Negative	Negative
3	Phenylalanine deaminase test	Positive	Positive
4.	Hugh-Leifson's Oxidation Fermentation test	Oxidative	Oxidative
5.	Indole test	Not produced	Indole produced
7.	Simmon'sCitrate Utilization test	Citrate utilized	Citrate utilized
8.	Christensen's Urease test	Urea hydrolyzed	Urea hydrolyzed
9.	Triple Sugar Iron agar test(TSI)	Acid butt/alkaline slant with H_2S production	Acid butt/alkaline slant with H_2S production
10.	Maltose	Not fermented	Fermented
11.	Arginine Dihydrolase test	Negative	Negative
12.	Lysine decarboxylation	Negative	Negative
13.	Ornithine decarboxylation	Positive	Negative

Pseudomonas aeruginosa. [Mackey

- Colony morphology –

  On MacConkey agar(MAC)-Large Spreading Lactose non- fermenting colonies

  On Nutrient Agar plate-Irregular colonies with metallic sheen and blue green diffusible pigment

- Gram staining showed Gram negative bacilli
- Motility test by Hanging Drop method-Motile
- Colonies were subjected to the following Identification tests.[Mackey

S.NO	TESTS	RESULTS
1.	Catalase	Positive
2.	Oxidase	Positive
3	Nitrate Reduction test	Positive
4.	Hugh-Leifson's Oxidation Fermentation test	Oxidative
5.	Indole test	Not produced
7.	Simmon's Citrate Utilization test	Citrateutilized
8.	Christensen's Urease test	Urea not hydrolysed
9.	Triple Sugar Iron agar test(TSI)	Alkaline butt/alkaline slant without gas or H_2S production
10.	Arginine Dihydrolase test	Positive
12.	Growth at 42ºC	Positive

Acinetobacter baumannii

- Colony morphology

  On MacConkey agar(MAC)-Large Lactose non- fermenting pale pink colonies

- Gram staining showed Gram negative cocco- bacilli
- Motility test by Hanging Drop method-Non-Motile
- Colonies were subjected to the following Identification tests.[Mackey

S.NO	TESTS	RESULTS
1.	Catalase	Positive
2.	Oxidase	Negative
3	Nitrate Reduction test	Negative
4.	Hugh-Leifson's Oxidation Fermentation test	Oxidative
5.	Indole test	Not produced
6.	Simmon'sCitrate Utilization test	Citrate utilized
7.	Christensen's Urease test	Urea not hydrolyzed
8.	Triple Sugar Iron agar test(TSI)	Alkaline butt/alkaline slant
9.	Growth at 42ºC	Positive
10.	10% OF Lactose Utilization test	Positive

ANTIBIOTIC SENSITIVITY TESTING

The colonies were identified as *Staphylococcus aureus*, beta hemolytic *Streptococci, Enterococcus faecalis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Klebsiella oxytoca, Proteus mirabilis, Proteus vulgaris, E. coli, and Acinetobacter baumannii* by gram staining and other biochemical reactions and antibiotic sensitivity testing was performed.

Antimicrobial susceptibility testing to be done for the isolates on Mueller HintonAgar by Kirby-Bauer Disc Diffusion method as per CLSI guidelines. For Streptococci and Enterococci sensitivity was performed on 5% Sheep blood agar.

DISC DIFUSSION METHOD

With a sterile loop, touch the tops of four or five similar-appearing, well-isolated colonies of the test organism from an overnight growth on primary agar plate and are suspended in 0.5ml of sterile saline.

The turbidity was matched with 0.5 Mc Farland turbidity standards.

A fresh sterile cotton tipped swab was dipped into the suspension and the excess of inoculum was removed by pressing it against the sides of the tube.

The dried surface of a Mueller–Hinton agar plate was brought to room temperature and inoculated by streaking the swab three times over the entire agar surface by rotating the plate approximately 60 degrees each time for even distribution of the inoculum. The rim of the agar was swabbed finally and the lid of the dish was replaced. Before adding the antibiotic disks allow the surface of the agar to dry for at least 3–5 minutes but no longer than 15 minutes.

The antibiotic discs were placed on the plate using sterile forceps so that even contact is ensured and incubated aerobically at 37°C.

After 18 to 24 hours of incubation the diameter of the clear zone around the disc was measured under transmitted light with measuring scale and results were interpreted as susceptible, intermediate or resistant as per the CLSI criteria.

The antibiotic disc was obtained from HI MediaLaboratories. The concentration of the antibiotic disc wasused as per CLSI guidelines.

The diameter of each zone (including the diameter of the disc) of inhibition was measured and recorded in millimeters and the result was then compared with the zone size interpretative chart according to CLSI guidelines.

The quality control for antimicrobial susceptibility testing was done with standard strains of S. aureus (ATCC 25923), E. coli (ATCC 25922) and Ps. aeruginosa (ATCC 27853)

Zone Diameter Interpretive Standards for *Staphylococcus species*

Antimicrobial Agent	Disc content	Zone Diameter Interpretive Criteria (nearest whole mm)		
		Sensitive	Intermediate	Resistant
Penicillin	10 units	≥ 29	-	≤ 28
Gentamycin	10 µg	≥15	13-14	≤12
Erythromycin	15 µg	≥23	14-22	≤13
Tetracycline	30 µg	≥19	15-18	≤ 14
Ciprofloxacin	5 µg	≥21	16-20	≤ 15
Trimethoprim/ Sulfamethoxazole	1.25/23.75 µg	≥16	11-15	≤ 10
Chloramphenicol	30 µg	≥18	13-17	≤12
Linezolid	30 µg	≥21	-	≤ 20

Zone Diameter Interpretive Standards for Enterobacteriaceae[clsi]

Antimicrobial Agent	Disc content	Zone Diameter Interpretive Criteria (nearest whole mm)		
		Sensitive	Intermediate	Resistant
Amikacin	30 µg	≥ 17	15-16	≤ 14
Gentamicin	10 µg	≥15	13-14	≤ 12

Ciprofloxacin	5 µg	≥21	16-20	≤ 15
Trimethoprim/ Sulfamethoxazole	1.25/ 23.75 µg	≥16	11-15	≤ 10
Tetracycline	30 µg	≥15	12-14	≤ 11
Cefotaxime	30 µg	≥26	23-25	≤ 22
Ceftazidime	30 µg	≥21	18-20	≤ 17
Piperacillin-Tazobactam	100/ 10 µg	≥21	18-20	≤ 17
Imipenem	10 µg	≥23	20-22	≤ 19

Zone Diameter Interpretive Standards for Gram Negative Non-Fermenter Bacteria

Antimicrobial Agent	**Disc content**	**Gram Negative Bacilli**	**Zone Diameter Interpretive Criteria (nearest whole mm)**		
			Sensitive	**Intermediate**	**Resistant**
Amikacin	30 µg	*Pseudomonas aeruginosa* and *Acinetobacter baumannii*	≥ 17	15-16	≤ 14
Gentamicin	10 µg	*Pseudomonas aeruginosa* and *Acinetobacter baumannii*	≥15	13-14	≤ 12
Trimethoprim/ Sulfamethoxazole	1.25/ 23.75 µg	*Acinetobacter baumannii*	≥16	11-15	≤ 10
Ciprofloxacin	5 µg	*Pseudomonas aeruginosa* and *Acinetobacter baumannii*	≥21	16-20	≤ 15
Ceftazidime	30 µg	*Pseudomonas aeruginosa* and *Acinetobacter baumannii*	≥18	15-17	≤ 14
Piperacillin-Tazobactam	100/ 10 µg	*Acinetobacter baumannii*	≥21	18-20	≤ 17
		Pseudomonas aeruginosa	≥21	15-20	≤ 14
Imipenem	10 µg	*Pseudomonas aeruginosa*	≥19	16-18	≤ 15
		Acinetobacter baumannii	≥22	19-21	≤ 18

PHENOTYPIC SCREENING TEST FOR METHICILLIN RESISTANCE

STAPHYLOCOCCUS AUREUS

Detection of mec A mediated oxacillin resistance using cefoxitin:

- Medium -MHA
- Antimicrobial concentration -30 µg cefoxitin disk
- Inoculum- Standard disk diffusion procedure
- Incubating condition- 33 to 35°C; ambient air (Testing at temperatures above 35°C may not detect MRSA.)
- Incubation length - 16–18 hours
- 24 hours (may be reported after 18 hours, if resistant)

RESULTS

S.aureus	**CONS**
≤ 21 mm = mecA positive	≤ 24 mm = mecA positive
≥ 22 mm = mecA negative	≥ 25 mm = mecA negative

Cefoxitin was used as a surrogate for mecA-mediated oxacillin resistance. Isolates that test as mecA positive should be reported as oxacillin (not cefoxitin) resistant; other β-lactam agents, except those with anti-MRSA activity, should be reported as resistant.

All the MRSA isolates were subjected to the following screening and confirmatory test for detection of Mupirocin resistance.

Detection of high level mupirocin resistance for MRSA isolates

Disk diffusion Test method -

- Medium- MHA
- Antimicrobial concentration –200-µg mupirocin disk
- Inoculum- Standard disk diffusion procedure
- Incubating condition- 33 to 35°C; ambient air (Testing at temperatures above 35°C may not detect MRSA.)
- Incubation length - 24 hours

`

RESULTS

Results Examined carefully with transmitted light for light growth within the zone of inhibition.

No zone = high-level mupirocin resistance.

Any zone = the absence of high-level mupirocin resistance.

E-test

Ezy MIC™ strip was useful for quantitative determination of susceptibility of bacteria toantimicrobial agents. The system comprises of a predefined quantitative gradient which was used to determine the Minimum Inhibitory Concentration (MIC) in mcg/ml of different antimicrobialagents against microorganisms tested on appropriate agar media, following overnightincubation. Mupirocin Ezy MIC Strip (MUP) (0.064-1024 mcg/ml)

It was a unique MIC determination paper strip which was coated with Mupirocin on a single paper strip in a concentration gradient manner, capable of

showing MICs in the range of 0.064 mcg/ml to 1024 mcg/ml, on testing against the test organism

Preparation of Inoculum

Direct colony suspension was prepared from18-24-hour old non-selective media agar plate in saline. Adjust the turbidity to that of standard 0.5 McFarland

Test Procedure

Plates with suitable make of Mueller Hinton Agar was prepared .A sterile non-toxic cotton swab on a wooden applicator was dipped into the standardized inoculum and rotate the soaked swab firmly against the upper inside wall of the tube to express excess fluid. The entire agar surface of the plate was streaked with the swab three times, turning the plate at 60° angle between each streaking.

Ezy MIC™ strip container was removed from cold and keep it at room temperature for 15minutes before opening. One applicator was removed from the self sealing bag stored at room temperature. Ezy MIC™ strip was lifted by holding the applicator in the middle and gently press its broader sticky side on the centre of the strip. The strip was placed at a desired position on agar plate pre-spread with test culture. Gently turnthe applicator clockwise with fingers to detach from the strip.DO NOT PRESS EZY MIC™ STRIP. Within 60 seconds, Ezy MIC™ strip was adsorbed and will firmly adhere to the agar surface.

Ezy MIC™ strip should not be repositioned or adjusted once placed. Transferthe plates to the incubator under appropriate conditions.

MIC Reading:

Read the plates only when sufficient growth was seen and read the MIC where the ellipse intersects the MIC scale on the strip.

For bacteriostatic drugs such Mupirocin, Chloramphenicol, Azithromycin, Fluconazole, and Trimethoprim/ sulphamethoxazole, read MICs at 80% inhibition forhomogenously sensitive strains such as QC control strains.

Isolated colonies, microcolonies and hazes appearing in the zone of inhibition areindicative of hetero nature of the culture having resistant subpopulation in it. In suchcases, consider reading for MIC determination at a point on the scale above which noresistant colonies are observed close to MIC strip (within 1-3 mm distance from thestrip).

Since Ezy MIC™ strip has continuous gradient, MIC values “in-between” two-fold dilutions can be obtained. Always round up these values to the next two-fold dilution before categorization. Forexample: mupirocin showing reading of 0.75mcg/ml should be rounded up to nextconcentration i.e. 1.0 mcg/ml. If the ellipse intersects the strip in between 2 dilutions, read the MIC as the value whichis nearest to the intersection.

When growth occurs along the entire strip, report the MIC as > the highest values on theMIC strip. When the inhibition ellipse is below the strip (does not intersect the strip), report the MIC < the lowest value on the MIC scale.

Ezy MIC™ Strip FEATURES AND ADVANTAGES

Ezy MIC™ strip exhibits several advantages over existing plastic strip.

Ezy MIC™ strip was made up of porous paper material unlike plastic non-porous material

Ezy MIC™ strip has MIC values printed on both sides identically.

The antimicrobial agent was evenly distributed on either side of the Ezy MIC™ strip and henceit can be placed by any side on the agar surface. For Ezy MIC™ strips, MIC values can be read without opening the lid of the plate as most commonly translucent medium such as Mueller Hinton Agar is employed.

Once placed, Ezy MIC™ strip is adsorbed within 60 seconds and firmly adheres to the agarsurface. Unlike the plastic material, it does not form air bubbles underneath and hence there is no needto press the strip once placed.

GENOTYPIC METHOD

PCR for mup A gene

Material & Methods:

Pure Fast® Bacterial DNA minispin purification kit [Kit contains Lysozyme, Lysozymedigestion buffer, Proteinase-K, Binding buffer, Wash Buffer-1, Wash Buffer-2, Spin columnswith collection tube and elution buffer. HELINI 2X ReDdye PCR Master Mix, Agarose gelelectrophoresis consumables and mupA and CFR Primers are from HELINI Biomolecules, Chennai, India.

2X Master Mix:

It contains 2U of Taq DNA polymerase, 10X Taq reaction buffer, 2mM MgCl2, 1µl of10mM dNTPs mix and RedDye PCR additives.

Agarose gel electrophoresis:

Agarose, 50X TAE buffer, 6X gel loading buffer and Ethidium bromide are from HELINIBiomolecules, Chennai.

PCR:

HELINI Ready to use mupA gene Primer mix - 5µl/reaction

PCR Product: 460bp

Bacterial DNA Purification

- 1ml of overnight culture was centrifuged at 6000rpm for 5min and the Supernatant was discarded. The Pellet was suspended in 0.2ml PBS, 180µl of Lysozyme digestion buffer and 20µl of Lysozyme [10mg/ml] was added and Incubated at 37C for 15min.

- 400µl of Binding buffer, 5µl of internal control template and 20µl of Proteinase K added, and mixed well by inverting several times and incubated at 56ºC for 15min.
- 300µl of Ethanol was added and mixed well, the entire sample was transferred into the PureFast® spin column and centrifuged for 1 min. Discard the flow-through and place the column back into the same collection tube.

- 500µl Wash buffer-1 was added to the PureFast® spin column and centrifuged for 30-60 seconds and discard the flow-through. Place the column back into the same collection tube.
- 500µl Wash buffer-2 was added to the PureFast® spin columnand Centrifuged for 30-60 seconds and the flow-through was discarded. Place the column back into the same collection tube.
- Discard the flow-through and centrifuged for an additional 1 min. This step was essential toavoid residual ethanol. Transfer thePureFast® spin column into a fresh 1.5 ml micro-centrifuge tube. And 100µl of Elution Buffer was added to the center of PureFast® spin column membrane. Incubated for 1 min at room temperature and then centrifuged for 2 min.
- Discard the column and store the purified DNA at -20°C. Quality and Quantity ofextracted DNA was checked by loading in 1% agarose gel and 5µl of extracted DNA wasused for PCR amplification.

PCR Procedure:

1.Reactions set up as follows;

Components	Quantity
HELINI RedDye PCR Master mix	10µl
HELINI Ready to use - Primer Mix	5µl
Purified Bacterial DNA	5µl
Total volume	20µl

2. Mixed gently and spin down briefly.

3. Placed into PCR machine and program it as follows;

Initial Denaturation	: 95ºC for 5 min
Denaturation	: 94ºC for 30sec
Annealing	: 58ºC for 30sec 35 cycles
Extension	: 72ºC for 30sec
Final extension	: 72º C for 5 min

Loading:

1. Prepared 2% agarose gel. [2gm of agarose in 100ml of 1X TAE buffer]
2. Run electrophoresis at 50V till the dye reaches three fourth distances and observe thebands in UV Transilluminator

Agarose gel electrophoresis:

Prepared 2% agarose. (2gm agarose in 100ml of 1X TAE buffer and melted using microoven)

When the agarose gel temperature was around 60ºC, 5µl of Ethidium bromide was added. Warm agarose solution was poured slowly into the gel platform and kept the gel set undisturbed till the agarose solidifies. 1XTAE buffer was poured into submarine gel tank. Place the gel platform carefully into the tank. Maintain the tank buffer level 0.5cm above than the gel.

PCR Samples were loaded after mixing with gel loading dye along with 10µl HELINI100bp DNA Ladder. [100bp, 200bp, 300bp, 400bp, 500bp, 600bp, 700bp, 800bp, 900bp,1000bp and 1500bp]

Run electrophoresis at 50V till the dye reaches three fourth distance of the gel. Gel was viewed in UV Transilluminator and the band pattern was observed.

Phenotypic confirmatory test for ESBL production- Combined discmethod

In this method, a lawn culture was madewiththe test isolates for discdiffusion method.

Ceftazidime (30µg) and ceftazidime-clavulanic acid(30µg/10µg) discs- HI media, were placed at a distance of 20mm center to center on the Mueller-Hinton agar plate, incubated at 37ºC for 20-24 hours. The testisolate was considered to produce ESBL if the zone of inhibition around the ceftazidime-clavulanic acid disc was ≥5mm than the zone around ceftazidime disc alone.

Results

RESULTS

The present study comprised of 200 randomly selected cases of clinically diagnosed Pyoderma, both primary and secondary, attended the Department of Dermatology, RGGGH, during the study period March 2017-February 2018.

Table 1: STUDY GROUP

Type of Pyoderma	Number of cases	Percentage
Primary Pyoderma	58	29%
Secondary Pyoderma	142	71%
Total	200	100%

Among 200 cases of Pyoderma, Primary pyoderma constitute 29% Secondary pyoderma constitute 71%, therefore Secondary pyoderma were more common than Primary pyoderma in this study.

FIGURE 1

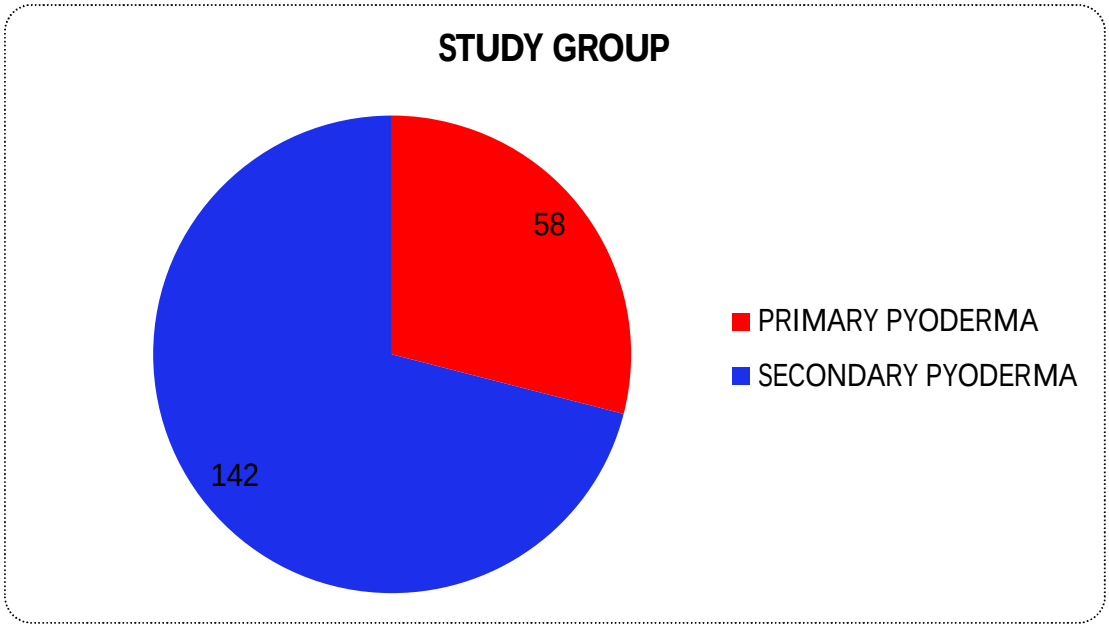
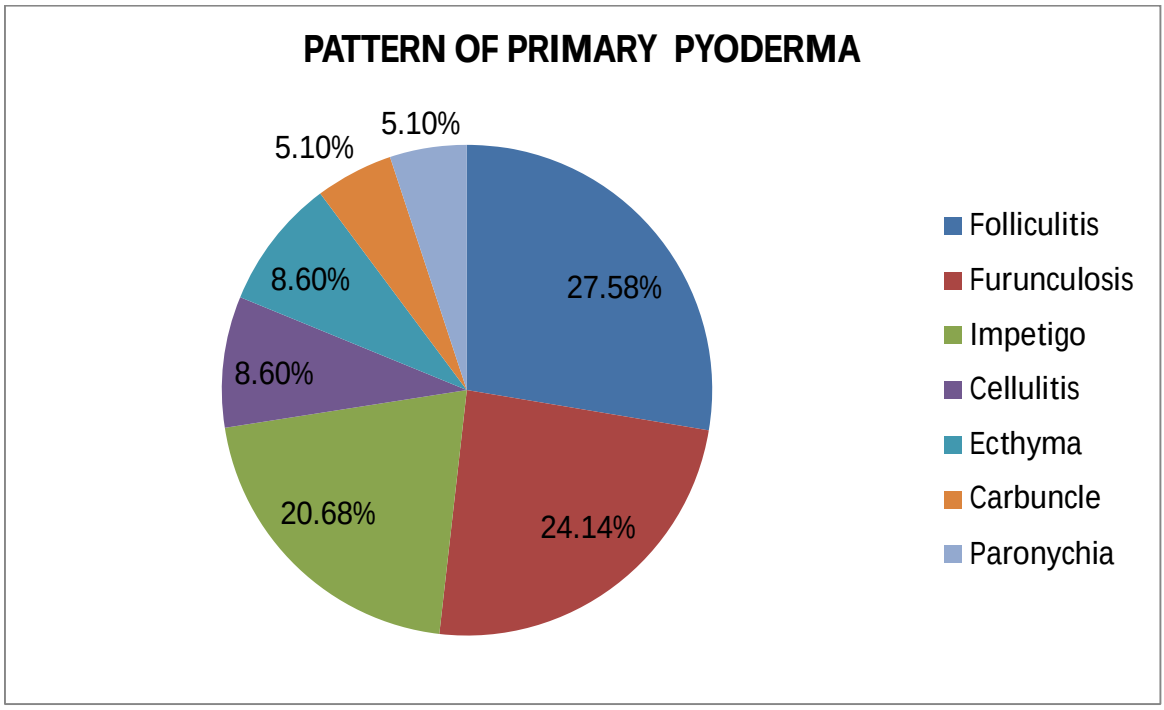


Table 2: PATTERN OF PRIMARY PYODERMA

S.No	Cinical diagnosis	No of cases	Percentage
1	Folliculitis	16	27.58%
2	Furunculosis	14	24.14%
3	Impetigo	12	20.68.%
4	Cellulitis	5	8.6%
5	Ecthyma	5	8.6%
6	Carbuncle	3	5.1%
7	Paronychia	3	5.1%
	Total	58	100 %

FIGURE 2

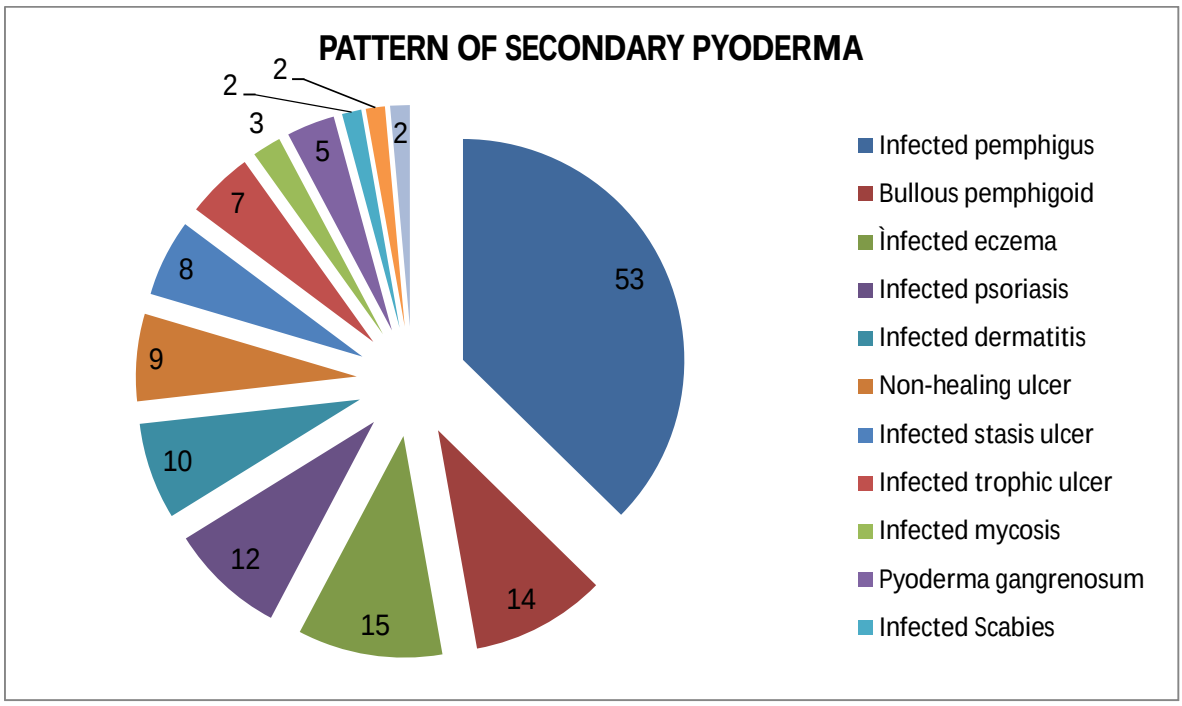


Among the primary pyoderma, Folliculitis(27.58%) was the most common clinical type followed by Furunculosis(24.14%), Impetigo(20.68%), Cellulitis(8.6%),ecthyma(8.6%), Carbuncle(5.1%) and Paronychia(5.1%).(table 2)

TABLE-3 PATTERN OF SECONDARY PYODERMA

S.no	Cinical diagnosis	No of cases	Percentage
1	Infected pemphigus	53	37.3%
2	Bullous pemphigoid	14	9.8%
3	Ìnfected eczema	15	10.5%
4	Infected psoriasis	12	8.4%
5	Infected dermatitis	10	7.0%
6	Non-healing ulcer	9	6.3%
7	Infected stasis ulcer	8	5.6%
8	Infected trophic ulcer	7	4.9%
9	Infected mycosis	3	2.1%
10	Pyoderma gangrenosum	5	2.1%
11	Infected Scabies	2	1.4%
12	Hidradenitissuppurativa	2	1.4%
13	Infected keloid	2	1.4%
	Total	142	100%

FIGURE 3

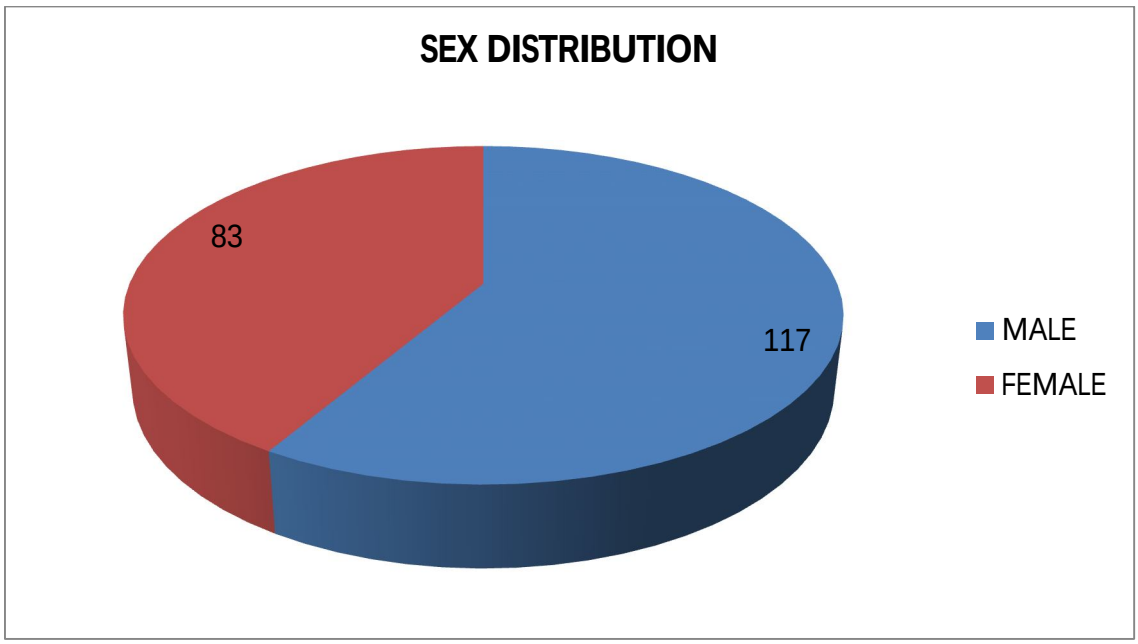


Among the secondary Pyoderma, Infected pemphigus (37.3%) was the most common followed by Eczema with secondary infection(10.5%),bullous pemphigoid (9.8%), infected Psoriasis (8.4%),infected dermatitis(7.0%), Non-healing ulcer(6.3%), infected trophic ulcer(4.9%),infected mycoses (2.1%),pyoderma gangrenosum (2.1%),Scabies with secondary infection(1.4%), Hidradenitis Suppurativa(1.4%) and infected keloid (1.4%).(table 3)

TABLE-4 SEX DISTRIBUTION

SEX	NO OF CASES	PERCENTAGE
MALE	117	58.5%
FEMALE	83	41.5%
TOTAL	200	100%

FIGURE 4



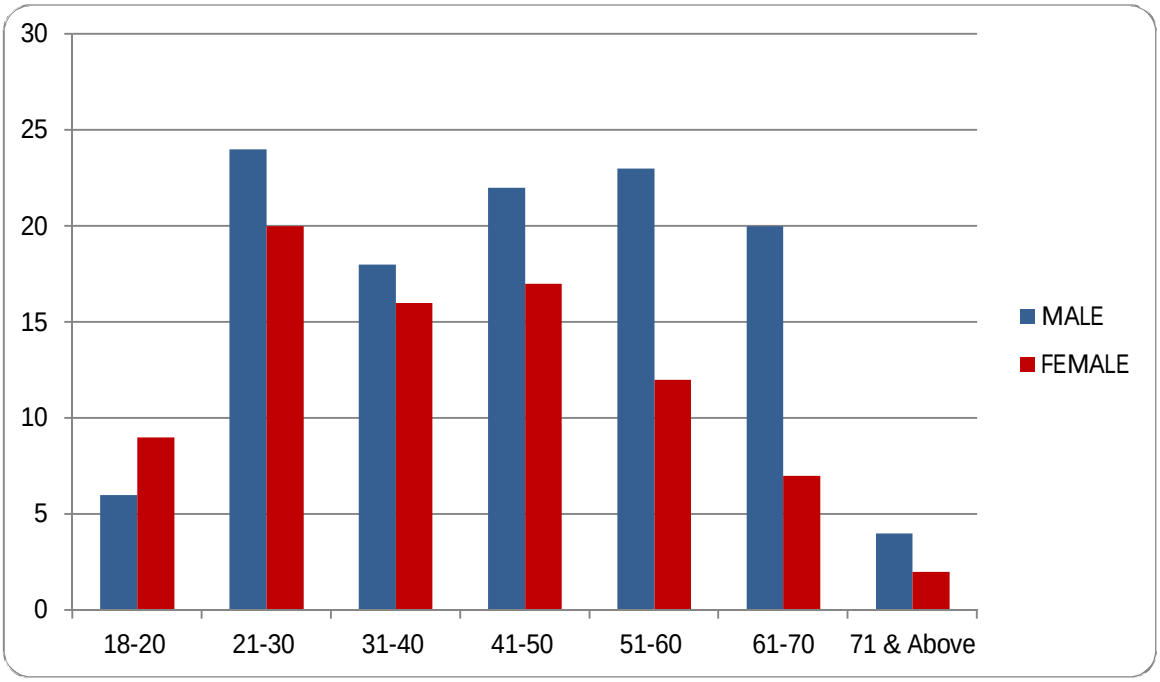
Out of the 200 cases, 117(58.5%) were males and 83 (41.5%) cases wereFemales. The male &female ratio was 1.4:1. (table 4).

TABLE-5 DISTRIBUTION OF AGE (IN YEARS)

AGE IN YEARS	NO OF PATIENTS		NO OF PATIENTS	PERCENTAGE
	MALE	FEMALE		
18-20	6	9	15	7.5%
21-30	24	20	44	22%
31-40	18	16	34	17%
41-50	22	17	39	19.5%
51-60	23	12	35	17.5%
61-70	20	7	27	13%
71 & Above	4	2	6	3%
TOTAL	117	83	200	100%
Mean age± SD			43.43 ±16.03	

Significant P-value <0.05

FIGURE 5

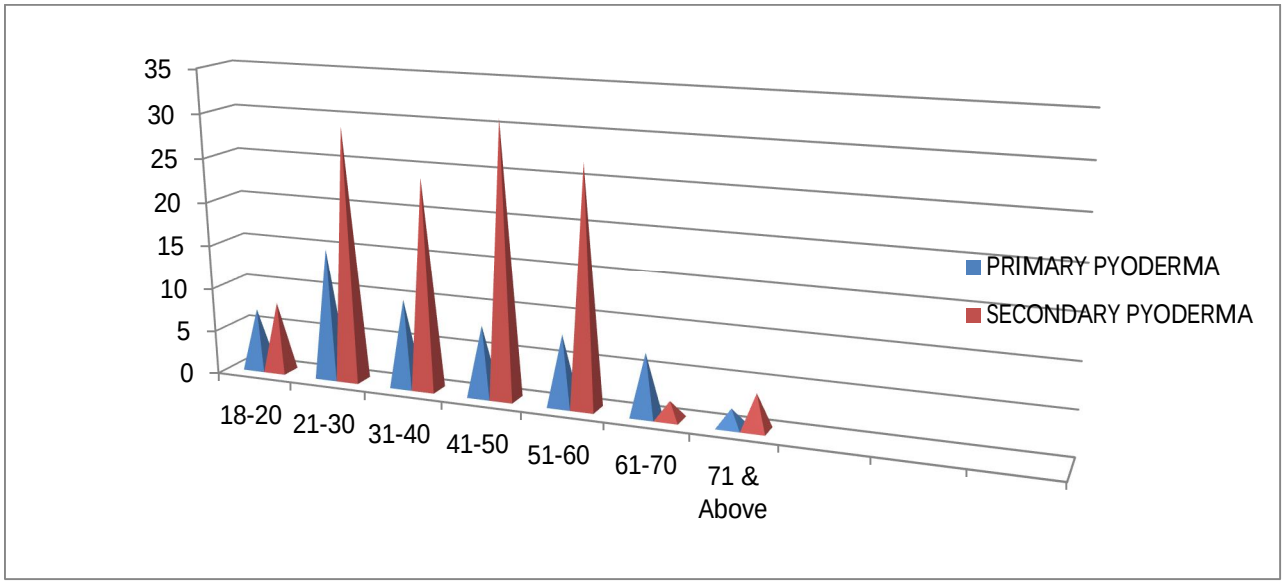


Pyodermas occurred most commonly in the age group between 21-30years (22%) followed by and 41-50 years age group (19.5%) and 51-60(17.5%). Half of total cases are in age group between 21-50 years. Pyodermas were less commonly occurring in age group 71years and above (4%).

TABLE-6A AGE DISTRIBUTION IN RELATION T0 TYPE OF PYODERMA

Age group	Primary pyoderma n (%)	Secondarypyoderma n (%)	Total n (%)
18-20	7(12.3)	8(5.6)	15(7.5)
21-30	15(26.3)	29(20.3)	44(22)
31-40	10(17.5)	24(16.8)	34(17)
41-50	8(14.0)	31(21.7)	39(19.5)
51-60	8(14.0)	27(18.9%)	35(17.5)
61-70	7(12.3)	20(14.0)	27(13.5)
71 & Above	2(3.5)	4(2.8)	6(3)
Total	57(100)	143(100)	200(100)

FIGURE 6A AGE DISTRIBUTION IN RELATION T0 PYODERMA



Primary Pyodermas were more common in the age group 21-30 years whereas secondary Pyoderma were more common in the age group 41-50years.

Table-6B SEX DISTIBUTION IN RELATION TO PYODERMA

Sex	Primary pyoderma	Secondary pyoderma	Total
Male	27(23.1%)	90(76.9%)	117(100%)
Female	30(36.1%)	53(63.9%)	83(100%)
Total	57(100%)	143(100%)	200(100%)

Significant P-Value- 0.05

FIGURE 6B

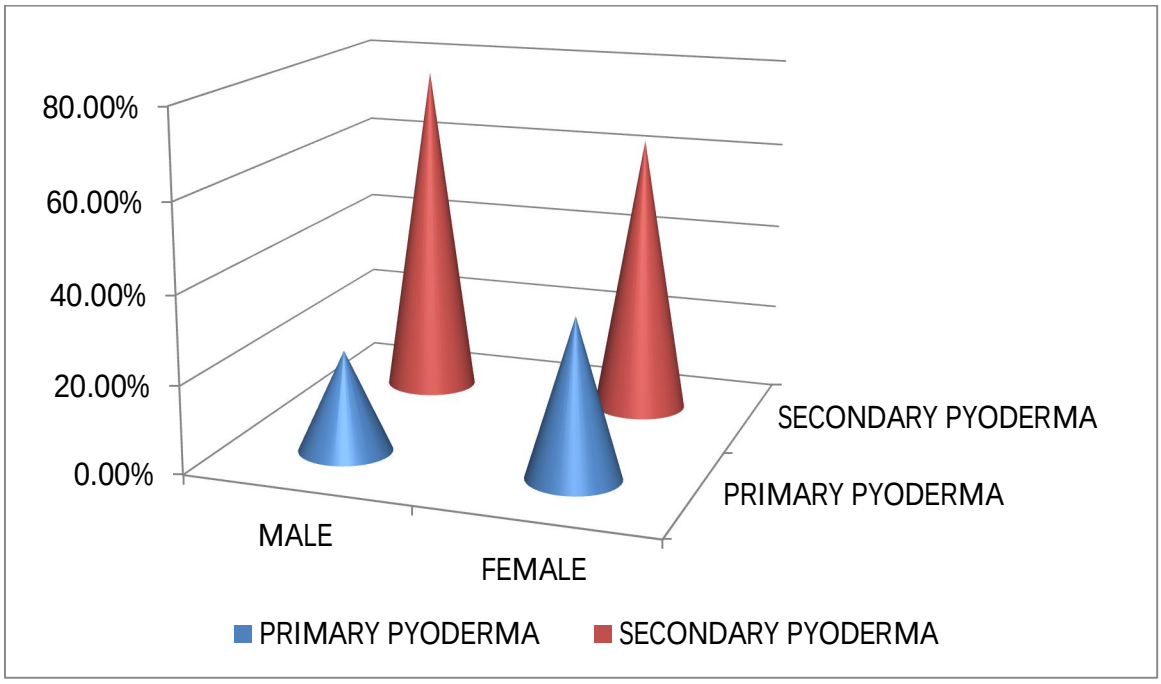
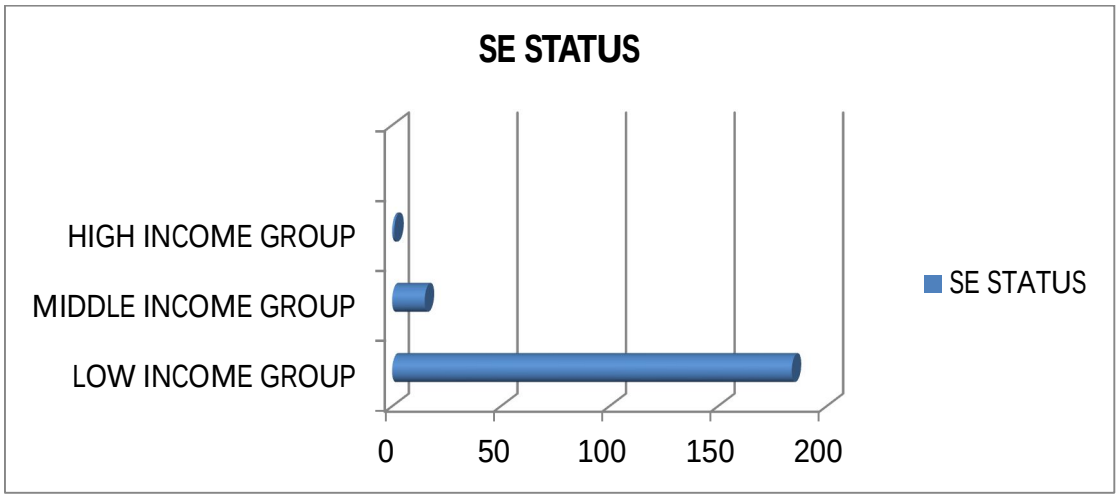


Table-7 SOCIO-ECONOMIC STATUS

Socio-economic status	No.of patients	Percentage(%)
Low income group	184	92
Middle income group	15	7.5
High income group	1	0.5
Total	200	100

FIGURE 7

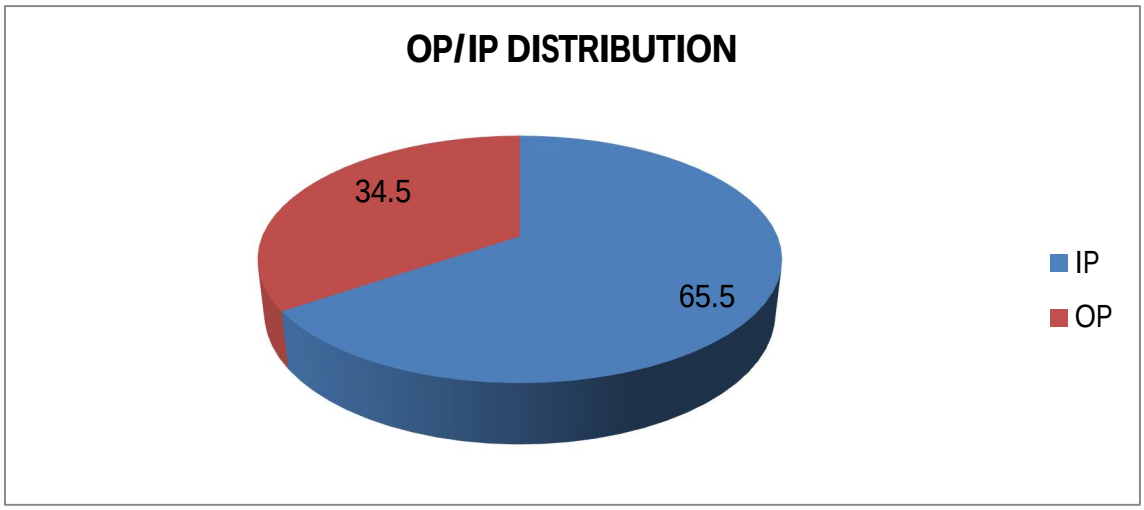


High incidence of pyoderma occurred among Low income group (92%) followed by middle income group (7.5%).

TABLE-8 OP/IP DISTRIBUTION

	No. of cases	Percentage
IP	131	65.5%
OP	69	34.5%
TOTAL	200	100%

FIGURE 8

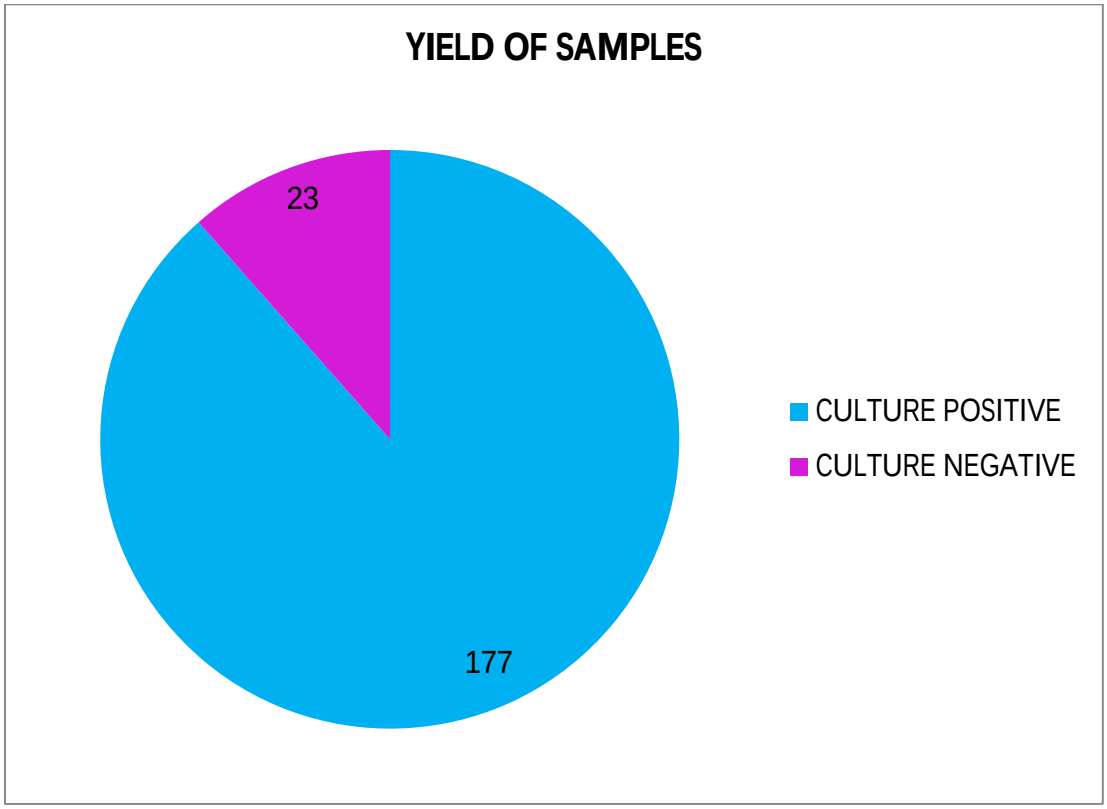


Incidence of pyoderma was more in patients admitted in wards than outpatients.

TABLE-9 YIELD OF SAMPLES

Yield of samples	No.of cases	Percentage
Culture positive	177	88.5
Culture negative	23	11.5
Total	200	100

Figure 9

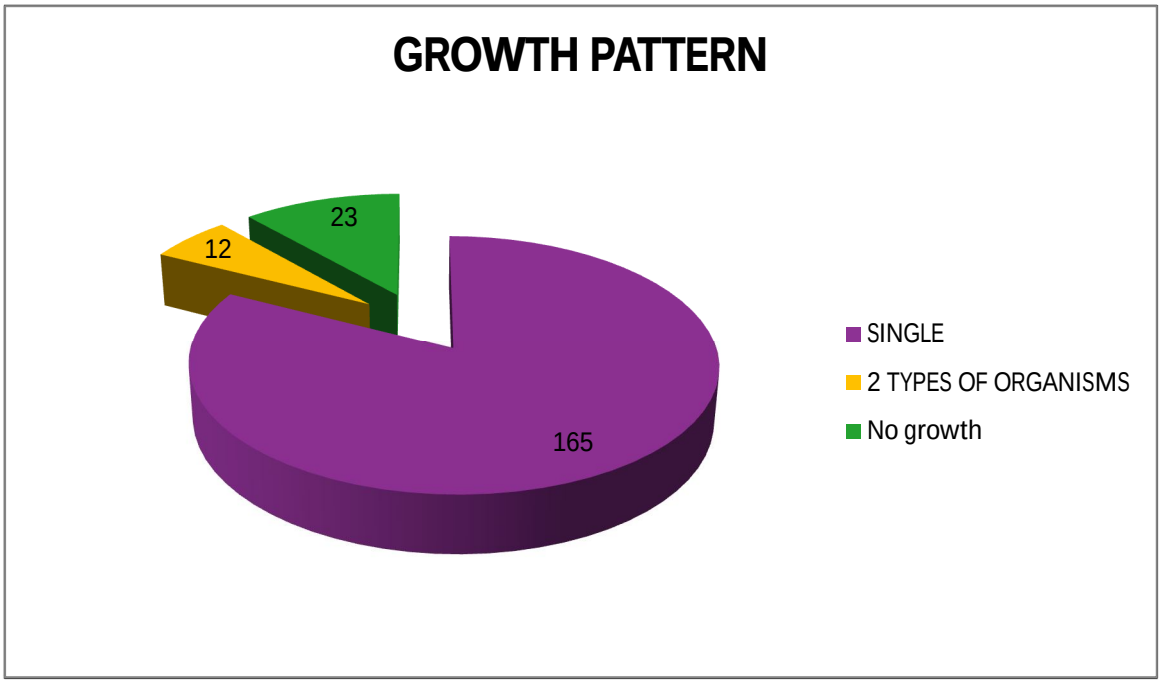


Among 200 samples, 88.5% were Culture positive and 11.5% were culture negative.

TABLE-10 GROWTH PATTERN

Type of isolate	Number of cases	Percentage
Single	165	83.5%
2 types of Organisms	12	5.7%
No growth	23	16.5%
Total	200	100 %

FIGURE 10

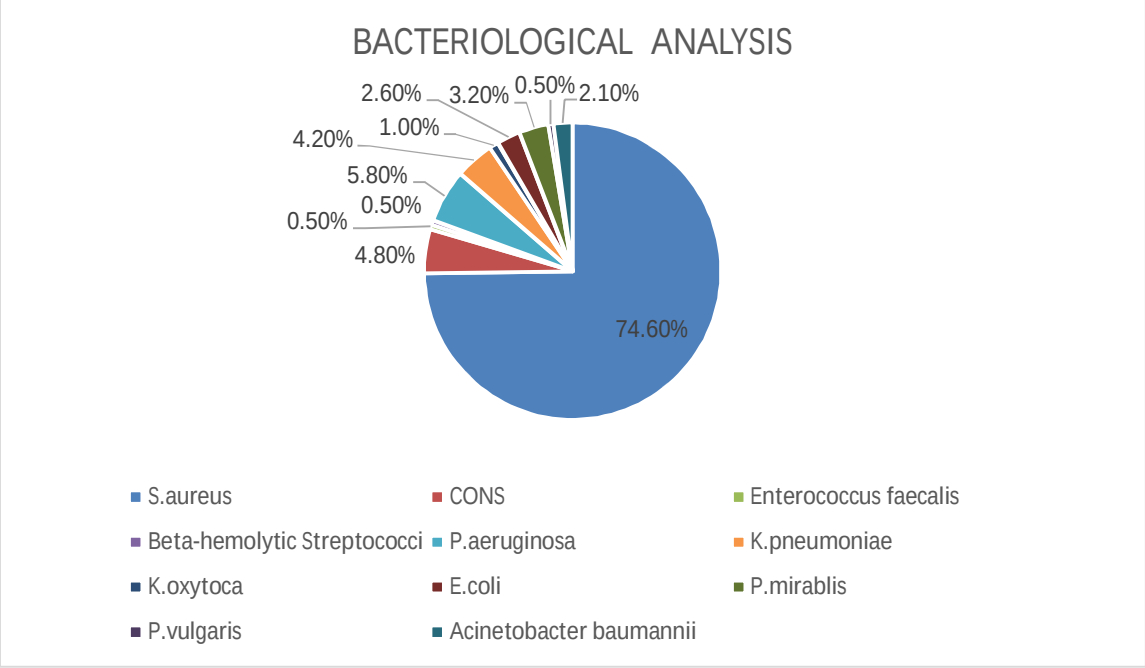


Single type of organism was grown in 165 samples (9%) and in 12 samples (6%) 2 types of organisms were grown and 23 samples showed no growth. (table no 10).

TABLE-11BACTERIOLOGICAL ANALYSIS

ISOLATES	TOTAL	PERCENTAGE
S.aureus	141	74.6%
CONS	9	4.8%
Enterococcus faecalis	1	0.5%
Beta-hemolytic Streptococci	1	0.5%
Ps.aeruginosa	11	5.8%
K.pneumoniae	8	4.2%
K.oxytoca	2	1.0%
E.coli	5	2.6%
P. mirabilis	6	3.2%
P.vulgaris	1	0.5%
Acinetobacter baumannii	4	2.1%
TOTAL	189	100

FIGURE 11



Staphylococcus aureus (74.6%) was the commonest organism isolated followed by Pseudomonas aeruginosa (5.8%).

TABLE -12 BACTERIAL ISOLATES FROM VARIOUS TYPES OF PYODERMA

Diagnosis	*S.aureus*	*CoNS*	*K. pneumoniae*	*E.feacalis*	*Klebsiella oxytoca*	*E.coli*	*Proteus vulgaris*	*Proteus mirabilis*	*Ps aeruginosa*	*Acinetobacter baumannii*	*BHS*	*S.aureus+Ps aeruginosa*	*S.aureus +Proteus mirabilis*	*S.aureus+ E.coli*	*S.aureus+K.oxytoca*	*S.aureus+K.pneumoniae*	*CONS+P.mirabilis*	*Ps.aeruginosa+K.pneumoniae*	*Ps.aeruginosa+E.coli*	*NG*	*TOTAL*
	n (percentage)																				
Folliculitis	9 (56.25)	0	0	0	0	0	0	1 (6.25)	0	0		0				1 (6.25)		1 (6.25)		4 (25)	16 (100)
Furunculosis	12 (85.7)																			2 (14.3)	14
Impetigo	10 (83.3)																			2 (16.7)	12
Cellulitis	3 (60)																			2 (40)	5
Ecthyma	3 (60)								1 (20)			1 (20)									5
Carbuncle	2 (66.7)																		1(33.3)		3
Paronychia	2 (66.7)																			1 (33.3)	3
Infected pemphigus	34 (64.2)	3 (5.7)	2 (3.8)	1 (1.9)		1 (1.9)				1 (1.9)	1 (1.9)	2 (3.8)	1 (1.9)				1 (1.9)			6 (11.1)	53
Bullous pemphigoid	11 (78.6)																			3 (21.4	14
Ìnfected eczema	13 (86.6)													1 (6.7)						1 (6.7)	15

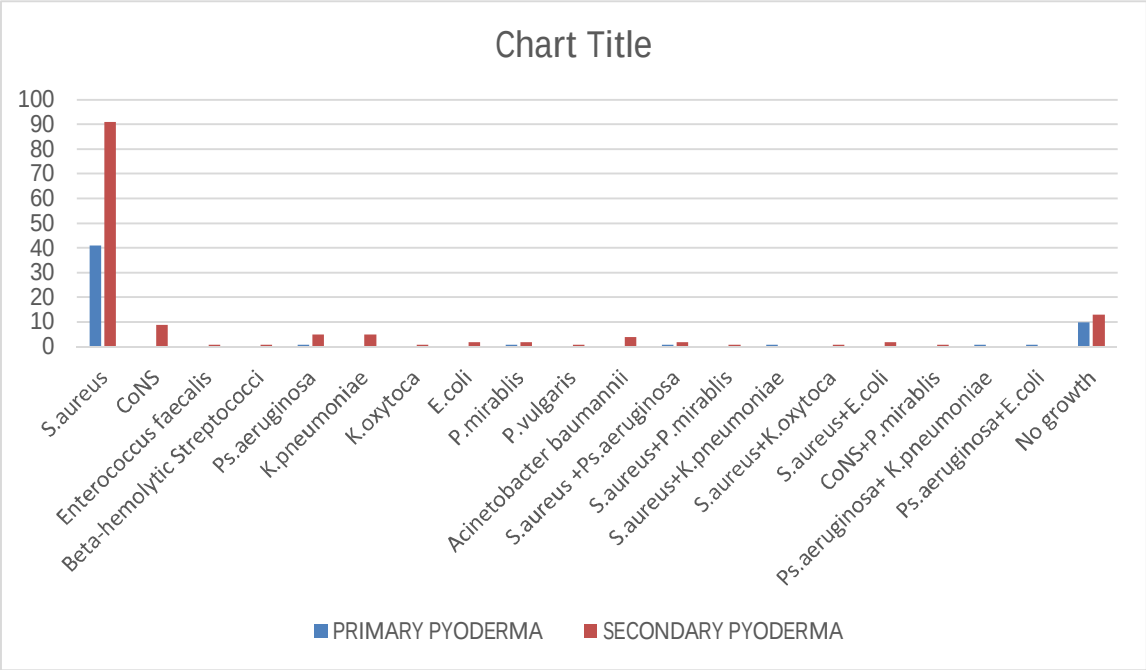
Diagnosis	***S.aureus***	***CoNS***	***K. pneumoniae***	***E.feacalis***	***Klebsiella oxytoca***	***E.coli***	***Proteus vulgaris***	***Proteus mirabilis***	***Ps aeruginosa***	***Acinetobacter baumannii***	***BHS***	***S.aureus+Ps aeruginosa***	***S.aureus +Proteus mirabilis***	***S.aureus+ E.coli***	***S.aureus+K.oxytoca***	***S.aureus+K.pneumoniae***	***CONS+P.mirabilis***	***Ps.aeruginosa+K.pneumoniae***	***Ps.aeruginosa+E.coli***	***NG***	***TOTAL***
	n (percentage)																				
Infected psoriasis	7 (58.3)	3 (25)													1 (8.3)					1 (8.3)	12
Infected dermatitis	9 (90)									1 (10)											10
Non-healing ulcer	6 (66.7)	1 (11.1)							2 (22.2)												9
Infected stasis ulcer	2 (25)		1 (12.5)		1 (12.5)			1 (12.5)	2 (25)				1 (12.5)								8
Infected trophic ulcer	3 (42.8)		2 (28.6)											1 (14.3)						1 (14.3)	7
Infected mycosis	1 (33.3)	1 (33.3)								1 (33.3)											3
Pyoderma gangrenosum	2 (40)						1 (20)	1 (20)		1 (20)											5
Infected Scabies	2 (100)																				2
Hidradenitis suppurativa	1 (50)					1(50)															2
Infected keloid	1 (50)								1(50)												2

Significant P –Value 0.020

TABLE-13 BACTERIOLOGICAL ANALYSIS IN RELATION TO TYPE OF PYODERMA

ISOLATES	***Primary pyoderma n (%)***	***Secondary pyoderma n (%)***
S.aureus	41(70.6)	91(64.1)
CoNS	-	9(6.3)
Enterococcus faecalis	-	1(0.7)
Beta-hemolytic Streptococci	-	1(0.7)
Ps.Aeruginosa	1(1.7)	5(3.5)
K.pneumoniae	-	5(3.5)
K.oxytoca	-	1(0.7)
E.coli	-	2(1.4)
P.mirabilis	1(1.7)	2(1.4)
P.vulgaris	-	1(0.7)
Acinetobacter baumannii	-	4(2.8)
S.aureus +Ps.aeruginosa	1(1.7)	2(1.4)
S. aureus+P.mirabilis	-	1(0.7)
S.aureus+K.pneumoniae	1(1.7)	-
S.aureus+K.oxytoca	-	1(0.7)
S.aureus+E.coli	-	2(1.4)
CoNS+P.mirabilis	-	1(0.7)
Ps.aeruginosa+ K.pneumoniae	1(1.7)	-
Ps.aeruginosa+E.coli	1(1.7)	-
No growth	10(17.2)	13 (9.1)
TOTAL	58(100%)	142(100%)

FIGURE 12

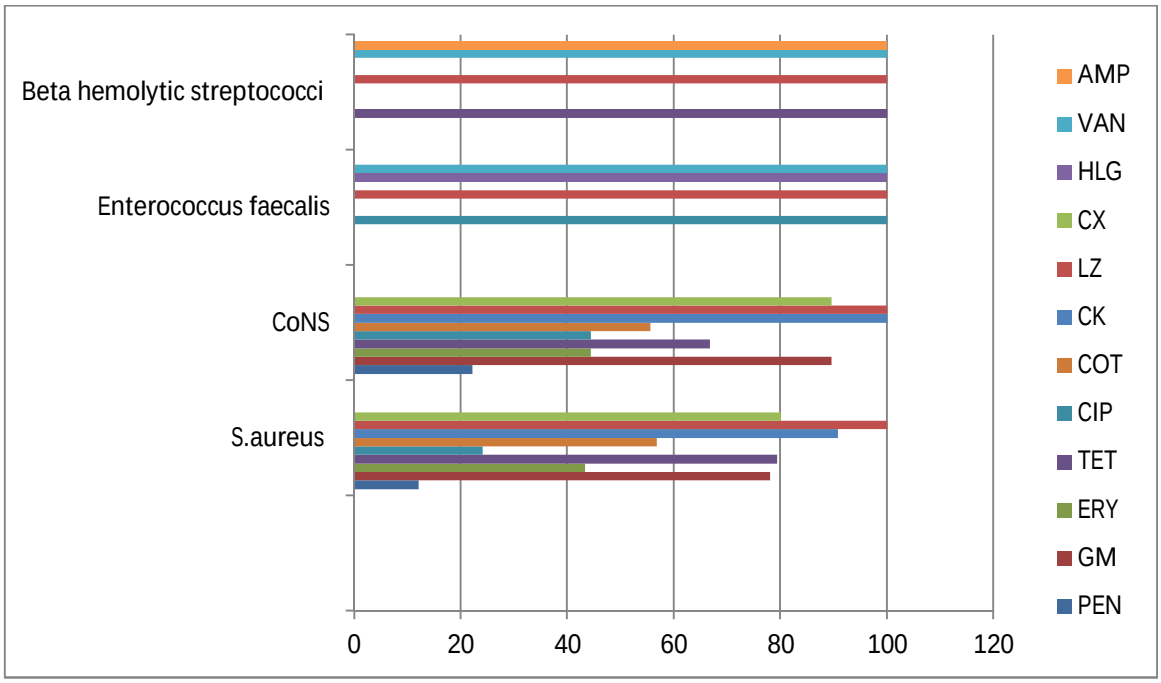


In both primary and secondary pyoderma, S. *aureus* was the commonest organism isolated, followed by *Pseudomonas aeruginosa***.**

TABLE-14 ANTIBIOTIC SUSCEPTIBITY PATTERN- GPC

ISOLATES	PEN %	GM %	ERY %	TET %	CIP %	COT %	CK %	LZ %	CX %	HLG %	VAN %	AMP %
S.aureus	12.1	78	43.3	79.4	24.1	56.7	90.8	100	80.1	-	-	-
CoNS	22.2	89.6	44.4	66.7	44.4	55.6	100	100	89.6	-	-	-
Enterococcus faecalis	0	-	0	-	100	-	0	100	-	100	100	0
Beta haemolytic streptococci	0	-	0	100	-	0	-	100	-	-	100	100

FIGURE 13



S.aureus was most sensitive to Linezolid(100%), chloramphenicol (90.8%) and tetracycline (79.4%), followed by Gentamicin (78%) and Cotrimoxazole (56.7%). It was least sensitive to Erythromycin (43.3%),ciprofloxacin And Penicillin (12.1%)Out of total 141 isolates of *S.aureus* 113 (80.1%) were sensitive to cefoxitin and 28 were resistant. Therefore percentage of MRSA isolated was 19.9%.

Among the CONS isolated, 100% were sensitive to chloramphenicol and Linezolid each. 89.6% isolates were sensitive to Gentamicin. They were least sensitive to Penicillin (22.2%) 2 out of 9 (71.4%) isolates were resistant to cefoxitin. Beta hemolytic Streptococci was most sensitive to Ampicillin, vancomycin,tetracycline and linezolid, showing 100% sensitivity each and

resistant to penicillin, erythromycin and cotrimoxazole. *Enterococcus faecalis* was most sensitive to Vancomycin (100%), linezolid, HLG and Ciprofloxacin and resistant to penicillin, Ampicillin, Erythromycin and chloramphenicol

TABLE-15 ANTIBIOTIC SUSCEPTIBITY PATTERN-ENTEROBACTERIACEAE

ISOLATES	AK %	GM %	OF %	TET	CTX %	CEC %	PT	AMP	IMP %
K.pneumoniae	87.5	87.5	87.5	62.5	37.5	100	100	-	100
K.oxytoca	100	100	50	50	50	100	100	-	100
Proteus mirabilis	83.3	66.7	83.3	83.3	50	100	100	66.7	100
Proteus vulgaris	100	0	0	100	100	100	100	0	100
E.coli	100	60	50	60	40	100	100	60	100

Klebsiellaspp were most sensitive to Imipenem (100%)and Piperacillin – tazobactam (100%), followed by Amikacin, Gentamicin and Ofloxacin. They were least sensitive to tetracycline and cefotaxime. Proteus spp were most sensitive to Imipenem (100%) and least sensitive to gentamicin (66.7%)and cefotaxime.

E. coli showed 100% sensitivity to Imipenem, Piperacillin –tazobactam and Amikacin. Least sensitive to Gentamicin, Tetracycline, ofloxacin and cefotaxime.

FIGURE 14

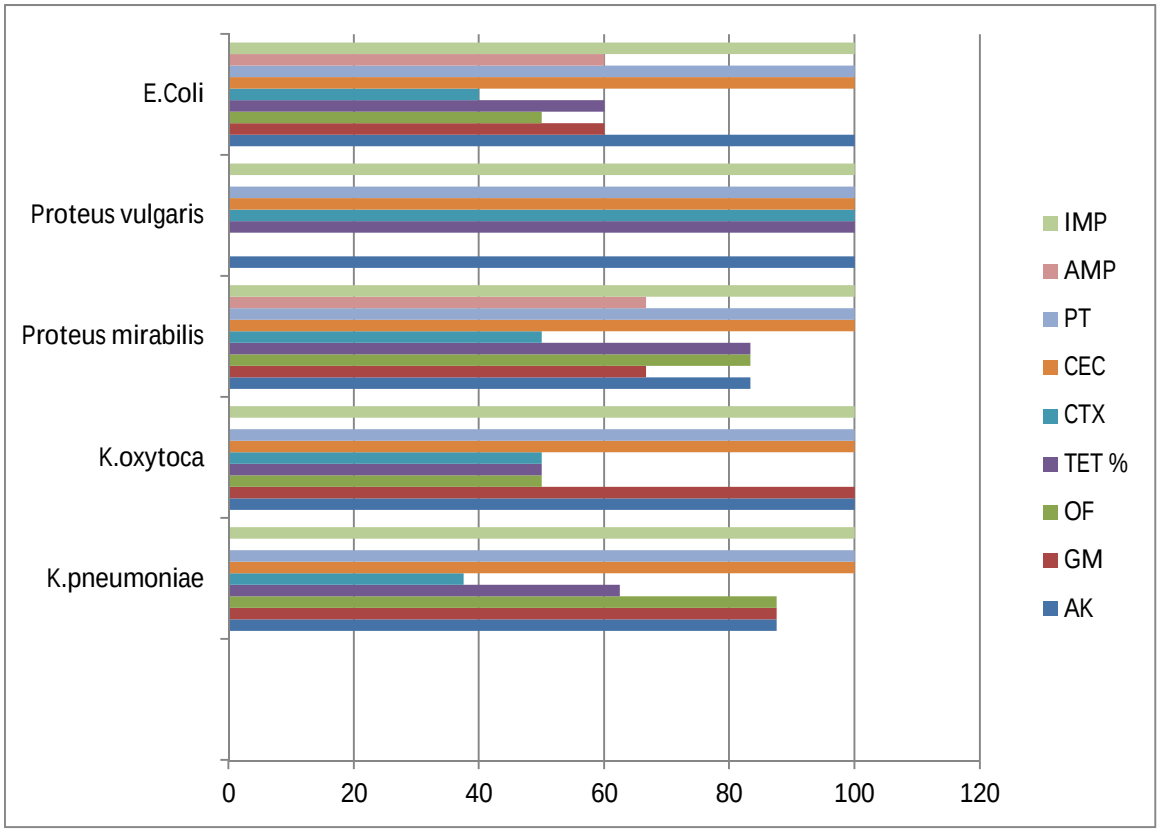

E.Coli
Proteus vulgaris
Proteus mirabilis
K.oxytoca
K.pneumoniae
0
20
40
60
80
100
120
IMP
AMP
PT
CEC
CTX
TET %
OF
GM
AK

TABLE-16 ANTIBIOTIC SUSCEPTIBITY PATTERN-NON-FERMENTERS

ISOLATE	AK	GM	CIP	COT	PT	CAZ	IMP	TET
Ps.aeruginosa	81.8%	72.7%	54.5%	37.5%	100%	60%	100%	72.7%
Acinetobacter baumannii	75%	75%	50%	75%	75%	75%	75%	50%

Ps. aeruginosa were most sensitive to Imipenem, Piperacillin-tazobactam followed by Amikacin, Gentamicin, Tetracyclines and least sensitive to Ciprofloxacin and Cotrimoxazole.

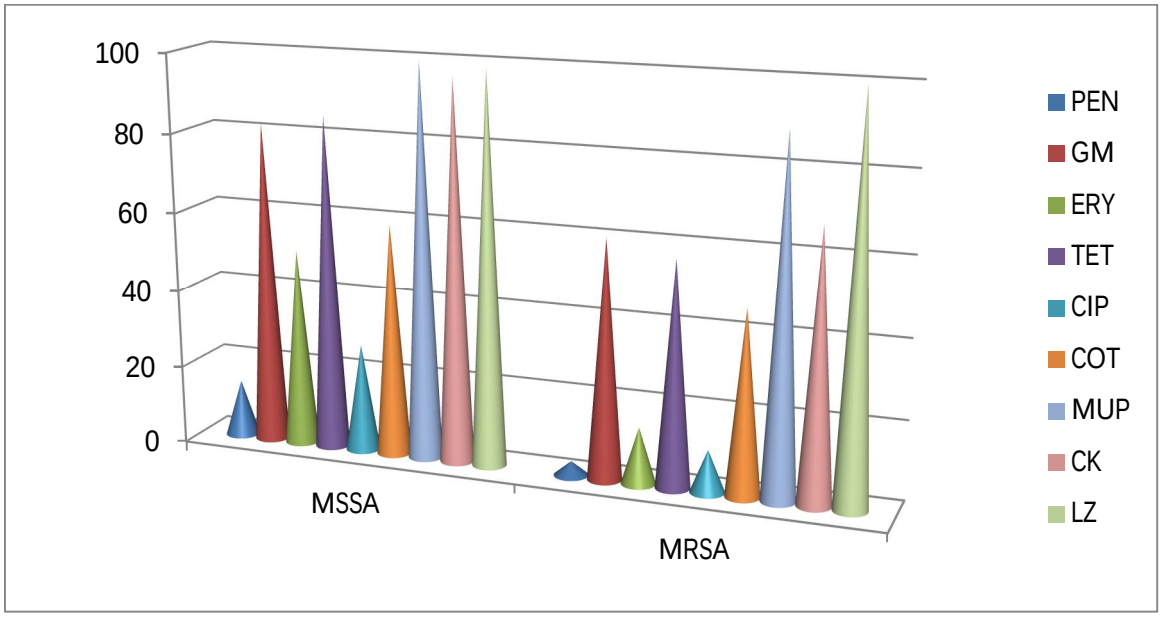
TABLE-17 ANTIBIOTIC SUSCEPTIBILITY PATTERN BETWEEN MSSA AND MRSA

Antibiotic	**MSSA (n-113)**		**MRSA(n-28)**	
	Sensitive n %	**Resistant %**	**Sensitive %**	**Resistant %**
PEN	14.3	85.7	3.6	96.4
GM	82.3	17.7	60.7	39.3
ERY	50.4	49.6	14.3	85.7
TET	85.0	15.0	57.1	42.9
CIP	27.4	72.6	10.7	89.3
COT	59.3	40.7	46.7	53.6
MUP	100%	0	89.3%	10.7%
CK	96.5	3.5	67.9	32.1
LZ	100	0	100	0

Significant P-value <0.05

Both MSSA and MRSA were most sensitive to linezolid, and least sensitive to penicillin. MSSA were more sensitive to chloramphenicol, tetracycline, gentamicin and erythromycin than MRSA.

FIGURE 15



MRSA VS MSSA

FIGURE 16

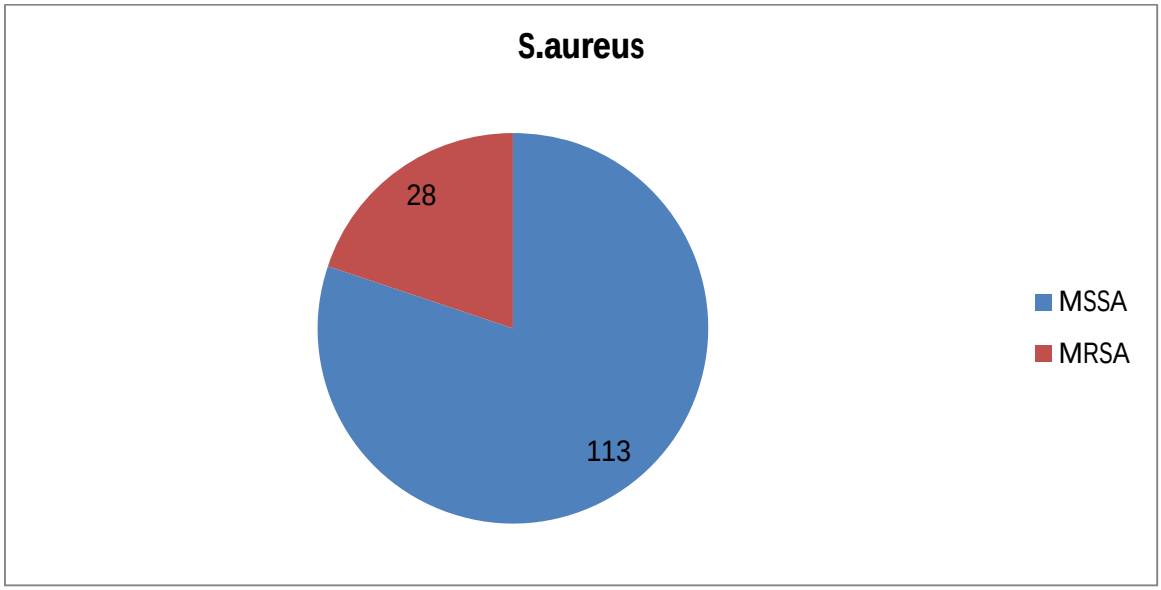
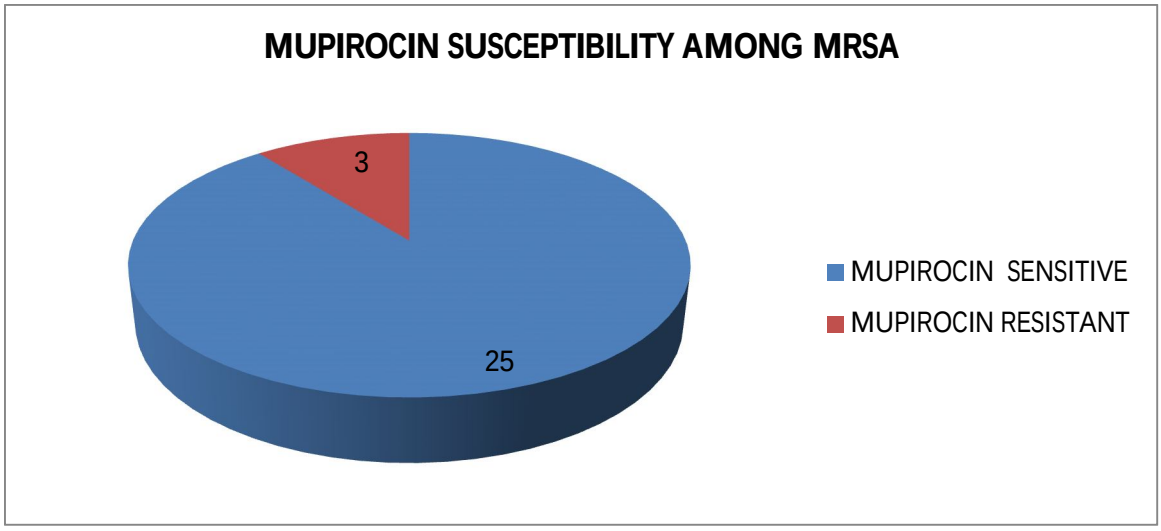


TABLE-18 MUPIROCIN SUSCEPTIBILITY AMONG MRSA ISOLATES

MRSA Isolates	Mupirocin						
	Disc Diffusion MUP(200mcg)		E-Test			PCR For mup A	
28	Sensitive	Resistant	≤1	2-256	≥256	Positive	Negative
	25(89.3%)	3(10.7%)	25	-	3	3(10.7%)	25(89.3%)

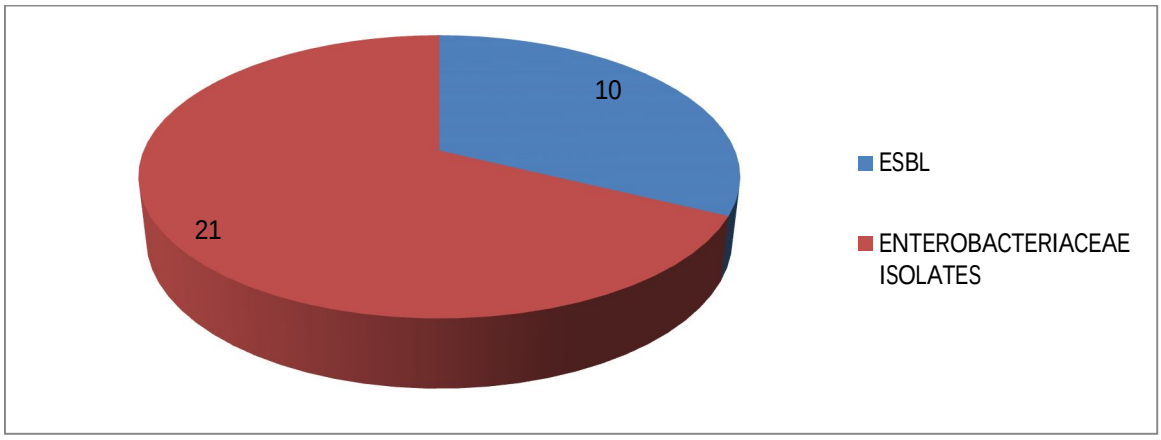
Among 28 MRSA isolates, 25 were mupirocin sensitive and 3 were resistant.

FIGURE 17



ESBL PRODUCERS AMONG ENTEROBACTERIACEAE ISOLATES

FIGURE 18



Among 21 Enterobacteriaceae isolates, 10(47.6%) were ESBL producers.

Discussion

DISCUSSION

Bacterial skin infections become one of the most common clinical problem encountered in many fields of clinical medicine[10].Pyoderma is the generic term used to describe any variant of superficial bacterial skin infection. [8] Pyoderma constitutes a major portion amongpatients attending dermatological clinics in India.

The present study comprised of 200 randomly selected cases of both primary and secondary Pyoderma, attended the Department of Dermatology, RGGGH, during the study period March2017 to February 2018, to identify the causative organism of both primary and secondary Pyodermas and also to determine their latest antibiotic susceptibility pattern.

In the present study, out of the 200 cases of Pyoderma, Secondary pyodermas constituted 71% of the cases and the remaining 29% constituted Primary pyoderma(Table-1). Thus, showing that secondary pyodermas were more common than primary pyoderma which is similar to the study conducted by Harshita et al which showed [68].13% secondary pyodermas and 31.87% of cases were primary pyodermas [96]. Malhotra SK et al which showed 80.33% cases of secondary pyodermas and 19.67% cases were primary pyoderma[10]. Sravani BVN, Kumar BS, Mavuri VVNR, et al. showed that secondary pyoderma accounted for 76% cases while 24% of cases were primary pyoderma.[94]

Few studies showed that primary pyodermas were more common than secondary pyoderma. In our study low incidence of primary pyoderma may be due to the fact that patients less than 18 years were excluded from this study and inpatients (65.5%) were more than outpatients (34.5%).

Incidence of pyoderma (table-4) was more in Males (58.5%) than females (41.5%) which correlates well with other studies.[94,95,96]

Present study showed that most of the patients belonged to the lower socioeconomic group (92%) followed by middle socioeconomic group(7.5%).only 0.5% of higher socioeconomic group presented with Pyoderma(table-8). This correlates well with many other studies.[24]

In this study, most of the cases of pyoderma i.e. 78(39%) belongs to the age group 21-40 years (Table-5). Mean age was found to be 43.43±16.03. This is similar to the study reported by Ghadage D P, Sali Y A. et al, which showed 42(36.5%) cases in the age group of20-40 years.[9]

Primary pyodermas occurred most frequently in the age group of 21-30 years(26.3%)(table-6), which is similar to the study conducted by Ashokan C et al, which showed Primary pyodermas were seen most frequently in the age group of 21-30 years with 28%(63/225 cases).[97]

Secondary pyodermas were common in the age group of 41-50 years (21.7%) followed by 21-30 years (20.3%) (table-6)in the present study.

In the present study, among the primary pyoderma, Folliculitis (27.58%) was the most common clinical type followed by Furunculosis (24.14%), Impetigo (20.68),Cellulitis (8.6%),ecthyma (8.6%), Carbuncle (5.1%)and Paronychia (5.1%). (table-2). This is similar to the study conducted by Paudel U et al showed that Folliculitis (26.7%) was the commonest followed by Furunculosis (22.7%)[95]. Although many studies have shown impetigo as the commonest lesion, however majority of our patients were adults which accounts for high frequency of folliculitis in this study.

In our study, among the secondary pyodermas, Infected pemphigus(vulgaris + foliaceus+ erythematosus) (37.3%) was the most common followed by Eczema with secondary infection(10.5%), infected bullous pemphigoid (9.8%), infected Psoriasis (8.4%),infected dermatitis(7.0%), Non-healing ulcer(6.3%), infected trophic ulcer(4.9%),infected mycoses (2.1%),pyoderma gangrenosum (2.1%),Scabies with secondary infection(1.4%), Hidradenitis Suppurativa(1.4%) and infected keloid (1.4%).(table-3).

Infected pemphigus (37.3%) was the commonest type which is similar to studies conducted by Sravani BVN, et al where Infected pemphigus constituted 44% of total cases[94] and also by Malhotra, et al. where Infected pemphigus constituted 39.34%[10].

Among 200 samples processed in our study,177 samples(88.5%) were culture positive(growth +)whereas 23(11.5%)samples were culture negative (no growth) (table-9).Among culture positive, single type of organism was grown in

165 samples(94%),more than 1 type of organism were grown in 12 samples(6%)(table-10).This correlates well with the study conducted by Rani SR et al which showed that among 135 cases which yielded growth, 97 cases showed monomicrobial infection while 38 cases showed mixed infection[98].

In bacteriological analysis of our study, a total of 189 isolates were obtained.Among which 50 isolates were obtained from 58 primary pyoderma cases and 139 isolates were obtained from 142 secondary pyoderma cases .We observed that *Staphylococcus aureus* (74.6%), was the most common organism isolated followed by *Pseudomonas aeruginosa* (5.8%), *Klebsiella*species(5.2%) , *Coagulase negative Staphylococci*(4.8%) , *Proteus species*(3.7%) , *Escherichia coli* (2.6%) , *Acinetobacter baumannii* (2.1%) *Enterococcus faecalis* and beta hemolytic *Streptococci* (0.5%) (table no.10)

In the present study among 200 cases, the most common pathogen isolated was *S.aureus* (74.6%). Similar findings have been reported by other workers. However, there was no significant difference between the isolation of S. *aureus* in primary and secondary pyoderma, the percentage being 75.4% and 68.5% respectively with a P value of 0.434, which correlates with the study of Paudel et al.[95]*Enterococcus faecalis* (0.5%) was isolated only in 1 patient in this study which is similar to the study of Paudel et al.[95]

There was low incidence of Streptococci which was isolated only in 1 patient (0.5%), which is similar to the study done by PihuSethi&JayadevBetkerur

et al[101]. But 2.3% to 9% of Beta- hemolytic Streptococci have been isolated by many others.

4.8% of Cons was isolated in the present study. However ,the percentage of isolation has been variable in other studies for example 23.61% were CoNS by Ghadage D P, Sali Y A. et al.[9]

Among the Gram-negative bacilli, *Pseudomonas aeruginosa* (5.8%) was the most common isolate in the present study, which is similar to the study conducted by Janardhan. B et al, they isolated (7%) Pseudomonas species[99].

Present study showed that *S.aureus* was the most common organism isolated which was 100% sensitive to linezolid and Chloramphenicol(90.8%) and tetracycline (79.4%), followed by Gentamicin (78%) and Cotrimoxazole (56.7%). It was least sensitive to Erythromycin (43.3%), ciprofloxacin (24.1) And Penicillin (12.1%)(table-14). This is similar to the study conducted by Prateek Kamble et al showed thatPenicillin was least sensitive (14.26%) probably due to the penicillinase producing strains. Similar findings have been shown by other workers also.[11]

Among the CONS isolated, 100% were sensitive to chloramphenicol and Linezolid each. 89.6% isolates were sensitive to Gentamicin. They were least sensitive to Penicillin (22.2%). This is similar to other studies conducted by Prateek Kamble et al showed sensitivity of aminoglycoside(80-90%)[11] and Malhotra et al (2012) also suggested high (77.7%) aminoglycoside sensitivity[10].

Beta hemolytic Streptococci was most sensitive to Ampicillin, Vancomycin, Tetracycline and Linezolid, showing 100% sensitivity each and resistant to Penicillin, Erythromycin and Cotrimoxazole.

Enterococcus faecalis was most sensitive to Vancomycin (100%), Linezolid, HLG and Ciprofloxacin and resistant to Penicillin, Ampicillin, Erythromycin and Chloramphenicol. This correlates with the study conducted by Harshita et al. showed that i.e. 100% sensitive to Linezolid and maximum 80% resistance to Ampicillin[96].

Among gram negative bacilli, Pseudomonas *aeruginosa* were most sensitive to Imipenem and Piperacillin-tazobactam (100%) followed by Amikacin (81.8), Gentamycin and Tetracyclines (72.7%)each and least sensitive to Ceftazidime (60%), Ciprofloxacin (54.5%) and Cotrimoxazole(37.5%)(table-16).This was similar to the study conducted by Ghadage D P, Sali Y A et al showed that Pseudomonas species were more sensitive to Amikacin (72%) and Carbenicillin (57%) and least sensitive to Cotrimoxazole (10%)[9].

In the present study, Klebsiellaspp were 100% sensitive to Imipenem and Piperacillin–tazobactam, followed by Amikacin, Gentamicin and Ofloxacin (87.5%)each. They were least sensitive to Tetracycline (62.5%) and Cefotaxime(37.5%)(table-15), which was similar to the study conducted by Malhotra SK et al, showed 50% resistant to Cefotaxime[10].

Proteus spp were more sensitive to Imipenem (100%) and least sensitive togentamycin (66. 7%). E. coli showed 100% sensitivity to Imipenem, Piperacillin–tazobactam and Amikacin. Least sensitive toGentamicin (60%), Tetracycline (60%),Ofloxacin (50%) and cefotaxime (40%) Acinetobacter baumannii were more sensitive to imipenem, piperacillin-tazobactam, ceftazidime, cotrimoxazole 75% each and least sensitive to ciprofloxacin and tetracycline. Amikacin, Gentamycin All the Gram-negative isolates showed 100%sensitivity to Imipenem and most of the strains were resistant to one or more antibiotics.

Out of total 141 isolates of *S. aureus* 113 (81%) were sensitive to cefoxitin and 28were resistant. Therefore, the percentage of MRSA isolated was 19.9%(table-14). 2 (22.2%)out of 9 isolates of CoNS were resistant to cefoxitin,22.2% of Marcon's were isolated.

Antibiotic resistance pattern among MRSA and methicillin sensitive *S. aureus* (MSSA) isolates were compared which showed that resistance to ciprofloxacin (89.3%), erythromycin (85.7%) and penicillin (96.4%) were significantly higher in MRSA isolates than MSSA isolates ($p<0.05$) (Table -17).

The present study shows a relatively high rate of susceptibility pattern among the clinically isolated MSSA to tetracycline (85%), Gentamycin (82.3%)and chloramphenicol (96.5%). Similar findings were observed in the study conducted by Prateek Kemble et al showed that resistance to

fluoroquinolones as well as to other antibiotics tested was significantly higher in MRSA than MSSA isolates[11].

In the present study among 28 MRSA isolates,3 (10.7%) isolates were found to be High level mupirocin resistant by disc diffusion test, E-test, and by PCR for mup A gene(table-18) and no discrepancies was observed between the disc diffusion and E-test MIC values and mup A PCR.MSSA isolates were 100% sensitive to mupirocin by disc diffusion method. Few studies had reported mupirocin resistance.

In a study conducted by Ravisekhar Gadepalli, and Benu Dhawan, they first reported the extent of mupirocin resistance in an Indian hospital, it was found that High-level mupirocin resistance was detected in 10(5%) of the 200 S. aureus isolates, among which 9 of 110 (8.2%) were MRSA isolates and 1 of 90 (1.1%) MSSA isolates[103]. Hodiwala Anahita et al. reported that 16.6% was high level resistant to mupirocin[100].

B. Madhumati et al states that out of 108 MRSA strains, 26 (24%) were mupirocin resistant Staphylococcus aureus. High-level mupirocin resistance was reported in 11%[21].

Summary

SUMMARY

- The present study was carried out on 200 randomly selected cases of pyoderma, both primary and secondary, attending dermatology outpatient and inpatient department in RGGGH, for bacteriological profile and antibiotic susceptibility pattern.
- Secondary pyoderma was more common than Primary pyoderma. Infected pemphigus was the commonest clinical type followed byfolliculitis.
- The incidence of pyoderma was found to be high in males than females, with the male to female ratio being 1.4:1.
- Most of the patients belonged to the adult age group. Maximum number of cases are in the age group 21-40 years (39%).
- Majority of the patients belongs to the lower income group.
- Among 200 samples processed 177 yielded growth whereas 23 samples showed no growth. Out of 177 cases that yielded growth 165 cases showed only one type of organism whereas 12 cases showed two types of organisms.
- *Staphylococcus aureus* (74.6%), was the most common organism isolated from both primary and secondary pyoderma followed by *Pseudomonas aeruginosa* (5.8%).
- *S. aureus* was the most common organism isolated which was 100% sensitive to linezolid followed by Chloramphenicol (90.8%), tetracycline (79.4%), Gentamicin (78%) Cotrimoxazole (56.7%)and Erythromycin

(43.3%). Most of the isolates were resistant to ciprofloxacin (75.9%) and penicillin (87.9%).

- Out of total 141isolates of *S. aureus* ,113(80.1%) were MSSA and 28(19.9%) wereMRSA. Among 28 MRSA isolates ,25(89.3%)were Mupirocin sensitive and 3 (10.7%) were high-level mupirocin resistant
- All the Gram negative isolates showed 100%sensitivity to Imipenem and most of the strains resistant to one or more antibiotics.

Conclusion

CONCLUSION

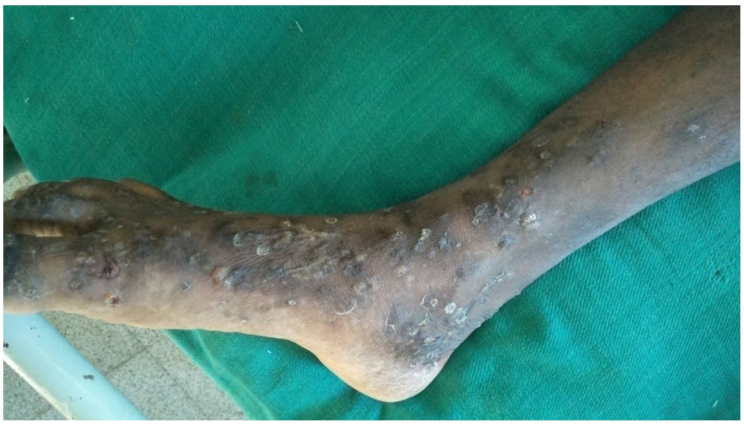
Bacterial skin infection is one of the commonest clinical problem encountered in day to day clinical practice. Among which Pyoderma or pyogenic infection of skin constitute major portion. Their management is complicated by the emergence of multidrug resistance among the commonly isolated etiological agents, thus limiting the treatment options. If not treated promptly they lead to recurrence of the disease and various other complications. With the advent of wide range of topical preparations containing broad spectrum antibiotics and chemotherapeutic agents, emergence of multidrug resistant organism become a great concern. Use of a particular antibiotic at therapeutic or sub- therapeutic levels were known to induce resistance by microorganism.

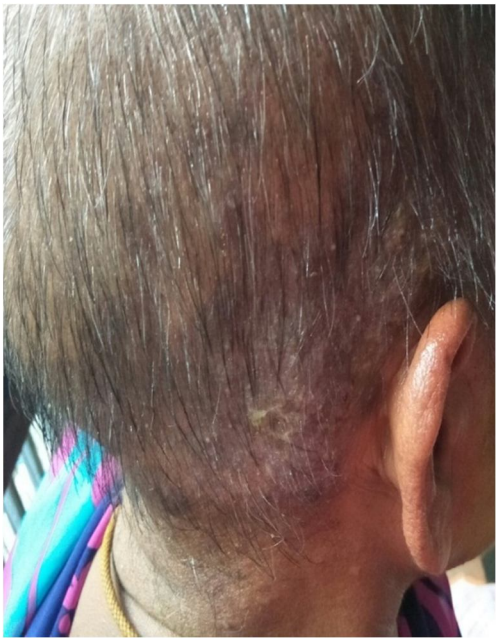
With the knowledge of common causative organism and their resistance pattern, proper antibiotic therapy can be given, thus avoiding unnecessary medication and also keep newer antibiotics in reserve for use only when necessity arises. Therefore, timely recognition, and prompt bacterial diagnosis and antibiotic susceptibility testing is very important for the management of pyoderma and also to prevent further major complications.

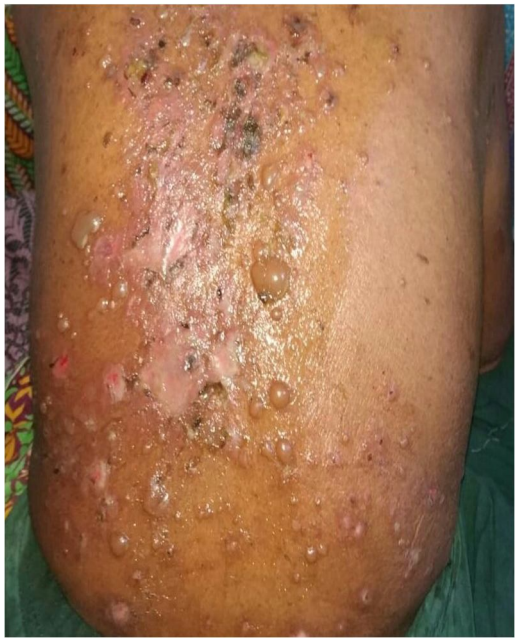
Colour Plates

COLOUR PLATE-1

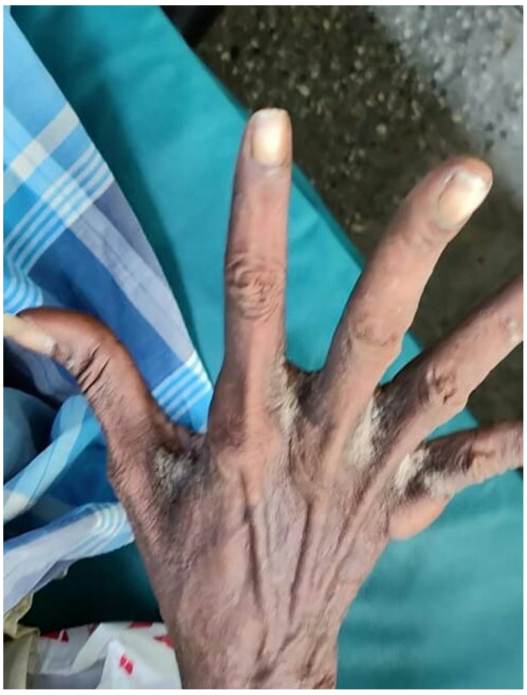
INFECTED PEMPHIGUS

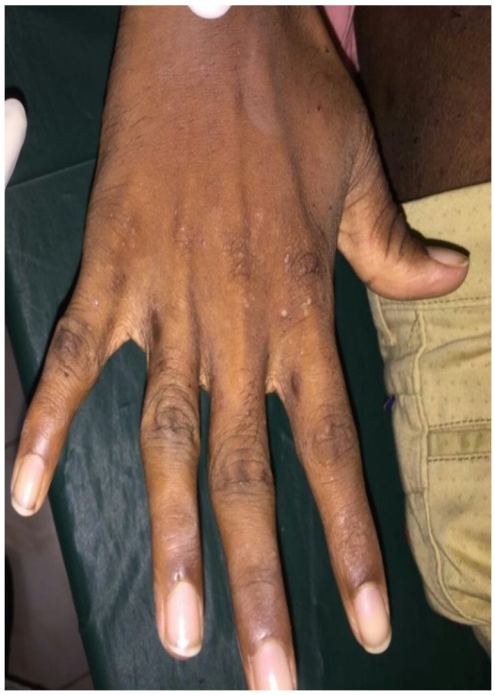




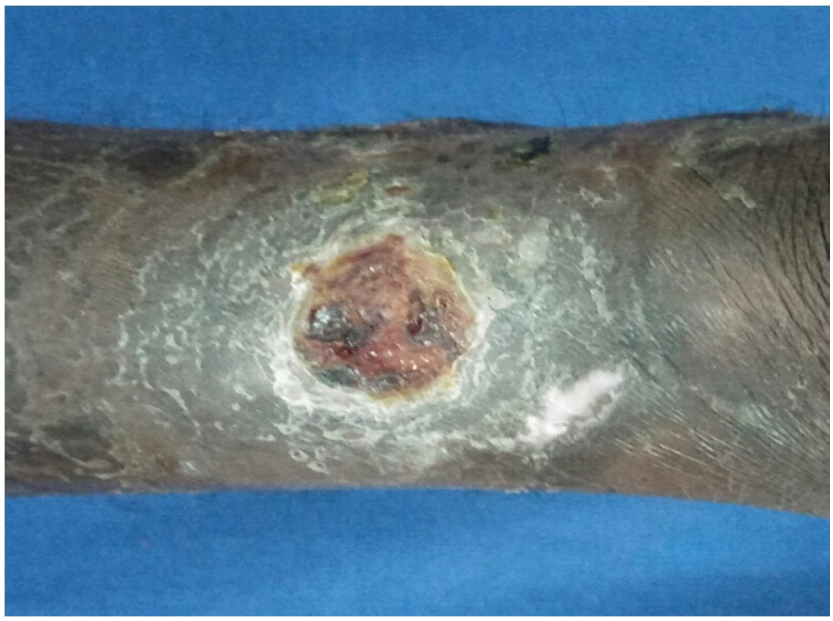


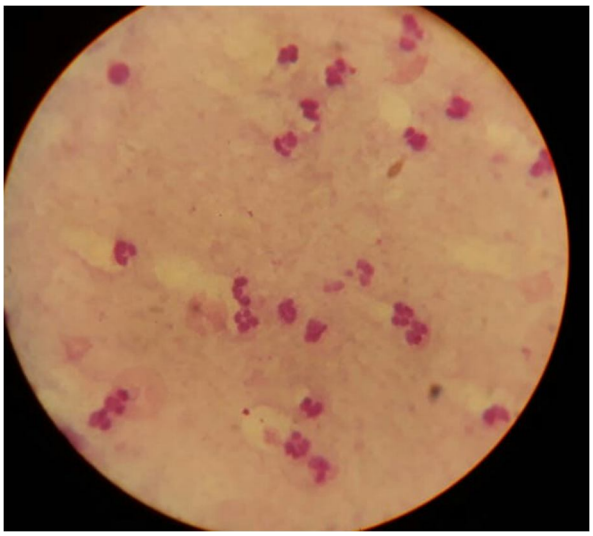
CP-2 INFECTED SCABIES



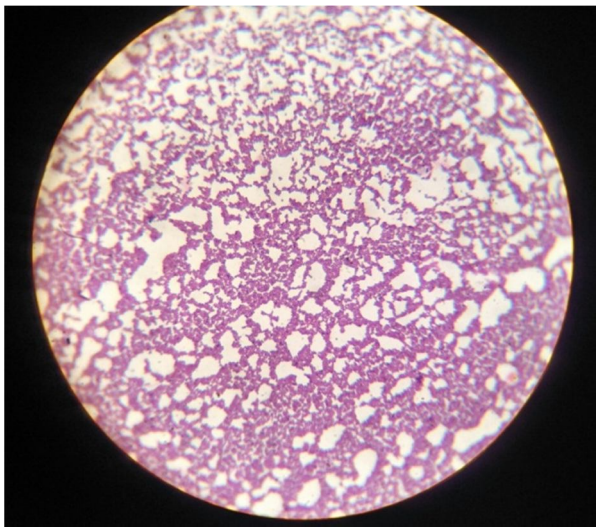


CP-3 NON-HEALING ULCER

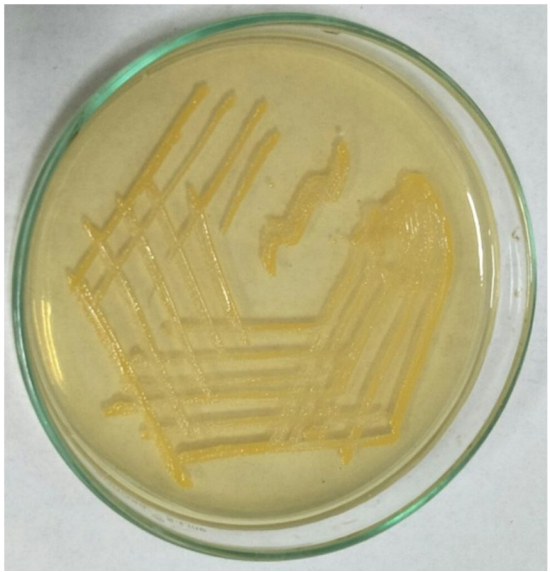




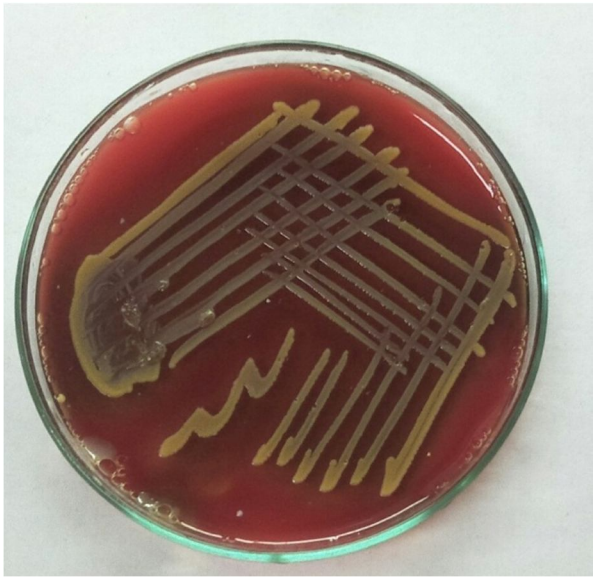
CP-4 DIRECT GRAM STAIN SHOWING PUS CELLS



CP-5 CULTURE SMEAR SHOWING GPC IN CLUSTERS

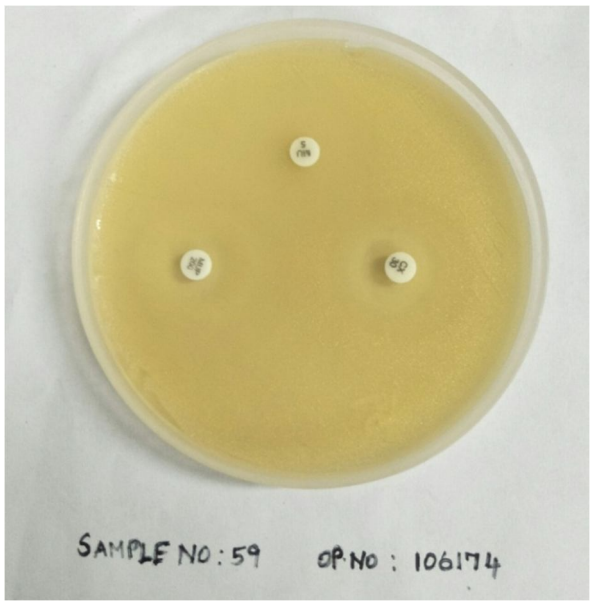


CP-6 NA SHOWING YELLOW PIGMENT COLONIES OF S.AUREUS

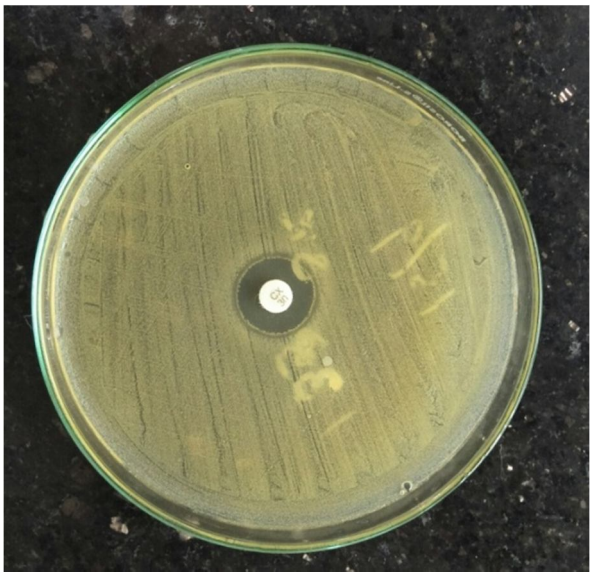


CP-7 BAP SHOWING BETAHEMOLYSIS OF S.AUREUS

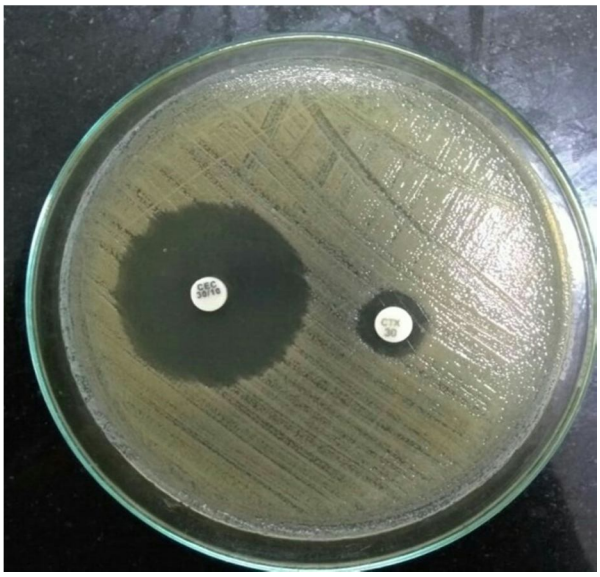
CP-8 MUPIROCIN SENSITIVE MRSA



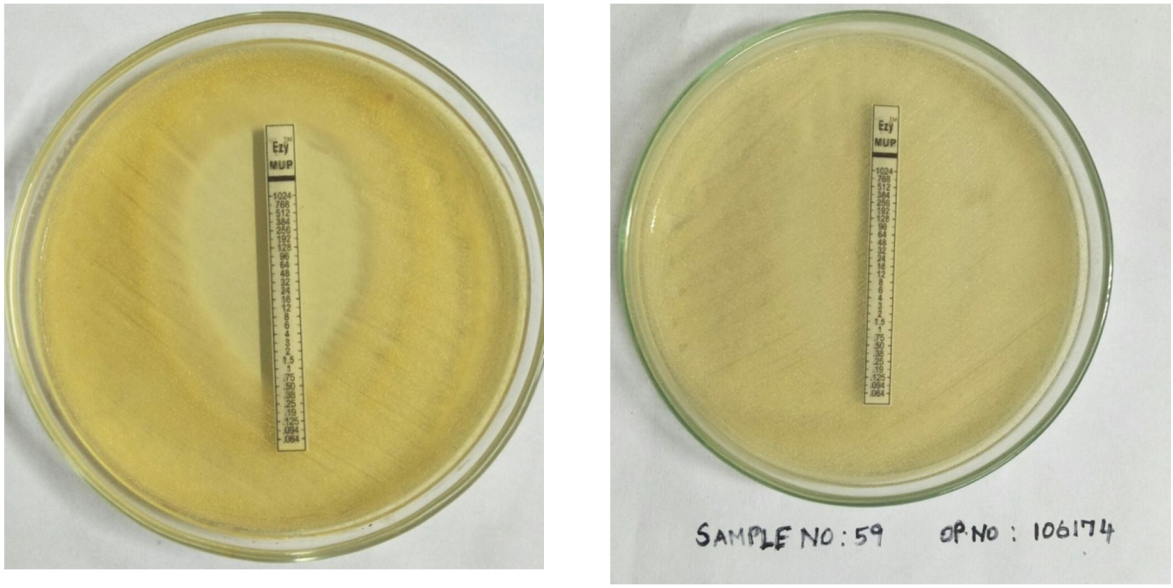
CP-9 MUPIROCIN RESISTANT MRSA



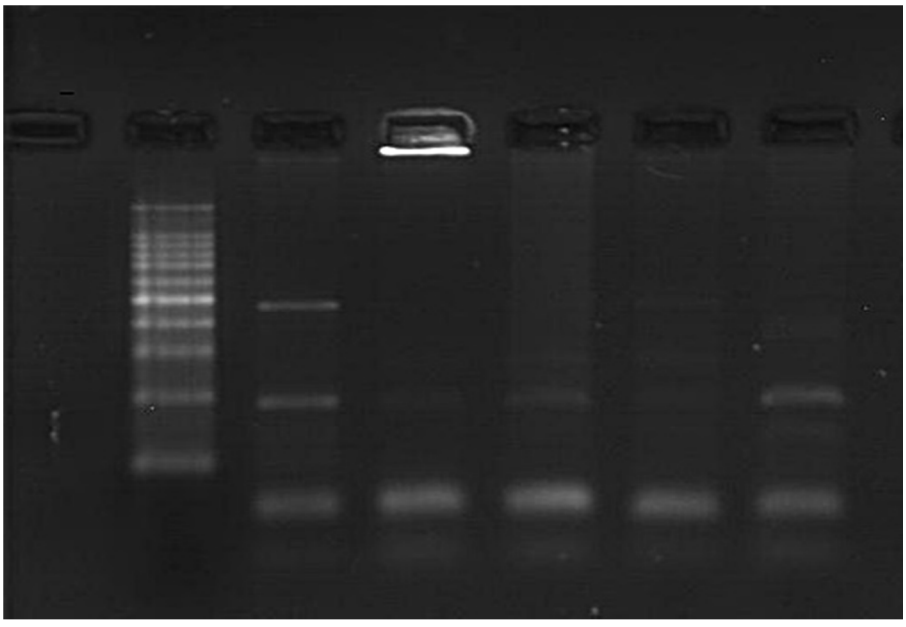
CP-10 MRSA



CP-11 ESBL



CP -12 MUPIROCIN MIC detection by E-test showing a) MIC-0.25 µg/ml b) MIC >1064µg/ml



CP- 13 Molecular identification of Mupirocin resistance mup A gene among MRSA

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Annexures

PROFORMA

- Name : IP NO:
- Age: Ward:
- Sex:
- Occupation:
- Address:

- Presenting complaints

- Personal history

- Past history

- Prior antibiotic therapy

- Clinical Diagnosis:

- Microbiological investigation:

- Direct Gram staining :

- Culture : Blood Agar-
  ChocolateAgar -
  Mac Conkey Agar-
- Organism identified/Speciation:

Antimicrobial susceptibility pattern –

PCR for mupA gene -

CONSENT FORM

STUDY TITLE :

A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA IN A TERTIARY CARE HOSPITAL

I……, hereby give consent to participate in the study conducted by Dr.S.KOKILA, Post graduate at Institute of Microbiology, Madras Medical College, Chennai and to use my personal clinical data and the result of investigations for the purpose of analysis and to study the nature of the disease, I also give consent to give my clinical Specimen for further investigations. I also learn that there is no additional risk in this study. I also give my consent for my investigator to publish the data in any forum or journal.

Signature/ Thumb impression Place Date

Of the patient/ relative

Patient Name & Address:

Signature of the Investigator:

Signature of the Guide:

INFORMATION SHEET

STUDY TITLE : A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA

IN A TERTIARY CARE HOSPITAL

INVESTIGATOR : **Dr.S.Kokila,**
I yr Post Graduate,
Institute of Microbiology,
Madras Medical College,
Chennai - 600003

GUIDE : **Dr. R.Vanaja M.D.,**
Professor of Microbiology,
Institute of Microbiology,
Madras Medical College,
Chennai - 600003

In recent times, the emergence of antibiotic resistance has significantly poses a serious threat to public health .For the successful treatment of pyodermas, various causative organisms and their sensitivity patterns in local area is essential. The present study aimed to find out the causative organisms and their antibiotic susceptibility patterns in pyodermas in the Dermatology department in a tertiary care hospital.

I am going to detect the prevalence of Mupirocin resistance among the MRSA isolates and their susceptibility in this tertiary hospital. I am going to collect two sterile swabs for this study and process them accordingly.200 patients are included in this study after getting informed consent only. This study is entirely voluntary and patient can withdraw any time from this study. Extra cost will not be incurred to the patients in this study. Any doubt regarding this study will be willingly clarified. Results of the study will be published. In case of any doubt please contact Dr.S.Kokila, Cell: 9626759625.

தகவல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு :

மூன்றாம் நிலை மருத்துவமனையில், தோல் சீழ் நோய் தொற்று காரண நுண்ணுயிரிகளின் விவரத்தினை கண்டறியும் ஆய்வு.

ஆய்வாளர் : **மரு. S. கோகிலா,**
முதலாம் ஆண்டு பட்டமேற்படிப்பு மாணவி,
நுண்ணுயிரியல் துறை,
சென்னை மருத்துவக் கல்லூரி,
சென்னை-600003.

பயோடெர்மா (Pyoderma) என்பது தோல்சீழ் தொற்று நோயாகும். இந்த மூன்றாம் நிலை மருத்துவமனையில் தோல்நோய் சிகிச்சை பிரிவில் தோல்சீழ் நோய் தொற்றால் பாதிப்படைந்த உள்நோயாளிகள் மற்றும் வெளிநோயாளிகளின் சீழ் மாதிரியை பெற்று நுண்ணுயிர் வளர்ப்பகுப்பாய்வு செய்து சரியான நுண்ணுயிர் காரணியை கண்டு அவற்றின விவரங்களை தொகுப்பு ஆய்வு செய்வதே இந்த ஆய்வின் நோக்கமாகும்.

இந்த ஆய்வில் உங்கள் முழு ஒப்புதலுடன் பெறப்படும் தோல்சீழ் மாதிரியைக் கொண்டு செய்யப்படும் பரிசோதனைகளால் உங்களுக்கு வழங்கப்படும் சிகிச்சை அல்லது இறுதியாக வழங்கப்படும் அறிக்கையில் எவ்வித பாதிப்பும் ஏற்படாது.

இந்த ஆய்வையொட்டி எந்த விதமான சந்தேகங்களுக்கும் விளக்கம் பெற பங்கேற்பாளர்களுக்கு உரிமை உள்ளது.

இந்த ஆய்வில் ஆகும் அதிகப்படியான செலவிற்கு நோயாளிகளிடமிருந்து பணம் பெற்றுக்கொள்ளப்படமாட்டாது.

இந்த ஆய்வின் முடிவுகள் இறுதியில் பிரசுரிக்கப்படும்.. இந்த ஆய்வை பற்றிய சந்தேகங்கள் முழுமையாக தங்களுக்கு விளக்கப்படும். தொடர்பு கொள்ள வேண்டியவர் மரு. S.கோகிலா , செல் : 9626759625.

ஆய்வாளர் கையொப்பம்

தேதி :

பங்கேற்பாளர் கையொப்பம் /
இடதுகை பெருவிரல் ரேகை

தேதி :

MASTER CHART

S. No.	IP No/OP No.	Wd. No./OP No.	AGE	SEX	SE status	Diagnosis	primary /secondary pyoderma	SINGLE/ TWO TYPE OF ORGANISM	organism isolated	PEN	GM	ERY	TET	CIP	COT	CK	LZ	CX	AK	HLG	VAN	AMP	OF	PT	CTX	CEC	CAZ	IMP	RESISTANCE PATTERN	HL MU	E TEST	PCR mup A
1	59896	44	34	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
2	58969	44	44	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	R	R	R	R	S	S	R											MRSA	S	S	NEGATIVE
3	59923	45	29	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
4	55144	44	60	M	LOW	CARBUNCLE	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
5	31477	45	57	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
6	29291	OP	52	M	LOW	DERMATITIS	SEC	1	S.aureus	R	S	R	S	S	S	S	S	S											MSSA	S		
7	72373	45	56	F	LOW	PEMPHIGUS VULGARIS	SEC	2	S.aureus	R	S	R	S	S	R	S	S	S											MSSA	S		
									Proteus mirabilis		S		S						S			S	S	S	S	S		S			
8	75303	44	75	M	LOW	PEMPHIGUS VULGARIS	SEC	1	CoNS	R	S	R	R	R	R	S	S	R											MRCoNS			
9	65088	44	47	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	R											MRSA	R	R	POSITIVE
10	129634	OP	35	M	LOW	ECZEMA	SEC	1	Klebsiella pneumoniae		S		S						S				S	S	S	S		S			
11	81913	45	37	F	LOW	PEMPHIGUS VULGARIS	SEC	1	Enterococcus faecalis	R		R		R		R	S			S	R										
12	82582	44	46	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
13	76222	45	35	F	LOW	PEMPHIGUS VULGARIS	SEC	2	CoNS	R	S	R	R	R	R	S	S	R											MRCoNS			
									Proteus mirabilis		R		S						R			R	S	S	R	S		S	ESBL			
14	709778	OP	29	F	LOW	FOLLICULITIS	PRI	2	S.aureus	R	S	R	S	R	S	S	S	R											MRSA	S	S	NEGATIVE
									Klebsiella pneumoniae		S		R						S				S	S	R	S		S	ESBL			
15	55517	OP	60	M	LOW	STASIS ULCER	SEC	2	S.aureus	R	R	R	R	R	R	R	S	R											MRSA	S	S	NEGATIVE
									Proteus mirabilis		S		S						S			S	S	S	S	S		S			
16	167370	OP	42	F	LOW	FOLLICULITIS	PRI		NG																							
17	120436	44	45	M	LOW	PSORIASIS VULGARIS	SEC	1	CoNS	S	S	R	S	S	S	S	S	S											MSCoNS			
18	121896	44	55	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
19	671123	OP	51	M	HIGH	FURUNCULOSIS	PRI	1	S.aureus	R	S	R	S	R	S	S	S	R											MRSA	S	S	NEGATIVE
20	12751	45	55	F	LOW	PSORIASIS VULGARIS	SEC		NG																							
21	105517	OP	40	M	LOW	ECZEMA	SEC	2	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
									E.coli		S		R						S				S	S	R	S		S	ESBL			

S. No.	IP No/OP No.	Wd. No./OP No.	AGE	SEX	SE status	Diagnosis	primary /secondary pyoderma	SINGLE/ TWO TYPE OF ORGANISM	organism isolated	PEN	GM	ERY	TET	CIP	COT	CK	LZ	CX	AK	HLG	VAN	AMP	OF	PT	CTX	CEC	CAZ	IMP	RESISTANCE PATTERN	HL MU	E TEST	PCR mup A
22	126574	44	75	M	LOW	TROPHIC ULCER	SEC	1	Klebsiella pneumoniae		S		S						S				S	S	S	S		S				
23	125036	44	55	M	MIDDLE	PEMPHIGUS VULGARIS	SEC	1	Klebsiella pneumoniae		S		R						S				S	R	R	S		S	ESBL			
24	171116	OP	51	M	LOW	STASIS ULCER	SEC	1	Klebsiella oxytoca		S		S						S				S	S	S	S		S				
25	47219	45	20	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus		R	S	S	S	R	S	S	S											MSSA	S		
26	14860	45	48	F	MIDDLE	FOLLICULITIS	PRI		NG																							
27	131426	45	60	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
28	556480	OP	18	F	LOW	IMPETIGO	PRI	1	S.aureus	R	R	R	R	R	S	S	S	S											MSSA	S		
29	936490	OP	18	M	LOW	INFECTED MYCOSES	SEC	1	CoNS	S	S	S	S	S	S	S	S	S											MSCoNS			
30	135690	44	36	M	LOW	DERMATITIS	SEC	1	S.aureus	S	S	S	S	S	R	S	S	S											MSSA	S		
31	135750	44	65	M	LOW	INFECTED MYCOSES	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
32	136838	45	55	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	S	S	S	S	R	S	S	S	S											MSSA	S		
33	128531	44	28	M	MIDDLE	HIDRADENITIS SUPPURATIVA	SEC	1	E.coli		R		S						S				S	S	R	S		S	ESBL			
34	137148	45	29	F	LOW	PYODERMA GANGRENOSUM	SEC	1	Proteus vulgaris		R		R						R				R	S	S	S		S				
35	504360	OP	47	M	LOW	STASIS ULCER	SEC	1	Proteus mirabilis		S		S						S			S	S	S	S	S		S				
36	520000	OP	18	M	LOW	PUSTULAR PSORIASIS	SEC	2	S.aureus	R	R	R	S	R	S	S	S	S											MSSA	S		
									Klebsiella oxytoca		S		R						S				R	S	R	S		S				
37	131675	44	60	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
38	29291	OP	52	M	LOW	DERMATITIS	SEC	1	S.aureus	R	S	R	R	R	R	S	S	S											MSSA	S		
39	25930	26	29	M	LOW	INFECTED WOUND	SEC	1	CoNS	R	S	S	S	S	S	S	S	S											MSCoNS			
40	118998	45	82	F	LOW	PEMPHIGUS FOLIAECEOUS	SEC		NG																							
41	111600	44	42	M	LOW	NON-HEALING ULCER	SEC	1	Pseudomonas aeruginosa		R		R	S	R				S					S			R	S				
42	109430	44	47	M	LOW	INFECTED WOUND	SEC	1	S.aureus	R	R	R	S	R	S	S	S	S											MSSA	S		
43	34280	44	60	M	LOW	PEMPHIGUS FOLIAECEOUS	SEC	1	S.aureus	R	S	R	R	R	R	S	S	S											MSSA	S		
44	131960	45	23	F	LOW	IMPETIGO	PRI	1	S.aureus	R	S	R	R	R	R	S	S	R											MRSA	S	S	NEGATIVE
45	59508	OP	27	M	LOW	DERMATITIS	SEC	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
46	365033	OP	48	F	LOW	VARICOSE ULCER	SEC	1	Klebsiella pneumoniae		S		S						S				S	S	R	S		S	ESBL			
47	144024	OP	52	M	MIDDLE	FURUNCULOSIS	PRI	1	S.aureus	S	S	S	S	R	S	S	S	S											MSSA	S		

S. No.	IP No/OP No.	Wd. No./OP No.	AGE	SEX	SE status	Diagnosis	primary /secondary pyoderma	SINGLE/ TWO TYPE OF ORGANISM	organism isolated	PEN	GM	ERY	TET	CIP	COT	CK	LZ	CX	AK	HLG	VAN	AMP	OF	PT	CTX	CEC	CAZ	IMP	RESISTANCE PATTERN	HL MU	E TEST	PCR mup A
48	48848	OP	53	F	LOW	TEN	PRI		NG																							
49	132674	44	61	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
50	132718	45	30	F	LOW	FOLLICULITIS	PRI	2	Klebsiella pneumoniae		R		S						S				R	S	R	S		S	ESBL			
									Pseudomonas aeruginosa		S		S	S	R				S					S			S	S				
51	10646	44	34	M	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
52	114528	OP	20	M	MIDDLE	ATOPIC DERMATITIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
53	11332	13	35	F	LOW	TROPHIC ULCER	SEC	2	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
									E.coli		S		S						S				S	S	S	S		S				
54	18696	44	45	M	LOW	PEMPHIGUS VULGARIS	SEC	1	Acinetobacter baumanii		R		R	R	R				R					R			R	R				
55	105231	OP	40	M	LOW	FURUNCULOSIS	PRI		NG																							
56	171500	OP	48	M	LOW	STASIS ULCER	SEC	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
57	17245	44	67	M	LOW	TEN	PRI	1	S.aureus	R	S	R	S	R	R	S	S	R											MRSA	S	S	NEGATIVE
58	68734	45	25	F	MIDDLE	INFECTED KELOID	SEC	1	Pseudomonas aeruginosa		S		S	S	S				S					S			S	S				
59	172367	OP	66	F	LOW	CELLULITIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
60	593450	44	25	M	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
61	62058	44	60	M	LOW	PEMPHIGUS FOLIAECEOUS	SEC	1	S.aureus	R	R	R	S	R	S	S	S	R											MRSA	S	S	NEGATIVE
62	105175	44	35	M	LOW	PSORIATIC ERYTHRODERMA	SEC	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
63	14239	44	39	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	S	R	R	S	S	S											MSSA	S		
64	106174	OP	18	M	LOW	IMPETIGO	PRI	1	S.aureus	R	R	R	S	S	R	S	S	R											MRSA	R	R	POSITIVE
65	14894	45	30	F	LOW	VARICOSE ULCER	SEC	1	Pseudomonas aeruginosa		S		S	S	S				S					S			S	S				
66	15298	45	30	F	LOW	ECTHYMA	PRI	1	S.aureus	R	S	R	R	R	S	S	R	S											MSSA	S		
67	14786	44	35	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	S	S	S	S	R	S	S	S	S											MSSA	S		
68	714123	45	52	F	LOW	PEMPHIGUS VULGARIS	SEC		NG																							
69	14789	44	31	M	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
70	15357	45	27	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
71	15710	44	45	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	R	R	S	R	S	R											MRSA	R	S	NEGATIVE
72	17239	44	70	M	LOW	DERMATITIS	SEC	1	S.aureus	S	S	R	S	S	S	S	S	R											MSSA	S		

S. No.	IP No/OP No.	Wd. No./OP No.	AGE	SEX	SE status	Diagnosis	primary /secondary pyoderma	SINGLE/ TWO TYPE OF ORGANISM	organism isolated	PEN	GM	ERY	TET	CIP	COT	CK	LZ	CX	AK	HLG	VAN	AMP	OF	PT	CTX	CEC	CAZ	IMP	RESISTANCE PATTERN	HL MU	E TEST	PCR mup A
73	180166	OP	30	F	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	R	S	S	S	R	R	S	R											MRSA	S	S	NEGATIVE
74	11168	45	25	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	R											MRSA	S	S	NEGATIVE
75	11237	OP	30	M	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	S	R	S	S	S											MSSA	S		
76	19538	30	18	M	LOW	CELLULITIS	PRI	1	S.aureus	R	R	S	R	R	S	S	S	R											MRSA	S	S	NEGATIVE
77	15941	OP	50	F	LOW	ECTHYMA	PRI	1	S.aureus	R	R	R	R	R	R	R	S	R											MRSA	S	S	NEGATIVE
78	19375	30	50	M	LOW	INFECTED WOUND	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
79	18325	OP	30	M	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
80	20703	44	70	M	LOW	PSORIASIS VULGARIS	SEC	1	CoNS	R	R	R	S	R	R	S	S	S											MSCoNS			
81	12550	45	25	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	S	R	S	S	S											MSSA	S		
82	12486	45	30	F	LOW	FOLLICULITIS	PRI		NG																							
83	962986	OP	29	M	LOW	DERMATITIS	SEC	1	S.aureus	R	R	R	R	R	R	R	S	S											MSSA	S		
84	45763	30	22	M	LOW	INFECTED WOUND	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
85	15358	45	27	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	S	R	R	S	S	S											MSSA	S		
86	17310	13	49	M	LOW	TROPHIC ULCER	SEC		NG																							
87	116566	OP	28	M	MIDDLE	FOLLICULITIS	PRI	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
88	106923	45	37	F	LOW	FOLLICULITIS	PRI	1	S.aureus	S	S	R	S	R	R	S	S	S											MSSA	S		
89	23694	44	34	M	LOW	FURUNCULOSIS	PRI	1	S.aureus	S	S	R	S	R	S	S	S	S											MSSA	S		
90	12430	OP	21	F	LOW	INFECTED SCABIES	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
91	23649	44	65	M	MIDDLE	FOLLICULITIS	PRI	1	S.aureus	S	S	R	R	R	S	S	S	S											MSSA	S		
92	31513	13	42	F	LOW	TROPHIC ULCER	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
93	26009	45	25	F	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
94	25095	45	25	F	MIDDLE	UNSTABLE PSORIASIS	SEC	1	S.aureus	R	R	R	S	R	S	S	S	R											MRSA	S	S	NEGATIVE
95	29012	45	30	F	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
96	30640	44	68	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
97	30613	45	50	F	LOW	PYODERMA GANGRENOSUM	SEC	1	Acinetobacter baumanii		S		S	R	S				S					S			S	S			
98	117947	45	35	F	LOW	PEMPHIGUS VULGARIS	SEC		NG																							
99	30343	45	65	F	LOW	PYODERMA GANGRENOSUM	SEC	1	Proteus mirabilis		R		R						S					S	R	S		S	ESBL			

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100	132711	OP	50	M	LOW	NON-HEALING ULCER	SEC	1	S.aureus	R	S	R	S	R	R	S	S	R											MRSA	S	S	NEGATIVE
101	63005	44	60	M	LOW	PEMPHIGUS VULGARIS	SEC	1	E.coli		R		S						S			S	S	S	S			S				
102	610258	OP	25	F	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
103	610044	OP	24	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
104	37490	44	58	M	LOW	PSORIATIC ERYTHRODERMA	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
105	32473	44	60	M	LOW	ECTHYMA	PRI	1	Pseudomonas aeruginosa		S		S	S	S				S					S			S	S				
106	90043	44	65	M	LOW	TEN	PRI	1	S.aureus	R	S	R	R	R	S	S	S	R											MRSA	S	S	NEGATIVE
107	94070	45	45	F	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
108	78628	45	68	F	LOW	PEMPHIGUS ERYTHEMATOSU	SEC	1	S.aureus	R	S	S	S	S	R	S	S	S											MSSA	S		
109	383955	OP	61	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
110	39477	44	54	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
111	70385	45	60	F	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	R	R	S	R	R	R	S	R											MRSA	S	S	NEGATIVE
112	92108	44	54	M	LOW	PSORIASIS VULGARIS	SEC	1	S.aureus	R	S	R	S	S	S	S	S	S											MSSA	S		
113	98663	45	62	F	MIDDLE	CARBUNCLE	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
114	137148	45	2	F	LOW	PYODERMA GANGRENOSUM	SEC	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
115	125485	45	70	F	LOW	BULLOUS PEMPHIGOID	SEC		NG																						
116	78364	44	69	M	LOW	ECZEMA	SEC	1	S.aureus	R	R	S	S	R	S	S	S	S											MSSA	S		
117	647110	OP	28	M	LOW	HIDRADENITIS SUPPURATIVA	SEC	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
118	67591	45	27	F	LOW	PEMPHIGUS FOLIAECEOUS	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
119	16023	OP	50	F	LOW	FOLLICULITIS	PRI	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
120	103576	OP	42	M	MIDDLE	FOLLICULITIS	PRI	1	S.aureus	R	R	R	S	R	R	S	S	R											MRSA	S	S	NEGATIVE
121	111952	OP	25	M	LOW	INFECTED SCABIES	SEC	1	S.aureus	R	S	R	S	R	R	R	S	S											MSSA	S		
122	169540	OP	66	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
123	25061	44	25	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	S	S	S	R											MRSA	S	S	NEGATIVE
124	93959	44	54	M	LOW	PEMPHIGUS VULGARIS	SEC		NG																						
125	58244	45	30	F	LOW	PUSTULAR PSORIASIS	SEC	1	S.aureus	R	S	R	S	S	S	S	S	S											MSSA	S		
126	30826	44	76	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	R	S	R	S	S	S	R											MRSA	S	S	NEGATIVE

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127	120356	45	45	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
128	120436	45	44	M	LOW	PSORIASIS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	R	S	S											MSSA	S		
129	356180	OP	54	M	LOW	FOLLICULITIS	PRI	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
130	127120	OP	48	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
131	8970	OP	37	F	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	S	R	S	S	S											MSSA	S		
132	124354	OP	54	M	LOW	DERMATITIS	SEC	1	S.aureus	S	S	S	S	S	R	S	S	S											MSSA	S		
133	37570	44	19	M	LOW	ATOPIC DERMATITIS	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
134	37557	45	30	M	LOW	PEMPHIGUS VULGARIS	SEC	1	Betahemolytic Streptococci	R	-	R	-	S	R		S				S	S								S		
135	39132	45	45	F	LOW	PEMPHIGUS VULGARIS	SEC	1	CoNS	R	S	S	S	S	S	S	S	S											MSCoNS			
136	87161	45	52	F	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	S	S	R	S	S	S	S	S	S											MSSA	S		
137	30613	45	50	F	LOW	PYODERMA GANGRENOSUM	SEC	1	S.aureus	R	R	R	R	R	R	R	S	R											MRSA	S	S	NEGATIVE
138	29012	45	31	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	R	R	R	S	S	S											MSSA	S		
139	38065	45	26	F	LOW	PEMPHIGUS VULGARIS	SEC	2	S.aureus	R	S	R	R	R	R	R	S	R											MRSA	S	S	NEGATIVE
									Pseudomonas aeruginosa		S		R	R	R				S					S			R	S				
140	39611	45	35	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	S	R	S	S	S	S											MSSA	S		
141	507344	OP	18	M	LOW	FURUNCULOSIS	SEC		NG																						
142	40050	45	50	F	LOW	PEMPHIGUS VULGARIS	SEC	2	S.aureus	R	R	R	S	R	R	S	S	S											MSSA	S		
									Pseudomonas aeruginosa		S		S	S	R				S					S			S	S				
143	45206	44	32	M	LOW	INFECTED WOUND	SEC	1	S.aureus	R	S	R	R	R	S	S	S	S											MSSA	S		
144	223874	OP	40	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	R	S	R	R	S	S	R											MRSA	S	S	NEGATIVE
145	1402	45	24	F	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	R	R	R	S	R	S	R											MRSA	S	S	NEGATIVE
146	1203671	OP	50	M	LOW	CELLULITIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
147	53208	OP	68	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	S	S	S	S	S	S	R											MRSA	S	S	NEGATIVE
148	575233	45	25	F	LOW	PEMPHIGUS VULGARIS	SEC	1	CoNS	R	S	R	R	R	R	S	S	S											MSCoNS			
149	40773	45	60	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
150	103735	44	35	M	LOW	DERMATITIS	SEC	1	Acinetobacter baumanii		S			S	S				S					S			S	S				
151	17314	45	20	F	LOW	FOLLICULITIS	PRI	1	Proteus mirabilis		S		S						S				S	S	S	S		S			

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152	37557	44	30	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
153	5136	OP	45	F	LOW	INFECTED MYCOSES	SEC	1	Acinetobacter baumanii		S		R	S	S				S					S			S	S				
154	11162	45	31	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
155	104865	OP	64	M	LOW	ECZEMA	SEC	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
156	21624	45	21	F	LOW	PEMPHIGUS VULGARIS	SEC	1	Klebsiella pneumoniae		S		R						S				S	S	R	S		S	ESBL			
157	123203	OP	46	M	LOW	CARBUNCLE	PRI	2	Pseudomonas aeruginosa		S		S	R	S				S					S			S	S				
									E.coli		S		R						S			S	S	S	R	S		S	ESBL			
158	122165	OP	53	F	LOW	FOLLICULITIS	PRI	1	S.aureus	R	R	R	S	R	R	S	S	S											MSSA	S		
159	42356	44	55	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	R	R	R	R	S	S	S											MSSA	S		
160	95373	44	40	M	LOW	PSORIASIS VULGARIS	SEC	1	CoNS	R	S	S	S	R	S	S	S	S											MSCoNS			
161	69044	44	28	M	LOW	NON-HEALING ULCER	SEC	1	S.aureus	R	S	S	S	R	R	S	S	S											MSSA	S		
162	77266	OP	62	M	LOW	TROPHIC ULCER	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
163	169540	OP	66	M	LOW	BULLOUS PEMPHIGOID	SEC	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
164	37389	OP	30	F	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	R	S	R	R	S	S	S											MSSA	S		
165	13913	44	38	M	MIDDLE	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
166	12204	OP	42	M	LOW	ECZEMA	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
167	106952	OP	63	F	LOW	ECTHYMA	PRI	1	S.aureus	R	S	R	S	S	S	S	S	S											MSSA	S		
168	1226421	OP	22	M	LOW	IMPETIGO	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
169	43591	45	34	M	LOW	TROPHIC ULCER	SEC	1	S.aureus	R	R	R	S	R	R	R	S	R											MRSA	R	R	POSITIVE
170	80627	45	41	F	LOW	PEMPHIGUS VULGARIS	SEC		NG																						
171	96048	44	62	M	LOW	BULLOUS PEMPHIGOID	SEC		NG																						
172	448888	44	46	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
173	40623	45	23	F	MIDDLE	PUSTULAR PSORIASIS	SEC	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
174	40785	45	37	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	S	S	R	S	S	S	S											MSSA	S		
175	1149198	OP	36	M	LOW	IMPETIGO	PRI		NG																						
176	160318	OP	70	M	LOW	STASIS ULCER	SEC	1	S.aureus	R	S	R	R	R	R	S	S	R											MRSA	S	S	NEGATIVE
177	121493	OP	37	M	LOW	INFECTED KELOID	SEC	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		

S. No.	IP No/OP No.	Wd. No./OP No.	AGE	SEX	SE status	Diagnosis	primary /secondary pyoderma	SINGLE/ TWO TYPE OF ORGANISM	organism isolated	PEN	GM	ERY	TET	CIP	COT	CK	LZ	CX	AK	HLG	VAN	AMP	OF	PT	CTX	CEC	CAZ	IMP	RESISTANCE PATTERN	HL MU	E TEST	PCR mup A
178	160436	OP	42	M	LOW	FURUNCULOSIS	PRI	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
179	46294	44	48	M	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
180	47698	45	40	F	MIDDLE	FURUNCULOSIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
181	1240771	OP	63	M	LOW	ECZEMA	SEC		NG																						
182	97674	OP	70	M	LOW	CELLULITIS	PRI		NG																						
183	208118	OP	47	M	LOW	NON-HEALING ULCER	SEC	1	Pseudomonas aeruginosa		R		R	R	R				R					S			R	S			
184	48215	45	42	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	S	R	S	S	S											MSSA	S		
185	122165	OP	53	M	LOW	FOLLICULITIS	PRI	1	S.aureus	R	R	R	S	R	R	S	S	S											MSSA	S		
186	4801	OP	83	F	LOW	CELLULITIS	PRI		NG																						
187	191118	OP	75	M	LOW	ECTHYMA	PRI	2	S.aureus	R	S	R	S	S	S	R	S	S											MSSA	S		
									Pseudomonas aeruginosa		R		R	R					R					S			S	S			
188	129340	OP	19	F	LOW	IMPETIGO	PRI	1	S.aureus	R	S	R	R	R	R	S	S	S											MSSA	S		
189	58968	44	70	M	LOW	BULLOUS PEMPHIGOID	SEC		NG																						
190	41629	45	39	F	LOW	IMPETIGO	PRI		NG																						
191	54924	44	56	M	MIDDLE	ECZEMA	SEC	1	S.aureus	S	S	S	S	R	R	S	S	S											MSSA	S		
192	143721	44	67	M	LOW	STASIS ULCER	SEC	1	Pseudomonas aeruginosa		S		S	R	R				S					S			R	S			
193	54574	45	38	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	R	R	R	R	R	S	S	S											MSSA	S		
194	54726	13	57	F	LOW	TROPHIC ULCER	SEC	1	Klebsiella pneumoniae		S		S						S				S	S	S	S		S			
195	106930	OP	18	F	LOW	FOLLICULITIS	PRI		NG																						
196	26765	44	18	M	LOW	PEMPHIGUS VULGARIS	SEC		NG																						
197	1271154	OP	50	M	LOW	ECZEMA	SEC	1	S.aureus	S	S	S	S	S	S	S	S	S											MSSA	S		
198	55843	45	31	F	LOW	FOLLICULITIS	PRI	1	S.aureus	R	S	S	S	R	S	S	S	S											MSSA	S		
199	55489	45	19	F	LOW	FURUNCULOSIS	PRI	1	S.aureus	R	S	S	S	S	S	S	S	S											MSSA	S		
200	40783	45	39	F	LOW	PEMPHIGUS VULGARIS	SEC	1	S.aureus	R	S	S	S	S	R	S	S	S											MSSA	S		

LEGENDS FOR MASTER CHART

PEN	-	PENICILLIN
GM	-	GENTAMICIN
ERY	-	ERYTHROMICIN
TET	-	TETRACYCLINE
CIP	-	CIPROFLOXACIN
COT	-	COTRIMOXAZOLE
CK	-	CHLORAMPHENICOL
LZ	-	LINEZOLID
CX	-	CEFOXITIN
AK	-	AMIKACIN
VAN	-	VANCOMYCIN
HLG	-	HIGH-LEVEL GENTAMICIN
AMP	-	AMPICILLIN
OF	-	OFLOXACIN
PT	-	PIPERACILLIN-TAZOBACTAM
CTX	-	CEFOTAXIME
CEC	-	CEFOTAXIME-CLAVULANIC ACID
CAZ	-	CEFTAZIDIME
IMP	-	IMIPENEM
R	-	RESISTANT
S	-	SENSITIVE
M	-	MALE
F	-	FEMALE
PRI	-	PRIMARY
SEC	-	SECONDARY
MRSA	-	METHICILLIN RESISTANT STAPHYLOCOCCUS AUREUS
MSSA	-	METHICILLIN SENSITIVE STAPHYLOCOCCUS AUREUS
ESBL	-	EXTENDEDED SPECTRUM BETALACTAMASE
MRCoNS	-	METHICILLIN RESISTANT COAGULASE-NEGATIVE STAPHYLOCOCCUS AUREUS
MSCoNS	-	METHICILLIN SENSITIVE COAGULASE-NEGATIVE STAPHYLOCOCCUS AUREUS

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr.S.Kokila
I Year PG in MD Microbiology
Institute of Microbiology
Madras Medical College
Chennai 600 003

Dear Dr.S.Kokila,

The Institutional Ethics Committee has considered your request and approved your study titled **"A STUDY ON BACTERIOLOGICAL PROFILE OF PYODERMA IN A TERTIARY CARE HOSPITAL" - NO.09032017(I)**

The following members of Ethics Committee were present in the meeting hold on **02.03.2017** conducted at Madras Medical College, Chennai 3

1.Dr.C.Rajendran, MD.,	:Chairperson
2 Dr. K.Narayanasamy,MD,DM.,Dean(FAC), MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	: Member Secretary
4.Prof.S.Suresh, MS, Prof. of Surgery,MMC,Ch-3	: Member
5.Prof.Baby Vasumathi,MD.,Director, Inst. of O & G	: Member
6.Prof.K.Ramadevi,MD.,Director,Inst.of Bio-Che,MMC,Ch-3	: Member
7.Prof.R.Padmavathy, MD, Director,Inst.of Pathology,MMC,Ch-3	: Member
8.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
9.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
10.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE
CHENNAI-600 003