"A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES"

BY

DR. U. JAGADISH, M.B.B.S

DISSERTATION SUBMITTED TO SRI DEVARAJ URS ACADEMY OF

HIGHER EDUCATION AND RESEARCH, KOLAR, KARNATAKA

In partial fulfilment of the requirements for the degree of

MASTER OF SURGERY
IN
ORTHOPAEDICS

Under the Guidance of
Dr. ARUN H.S
MBBS, MS ORTHOPAEDICS
PROFESSOR & HOD
And
Dr. ARVIND NATARAJAN
PROFESSOR & HOD
DEPARTMENT OF MICROBIOLOGY.

DEPARTMENT OF ORTHOPAEDICS
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA, KOLAR-563101

June 2023

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND

RESEARCH, TAMAKA, KOLAR, KARNATAKA

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. ARUN HS**, Professor and Head of Department, Department of Orthopaedics, **Dr. ARVIND NATARAJAN**, Professor and Head of Department, Department of Microbiology, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of University regulation for the award **"M.S.DEGREE IN ORTHOPAEDICS"**, the examination to be held in May/June 2023 by SDUAHER. This has not been submitted by me previously for the award of any degree or diploma from the university or any other university.

Date :

Place:

Dr. JAGADISH U
Postgraduate
Department of Orthopaedics
Sri Devaraj Urs Medical College
Tamaka, Kolar.

Sri Devaraj Urs Academy of Higher Education and Research Centre

(Deemed University), Tamaka, Kolar.

CERTIFICATE BY THE GUIDE

This is to certify that the dissertation entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities"** is a bonafide research work done by **Dr. JAGADISH U**, under my direct guidance and supervision at Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of the requirement for the degree of "M.S. IN ORTHOPAEDICS"

Date:

Place:

Dr. ARUN HS
Professor and Head of
Department of Orthopaedics
Sri Devaraj Urs Medical College
Tamaka, Kolar – 563101

Sri Devaraj Urs Academy of Higher Education and Research Centre

(Deemed University), Tamaka, Kolar.

CERTIFICATE BY THE CO-GUIDE

This is to certify that the dissertation entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities"** is a bonafide research work done by **Dr. JAGADISH U**, under my direct guidance and supervision at Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of the requirement for the degree of "M.S. IN ORTHOPAEDICS"

Date:

Place:

Dr. ARVIND NATARAJAN,
Professor and Head of Department,
Department of Microbiology,
Sri Devaraj Urs Medical College
Tamaka, Kolar – 563101

Sri Devaraj Urs Academy of Higher Education and Research Centre

(Deemed University), Tamaka, Kolar.

CERTIFICATE BY THE HEAD OF DEPARTMENT

This is to certify that the dissertation entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities**" is a bonafide research work done by **Dr. JAGADISH U,** under direct guidance and supervision of **Dr. ARUN HS,** Professor and Head of Department, Department of Orthopaedics, **Dr. ARVIND NATARAJAN**, Professor and Head of Department, Department of Microbiology, at Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of the requirement for the degree of "M.S. IN ORTHOPAEDICS".

Date:

Place:

Dr. ARUN.H. S

Professor & HOD

Department of Orthopaedics

Sri Devaraj Urs Medical College

Tamaka, Kolar

Sri Devaraj Urs Academy of Higher Education and Research Centre (Deemed University), Tamaka, Kolar.

ENDORSEMENT BY THE HEAD OF THE DEPARTMENT OF ORTHOPAEDICS & PRINCIPAL

This is to certify that the dissertation entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities "** is a bonafide research work done by **Dr. JAGADISH U**, under the direct guidance and supervision of **Dr. ARUN HS,** Professor and Head of Department, Department of Orthopaedics, **Dr. ARVIND NATARAJAN,** Professor and Head of Department, Department of Microbiology, Sri Devaraj Urs Medical College, Kolar, in partial fulfilment of university regulation for the award "M.S. DEGREE IN ORTHOPAEDICS".

Signature of the Head of Department

Dr. ARUN H S

Professor

Department of Orthopaedics

Sri Devaraj Urs Medical College

Tamaka, Kolar – 563101

Date:
Place: Kolar

Signature of the Principal

Dr. SREERAMULU P N

Principal

Sri Devaraj Urs Medical College

Tamaka, Kolar – 563101

Date:
Place: Kolar

Sri Devaraj Urs Academy of Higher Education and Research Centre

(Deemed University) Tamaka, Kolar

ETHICAL COMMITTEE CERTIFICATE

This is to certify that the Ethical Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved **Dr. JAGADISH U**, student in the Department of Orthopaedics at Sri Devaraj Urs Medical College, Tamaka, Kolar to take up the dissertation work entitled "**A Prospective Comparative Study of Pre-Debridement and Post-Debridement Culture in Open Fractures of the Extremities"** to be submitted to the Sri Devaraj Urs Academy of Higher Education and Research Centre, Tamaka, Kolar**.**

Signature of the Member Secretary

Ethical Committee

Sri Devaraj Urs Medical College

Tamaka, Kolar – 563101

Date:

Place: Kolar

Sri Devaraj Urs Academy of Higher Education and Research Centre

(Deemed University) Tamaka, Kolar

COPYRIGHT DECLARATION BY THE CANDIDATE

I hereby declare that Sri Devaraj Urs Academy of Higher Education and Research, Kolar, Karnataka shall have the rights to preserve, use and disseminate this dissertation in print or electronic format for academic / research purpose.

Date: Signature of the Candidate

Place: Kolar **Dr. JAGADISH U**

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH

SRI DEVARAJ URS MEDICAL COLLEGE

Tamaka, Kolar

INSTITUTIONAL ETHICS COMMITTEE

Members

1. **Dr. D.E.Gangadhar Rao, (Chairman)** Prof. & HOD of Zoology, Govt. Women's College, Kolar,
2. **Dr. Sujatha.M.P, (Member Secretary),** Assoc. Prof. of Anesthesia, SDUMC,
3. Mr. Gopinath Paper Reporter, Samyukth Karnataka
4. Mr. G. K. Varada Reddy Advocate, Kolar
5. Mr. Nagesh Sharma Priest, Sanskrit Scholar and School Teacher
6. Dr. Hariprasad, Assoc. Prof Department of Orthopedics, SDUMC
7. Dr. Mahendra.M , Asst. Prof. of Community Medicine, SDUMC
8. Dr. Harish Asst. Prof. of Pharmacology, SDUMC
9. Dr. Vinay Kulkarni Lecturer, Dept. of Anatomy, SDUMC
10. Dr. Ruth Sneha Chandrakumar Asst. Prof. of Psychiatry, SDUMC
11. Dr. Shiva Kumar C S Asst. Prof. Dept. of Clinical Nutrition and Diabetics, SDUMC
12. Dr. Munilakshmi U Asst. Prof. of Biochemistry, SDUMC

No. SDUMC/KLR/IEC/633/2020-21 **Date: 24-12-2020**

PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"A prospective comparative study of pre-Debridement and post - Debridement culture in open fractures of the extremities"** being investigated by **DR. U.JAGADISH**, Dr. Arun H S & Dr. Arvind Natarajan1 in the Departments of Orthopedics & Microbiology1 at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study**.

Sujatha MP

Member Secretary

Member Secretary
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar.

Chairman

CHAIRMAN
Institutional Ethics Committee
Sri Devaraj Urs Medical College
Tamaka, Kolar

PLAG CERTIFICATE

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH
Tamaka, Kolar 563103

Certificate of Plagiarism Check

Title of the Thesis/Dissertation	A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES
Name of the Student	DR. U.JAGADISH
Registration Number	20OR1071
Name of the Supervisor / Guide	DR. ARUN HS
Department	ORTHOPAEDICS
Acceptable Maximum Limit (%) of Similarity (PG Dissertation /Ph.D. Thesis)	10%
Similarity	9%
Software used	Turnitin
Paper ID	1990645905
Submission Date	10-01-2023

Signature of Student

Signature of Guide/Supervisor

Professor & HOD
Dept. of Orthopaedics
Sri Devaraj Urs Medical College
Tamaka, Kolar-563103.

HOD Signature

PROFESSOR & HOD
DEPARTMENT OF MICROBIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE
TAMAKA-KOLAR, KARNATAKA-563103

University Librarian

University Library
Learning Resource Centre
SDUAHER, Tamaka
KOLAR-563103

Coordinator UG and PG Program

Co-Ordinator,
UG&PG Program ,Faculty of Medicine,
Sri Devaraj Urs Medical College ,
Tamaka, Kolar- 563103

turnitin

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	Jagadish U
Assignment title:	PG dissertation 2023
Submission title:	A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT...
File name:	jagadish_thesis_edit_main_with_micro_notes.docx
File size:	2.46M
Page count:	85
Word count:	14,001
Character count:	79,177
Submission date:	10-Jan-2023 02:10PM (UTC+0530)
Submission ID:	1990645905

"A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES"

DR. ARVIND .N
Co-GUIDE
PROFESSOR & HOD
DEPARTMENT OF MICROBIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE

DR. ARUN H.S
PROFESSOR AND HOD
DEPT. OF ORTHOPAEDICS
KMC NO : 46362

Turnitin Originality Report

Processed on: 10-Jan-2023 14:18 IST
ID: 1990645905
Word Count: 14001
Submitted: 1

A PROSPECTIVE COMPARATIVE STUDY OF P... DEBRID... By Jagadish U

Similarity Index
9%

Similarity by Source
Internet Sources: 7%
Publications: 4%
Student Papers: 4%

include quoted | include bibliography | excluding matches < 10 words | mode: quickview (classic) report | print | refresh | download

<1% match (Internet from 13-Dec-2020)
https://microbenotes.com/bacterial-growth-and-factors-affecting-growth-of-bacteria/

<1% match (Internet from 13-Aug-2020)
https://www.slideshare.net/DineshJain4/staining-of-microorganisms

<1% match (student papers from 18-Jun-2018)
Submitted to MAHSA University on 2018-06-18

<1% match (student papers from 18-Jun-2018)
Submitted to MAHSA University on 2018-06-18

<1% match (student papers from 09-Sep-2017)
Submitted to MAHSA University on 2017-09-09

<1% match (student papers from 10-Aug-2020)
Submitted to MAHSA University on 2020-08-10

<1% match (Internet from 08-Dec-2020)
https://openventio.org/erythema-annulare-centrifugum-deep-type-a-rare-case-report/

<1% match (Internet from 12-Dec-2022)
https://tett-mindjart.com/doi/10yrcs432j03z.1177/0954411914556137

<1% match (student papers from 02-Nov-2021)
Submitted to Roberts Wesleyan College on 2021-11-02

<1% match (Internet from 28-Feb-2019)
http://upendrats.blogspot.com

<1% match (Internet from 05-Apr-2019)
http://lilymarietodd.com

<1% match (Internet from 05-Mar-2021)
https://archive.org/details/pee?and%5B%5D=firstTitle%3A3&sort=titleSorter

<1% match (Internet from 08-Oct-2015)
http://www.ncbi.nlm.nih.gov

<1% match (Jonna Malathi, Venigalla Sunita. "OBSTETRIC OUTCOME IN WOMEN WITH PRELABOUR RUPTURE OF MEMBRANES AT TERM PREGNANCY", Journal of Evolution of Medical and Dental Sciences, 2015)
Jonna Malathi, Venigalla Sunita. "OBSTETRIC OUTCOME IN WOMEN WITH PRELABOUR RUPTURE OF MEMBRANES AT TERM PREGNANCY", Journal of Evolution of Medical and Dental Sciences, 2015

<1% match (Internet from 25-Nov-2022)
https://medium.com/@bscmicronotes/useful-and-point-to-point-notes-on-prokaryotes-cell-structure-and-function-8b9f17a6f200

<1% match (student papers from 09-Jan-2023)
Submitted to Sri Devraj Urs Academy of Higher Education and Research, Kolar on 2023-01-09

<1% match (student papers from 30-Mar-2012)
Submitted to University of Sunderland on 2012-03-30

<1% match (student papers from 24-Jun-2022)
Submitted to University of Sunderland on 2022-06-24

<1% match (Maroti R. Koichade, Abhishek P. Bhalotia, Milind V. Ingle. "Orange city tapping nail: An innovative implant for humeral diaphyseal fractures", Journal of Clinical Orthopaedics and Trauma, 2018)
Maroti R. Koichade, Abhishek P. Bhalotia, Milind V. Ingle. "Orange city tapping nail: An innovative implant for humeral diaphyseal fractures", Journal of Clinical Orthopaedics and Trauma, 2018

<1% match (student papers from 29-Jul-2022)
Submitted to University of New England on 2022-07-29

<1% match (Paul H. Kim, Seth S. Leopold. "Gustilo-Anderson Classification", Clinical Orthopaedics & Related Research, 2012)
Paul H. Kim, Seth S. Leopold. "Gustilo-Anderson Classification", Clinical Orthopaedics & Related Research, 2012

<1% match (Internet from 22-Jul-2022)
https://jnsbm.com/jnsbmsite/wp-content/uploads/2021/07/JNatScBiolMed-6-1s-63-1.pdf

<1% match (S. Rajasekaran, S. R. Sabapathy, J. Dheenadhayalan, S. R. Sundararajan, H. Venkatramani, A. Devendra, P. Ramesh, K. P. Srikanth. "Ganga hospital open injury score in management of open injuries", European Journal of Trauma and Emergency Surgery, 2014)

DEPARTMENT OF MICROBIOLOGY, SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR, KARNATAKA-563103

DR. ARUN H.S PROFESSOR AND HOD DEPT. OF ORTHOPAEDICS KMC NO : 48362

University Library Learning Resource Centre SDUAHER, Tamaka KOLAR-563103

U. Jagadish

ACKNOWLEDGEMENT

First and foremost, I express my profound gratitude to my beloved parents **SRI. UDAYAKUMAR P** and **SMT. SUMATHI B**, my sister **Dr. PADMAVATHY U** for giving me continuous encouragement, unfailing support and unconditional love throughout my life.

I would like to acknowledge all those who have supported me, not only to complete my dissertation, but throughout my Postgraduation course.

I want to express my heartful indebtedness and deep sense of gratitude to my mentor and guide **DR. ARUN HS**, Professor and Head of the Department, Department of Orthopaedics, for being very helpful throughout the study and offered his invaluable guidance and support to fully understand and complete this study. Through his vast professional knowledge and expertise, he ensured that I understand everything before I apply the information in my study. Without his constant supervision and advice, completion of this dissertation would have been impossible.

I am extremely thankful to **Dr. NAGAKUMAR JS**, Professor of Orthopaedics, for encouraging me to the highest peak, paying close and continuous attention towards met to finish all tasks and providing his kind support, valuable suggestions, immense patience and great care. His sense of punctuality, strict adherence to academic schedule, humility and knowledge have been highly inspirational for the whole of my postgraduation period.

I wish to express my heartful sense of gratitude to my mentor **Dr. PRABHU E**, Professor, Department of Orthopaedics for being helpful throughout the study. He had offered his invaluable guidance and moral support during my entire post-graduate course which enabled me to complete my work.

I wish to express my heartful sense of gratitude to my mentor **Dr. ARVIND NATARAJAN**, Professor, Department of Microbiology for being helpful throughout the study. He had offered his invaluable guidance and moral support during my entire post-graduate course which enabled me to complete my work.

It gives me immense pleasure to extend my sincere thanks to Professor **Dr. HARIPRASAD S** and Associate Professor **Dr. SAGAR V** for their guidance, motivation and moral support during my entire postgraduate course which enabled to complete my work.

I am extremely thankful to **Dr. ARUN PRASAD P, Dr. VINOD KUMAR K, Dr. AJAY SS, Dr. SANDESH AGARAWAL** for their constant help and guidance throughout the course. They were the source of encouragement, support and for patient perusal to which I am deeply obliged. My heartful thanks to my seniors **Dr. KARTHIK SJ, Dr. NANDINI, Dr. DHARSHAN, Dr. ABHI SHARMA, Dr. ANIL KUMAR, Dr. SAI GANESH, Dr. ARUN KUMAAR, Dr. MADHAVAN** for their support and help in carrying this study and throughout the graduation course. I express my sincere thanks to my colleagues and dear friends **Dr. HRUSHIKESH, Dr. SIYAD, Dr. VYSHNAV, Dr. VISHNU, Dr. KIRAN, Dr. TARUN, Dr. HARSHA** for their constant support.

I thank my juniors **Dr. NAVIN BALAJI, Dr. BASANTH REDDY, Dr. GOWTHAM, Dr. ARYADEV, Dr. ROHITH, Dr. GILS THAMPI, Dr. AKSHAY, Dr. AYUSH AGARAWAL** for providing support throughout the study. I am also thankful to all the **INTERNS, OT, OPD** and **PARAMEDICAL STAFF** for their valuable help while performing this study.

I express my special thanks to all my patients and their families, who are the best teachers in the final conclusion and without whom this study would have been impossible.

Last but not least I would be failing in duty if I do not express my gratefulness to the **ALMIGHTY** who helped me mentally and physically not only during this study, but throughout the post-graduation course.

Date:

Place: Kolar **DR. JAGADISH U**

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

S. No	Abbreviation	Explanation
1	LPS	Lipopolysaccharide
2	NAM	N-acetyl muramic acid
3	NAG	N-acetyl glucosamine
4	H_2O_2	Hydrogen peroxide
5	TSI	Triple sugar iron agar test
6	(K/K)	Alkaline slant/Alkaline butt
7	(K/A)	Alkaline slant/Acidic butt
8	(A/A)	Acidic slant/ Acidic butt
9	H_2S	Hydrogen sulphide
10	WHO	World Health Organisation
11	MESS	Mangled Extremity Severity Scale
12	GHOIS	Ganga Hospital Open Injury Score
13	ESBL	extended spectrum beta lactamase
14	MRSA	Methicillin-resistant Staphylococcus aureus
15	BOA	British Orthopaedic Association
16	BAPRAS	British Association of Plastic Reconstruction and Aesthetic Surgeons

17	SPRINT	Study to Prospectively assess Reamed Intramedullary Nails in Tibial Fractures
18	ATLS	Advanced Trauma Life Support
19	CRO	Carbepenem resistant organisms
20	MDR	Multidrug-resistant

ABSTRACT

Background: Amputations and recurrent infections are two terrible outcomes of open fractures that can leave patients with permanent impairments. Rapid and effective treatment can protect patients from open fracture sequelae and the long-term financial burden these injuries frequently cause. High energy trauma is the most frequent cause of damage for open fractures, with over 50% occurring in auto accidents or falls from great heights. The first bacterial ecology of open fracture wounds in the Indian environment has not been extensively studied. Therefore, the need of the current assignment was to assess the effectiveness of pre-debridement and post-debridement culture in open fractures of the extremities.

Aim and objectives:

To assess pre-debridement and post-debridement culture in open fractures of the extremities.

Methodology: A prospective comparative study was conducted among 65 patients admitted from Casualty and OPD at R. L. Jalappa Hospital and Research center, attached to Sri Devaraj Urs Medical college, affliated to SDUAHER were included during the period between December 2020 and July 2022.

Results: Among the study participants, majority of cases (26.15%) were belonged to 21-30 years of age group. Total 14 participants belonged to 41-50 years of age group.

Out of total, 9 patients were aged less than 20 years. The majority of the pre-debridement cultures yielded *Staphylococcus aureus* followed by *Acinetobacter species, Enterobacter species, Pseudomonas species and Klebsiella Species.* The post debridement and treatment for bacterial infections, on subsequent cultures yielded only 4 positive growth of *Pseudomonas species, Enterobacter species and Proteus Species* and no growth was observed in 61 patients.

Conclusion: The study demonstrated that debridement cultures have a significant impact in the prediction of postoperative infection. Debridement culture is therefore advised to offer information about the selection of antimicrobial medication, which when paired with a complete wound debridement will permit an early wound closure and better overall outcome functionally.

Keywords: Open Fracture, bacterial infection, debridement, surgical site infection.

INTRODUCTION

INTRODUCTION

Amputations and recurrent infections are two terrible outcomes of open fractures that can leave patients with permanent impairments. Rapid and effective treatment can protect patients from open fracture sequelae and the long-term financial burden these injuries frequently cause. High energy trauma is the most frequent cause of damage for open fractures, with over 50% occurring in auto accidents or falls from great heights [1, 2]. High-energy open fractures are more common in young male patients than female patients, and these patients usually have concurrent injuries that are compounded by significant soft tissue damage. If open fractures are not treated properly from the onset, they might result in substantial morbidity. Unfortunately, this is still the situation in several impoverished nations. In the past, these wounds would leave patients dealing with persistent infections, agony, and impairment, with many patients finally needing an amputation [3, 4]. The care of patients with open fractures has significantly improved over time, mostly as a consequence of a greater understanding of the need of early therapy in addressing contamination and achieving early definitive closure and fixation.

Open fracture wounds are contaminated wounds, and the most common problem is postoperative period which presents as infection [5]. The fracture pattern, the patient's co-morbidities, the presence of traumatized soft tissue, and the interval between the injury and therapy all affect the likelihood of bacterial infection. In the seminal 1976 paper, Gustilo and Anderson found that 70.3% of 158 open long-bone fracture wounds had a positive bacterial culture [6]. Eighty-three to eighty-nine

percent of fracture sites presented with first cultures were contaminated by bacteria, according to many investigators [7, 8].

It has been quoted that determining the bacterial flora of the fracture site would enable logical and effective antibiotic treatment prior to starting antibiotic medication and making a final decision regarding wound management [9, 10]. Because they are acquired in the community, contaminating bacteria in open fractures ought to be treatable by the majority of conventional antibiotics. In general, it is advisable to use an empiric antibiotic which has a broad spectrum activity against both Gram-positive and Gram-negative bacteria.

Delayed wound culture may reveal the original contaminating organism, which might indicate a technical debridement failure and enormous risk of postoperative infection. Reports, contend hospital-acquired microorganisms to be blamed for surgical site infections [11]. This sparked a debate on the necessity and justification for acquiring first wound cultures, with some writers speculating that these cultures have little prognostic value for postoperative infection [12]. Others, however, assert that despite their lack of specificity, they have great sensitivity in detecting wounds that might get infected after surgery [13, 14].

The first bacterial ecology of open fracture wounds in the Indian environment has not been extensively studied. Therefore, the need of the current assignment was to assess the effectiveness of pre-debridement and post-debridement culture in open fractures of the extremities.

AIM AND OBJECTIVES

AIM AND OBJECTIVES

AIM:

- To assess pre-debridement and post-debridement culture in open fractures of the extremities.

OBJECTIVE:

- To analyze pre-debridement and post-debridement culture organisms in open fractures and tabulate the findings.
- To assess antibiotic sensitivity among various infective organisms in my study and tabulate the findings.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Problem Statement

As stated by the WHO, 5.8 millions of people die each year as a consequence of injuries (WHO 2014). These deaths make up a minor percentage of the total number of people harmed. Abrasions as well as small skin incisions or lacerations (tears) to wounds with severe tissue damage or loss, traumatic wounds (wounds induced by injury) can be coupled with injury to underlying tissues such as bone or viscera (internal organs). The mechanism of wound influences the degree of tissue damage: blunt trauma, penetrating injury, throng injury, explosion injury, scalds, de-gloving wound and animal bites are all examples of traumatic wounds. The requirement for immediate assessment and need for management of simultaneous severe, life-threatening injuries frequently dictates initial treatment of traumatic wounds [15].

An open fracture is characterised as a wound where the fracture site and/or fracture hematoma interact with the outside world. Low energy injuries are frequently the cause of open fractures in the long bones of the lower leg, which are frequently accompanied by other potentially fatal disorders as a result of poly-trauma. They also carry other significant hazards such as soft tissue lesions, neurovascular injuries, skin de-gloving and wound contamination, all will increase the likelihood of long-term consequences like delayed unions, non-unions, and amputations as well as persistent bone infections. With an annual frequency of 3.4 per 1,00,000 open tibia fractures are the most frequent open long bone fractures [1].

Open tibia fractures most usually affect young adult males and elderly females, with a mean age of 43.3 years. The main mechanism of damage is high energy trauma, with road traffic accidents and high-altitude falls accounting for more than 50% of occurrences. It should be noted that the majority of proximal and distal tibia fractures have severe soft tissue damage, which makes treating the injury more difficult as stated by **Jenkins PJ et al** [16].

Classification Systems

The evolution of system of grading that categorises open fractures based upon increasing severity of the traumatized soft tissue injuries was prompted by variety of outcomes among variety of patterns of open fractures with varying severities, despite overall improvement in outcome after open fractures. These grading schemes aim to aid in treatment direction, enhance research and communication, and forecast results. These classifications have been around for a while, but the Gustilo and Anderson system has emerged as the most used one for describing open fractures. Gustilo and Anderson improved on early Veliskakis attempts to grade open fractures in 1976. Gustilo et al. revised their first categorization of the most serious open injuries and later changed it into its present form in 1984.

Open fractures are often high-energy wounds. This increases the risk of infection, wound problems, and non-union along with exposing bone and deep tissue to the environment. The underlying principles for treating open fractures have not changed since World War I: adequate debridement, primary asepsis, immobilisation, and protection of wounds against disturbance and reinfection. Despite the significant

improvements brought about by antibiotics, surgical debridement, and internal fixation, open fracture management still relies on these fundamental principles.

Gustilo et al. ultimately described main properties of care for open fractures and helped define the modern approach to the management of open fractures via their research in avoidance of infection in open long bone fractures.

Based on the size of the wound, the degree of contamination, and the extent of osseous damage, they divided open injuries into the well-known three groups as follows: Type I open fractures have clean, less than 1 cm long wounds; Type II open fractures have wounds longer than 1 cm but don't have flaps or avulsions; and Type III open segmental fractures, open fractures with significant soft tissue injury, or traumatic amputations. Gunshot wounds, any open fracture brought on by a farm accident, and any open fracture with a vascular damage that needs to be repaired were special categories in Type III.

Wagirayezu, E et al [17], stated in his study due to their diverse injury patterns, increased morbidity from accompanying injuries, extensive soft tissue loss or damage around the fracture sites, compromised vascularity, wound contamination, and fracture instability, Type III open fractures have proven to be the most challenging to diagnose and treat. High-energy trauma, independent of the size of the wound, or open fractures with sufficient soft tissue coverage of a broken bone; Type IIIA, Type IIIB, open fractures with severe soft tissue damage loss with periosteal stripping and exposed bone. This is typically accompanied by severe contamination [17], and Type

IIIC fractures are those that are open and have vascular damage that needs to be repaired.

Table 1: Gustilo and Anderson open fracture classification system [18]

Gustilos' type	Definition
I	Open fracture, clean wound, wound length <1 cm
II	Open fractures, wounds longer than 1 cm without severe soft-tissue injury, flaps and avulsions are all examples of open fractures.
III	An open or segmental compound fracture with substantial soft-tissue injury, laceration or loss. Such as farm injuries, fractures that requires vascular-repair and fractures that have been open for more than 8 hours previous to management are all included in this category.
III A	Despite severe soft-tissue laceration or destruction, Type III fractures have adequate periosteal covering of the fracture bone.
III B	Soft-tissue loss, periosteal stripping and bone destruction characterises type III fractures. It's usually linked to a lot of pollution. Will almost always necessitate a second soft-tissue covering operation (i.e. free or rotational flap)
III C	Regardless of the degree of soft-tissue injury, type III fractures are accompanied with an artery injury that requires treatment.

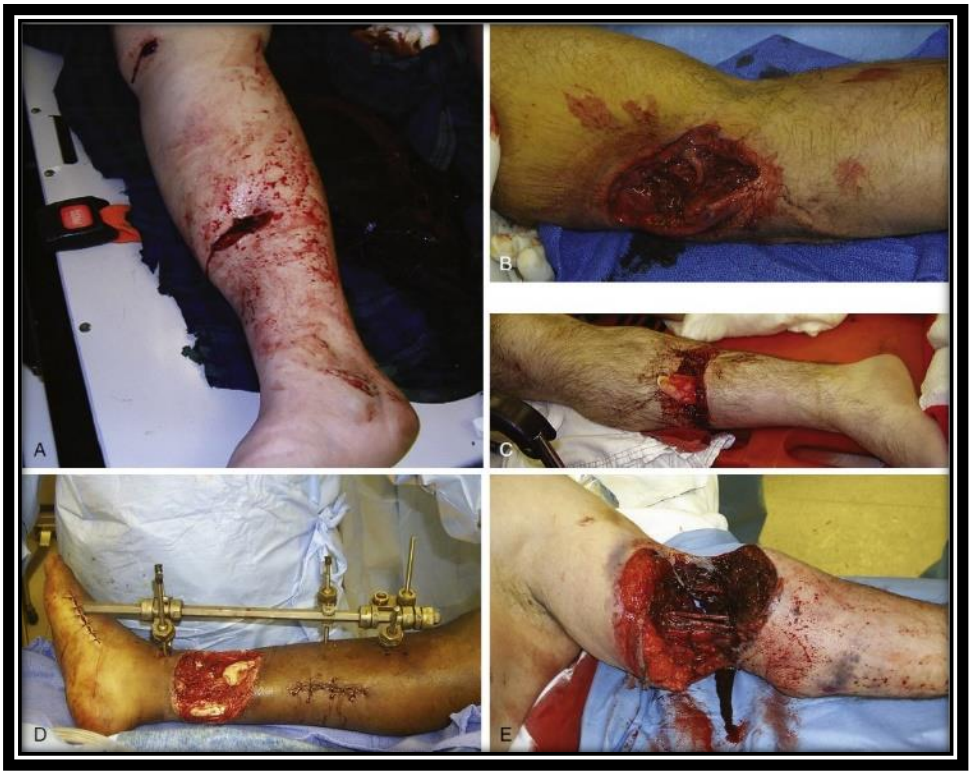

Figure 1: Gustilo and Anderson Open fracture [18]

Table 2: Tscherne classification for open fractures [19]

Grading	**Definition**
I	Small puncture wound without associated contusion, negligible bacterial contamination, low-energy mechanism of fracture
II	Small laceration, skin and soft tissue contusions, moderate bacterial contamination, variable mechanisms of injury
III	Large laceration with heavy bacterial contamination, extensive soft tissue damage, with frequent associated arterial or neural injury
IV	Incomplete or complete amputation with variable prognosis based on location and nature of injury (e.g., cleanly amputated middle phalanx vs. crushed leg at the proximal femoral level)

The result in type III B tibia injuries without vascular insufficiency is the focus of the Ganga Hospital Open Injury Score (GHOIS), which was introduced in 2004. It assigns a score from 0 to 5 depending on the degree of the injury to each of the three limb parts, including the skin, bone, and musculotendinous tissues. The score includes seven comorbid criteria with two points each that affect the course of therapy and the final result. The management of III B injuries using both the overall score and the specific tissue scores [20].

Table 3: Ganga Hospital scoring system [20]

Covering tissues: skin & fascia	Wound with no skin loss and not over fracture site	1
	Wound with no skin loss and over fracture site	2
	Wound with skin loss and not over fracture site	3
	Wound with skin loss and over fracture site	4
	Wound with circumferential skin loss	5
Functional tissues: musculotendinous and nerve units	Partial injury to musculotendinous unit	1
	Complete but repairable injury to musculotendinous unit	2
	Irreparable injury to musculotendinous unit, a partial loss of a compartment or complete injury to posterior tibial nerve	3
	Loss of one compartment of musculotendinous unit	4
	Loss of two or more compartment or subtotal amputation	5
Skeletal structures	Transverse or oblique fracture or butterfly fragment < 50% circumference	1

	Large butterfly fragment >50% circumference	2
	Comminution or segmental fractures without bone loss	3
	Bone loss < 4 cm	4
	Bone loss > 4 cm	5
Co-morbid conditions (add 2 points for each conditions)	Injury to debridement time interval > 12 hours	
	Sewage or organic contamination or farmyard injuries	
	Age >65 years	
	Drug dependent diabetes or cardiorespiratory diseases leading to increased anesthetic risk	
	Poly-trauma with injury severity score>25 or fat embolism	
	Hypotension with systemic blood pressure lesser than 90mmHg at the time of presentation	
	Another injury to the same limb or compartment syndrome	

The Mangled Extremity Severity Score (MESS) was initially developed as a guideline for determining whether to amputate severely injured lower limbs. The amount of soft tissue or skeletal damage, limb ischemia, shock using a cut-off of 90 mmHg for systolic blood pressure, and patient age are the four injury-related factors that receive points. Its application has been extended in recent years to the upper limb, albeit without a thorough analysis of its applicability [21].

Table 4: MESS- Mangled extremity severity score [21]

Criteria	Value	Points
Limb ischemia [Ischemia present >6 hours, points are multiplied by 2]	Reduced pulse but normal perfusion	+1
	Pulseless, Paraesthesia, Slow capillary refill	+2
	Cool, Paralysis, Numb/Insensate	+3
Patient age range	<30	0
	30-50	+1
	>/=50	+2
	SBP> 90mmHg consistently	0
Shock	Hypotension transiently	+1
	Persistent hypotension	+2
	Low energy (stab, gunshot, simple fractures)	+1
	Medium energy (dislocations, open/ multiple fractures)	+2
Injury mechanism	High energy (high speed MVA or rifle shot)	+3
	Very high energy(high speed trauma with gross contamination)	+4

Microbiology of open wounds and antibiotic prophylaxis

Brilliant Scottish physician, pharmacologist, and biologist Alexander Fleming was all three. His contributions to bacteriology, which was especially important during the First World War, are recognised. Using solely local antiseptics to heal wounds was rejected by him since it only touched surface tissue and was inefficient for treating deeper injured tissue and contused muscle. The majority of the combatants who were hurt were not able to receive medical attention right once and were left to rot in their trenches or on the "no man's land" of the battlefield for days. Aware that bacterial infection of wounds related to mortality, Antoine Depage took a particular interest in the research of wound treatment and debridement at the same time.

Fleming collaborated with Depage and found out *Clostridium perfringens, Clostridium welchii, Clostridium tetani, streptococcus*, and *staphylococcus species* were among the organisms developed when he analysed cultures taken from the clothes of 12 soldiers who had been injured during a conflict [22]. He came to the conclusion that these 30 organisms were in charge of causing wound infections and that they flourished in the dirt, an anaerobic environment where the injured troops were lying. Increased time for bacterial colonisation suggested by a delay in wound cleaning and treatment. However, his greatest contribution was the 1927 discovery of Penicillin and the area of antibiotic prophylaxis.

During this period, it was noted that 10% of all patients passed away with gangrene gaze use, or gas gangrene, a deadly type of the disease. In 1916, Chalier and Glenard identified four different forms of gas-forming infections: Fournier's gangrene,

wet gangrene, necrotizing fasciitis, and moist gangrene. *Beta-hemolytic streptococci, clostridium species, anaerobic streptococci, staphylococci,* and other gram-negative bacteria were the causative agents. Throughout World War II and the Vietnam War, use of Penicillin spread widely.

As a result of improvements in fracture management, triage, transport, and wound care, death rates from gas-gangrene during the Vietnam War were as low as 0.16% [23, 24]. The most frequent species known to exist are *staphylococcal species*. According to the results of Gustilos' prospective research from 1976, Cephalosporins were regarded as the best antibiotic. However, gram negative organisms were becoming more common in their follow-up investigation in 1984. Following that, it was suggested that Cephalosporins can be used in conjunction with an Aminoglycoside. Patzakis was the first to note in 1974 that only 18% of infections included organisms that were comparable to those discovered in first wound cultures [25].

Hospital acquired infections became a thing as a result, and they are now an increasing problem. Later, Glass et al studied a group of 52 grade 3B fractures at a tertiary care facility in London and observed an incidence rate of infection to be 17% [26]. *Methicillin-resistant Staphylococcus aureus, Pseudomonas, Enterobacter, and Enterococci* were among the organisms that were identified (MRSA). The majority of these pathogens were hospital-acquired. It was discovered that the current recommendations for antibiotic prophylaxis (Cephalosporins with an Aminoglycoside like Gentamycin or a beta-lactum) were insufficient. When a specific wound was

being covered, they recommended using Teicoplanin and Gentamycin as a single dose blanket treatment to combat hospital acquired and resistant germs. Recent research has demonstrated that industrialised nations have higher rates of nosocomial infections and antibiotic resistance, and that the microbiologic makeup of wounds changes depending on the host, mode of damage, degree of stress, and environment [27]. There has been an increasing trend for the development of resistant strains, such as Extended spectrum beta lactamase (ESBL) resistant organisms, Carbepenem resistant organisms (CRO), and *Methicillin resistant Staphylococcus aureus,* in high-income, developed nations where the cost of antibiotics is not a concern (MRSA). Contrarily, patients in underdeveloped nations like India, China, Africa, and Israel struggle to pay for second-line medications, which prevents the widespread use of high-dose antibiotics.

Additionally, it has been discovered that antibiotic resistance itself is linked to a higher risk of infection, many re admissions, and hospital stays. At an Israeli tertiary hospital, 89 fractures were prospectively studied by Robinson et al. According to him, the majority of wounds were infected when they were revived, and positive cultures collected more than 24 hours after debridement suggested that the surgical procedure had failed [28]. This research emphasised the significance of the surgeon's expertise and the appropriateness of wound debridement.

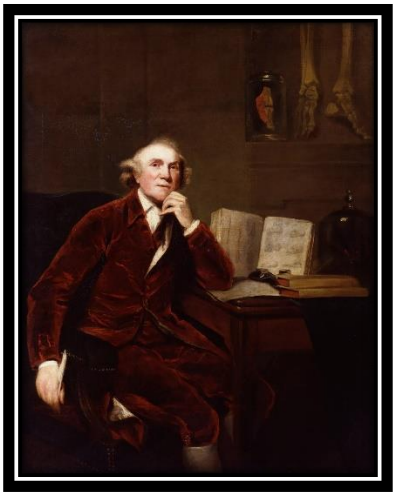

Figure 2: Image of Alexander Fleming [24]

In their prospective series examination of open fractures, Alonge et al. discovered that whereas wounds debrided after 48 hours were positive for poly-microbial or mixed organism development, wounds treated within 6 hours only isolated single culture organisms. He also demonstrated that the organisms from superficial and deep wound cultures were identical in more than 90% of the instances [29]. All of these studies from underdeveloped nations demonstrate that the majority of bacteria are responsive to common antibiotics, hence antibiotic prophylaxis is not the main concern in wound infections.

The timing and technique of wound debridement have a significant impact on the risk of infections. This emphasises how important it is to debride wounds thoroughly and meticulously as soon as possible in order to get rid of the infectious organism. There is controversy over the length of time that antibiotics must be taken. Antibiotics should be administered for at least 24 to 48 hours following Gustilos' grade I injuries, according to British Orthopaedic Association/British Association of

Plastic Reconstruction and Aesthetic Surgeons (BOA/BAPRAS) recommendations. They advise giving antibiotics to patients with injuries of grades 2 and 3 for a maximum of 72 hours, or until soft tissue has fully recovered [30].

During this time, the word "debridement" and the idea behind it were first used in relation to open wounds. The phrase "débrider" in French literally translates to "unbridling a horse." In order to unbridle or drain away undesired foreign material and pus from wounds, it refers to incising and de-tensioning of the soft tissue and fascia. Incising a wound was thought to relieve pressure and lessen swelling, so avoiding gangrene. Bleeding was thought to be a sign of healthy muscle. Larry used this method to expose and ligate bleeding arteries as well as to decompress soft tissue and remove debris. He understood the value of early wound care, surgical debridement, and triage. He cited debridement as one of surgery's most important discoveries in his 1814 book "Memoirs of a Military Surgeon." However, following his passing, the practise of surgical wound treatment gradually fell out of favour. The rationale for surgeons' reluctance to incise wounds was that they were thought to represent a risk to soft tissue and underlying blood vessels during surgical wound therapy. Only open fractures with severe soft tissue injury were treated with radical soft tissue excision and, if required, amputations [31].

Only 3% of wounds suffered during the American Civil War were treated medically. The overall mortality recorded was 12%. After the development of antiseptics in the latter half of the 20th century, medical care began to replace surgical treatment for wounds. In 1867, Lister popularised the older view of John Hunter

(1728–1793) that incising wounds would cause more inflammation and were best left alone by introducing the use of carbolic acid as an antiseptic in open fractures. In order to facilitate drainage and the elimination of foreign objects, Pilcher recommended the use of antiseptics in 1833 and slightly expanding wounds only if significant soft tissue damage was predicted with stable skeletal structure [32]. The two most important aspects of managing open fractures are the restoration of bone morphology and skeletal stabilization [33, 34].

Immobilization from outside Splinting and immobilisation of the severely damaged limb have been practised since the time of the Chinese, Egyptians, and Indians. Pirogov and Mathijsen (1805–1878) invented Plaster of Paris (1810- 1881). They discovered that this material was very conformable to the wounded limb and also helped to stabilise the area, promoting bone and soft tissue healing. Along with Carrel, orthopaedic physician H. Winnett Orr (1877–1956) practised wound care in open fractures by cleansing wounds, packing them with petroleum-soaked gauze, then stabilising and immobilising the fracture using plaster-soaked bandages.

After an exothermic reaction caused this to solidify, the cast was removed after three weeks. Low infection rates were recorded. Thomas splint was the most successful and efficient splinting device for open femoral fractures by **Robinson PM et al** [35]. Hugh Owen Thomas first wrote about it in 1875. The Ilizarov ring fixator, a device that transformed the way orthopaedic surgeons treat fractures, was created during this time period and was first reported for complicated open fractures. Four rings were originally intended for the fixator. Crossed tensioned Kirschner wires were

used to secure two of these rings to the bone on either side of the fracture. Threaded rods were used to join the rings. This layout gave the limbs strong circumferential support. Ilizarovs' main principles of management included adequate support of the fracture, minimize surgery, prompt weight bearing and early joint mobilization in the study by **Paul GW et al** [36].

The rods connecting the rings were compressed to address fracture non-unions. When a patient unintentionally extended the rods and a callus developed in the fracture gap, distraction osteogenesis was identified. The law of tension stress and a novel strategy for limb salvaging are products of further research. Numerous studies have shown that Ilizarov fixators are useful in treating acute trauma. In 24 segmental tibia shaft fractures, 17 of which were open fractures which are primarily grade III injuries, Yusuf et al. assessed the outcomes of acute application of the Ilizarov fixator in 2009. **Uhthoff HK et al**. in his study analyzed the average amount of time between an injury and operation was 14 hours [37]. The average time for union was 36.8 weeks, and none of the patients experienced persistent osteomyelitis or deep infections. They concluded that the Ilizarov fixator was especially beneficial when the distal fracture segment was less than 3cm with severe soft tissue compromise.

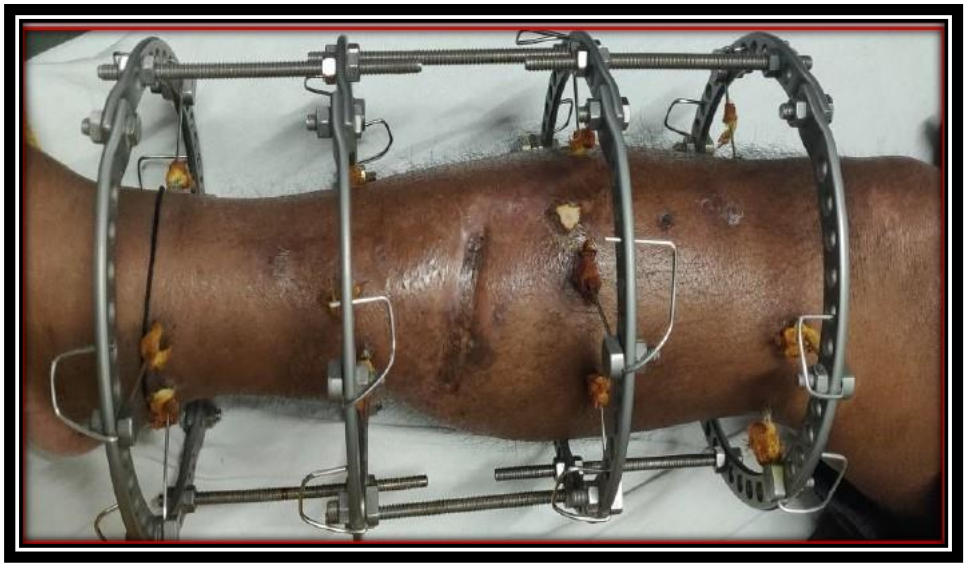

Figure 3: Acute trauma immobilised with Ilizarovs' ring fixator

The 400 open fractures in the SPRINT (Study to Prospectively assess Reamed Intramedullary Nails in Tibial Fractures) randomised controlled study were compared for effectiveness between reamed and unreamed nails by **Cronier P, et al. and Bhandari M, et al**. [38,39,40]. Up until a subsequent re-operation procedure was carried out, all patients were monitored. Although there was a slightly rising trend for patients in the repeat group to have further procedures, there was no statistically significant difference in the re-operation rates between the two groups. The reported total infection rate was 27%, flaps and soft tissue coverage. Complex fracture triage is "very difficult, exceedingly technical, and team oriented," and is "to an orthopaedic traumatologist with plastic surgery assistance." Hansen, S. 1991 stated that the value of soft tissue covering in open wounds was initially recognised by the ancient Egyptians. The Smith Papyrus stated that ‘‘whenever there is a gaping wound, such as that perpetrated by the mouth of a crocodile it should be shielded with meat’ by **Bhandari M et al.** [40].

Based on his observations from World War 1, Baer argued that wounds that are debrided within 12 hours had a better likelihood of success in primary wound closure. He claimed that early debridement of wounds had an 85–90% success rate. Harold Gilles was the first to use flaps to patch infected fractures with soft tissue around this time. The use of pedicle tubed flaps successfully provided soft tissue cover in the significant percentage of chronic osteomyelitis patients in limbs saved. Slovenian plastic surgeon Marco Godina (1943–1966) was the first to use free flaps to cover soft tissue. He made the use of Latissimus dorsi flaps in commonplace. He further demonstrated that early soft tissue cover within the first 48 hours was essential for limb salvage and also decreased the risk of amputations [41]. Gustilo and Anderson stated that all Type 3 injuries should have delayed wound closure either in the form of skin grafting or flaps to reduce the risk of infections [42, 43].

A British military surgeon named John Trueta promoted early wound debridement in 1942 and held that the muscle, not the bone, was more susceptible to infection. His next step was to implement a 5-point protocol for treating open fractures, which comprises immediate surgery, wound cleanliness, wound excision, drainage, and immobilisation of the fracture in a plaster cast. In 1973, Robson assessed the severe wounds of 80 people. According to his findings, infections formed in every wound that had > 105 bacterial organisms per gram of tissue before healing. He also discovered that the colonisation threshold reached its maximum 5.17 hours after the damage on average. The incidence of major limb amputations was significantly lowered in the latter decades of the 20th century thanks to immediate

prompt transport from the battlefield, knowledge and application of treatment principles for open fractures, antibiotic prophylaxis, early debridement, and meticulous repair of neurovascular injuries [44].

As a result, Friedrich's 6-hour rule of early quick debridement acquired more and more attraction as an established standard recommendation for orthopaedic surgeons in the therapy of open fractures. Later, a number of studies examined the reliability of the 6-hour rule. In 1995, Kindsfater and Johansson examined 47 open type 2 and 3 tibia fractures and discovered that an infection was substantially more likely to occur when more than 5 hours had passed since the injury. **Kindsfater K et al** [45], Additionally noted that profound infections and osteomyelitis did not manifest until at least 4.8 months after the accident, and that only 25% of patients showed a positive first culture association with the causative bacteria. The probability of an infectious sequelae was not ruled out by negative cultures. This study is important in literature as it was the first to highlight that deep infections in open fractures may remain occult in the early post-operative period and can manifest later as long as the infecting organism in the wound was inherent as stated by **Kreder HJ** et al [46].

Management:

Initial treatment -

In one-third of instances with multiple injuries from trauma, open fractures typically develop from high energy trauma. Injury to soft tissues and bone is equal to

kinetic energy (KE=12mv2), where m is the body's mass and v is its velocity. It is essential to manage the patient first in line with Advanced Trauma Life Support (ATLS) standards, excluding injuries to the head, chest, abdomen, and pelvis that pose a serious risk of death. As soon as possible, resuscitation was started, and any related injuries were treated according to their priority. Diabetes mellitus, malnutrition, liver disease, peripheral vascular disease, extremes of age, immune deficiency syndromes, smoking, and use of steroids are co-morbidity variables that should be taken into account early in care since they are linked to delayed recovery [47, 48].

The degree of the wound, soft tissue damage, and contamination are evaluated on the wounded extremities. Additionally evaluated and documented, the limb's neurovascular condition should rule out compartment syndrome. In order to determine the best strategy for immobilising a fracture and the danger of infection, soft tissues are examined for contamination, stripping, de-vascularization and degree of loss. Tscherne initially divided soft tissue injuries into groups based on severity for closed fractures, but as knowledge of the pathophysiology of soft tissue injury increased, more sophisticated and detailed grading systems, such as the Hannover fracture scale and the AO soft tissue grading system, were created. These systems can be useful in treating severe open fractures by **Tscherne H et al** [49].

If surgical intervention is intended to reduce the danger of additional contamination, exploration of the wound in emergency rooms is not advised. Wounds are bandaged with saline-soaked gauze after any obviously foreign bodies have been removed and trauma radiographs have been taken. Tetanus prophylaxis and enough

antibiotic coverage should be administered. Regardless of age, the current toxoid dosage is 0.5ml, and the recommended immune globulin dosage is 75U for 10 years. They should each be intramuscularly administered using a separate syringe and location. Whether a wound is tetanus-prone or not depends on the patient's vaccination history and the type of wound. A clean, small wound that heals within six hours is not tetanus-prone.

Tetanus-prone individuals have wounds that are irregular in shape, > 1 cm deep, and the result of a projectile, crush, burn, or frostbite [50].

The use of antibiotics since open fractures get infected, antibiotic coverage is necessary. The incidence of infection is greatly decreased when antibiotics are used in the treatment of open fractures. Delays longer than three hours increase the risk of infection; if treated promptly, the risk is reduced by six times. Giving the first dose as soon as feasible is advised. The length of time that antibiotics are administered in open fractures is still up for debate. According to the most recent research, three days should be given repeatedly when a wound is closed, the bone is stabilised, or bone grafting is done [51].

Cephalosporin alone is advised for Gustilo type I injuries, Cephalosporin plus aminoglycoside for type II injuries, and Cephalosporin, Penicillin, and Aminoglycoside for type III injuries. The contaminating organisms should be the focus of the prescribed antibiotics. Staphylococcus aureus and coagulase-negative staphylococcus are the most typical bacteria that infect open fractures. Nosocomial infections that develop in open fractures later in the hospital are typically brought on

by gram negative bacteria. Pseudomonas aeruginosa is frequently seen in foot puncture wounds. Because they are susceptible to Clostridial infections, open fractures that develop on farms or those with deep tissues without debridement should be taken seriously. As a nosocomial and community-acquired infection, *Methicillin-resistant staphylococcus aureus* (MRSA) has been isolated in patients, and its occurrence is linked to morbidity, death, and higher treatment costs as described by **Brumback RJ et al** [52].

Intensive care facilities and institutionalised patients frequently have outbreaks of it. There is limited information on the effectiveness of Daptomycin in treating orthopaedic surgical infections in a randomised control study, despite the fact that it has been approved for use against MRSA in Europe and America and that it has been successfully demonstrated to be effective against MRSA osteomyelitis. Although culture and sensitivity patterns should be followed, Linezolid and Vancomycin are effective against MRSA as well as other older antibiotics including Tetracycline, Rifampicin, Clindamycin, and Trimethoprim-sulphamethoxazole. Due to the quick emergence of resistance, Quinolones like Ciprofloxacin and others should only be used in conjunction with other antibiotics for treating MRSA. **Johansen K Et al and Slauterbeck JR et al** [53, 54].

Irrigation and Debridement One of the most crucial guidelines for managing open fractures is the debridement of fractures. Desault was the first to describe it, and nowadays extensive lavage is used. Sharp dissection should be used, and it should be done carefully. Remove any trash, dead tissue, and loose cortical bone pieces.

Dissection moves on to the boundaries of living tissues, which may be recognised by their consistency, colour and contractility of the tissue. Grade I and II wounds must be lengthened in order to properly remove them. Debridement within six hours has always been advised as being crucial to infection prevention as observed by **Hansen ST et al** in his study [55].

It helps to understand perforators and anastamosis while placing incisions correctly. If all conditions are met and the necessary skill is available, debridement should be carried out as soon as feasible before we have convincing proof against the 6-hour time before debridement. If more debridement is required, it should be performed within 24 to 48 hours. Although irrigation is a crucial component in managing open fractures, there is ongoing debate about the best delivery method, volume, and irrigation solution. For Grade I wounds, 3 litres, Grade II wounds, 6 litres, and Grade III wounds, 10 litres have been suggested. Although there isn't much research to back it, antibiotic treatments seem more effective than saline. In one investigation, detergents (soap) were shown to be as effective at lowering infection risk as antibiotics at removing germs. **Lange RH et al** [56].

Because they are hazardous to tissues, antiseptics should be avoided. The necessary pressure is still debatable; high pressure enhances bacterial clearance but harms soft tissues and bone. Suction and low pressure devices like bulb syringes are sufficient. When washing wounds, there is no discernible difference between using water and saline. Saline is still used by most surgeons.

Closure of wounds

In open fractures, delayed primary closure is used to seal the majority of wounds. The original open fracture site might be left open while the surgical incisions made during the initial debridement are mostly healed. Useful techniques for aided wound closure include vacuum assisted dressings and antibiotic bead pouches. Early primary closure of the open wound, split skin grafts (SSG), fasciocutaneous flaps, rotating muscle flaps, and free muscle flaps may be necessary for the management of wounds in open fractures. Godina demonstrated that skilled hands may prefer wide early thorough debridement to achieve healthy tissues and early rotating or muscle flap cover over sequential debridement and delayed closure. **Bosse MJ et al** [57].

Early closure has been demonstrated to reduce the risk of nosocomial infections, which are frequent after several debridement and delayed closure. The goal should be to close the deal within 72 hours. Fracture prevention Stabilization of the fracture may be permanent or temporary. Skeletal traction and occasionally external fixation are included in temporary fixation. External fixation, plate fixation, and intramedullary nailing are all examples of definitive fixation. Each of these methods has benefits and drawbacks. In cases of pelvic fractures and femoral fractures, skeletal traction may be temporary helpful. The majority of therapy for severe open fractures like IIIA and IIIB is external fixation, which has the benefit of making it simple to control soft tissue infections and bone transport and can be substituted by an internal fixation. **Taufik A et al** [58].

Within fourteen days, there should be no pin tract infection for safe exchange. The primary issues with external fixators include loosening, delayed or non-union union, and pin tract infections. Open fractures with plate and screw fixation had increased infection rates and is prioritized for certain peri-articular fractures. The use of intramedullary nails is most common in grade I open tibia fractures, radius and ulna gunshot wounds, and open femur fractures. Additionally, these are substituted for open type II tibia fractures. Whether to use reamed or undreamed nails has generated debate. Reaming provides the benefit of a quicker healing process, a lower non-union rate, and fewer screw breaks. Un-reamed nails have been demonstrated to be beneficial in patients who have had many injuries because they cause less pulmonary problems. The superiority of unreamed nails in multiply injured patient have been shown as it decreases pulmonary complications and spares endosteal blood flow, well knowing that the soft tissue blood flow is already compromised [59].

Contamination in Open fracture of Extremities: **Naique SB et al** [59].

Cefazolin and Amikacin resistance has been documented in several research from various nations, which are common antibiotic regimens for preventive treatment in open fractures. In Texas in the 1980s, Johnson described the prevalence of *methicillin-resistant Staphylococcus aureus*. Patients with post-implantation osteomyelitis have been shown to have *Staphylococcus epidermidis* that is resistant to Ampicillin, Penicillin, Cefazolin, and Chloramphenicol, according to Arcilla et al. Amikacin resistance in *E. Coli* was also written by SA Lerner and MH Perlin. Methicillin, Vancomycin, third-generation Cephalosporins, and Fluoroquinolone

resistance in *Staphylococcus aureus, Coagulase-negative Staphylococcus, Pseudomonas aeruginosa,* and *Escherichia coli* has been widely reported over the past ten years.

Gram-positive bacteria predominated in the pattern of bacteria before debridement was performed, according to Herlambang's investigation at the Emergency Room (IRD), Dr. Soetomo Hospital Surabaya. After debridement, *Pseudomonas aeruginosa* (43.75%) and *Staphylococcus aureus* (18.75%) were the most prevalent gram-negative bacteria. These bacteria were primarily *Staphylococcus aureus* (49.523%) and *Pseudomonas aeruginosa* (20%). Cefazolin resistance was also discovered to be 100% in *Pseudomonas* and 19.4% in *Staphylococcus aureus*. *Pseudomonas aeruginosa* and *Staphylococcus aureus* both have 10% and 5% Amikacin resistance, respectively. **Zalavras CG et al** [60].

Prior to debridement, gram-positive bacteria predominated over gram-negative bacteria. Most infections in open fractures are brought on by gram-negative bacteria. It was discovered through culture that the bacteria causing the disease did not correspond to the germs that first contaminated the patient's open fractures. It was shown that nosocomial infections accounted for 92% of the causes of infection. Based on this, it is necessary to re-evaluate the use of the antibiotics Cefazolin and Amikacin in following therapy given in the room for up to 5 days. This is because resistance to common antibiotics was discovered during the examination two days after debridement. Researchers believe that once the patient receives treatment in the room, antibiotic resistance increases even worse.

Table 5: Various etiological agents of Wound infections [61]

Gram positive cocci	*Staphylococcus*
	Streptococcus
	Enterococcus
Gram positive bacilli	*Corynebacterium*
	Bacillus
	Clostridium
	Mycobacterium
Gram negative bacilli	*Escherichia coli*
	Klebsiella
	Enterobacter species
	Proteus species
	Pseudomonas and other non-fermenters

Morphology of bacteria [61]:

Depending on their shape, bacteria are classified into:

- Cocci (singular coccus, from; kokkos., meaning berry) are oval or spherical cells and
- Bacilli or rods (singular bacillus, meaning rod shaped).

Cocci are arranged in groups (clusters), pair or chains. Similarly, bacilli can be arranged in chain, pair, and some bacilli are curved, comma shaped, or cuneiform shaped.

Both cocci and bacilli are further classified based on Gram staining property into

- Gram-positive cocci
- Gram-negative cocci
- Gram-positive bacilli
- Gram-negative bacilli

Bacterial cell anatomy comprises of the following structures.

The outer layer or the envelope of a bacterial cell consists of

- A rigid cell wall
- Underlying plasma membrane.

The cytoplasm contains cytoplasmic inclusions (mesosomes, ribosomes, inclusion granules, vacuoles) and a diffuse nucleoid containing single circular chromosome. Some bacteria may possess additional cell wall appendages such as capsule, flagella and fimbriae.

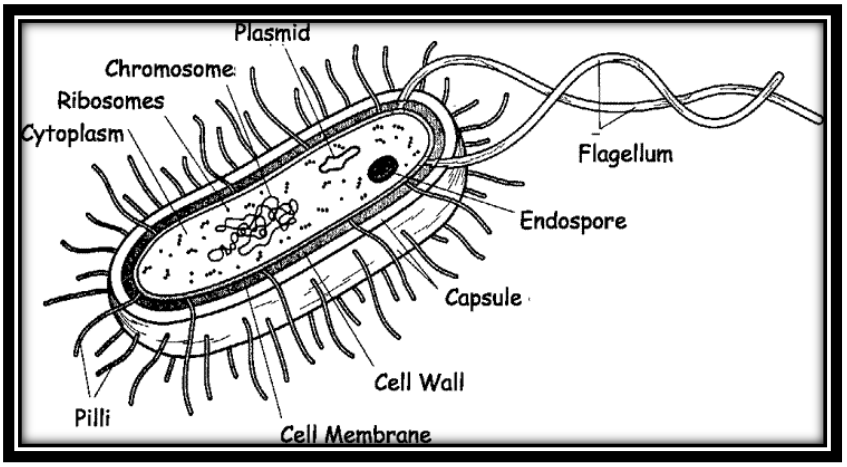

Figure 4: Morphology of bacteria [61]

Bacterial Cell Wall:

The cell wall is a tough and rigid structure, surrounding the bacterium. It is 10-25 nm in thickness and weighs about 20-25% of the dry weight of the cell.

The cell wall has following functions:

- It provides protection to the cell against osmotic-lysis.
- It confers rigidity upon bacteria due to presence of peptidoglycan layer in the cell wall.

It accounts for the shape of the cell.

- It takes part in cell division.
- The cell wall can protect a cell from toxic substances and is the site of action of several antibiotics.
- Virulence factors- Bacterial cell wall contains certain virulence factors (e.g. endotoxin), which contribute to their pathogenicity.
- Immunity: Antibody raised against specific cell wall antigens (e.g. antibody to LPS) may provide immunity against some bacterial infection.

Gram-positive Cell Wall

Peptidoglycan:

- In gram-positive bacteria, the peptidoglycan layer is much thicker (50- 100 layers thick, 16-80 nm) than gram-negative cell wall.
- Each layer is a mucopeptide (murein) chain, composed of alternate units of N-acetyl muramic acid (NAM) and N-acetyl glucosamine (NAG) molecules; cross linked to each other via tetra peptide side chains and penta glycine bridges.
- A tetra peptide side chain ascended from NAM molecule is composed of L-alanine-D-glutamine-L-lysine-D- alanine.
- The L-lysine of one tetra peptide chain is covalently linked to the terminal D-alanine of the adjacent chain via a penta glycine bridge.

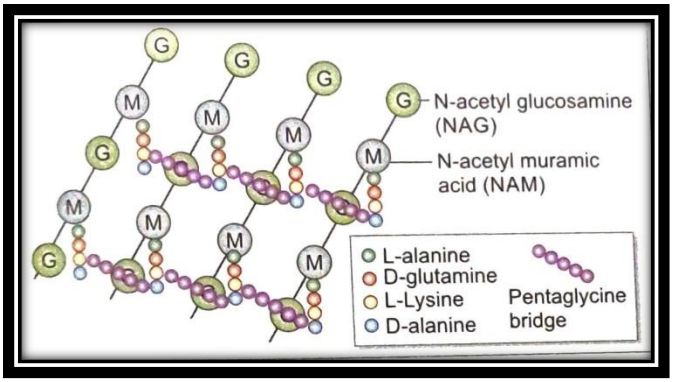

Figure 5: Peptidoglycan layer of Gram-Positive cell wall [61]

Teichoic Acid:

- Gram-positive cell wall contains significant amount of teichoic acid which is absent in gram-negative bacterial cell wall.
- They are polymers of glycerol or ribitol joined by phosphate groups. The functions of these molecules are still unclear, but they may be important in maintaining the structure of the cell wall.

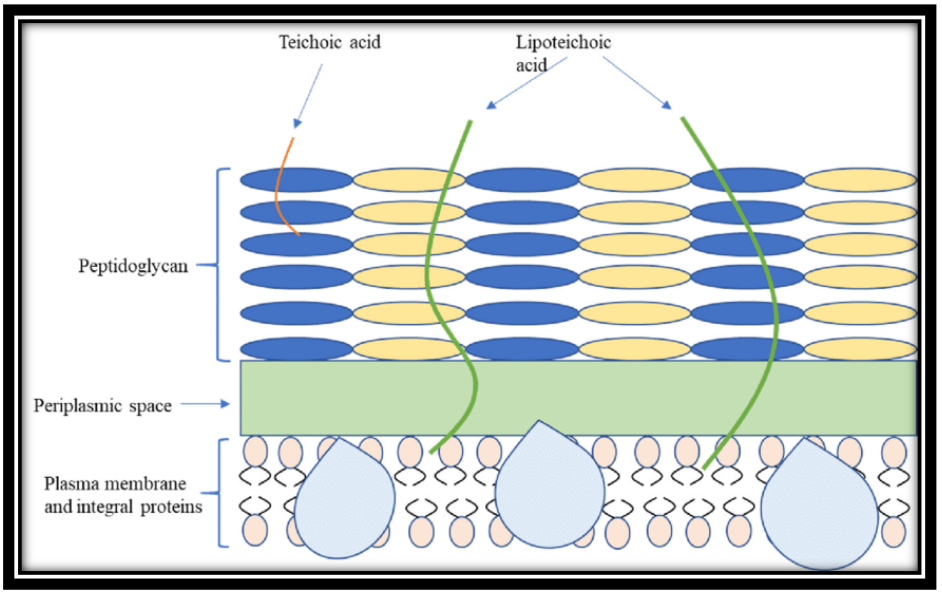

Figure 6: Gram-Positive cell wall [61]

Gram-negative Cell Wall

- Gram-negative cell wall is thinner and more complex than the Gram-positive cell wall, comprises of the following components.

Peptidoglycan Layer:

- It is very thin (1-2 layer, 2nm thick), composed of a mucopeptide chain similar to that of gram-positive cell wall, and consists of alternate NAM and NAG molecules.

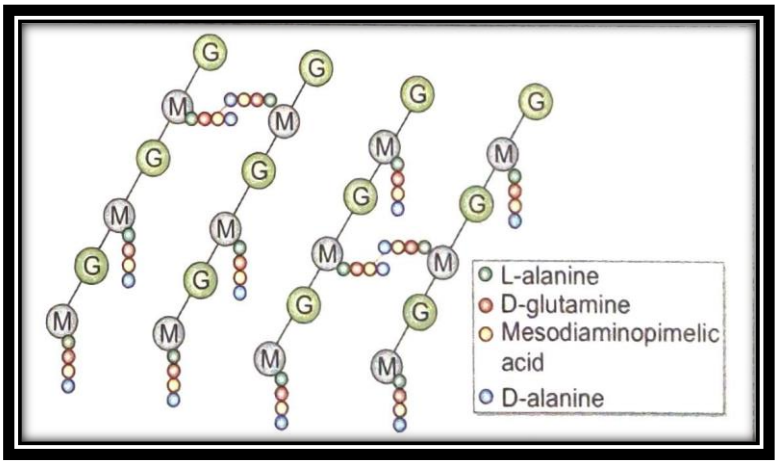

Figure 7: Peptidoglycan layer of Gram-Negative cell wall [61]

Outer Membrane:

- This is a phospholipid layer which lies outside to the thin peptidoglycan layer; firmly attached to the later by covalent linkage of membrane protein called Braun's lipoprotein.
- It serves as a protective barrier to the cell.
- Outer membrane proteins (OMP) or porin proteins. They are the specialized proteins present in outer membrane. Three porin molecules cluster together and span the outer membrane to form a narrow channel through which molecules

smaller than about 600-700 Daltons can pass.

- The outer membrane also prevents the loss of constituents such as periplasmic enzymes.

Lipopolysaccharide (LPS):

- This layer is unique to gram-negative bacteria which is absent in gram-positives. It consists of three parts:
- Lipid A or the endotoxin: It has endotoxic activities, such as pyrogenicity, lethal effect, tissue necrosis, anti-complementary activity, B cell mitogenicity, adjuvant property and antitumour activity.
- It consists of two glucosamine sugar derivatives, each with three fatty acids and phosphate attached.
- It is buried in the outer membrane and the remainder of the LPS molecule projects from the surface
- Core polysaccharide: It is projected from lipid A region. It is composed of 10-12 sugar moieties.
- O side chain (or O antigen): It is a polysaccharide chain extending outwards from the core polysaccharide region. It is made up of several sugar moieties and it greatly varies in composition between bacterial strains.
- O antigen is a major surface antigen (called somatic antigen), induces antibody formation. It is also used for serotyping.

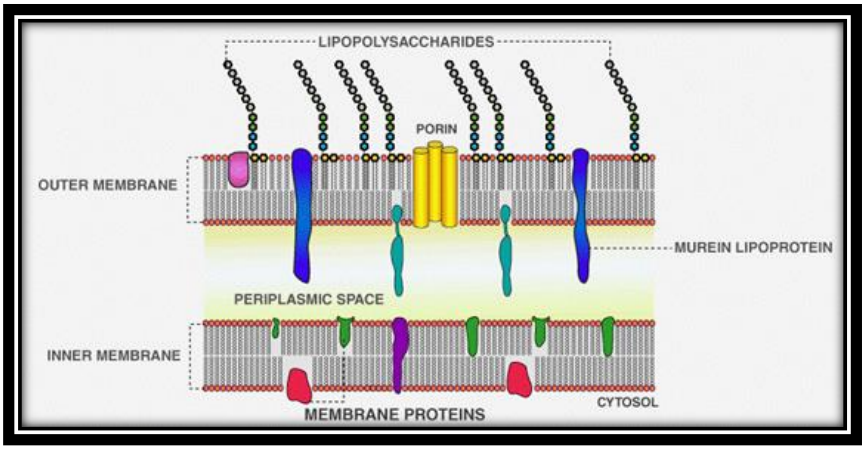

Figure 8: Gram-Negative cell wall [61]

Table 6: Differences between Gram-Positive and Gram-Negative cell wall [61]

Characters	**Gram- Positive cell wall**	**Gram-Negative cell wall**
Peptidoglycan layer	Thicker (15-80nm)	Thinner (2 nm)
At third position of tetra peptide side chain	L-Lysine present	Mesodiaminopimelic acid present
Penta glycine bridge	Present	Absent
Lipid content	Nil or scanty(2-5%)	Present(15-20%)
Lipopolysaccharide	Absent	Present (endotoxin)
Teichoic acid	Present	Absent
Variety of amino acids	Few	several
Aromatic amino acids	Absent	Present

Periplasmic Space

It is the space between the inner cell membrane and outer membrane. It encompasses the peptidoglycan layer.

CELL MEMBRANE

The plasma membrane is essential for the survival of the bacteria.

- Fluid mosaic model is the most widely accepted current model to describe the membrane structure.
- It is 5-10 nm thick, composed of bi-layered phospholipid in which several proteins are embedded, such as integral proteins and peripheral proteins
- It differs from eukaryotic membranes in lacking sterols, such as cholesterol (except in Mycoplasma*)*. However, many bacterial membranes do contain penta-cyclic

  sterol-like molecules called Hopanoids.
- Carbohydrate: Some carbohydrates are often attached to the outer surface of plasma membrane proteins.

It is a semi permeable membrane acting as an osmotic barrier; allows selectively only particular ions and molecules to pass, either into or out of the cell, while preventing the movement of others.

Transport system: Proteins and enzymes present in cell membrane are involved in nutrient uptake, and waste excretion.

Site for metabolic processes: Bacterial cell membrane is the site of a variety of crucial metabolic processes such as: Respiration, the synthesis of lipids and cell wall, and probably chromosome segregation.

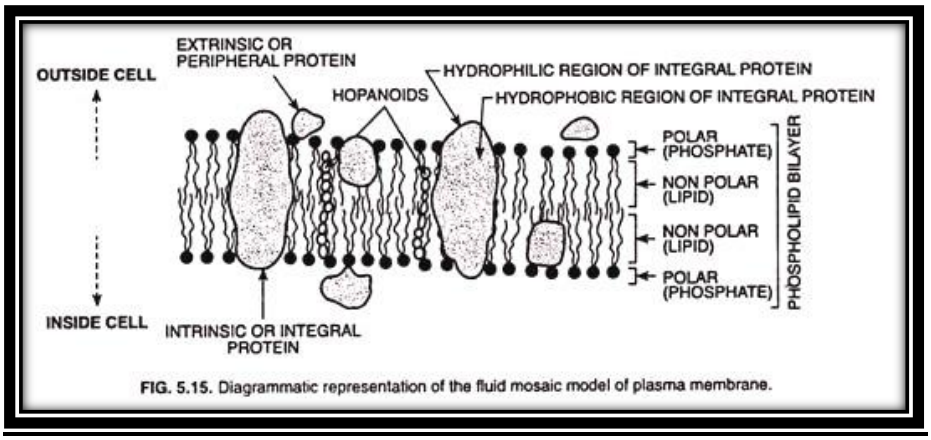

Figure 9: Structure of Bacterial cell membrane [61]

Cell wall appendages

Capsule and Slime Layer

Some bacteria possess a layer of amorphous viscid material lying outside the cell wall called glycocalyx. When the glycocalyx layer is well organized and not easily washed off, it is called capsule.

When the glycocalyx layer is in the form of diffuse, unorganized loose material that can be removed easily, it is called slime layer.

The capsule has various functions as follows:

- Contribute to bacterial virulence:
- Capsule protects the bacterium from phagocytosis.
- It can also prevent complement-mediated bacterial cell lysis
- Prevent cell from drying out (desiccation)
- It protects the bacterium from the action of lysozyme and bacteriophages.

- Biofilm formation and adhesion

Biofilm Formation

A biofilm is a living ecosystem made of millions of adherent bacterial cells embedded within a self-produced matrix of extracellular polymeric substance (i.e. the polysaccharide slime layer).

Persistent biofilms containing pathogenic bacteria are capable of adherence to damaged tissues and plastic surfaces (e.g. medical devices, such as catheters and pacemakers).

This is the first step in bacterial colonization and sometimes it leads to disease, e.g. prosthetic valve endocarditis and catheter related urinary tract infection.

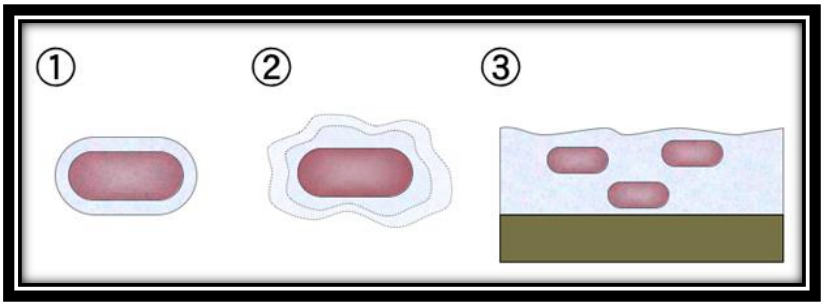

Figure 10: Capsule, Slime and Biofilm [61]

BACTERIAL GROWTH [61]:

Bacterial Growth Requirement:

Water constitutes about 80% of total bacterial cell. The minimum nutritional requirements that are essential for growth and multiplication of bacteria include sources of carbon, nitrogen, hydrogen, oxygen and some inorganic salts (such as small amounts of sulphur, phosphorus and other elements like sodium, potassium, magnesium, iron and manganese).

Bacterial Vitamin:

Some fastidious bacteria do not grow in the routine culture medium unless certain organic compounds (that are essential to those bacteria) are added to the medium. These are known as growth factors or bacterial vitamins. In most instances, bacterial vitamins are same as the vitamins necessary for mammalian nutrition, particularly those belonging to the vitamin B group-thiamine, nicotinic acid, riboflavin, pyridoxine, folic acid and vitamin B12.

Table 7: Bacterial vitamins [61]

Vitamins	**Bacteria requiring Vitamin**
Biotin	*Leuconostoc species*
Cyanocobalamin (B12)	*Lactobacillus species*
Folic acid	*Enterococcus faecalis*
Pantothenic acid	*Morganella morganii*
Pyridoxine (B6)	*Lactobacillus species*
Niacin (nicotinic acid)	*Brucella abortus, Haemophilus jnfluenzae*
Riboflavin (B2)	*Bacillus anthracis*

Rate of Multiplication in Bacteria:

Generation time is the time required for a bacterium to give rise to two daughter cells under optimum condition.

The generation time for different bacteria is as follows:

Escherichia coli and most of the other pathogenic bacteria takes 20 minutes.

- *Mycobacterium tuberculosis*-20 hours
- *Mycobacterium leprae* -20 days

As bacteria grow so rapidly and by geometric progression, a single bacterium can theoretically give rise to 10^{21} daughter cells in 24 hours. Fortunately, it does not happen in reality, because the bacterial multiplication is arrested after a few cell divisions due to exhaustion of nutrients and accumulation of toxic products.

Table 8: Generation times for some common bacteria under optimal growth conditions [61]

Bacterium	Medium	Generation Time (minutes)
Escherichia coli	Glucose-salts	17
Staphylococcus aureus	Heart infusion broth	27-30
Mycobacterium tuberculosis	Synthetic	792-932

Bacterial Count:

Bacterial count may be expressed in terms of total count and viable count.

- Total count: It indicates total number of bacteria (live or dead) in the specimen. This is done by counting the bacteria under microscope using counting chamber.
- Viable count: It measures the number of living (viable) cells in the given specimen

Bacterial Growth Curve

When a bacterium is inoculated into a suitable liquid culture medium and incubated, its growth follows a definite course. When bacterial count of such culture is

determined at different intervals and plotted in relation to time, a bacterial growth curve is obtained comprising of four phases.

Lag phase: It is the period between inoculation and beginning of multiplication of bacteria. After inoculating into a culture medium, bacteria do not start multiplying immediately, but take some time to build-up enzymes and metabolites.

- Bacteria increase in size due to accumulation of enzymes and metabolites.
- Bacteria reach their maximum size at the end of lag phase.

Log phase: In this phase bacteria divide exponentially so that the growth curve takes a shape of straight line. At this stage, the bacterium is:

- Smaller in size
- Biochemically active: It is the best stage to perform the biochemical reactions.
- Uniformly stained: It is the best time to perform the Gram stain.

Stationary phase: After the log phase, the bacterial growth ceases almost completely due to exhaustion of nutrients, accumulation of toxic products and autolytic enzymes. The number of progeny cells formed is just enough to replace the number of cells that die.

Hence, the number of viable cells remain stationary as there is almost a balance between the dying cells and the newly formed cells. But the total count keeps rising. In this phase:

- Bacterium becomes Gram variable
- More storage granules are formed
- Sporulation occurs in this phase

- Bacteria produce exotoxins, antibiotics and bacteriocins.

4. Decline phase: Gradually, the bacteria stop dividing completely; while the cell death continues due to exhaustion of nutrients, and accmulation of toxic products.

• There is decline in viable count and not in total count.

• Involution forms are seen.

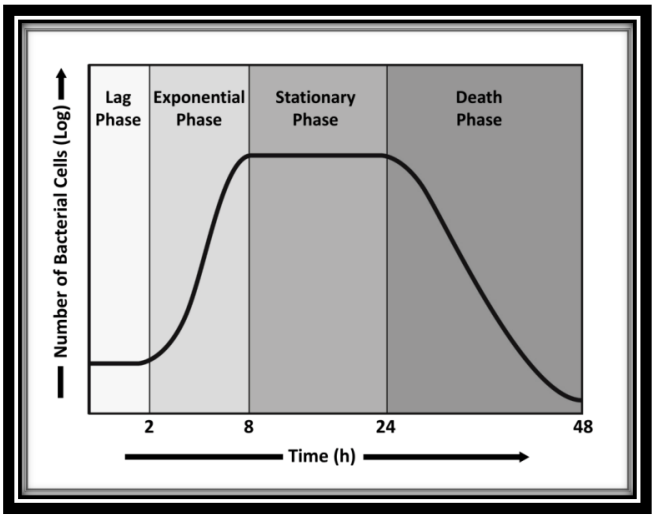

Figure 11: Bacterial Growth curve [61]

CULTURE MEDIA [62]

Culture media are required to isolate the bacteria from the clinical specimens; following which the appropriate biochemical tests can be performed to identify the causative agent.

- Water: Distilled water or potable water with low mineral content is suitable for culture media preparation. Water serves as the source of hydrogen and oxygen.
- Electrolytes: Sodium chloride or other electrolytes.
- Peptone: It is a complex mixture of partially digested proteins.

Agar: It is used for solidifying the culture media. It is commercially available in powder form; melts in water after boiling and jellifies after cooling.

TYPES OF CULTURE MEDIA

Bacteriological culture media can be classified in two ways.

Based on consistency, culture media are grouped into:

1. Liquid media (or broth)

2. Semisolid media

3. Solid media

Based on the growth requirements, culture media are classified as:

1. Routine laboratory media: They are prepared from nutrients, such as aqueous extract of meat, peptone, etc. They can further be classified into various types based on functional use or application, as follows-

- Simple/ basal media
- Enriched media
- Enrichment broth
- Selective media
- Differential media
- Transport media
- Anaerobic media

2. Defined or synthetic media: They are prepared from pure chemical substances and the exact composition of the media is known.

- Simple synthetic media
- Complex synthetic media

Identification of organisms:

GRAM STAIN [62]:

It is staining technique which was originally developed by Hans Christian Gram (1884). Even after more than 130 years of its discovery and even if the newer modern diagnostic facilities are available, still Gram stain remains the most widely used stain in diagnostic bacteriology.

Procedure

- Fixation: The smear made on a slide from bacterial culture or specimen, is air dried and then heat fixed.
- Step 1(Primary stain): The smear is stained with pararosaniline dyes such as crystal violet (or gentian violet or methyl violet) for one minute. Then the slide is rinsed with water. Crystal violet stains all the bacteria violet in colour (irrespective of whether they are gram-positive or negative).
- Step 2 (Mordant): Gram's iodine (dilute solution of iodine) is poured oven the slide for one minute. Then the slide is rinsed with water. Gram's iodine acts as a mordant.
- Step 3 (Decolourization): Next step is pouring of few drops of decolourizer to the smear: e.g. acetone (for 1-2 sec) or ethyl alcohol (20-30 sec) or acetone alcohol (for 10 sec) or iodine acetone. The slides were immediately rinsed with water. Decolourizer removes the primary stain from gram-negative bacteria while the gram-positive bacteria retain the

primary stain.

- Step 4 (Counter Stain): Secondary stains such as safranin or diluted carbol fuchsin is added for 30 seconds. It imparts pink or red colour to the gram-negative bacteria. Alternatively, neutral red may also be used as a counter stain, especially for gonococci. The slide is rinsed in tap water, dried, and then examined under oil immersion objective.

Uses of Gram Stain

- Differentiation of bacteria into gram-positive and gram-negative: It is the first step towards the identification of bacteria.
- For identification: Gram staining from bacterial culture gives an idea to put the corresponding biochemical test for further identification of bacteria
- To start empirical treatment: Gram stain from specimen gives a preliminary clue about the bacteria present (base on the shape and Gram staining property of the bacteria) so that the empirical treatment with a broad-spectrum antibiotics can be started early before the culture report is available.
- For fastidious organisms, such as *Haemophilus* which takes time to grow in culture; Gram stain helps in early presumptive identification.
- Anaerobic organisms, such as *Clostridium*, which do not grow in routine culture. Hence, Gram stain gives a preliminary clue to put anaerobic culture.

- Yeasts: In addition to staining the bacteria, Gram stain is useful for staining certain fungi such as *Candida* and *Cryptococcus* (appear gram-positive).

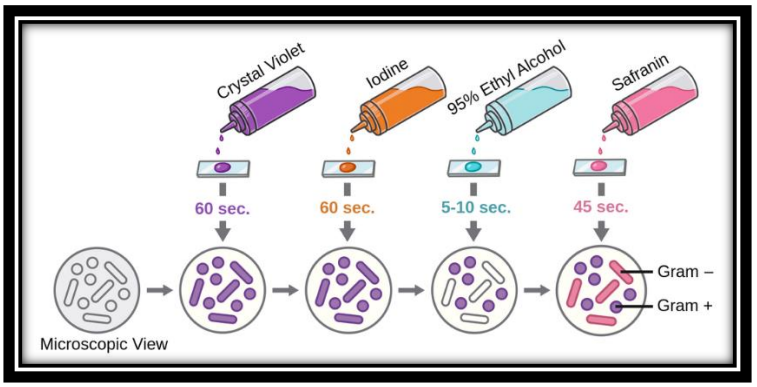

Figure 12: Procedure and Principle of Gram staining [62]

<u>Biochemical tests for the identification of organisms [62]</u>

<u>Catalase Test:</u>

When a drop of hydrogen peroxide (3% H_2O_2) is added to a colony (or when the colony is mixed to a drop of H_2O_2 placed on a slide) of any catalase producing bacteria, effervescence or bubbles appear due to breakdown of H_2O_2, by catalase to produce oxygen.

• Catalase test is primarily used to differentiate between *Staphylococcus* (catalase positive) from *Streptococcus* (catalase negative).

• It is also positive for members of the families *Enterobacteriaceae, Vibrionaceae*, etc.

• False-positive: Since blood contains catalase, colonies from blood agar may result in false-positive reaction. Use of iron wire/loop for picking up colonies may also produce false-positive test

• Nutrient agar is the ideal medium to perform the catalase test and the colonies should be picked by glass/wooden sticks (e.g. tooth picks).

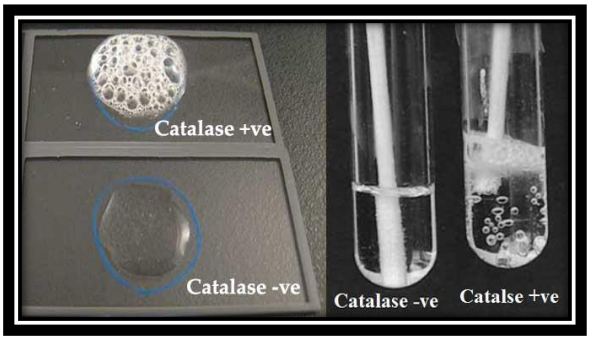

Figure 13: Interpretation of Catalase test [62]

Oxidase Tests:

It detects the presence of cytochrome oxidase enzyme in bacteria, which catalyzes the oxidation of reduced cytochrome by atmospheric oxygen.

• When a filter paper strip or disk, soaked in oxidase reagent (l % tetra methyl para-phenylenediamine dihydrochloride), is smeared with a bacterial colony producing cytochrome oxidase enzyme, the smeared area turns deep purple within 10 seconds due to oxidation of the dye to form a purple-coloured compound indophenol blue.

Interpretation and examples:

Oxidase positive (deep purple): Examples include *Pseudomonas, Vibrio, Neisseria, Bacillus*, etc.

Oxidase negative (no colour change): Examples include; members of family *Enterobacteriaceae*, etc.

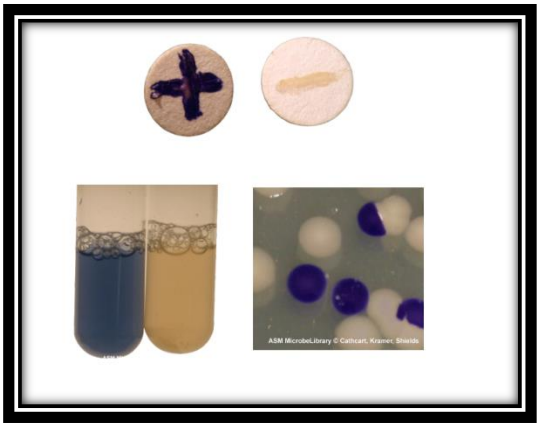

Figure 14: Interpretation of Oxidase test [62]

Indole Test:

It detects the ability of certain bacteria to produce enzyme tryptophanase that breaks down amino acid tryptophan present in the medium into indole.

• When Kovac' reagent (para-dimethylaminobenzaldehyde) is added to an overnight incubated broth of a bacterial colony, it complexes with indole to produce a cherry red colour ring near the surface of the medium.

• Indole positive: A red coloured ring is formed near the surface of the broth. Examples include *Escherichia coli, Proteus vulgaris, Vibrio cholerae*, etc.

• Indole negative: Yellow coloured ring is formed near the surface of the broth, e.g. *Klebsiella, Proteus mirabilis, Pseudomonas, Shigella, Salmonella*, etc.

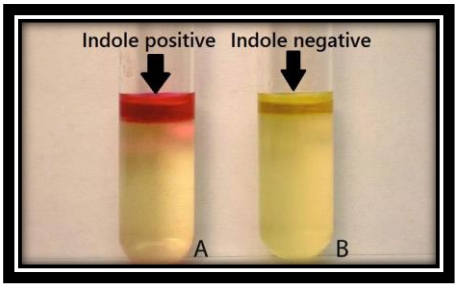

Figure 15: Interpretation of Indole test [62]

Citrate Utilisation Test

It detects the ability of a few bacteria to utilize citrate as the sole source of carbon for their growth, with production of alkaline metabolic products. Citrate test is performed on citrate containing medium, such as Simmon's (solid) or Koser's (liquid) medium.

- Simmon's citrate medium: Citrate utilizing bacteria produce growth and a colour change i.e. original green colour changes to blue. Here bromothymol blue is used as an indicator
- Koser 's (liquid) medium: It becomes turbid, by the growth of citrate utilizing bacteria.
- Citrate test is positive for *Klebsiella pneumoniae, Citrobacter, Enterobacter,* etc.
- The test is negative for *Escherichia coli, Shigella,* etc.

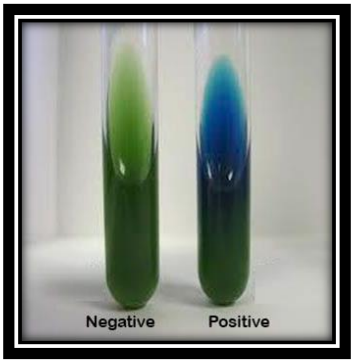

Figure 16: Citrate utilisation test [62]

Urea Hydrolysis Test

Urease-producing bacteria can split urea present in the medium to produce ammonia that makes the medium alkaline.

- Test is done on Christensen's urea medium, which contain phenol red indicator that changes to pink colour in alkaline medium.

- Urease test is positive for: *Klebsiella pneumoniae, Proteus species, Helicobacter pylori, Brucella*, etc.
- Urease test is negative for: *Escherichia coli, Shigella, Salmonella,* etc.

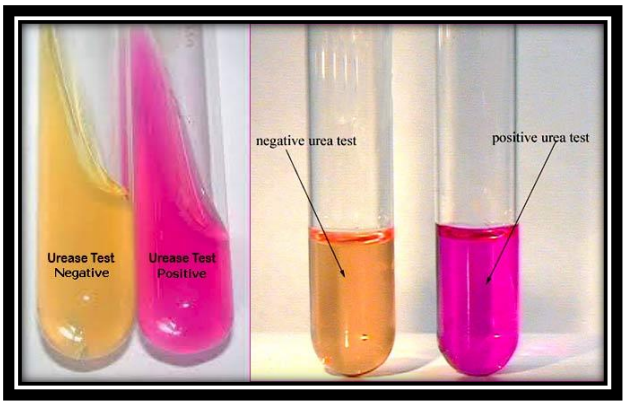

Figure 17: Interpretation of Urea hydrolysis test [62]

Triple Sugar Iron (TSI) Agar Test

TSI is a very important medium employed widely for identification of gram-negative bacteria.

Composition:

It is a composite solid agar medium in a 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 salts as an indicator of hydrogen sulfide (H2S) production.

Procedure:

Medium is 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 al 37^0C for 18- 24 hours. Under incubation or over incubation may lead to false interpretation of result.

Interpretation:

TSI detects three properties of bacteria, such as fermentation of sugars to produce acid and/or gas and production of H2S.

Ability to ferment sugars to produce acid:

Uninoculated TSI medium is red in colour and on acid production the colour changes to yellow. Based on fermentation of sugar present in TSI, the organisms are categorized into three groups.

I. Non-fermenters: They do not ferment any sugars, hence an alkaline slant and alkaline butt (no change) reaction is observed, (K/K reaction or alkaline (red) slant/ alkaline (red) butt.

2. Glucose only fermenters: They ferment only glucose and produce little acid. Initially at 8 hours, the whole medium turns acidic (yellow). Later on, the organism begins oxidative degradation of the peptones present in the slant, resulting in alkaline by-products in slant, which change the indicator back to red colour. At 18-24 hours, the medium appears alkaline (red) slant/ acidic (yellow) butt or (K/ A reaction).

3. Lactose and/or sucrose fermenters: They ferment glucose and also ferment lactose and/or sucrose to produce large amounts of acid so that the medium turns acidic at 8 hours. At 18-24 hours, the medium maintains acidic pH both in slant and butt and gives an acidic (yellow) slant/ acidic (yellow) butt or (A/A reaction).

Ability to produce gas: Some bacteria produce gas by sugar fermentation; which is denoted by breaks/ cracks in the medium or the medium is lifted up.

Ability to produce H_2S: Certain bacteria produce hydrogen sulphide (H_2S) which is a colourless gas. H_2S combines with ferric ions (from ferric salts present in the medium) to form ferrous sulphide, that produces blackening of the medium.

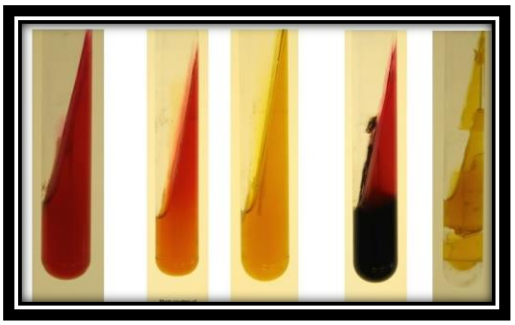

Figure 18: Interpretation of Triple sugar iron test [62]

Table 9: Various classes of Antibiotics and their properties [62]

Chemical class	Examples	Biological source	Spectrum (effective against)	Mode of action
Beta-lactams (Penicillins and Cephalosporins)	Penicillin G, Cephalothin	*Penicillium notatum and Cephalosporium species*	Gram-positive bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Semisynthetic Penicillin	Ampicillin, Amoxycillin		Gram-positive andGram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Clavulanic Acid	Clavamox is clavulanic acid plus amoxycillin	*Streptomyces clavuligerus*	Gram-positive andGram-negative bacteria	Suicide inhibitorof beta-lactamases
Monobactams	Aztreonam	*Chromobacter violaceum*	Gram-positive andGram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly

Carboxypenems	Imipenem	*Streptomyces cattleya*	Gram-positive andGram-negative bacteria	Inhibits steps in cell wall (peptidoglycan) synthesis and murein assembly
Aminoglycosides	Streptomycin	*Streptomyces griseus*	Gram-positive andGram-negative bacteria	Inhibit translation (protein synthesis)
	Gentamicin	*Micromonospora species*	Gram-positive andGram-negative bacteria esp. *Pseudomonas*	Inhibit translation (protein synthesis)
Glycopeptides	Vancomycin	*Streptomyces orientales*	Gram-positive bacteria, esp. *Staphylococcus aureus*	Inhibits steps in murein (peptidoglycan) biosynthesis andassembly
Lincomycins	Clindamycin	*Streptomyces lincolnensis*	Gram-positive andGram-negative bacteria esp. anaerobic Bacteroides	Inhibits translation (protein synthesis)
Macrolides	Erythromycin	*Streptomyces erythreus*	Gram-positive bacteria, Gram-negative bacteria not enterics, *Neisseria, Legionella, Mycoplasma*	Inhibits translation (protein synthesis)
Polypeptides	Polymyxin	*Bacillus polymyxa*	Gram-negative bacteria	Damages cytoplasmic membranes
	Bacitracin	*Bacillus subtilis*	Gram-positive bacteria	Inhibits steps in murein (peptidoglycan) biosynthesis andassembly
Polyenes	Amphotericin	*Streptomyces nodosus*	Fungi	Inactivate membranes containing sterols

	Nystatin	*Streptomyces noursei*	Fungi (*Candida*)	Inactivate membranes containing sterols
Rifamycins	Rifampicin	*Streptomyces mediterranei*	Gram-positive andGram-negative bacteria, *Mycobacterium tuberculosis*	Inhibits transcription (eubacterial RNA polymerase)
Tetracyclines	Tetracycline	*Streptomyces species*	Gram-positive andGram-negative bacteria, Rickettsias	Inhibit translation (protein synthesis)
Semisynthetic Tetracycline	Doxycycline		Gram-positive andGram-negative bacteria, *Rickettsias Ehrlichia, Borellia*	Inhibit translation (protein synthesis)
Chloramphenicol	Chloramphenicol	*Streptomyces venezuelae*	Gram-positive andGram-negative bacteria	Inhibits translation (protein synthesis)

MATERIALS
&
METHODS

MATERIALS AND METHODS

STUDY METHOD:

Prospective comparative study

STUDY LOCATION:

Department of Orthopaedics & Central Diagnostic Laboratory Services, Microbiology section of R. L. Jalappa hospital attached to Sri Devaraj Urs Academy of Higher Education and Research Tamaka, Kolar.

STUDY PERIOD AND DURATION:

From December 2020 to July 2022 for a duration of one year eight months

STUDY POPULATION:

The patients admitted from Casualty and OPD at R. L. Jalappa Hospital and Research center, attached to Sri Devaraj Urs Medical college, affliated to SDUAHER were included during the period between December 2020 and July 2022.

SAMPLE SIZE CALCULATION

Pre-debridement and post debridement difference in infection rate of 9% as reported in a study **Naique et al**, [61] considering an alpha error of 1% with power of 90% and assuming the proportion expected in the population as 30%.

$$\text{Sample size} = \frac{z^2 1-\alpha/2 (1-p)}{d^2}$$

p: Expected proportions of 5.4% infection rated: Absolute precision = 8%

1-α/2: desired confidence level = 1.96 (95% confidence level)

Z : confidence interval

The estimated sample size will be 54, expecting a dropout rate of 20% during the study. The final sample size was 54 +11 = 65

SAMPLING METHOD:

Purposive Sampling done for all patients admitted in RLJH between December 2020 to July 2022

INCLUSION CRITERA:

1. Patients presenting with open fractures of upper and lower extremities (Gustilo-Anderson classification I, II, IIIA, IIIB).
2. Patients presenting to Emergency Medicine Department within 6 hours of trauma.

EXCLUSION CRITERIA:

1. Patients who had undergone surgical procedure or wound debridement before reaching the hospital.
2. Patient already underwent an antiseptic wound dressing
3. Patient already received antibiotics.

METHOD OF DATA COLLECTION

Patients whose profile fitted that of the inclusion criteria were considered in our study. The patient and their attendants provided the working proforma with the following information: demographic information, the date and time of the accident, the mechanism of the injury, Time since the patient's injuries and admission to the RLJH hospital.

The injured limb(s) were splinted, the wound was examined for size and extent, both soft tissue and bone health was checked, and the amount of contamination was noted for all patients who had trauma assessment and appropriate management in the emergency room. Gustilo Anderson's classification of open fractures served as the basis for a tentative classification of all wounds. All the patients were given tetanus toxoid and the limb was splinted. The wound was covered by a sterile saline soaked gauze. Under complete aseptic precautions the wound was first cleaned with sterile

normal saline and then a wound swab was taken and sent to Central Diagnostic Laboratory Services, Microbiology section. This was considered to be the pre-debridement sample. Then a thorough wound toileting is carried out in the operation theatre using 6-10 liters of saline under complete asepsis. Following which another swab was taken deep within the wound. This was considered to be the post-debridement sample which was also sent to Central Diagnostic Laboratory Services, Microbiology section. The wound was then debrided under anesthesia and the open fracture was classified depending on findings. The bony injury is stabilized depending on criteria such as soft tissue coverage, comminution, contamination, and periosteal stripping. The soft tissue wounds were dressed as necessary. Further management of fracture was carried out based on standard protocol /guidelines.

Swabs were immediately streaked onto Blood agar and MacConkey agar medium upon receipt (both pre- and post-debridement), incubated aerobically at 37°C for 24–48 hours, and checked for any bacterial growth.

Figure 19: Processing of swabs onto respective agars being done in a Bio Safety Cabinet.

Figure 20: Inoculated plates are placed in incubator to provide optimal environment.

Standard bacteriological techniques were used to further identify any bacterial growth, and relevant biochemical assays were run in accordance with the standard protocol (like Gram stain, catalase, coagulase, oxidase, indole, citrate, urease, mannitol motility and triple sugar iron tests) [63, 64].

All patients were started on antibiotics after pre-debridement culture samples based on hospital protocol. Prophylactic antibiotics were given for 3 days and later escalate /de-escalate antibiotics based on the culture sensitivity report of pre and post-debridement cultures. If no growth occurred antibiotics are stopped on 5th day.

The patient during hospital stay was assessed clinically for signs of infection and repeat cultures were sent if infection was found to be present. Clinical indicators taken into account that suggested a wound infection were localized temperature increase, pain, sero sanguinous discharge, abscess collection, frank pus, foul odour, necrosis of the graft or flap, and fever with chills.

The patient wound was inspected regularly and dressing was done using aseptic measures. If there was any evidence of infection the wound sample was again sent for culture and antibiotics started and secondary definitive soft tissue and bony procedure was done to obtain coverage. Once soft tissue coverage was established and patient has no evidence of infection, the patient was discharged from hospital. The microbial profile & antibiogram pattern was documented and analyzed.

ETHICAL CONSIDERATION

The Institutional Ethics Committee granted its ethical approval. (SDUMC/KLR/IEC/633/2020-21). Written informed consent was taken prior to the study of each participants. All ethics morals were followed in the study. The composed data was utilized only for the anticipated purpose of the study. The dignity and welfare of participants were shielded at all times from ethics point of view. The authors got the study participants' permission to use their true identities in the report analysis, and the research data was kept suppressed throughout the study.

STATISTICAL ANALYSIS:

- Data was collected by case record form and entered into MS excel 2016.
- Data analysis was done in SPSS Software version 26.

RESULTS

RESULTS

1) Descriptive statistics:

Socio-demographic profile:

Table 10: Age-wise distribution among study participants (n=65)

Age group (in years)	Frequency (%)
≤ 20	9 (13.84)
21-30	17 (26.15)
31-40	10 (15.3)
41-50	14(21.5)
51-60	7(10.7)
61-70	4(6.15)
71-80	2(3)
>80	2(3)

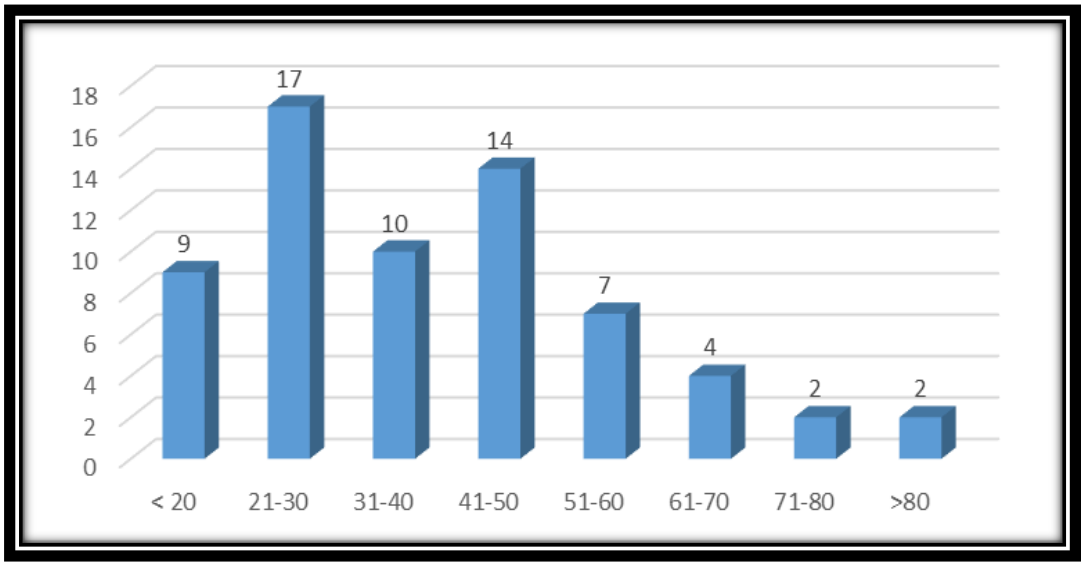

Figure 21: Age-wise distribution among study participants

The majority of instances (26.15%) among the study participants were in the 21–30 age range. There were 14 individuals overall who were between the ages of 41 and 50. Nine patients, out of the total, were under 20 years old. Only 2 of the patients were above 80 years old. The average age of research participants was 40.11+17.5 years.

Table 11: Gender-wise distribution among study participants (n=65)

Gender	Frequency (%)
Male	55(85)
Female	10(15)

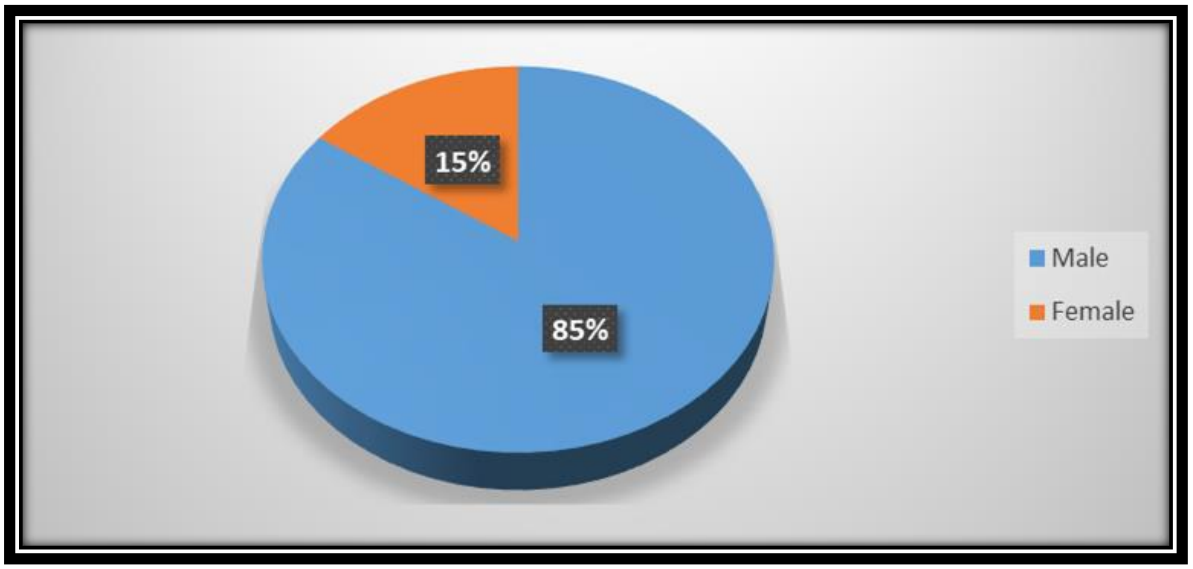

Figure 22: Gender-wise distribution among study participants

Out of total, 85% were males and 15% were females in the study.

Table 12: Mode of injury among study participants (n=65)

Mode of injury	Frequency (%)
Fall from Height	1 (1.5)
Fall of heavy Object	1(1.5)
RTA	52 (80)
Work Place injury	11 (17)

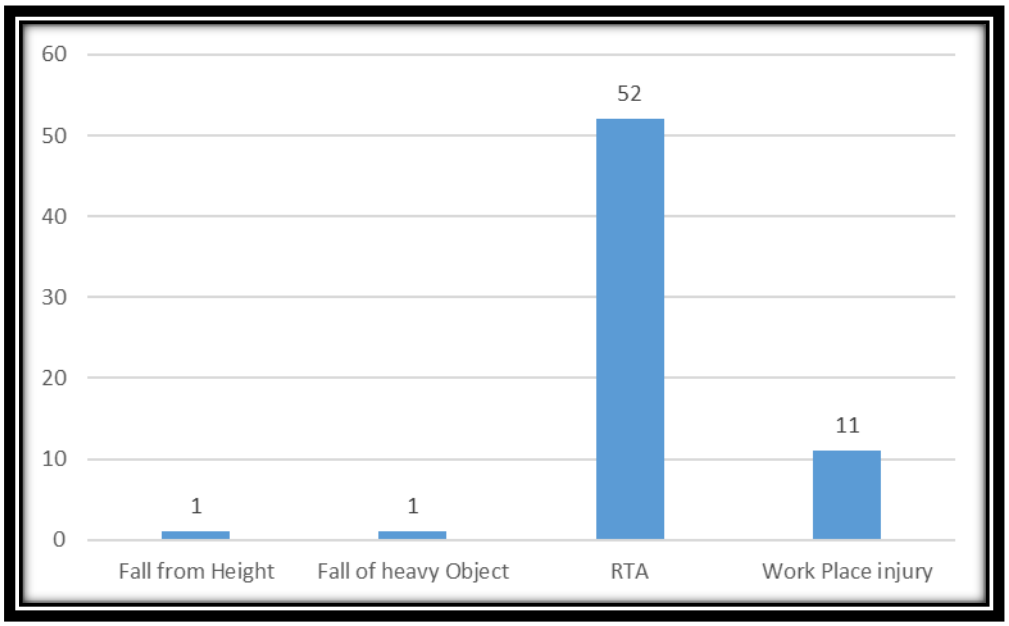

Figure 23: Mode of injury among study participants

One patient experienced a fall from a height as the cause of their injury, while road traffic accidents accounted for 80% of participant injuries and 11 patients had workplace injuries. One patient was still hurt as a result of a hefty object falling on them.

Table 13: Comorbidities among study participants (n=65)

Comorbidities	Frequency (%)
Hypertension	1 (1)
Diabetes Mellitus	3 (5)
Hypertension + Diabetes mellitus	18 (28)
Thyroid disorder	2 (3)
None	41 (63)

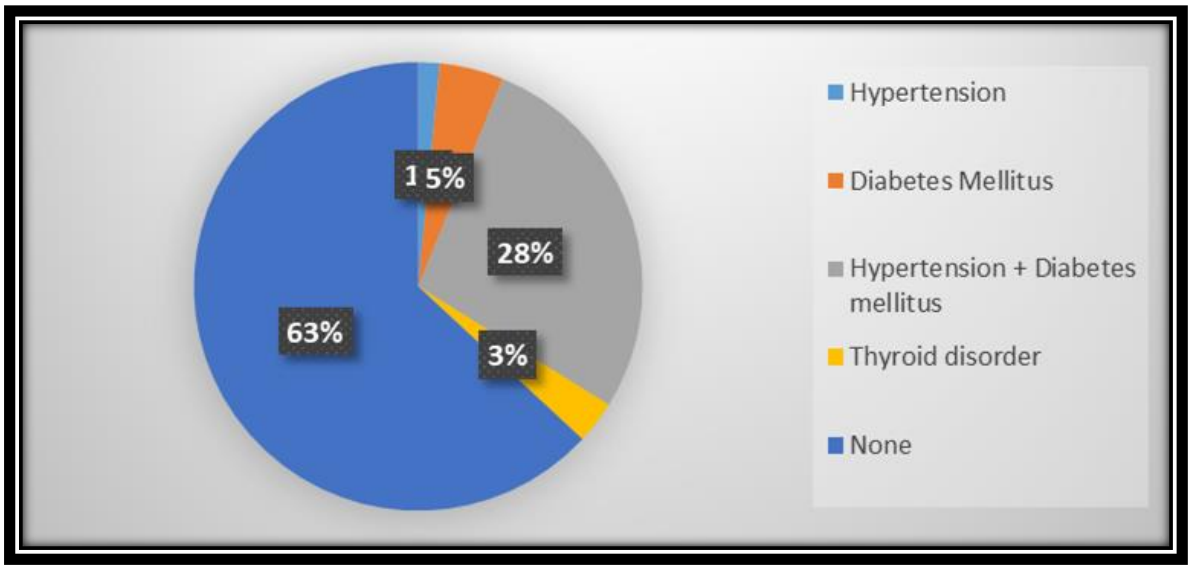

Figure 24: Comorbidities among study participants

Among the study participants, 28% patients had history of hypertension as well as diabetes mellitus. While in 1 patient had history of hypertension only, 2 patient had history of Diabetes mellitus. Total 2 patient had history of thyroid disorder.

Table 14: Side of injury among study participants (n=65)

Side of injury	Frequency (%)
Left	34 (52.3)
Right	31(47.7)

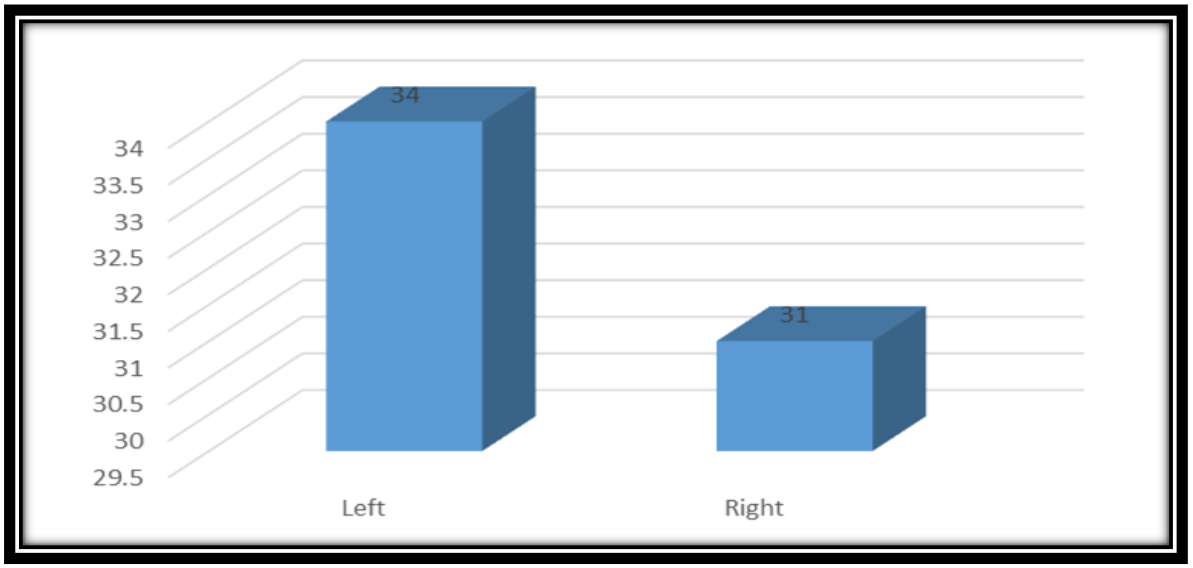

Figure 25: Side of injury among study participants

34 patients in the study had left-side involvement, while 31 patients had right-side involvement.

Table 15: Distribution as per Gustilo-Anderson Classification (n=65)

Gustilo Anderson Classification	Frequency (%)
1	22(33.84)
2	28(43.1)
3A	10(15.4)
3B	4(6.2)
3C	1 (1.5)

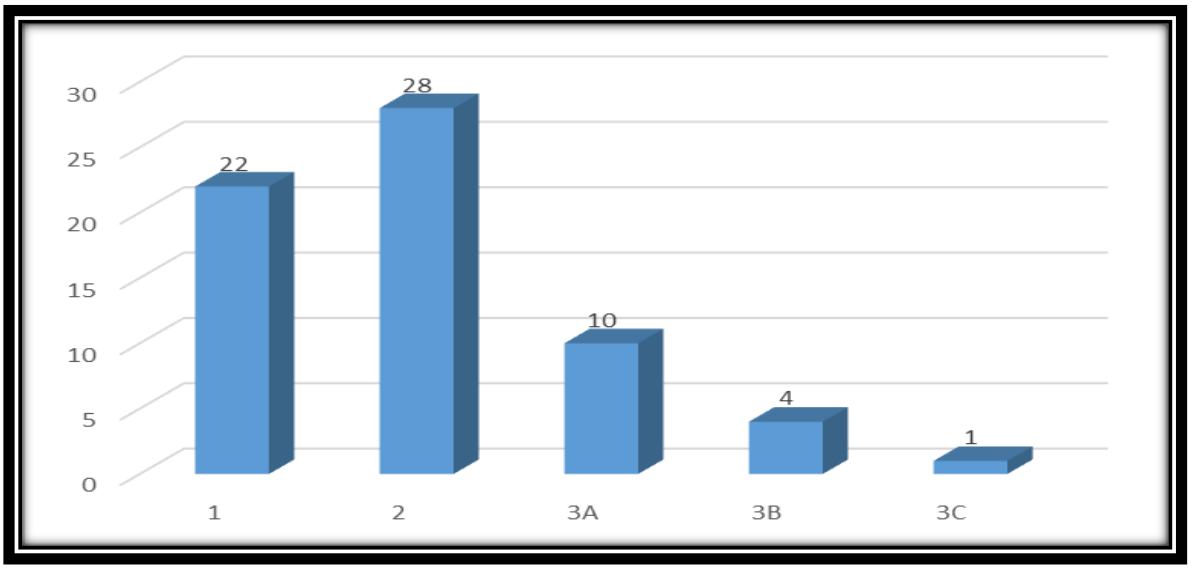

Figure 26: Gustilo and Anderson classification among study participants

In the study, 28 patients had grade 2, while in 22 patients had grade 1 Open fracture as per Gustilo Anderson classification. Only single patient had grade 3C Open fracture among study participants.

Table 16: Affected limb among study participants (n=65)

Affected limb	Frequency (%)
Upper	20(31)
Lower	45(69)

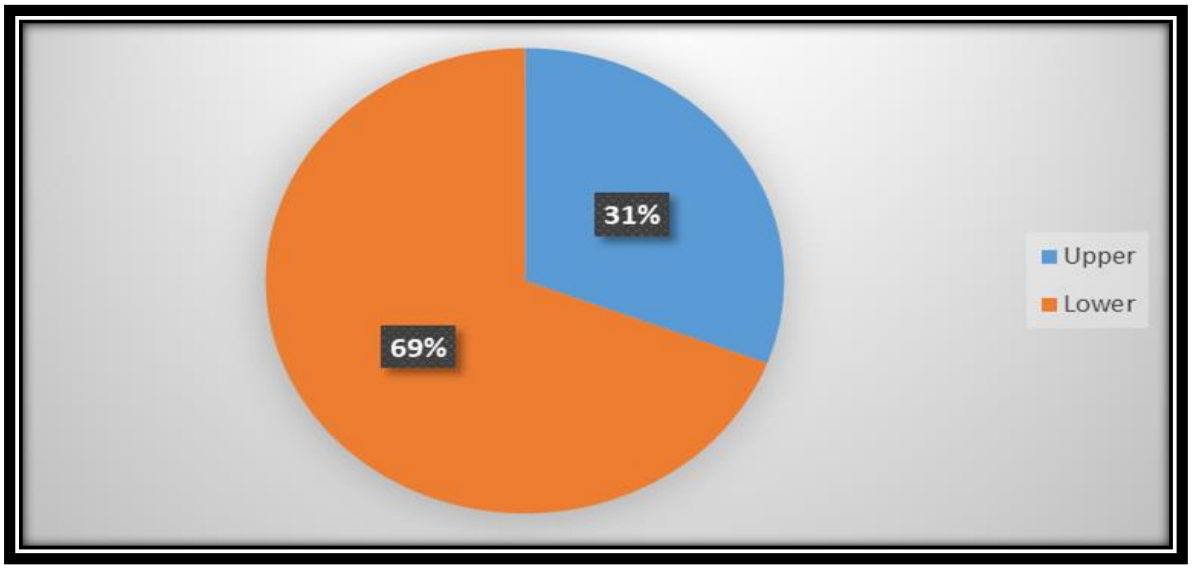

Figure 27: Affected limb among study participants

Among the study participants, 31% were affected on upper limb, while remaining 69% were affected on a lower limb.

Table 17: Pre-debridement Growth of microorganism (n=65)

Pre-debridement Growth	Frequency (%)
Acinetobacter species	9 (13.8)
Enterobacter species	9 (13.8)
Staphylococcus aureus	10 (15.3)
Klebsiella species	6 (9.2)
Pseudomonas species	9 (13.8)
None	22 (33.8)

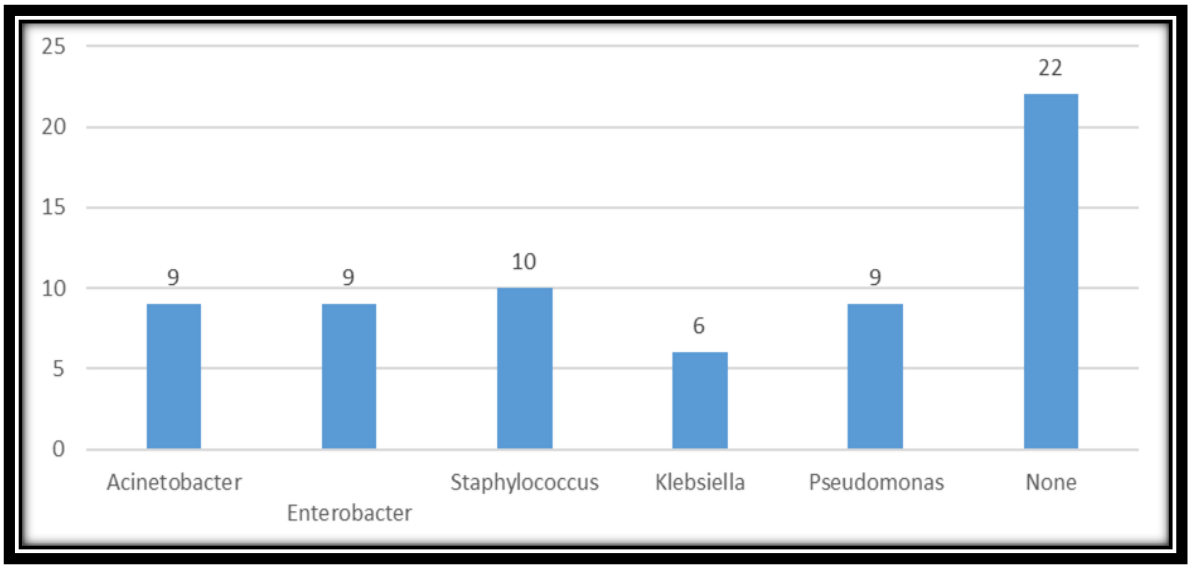

Figure 28: Pre-Debridement growth of micro-organisms among study participants

Out of total, in Pre-debridement culture majority cases had presence of growth of S*taphylococcus* followed by *Acinetobacter, Enterobacter, and Pseudomonas*. Only in 6 patients had growth of *Klebsiella.*

Table 18: Post-Debridement Growth of microorganism (n=65)

Post debridement Growth	Frequency (%)
Acinetobacter species	2(3)
Enterobacter species	4 (6.2)
Proteus vulgaris	2 (3)
Klebsiella species	2 (3)
Pseudomonas species	3 (4.6)
None	52 (80)

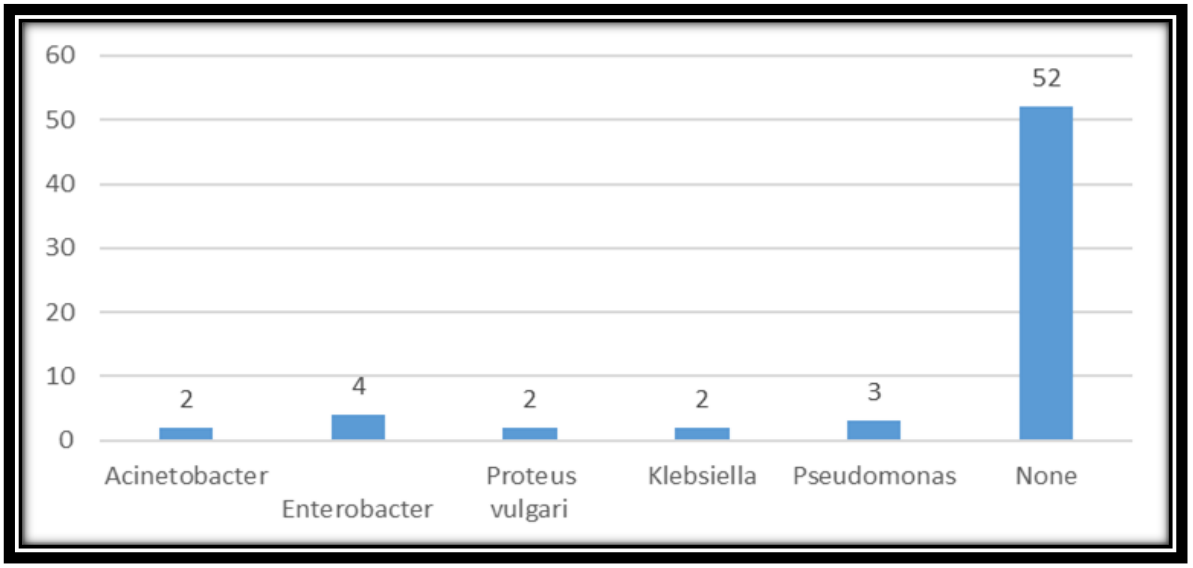

Figure 29: Post-Debridement growth of micro-organisms among study participants

After post debridement, there was only in 13 patients culture found the growth of microorganisms. Most common species found was *Pseudomonas* followed by *Acinetobacter, Proteus vulgaris, Klebsiella,* and *Enterobacter.*

Table 19: Growth of Microorganism in subsequent Culture (n=65)

Growth of Microorganism	Frequency (%)
Pseudomonas species	2 (3)
Enterobacter species	1 (1.5)
Proteus	1 (1.5)
None	61 (94)

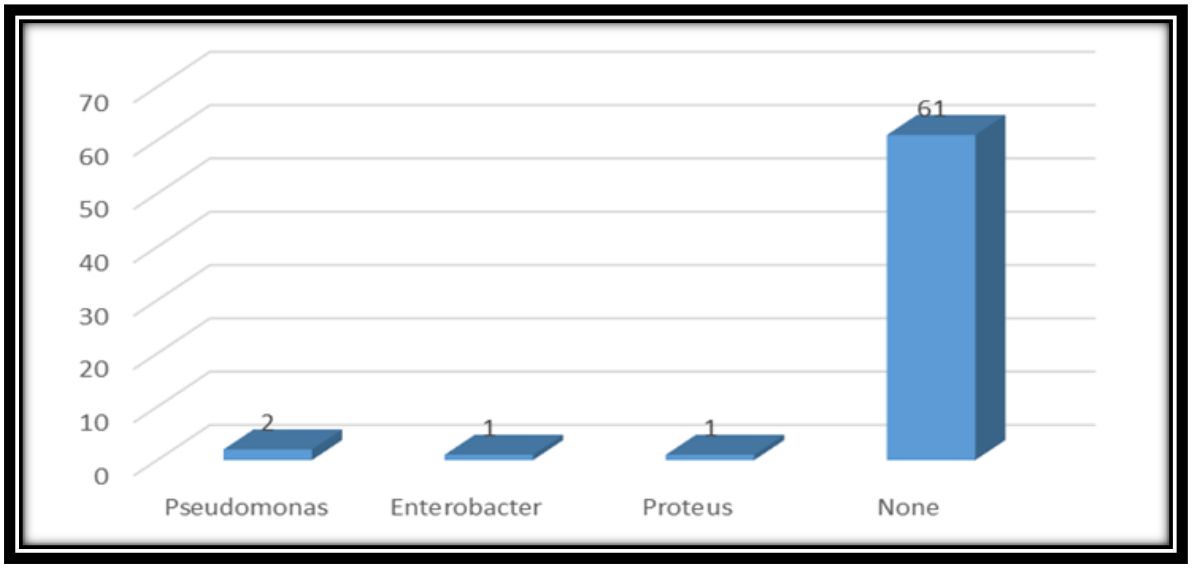

Figure 30: Subsequent culture findings among study participants

After debridement treatment for bacterial infection, on subsequent culture examination, no growth found among 61 patients. Although in 4 patients, there was presence of *Pseudomonas, Enterobacter and Proteus microorganism.*

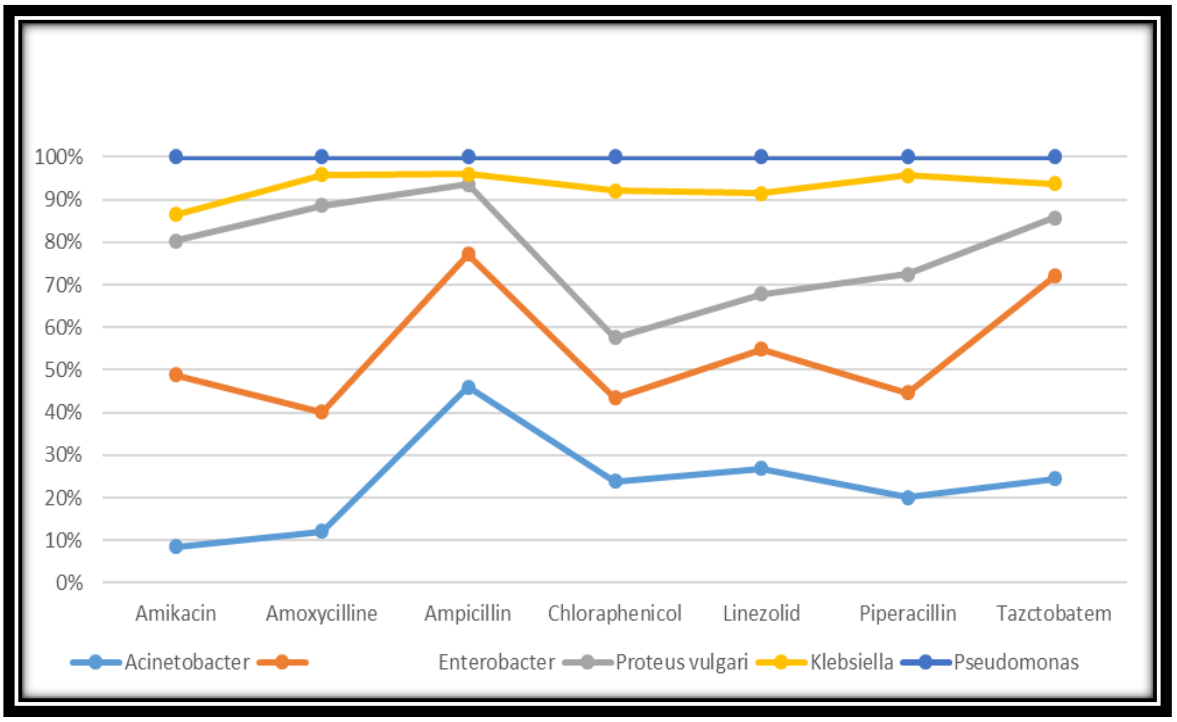

Figure 31: Antibiotic sensitivity among study participants

Overall the sensitivity was higher in Piperacillin and Tazobactam followed by less sensitivity was found in Amikacin.

Figure 32: Growth of *Staphylococcus aureus* on Blood Agar displaying beta haemolytic properties

Figure 33: Growth of *Klebsiella species*- Mucoid lactose fermenting colonies

Figure 34: Growth of *Acinetobacter species* – Non lactose fermenting colonies

Figure 35: Growth of *Pseudomonas species*– Non lactose fermenting colonies

DISCUSSION

DISCUSSION

Open fractures are often caused by extreme-velocity injury and can range in severity from mild to severe soft tissue and skeletal injury, both of which impair the local tissue's vascularity [65]. Due to the transmission of the bacteria, all open fracture wounds should be considered infected fracture site and the environment beyond [66]. As a result of the microbial contamination of the wound and the region's weakened vascular supply, there is a higher risk of infection and difficulties with healing [67]. Exogenous or endogenous wound contamination can happen in traumatic wounds. Depending on whether contamination occurs at the moment of injury, immediately after damage, or occurs 24 hours or more after injury, it may be categorised as primary or secondary [68].

Various genera of organisms have been recovered from cultures that were collected from different fracture sites. However, the repeated isolation of certain aerobic or anaerobic microbial isolates suggests that fracture sites may have been contaminated by skin, faeces, or the environment. Based on the types of organisms generating infections compared to those discovered on initial wound cultures, numerous articles have suggested that many infections in open fractures are nosocomial [69]. The results of the test should enable the surgeon to modify the course of therapy in some manner to enhance the outcome, or at the very least, to predict the severity or future course of the patient's illness. Due to the variance in bacterial frequency in various countries, as well as between hospitals within the same nation, wound-infecting pathogens vary [70].

Compound fractures are fractures that expose the bone and connect with the outside world through a wound. According to reports, bacterial contamination happens in 60–70% of the instances, which may be the cause of the infectious issues these patients experience. The exposed fracture site, the presence of devascularized cells, the extent of the external wound, the presence of coexisting conditions in the participant, the patient's immune status, the patient's delayed admission to the hospital, and the prolongation in starting treatment are a few factors that influence the likelihood of infection. Due to the breakdown of skin integrity and exposing of the subcutaneous tissue, microorganisms can colonise and grow more quickly in warm, favourable settings [71].

Without intervention and treatment with preventive antibiotics and surgical debridement, the presence of a foreign body, dead tissue, and devitalized tissue in a traumatic fracture wound create a perfect habitat for microbial growth and the formation of infection. Once an infection is developed, wound healing is slowed down, medical expenses increase, and wound care procedures get trickier [72]. The avoidance of infection, successful bone union, and function restoration are the aims of open fracture care.

Even if it merely affects the epidermal layer, a wound compromises the soft tissue integrity [73]. An open fracture might result from a soft tissue breach that exposes the underlying bones or joints to the outside environment. The main goals of treating open fractures are to treat the whole wound and stop initial contamination from turning into an infection [74].

For Gustilos' type I fractures, positive culture rates in open fractures range from 0% to 2%, for type II fractures, from 2% to 10%, and for type III fractures, from 10% to 50%. [75]. In the past, open fractures might be quite fatal and necessitated an immediate amputation. Without antibiotics, relatively few people could survive death after amputation [76].

When treating open fractures, initial antibiotic treatment is crucial, and the infection incidence can be greatly decreased when combined with prompt and thorough debridement [77]. Debridement is the process of removing dead or lifeless tissue from a wound [78].

Over the last century, effective antibacterial development has decreased the frequency of fatal illnesses, but the emergence of resistance has hidden this accomplishment [79]. Although the concept of the prudent use of antibiotics and recommendations for infection management have been extensively disseminated, recommendations are frequently ignored.

Antibiotic resistance, according to the World Health Organization, poses a serious concern today and may signal the beginning of a post-antibiotic age in which common diseases and minor injuries may once again endanger human life [80]. Multidrug-resistant (MDR) super infections and an increase in antibiotic-resistant microbial ecosystems have resulted from the indiscriminate, excessive, and inappropriate use of antibiotics globally [81].

The combination of this infectious consequence and antibiotic resistance poses a serious danger to the healthcare system. For the treatment of open fractures, it is

crucial to have current information about the range of pathogenic organisms as well as their current pattern of resistance. In adults, tibia shaft fractures make up 44.4% of all open long-bone fractures and 2% of all fractures [82, 83].

In present study, majority of cases (26.15%) were belonged to 21-30 years of age group. Total 14 participants belonged to 41-50 years of age group. Out of total, 9 patients were aged less than 20 years. Only 2 patients were aged more than 80 years. Participants in the study had an average age of 40.11 + 17.5 years. While in **Singh G et al**, [77] study, the median patient age was 36.98 years old, with a range of 7 to 70. In research of **Khatod M et al**, [78] the mean age of study participants was 29.7 $\pm$ 15.4 years.

Table 20: Comparison of mean age of patients among various studies

	Mean age (in years)
In present study	40.11
Singh G et al in his study, [77]	36.98
Khatod M et al in his study, [78]	29.7

In the study, 15% of participants were women and 85% of participants were men. Compared to women, there were more men. Studies by **Singh J et al**. [79] and **Fernandes MC et al**. [80] found similar results.

Table 21: Comparison of Gender among various studies

	Male	**Female**
In present study	85%	15%
Singh J et al in his study, [79]	87.4%	12.6%
Fernandes MC et al in his study [80]	86.3%	13.7%

One patient out of the total had a fall from a height as their cause of injury, road traffic accidents caused 80% of patient injuries, and 11 patients had workplace injuries. Remaining 1 patient was injured due to fall of heavy object. Although **Roth et al**. [81] indicated that assault (3.3%), falls from height (6.7%), fire arm injuries (15%), and roadside accidents (68%) were the most common modes of injury.

Table 22: Comparison of Mode of injury among various studies

Mode of Injury	**In present study**	**Roth et al in his study [81]**
Fall from Height	3.1%	6.7%
Road traffic accident	80%	68%
Injured at work place	16.9%	18.3%

In current research, 28% patients had history of hypertension as well as diabetes mellitus. While in 1 patient had history of hypertension only, 2 patient had history of Diabetes mellitus. Total 2 patient had history of thyroid disorder. 34

patients in the study had left-side involvement, while 31 patients had right-side involvement. In the study, 28 patients had grade 2, while in 22 patients had grade 1 Open fracture as per Gustilo & Anderson classification. Only one patient had grade 3C Open fracture among study participants. In the study by **Fred Sitt et al**, [82] of the fractures, 18 (18.36%) were Gustilo I, 38 (39.8%) were grade II, 22 (22.4.0%) were grade IIIA, and 19 (19.4%) were grade IIIB. Tibia/fibula fractures accounted for 43.9% of all fractures.

According to **Cherian JJ et al** in his study, [83] The majority of open fractures in the study (63.3%) were of type 2, followed by type 3A (20%) and 3B (16.7%). Among the study participants, 31% were affected on upper limb, while remaining 69% were affected on a lower limb in this study. In study of **Dellinger et al**, [84] total 50.6% cases were affected in lower limb as well as in study of Fernandes et al [80] also observed most common site of injury to be lower limb in 62.2% of cases studied.

Table 23: Comparison of Grade of fracture as per Gustilo Anderson Classification among various studies

Gustilo Anderson Classification	In present study	Fred Sitt et al in his study [82]	Cherian JJ et al in his study [83]
1	33.84%	18.36%	-
2	43.1%	39.8%	63.3%
3A	15.4%	22.4%	20%
3B	6.2%	19.4%	16.7%
3C	1.5%	-	-

Table 24: Comparison of affected limb among various studies

Affected limb	In present study	Fernandes et al in his study [80]	Dellinger et al in his study [84]
Upper	31%	37.8%	48.4%
Lower	69%	62.2%	50.6%

In present study, in pre-debridement culture majority cases had presence of growth of *Staphylococcus* followed by *Acinetobacter, Enterobacter, and Pseudomonas.* Only in 6 patients had growth of *Klebsiella*. After post debridement, there was only in 13 patients culture found the growth of microorganisms. Most common species found was *Pseudomonas* followed by *Acinetobacter*, *Proteus vulgaris*, *Klebsiella*, and *Enterobacter*. After debridement treatment for bacterial infection, on subsequent culture examination, no growth found among 61 patients. Although in 4 patients, there was presence of *Pseudomonas*, *Enterobacter* and *Proteus* microorganism.

E. coli made up the majority of the gram-negative bacteria in the study by Lakshminarayan et al. [85], with *Pseudomonas* (13.6%), *Proteus* (4.6%), and *Klebsiella* (10.2%) of the population growing next, *S. aureus and hemolytic streptococci*, on the other hand, were isolated from 40.9% and 2.3% of the population, respectively. **Johnson et al.** [86] detected *S aureus* in 18.2% of the gram-positive population. *Acinetobacter* (19.7%) was the most abundant gram-negative bacterium, followed by *Pseudomonas* (12.1%). In comparison to 4.54% of samples that had *Klebsiella* and *E. coli*, 9.09% of samples had *Enterobacter* growth. According to **Agrawal et al**. [87], the two most common gram-negative bacteria were *E Coli*

(34.2%) and *Pseudomonas* (26.1%). *Klebsiella* growth was found in 8% of cases, followed by *Proteus* in 6.3%.

Table 25: Comparison of Pre-Debridemental Growth of microorganisms among various studies

Pre-debridement Growth	**In present Study**	**Lakshminarayan et al in his study [85]**	**Johnson et al. in his Study [86]**	**Agrawal et al in his study [87]**
Acinetobacter species	13.8%	-	19.7%	-
Enterobacter species	13.8%	-	9.09%	34.2%
Staphylococcus aureus	15.3%	40.9%	18.2%	-
Klebsiella species	9.2%	10.2%	4.54%	8%
Pseudomonas species	13.8%	13.6%	12.1%	26.1%
Proteus	-	4.6%	-	6.3%
None	33.8%	-	-	-

CONCLUSION

CONCLUSION

- There is a considerable danger of infection and other problems from open fracture wounds.
- The treatment of open fractures focuses on timely wound closure, proper antibiotic medication, and efficient wound debridement.
- The management of infection depends heavily on diagnostic microbiology. When compared to pre-debridement cultures, cultures collected during debridement are found to be more sensitive in predicting the infection rate.
- Although the validity of sequential cultures has been questioned in a number of investigations, this study has demonstrated that debridement cultures have a significant impact in the prediction of postoperative infection.
- Debridement culture is therefore advised to offer information about the selection of antimicrobial medication, which when paired with a complete wound debridement will permit an early wound closure and better functional outcome.

LIMITATIONS
&
RECOMMENDATIONS

LIMITATIONS AND RECOMMENDATIONS

- This study was conducted among smaller sample size so, observations will not be generalized. Large scale study will be required.
- No long term follow-up of patients was done, therefore consequences will not be clear.
- It is a single center study.
- All patients of history of open Fracture and admitted within 6 hours included, if patient had history of Comorbidity like diabetes and immunocompromised state then there is more chances of infection.

SUMMARY

SUMMARY

A prospective Comparative study was conducted among 65 patients of open fractures admitted at Department of Orthopaedics, R. L. Jalappa hospital attached to Sri Devaraj Urs Academy of Higher Education and Research Tamaka, Kolar.

- Among the study participants, majority of cases (26.15%) were belonged to 21-30 years of age group. Total 14 participants belonged to 41-50 years of age group. Out of total, 9 patients were aged less than 20 years.
- Out of total, 85% were males and 15% were females in the study.
- Out of total, in 1 patient mechanism of injury was fall from height, In 80% patients mechanism was road traffic accident and 11 patients had injured at work place. Remaining 1 patient was injured due to fall of heavy object.
- Among the study participants, 28% patients had history of hypertension as well as diabetes mellitus. While in 1 patient had history of hypertension only, 2 patient had history of Diabetes mellitus. Total 2 patient had history of thyroid disorder.
- In the study, 28 patients had grade 2, while in 22 patients had grade 1 Open fracture as per Gustilo Anderson classification. Only one patient had grade 3C Open fracture among study participants.
- After post debridement, there was only in 13 patients culture found the growth of microorganisms. Most common species found was *Pseudomonas* followed by *Acinetobacter species, Proteus vulgaris, Klebsiella species, and Enterobacter species.*
- After debridement treatment for bacterial infection, on subsequent culture

examination, no growth found among 61 patients. Although in 4 patients, there was presence of *Pseudomonas species, Enterobacter species* and *Proteus* species.

- Overall the sensitivity was higher in Piperacillin and Tazobactam followed by less sensitivity was found in Amikacin.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Court-Brown CM, KE Bugler, ND Clement, AD Duckworth, MM McQueen. The epidemiology of open fractures in adults A 15-year review. Injury. 2012;43(6):891–7.
2. Larsen P, R Elsoe, SH Hansen, T Graven-Nielsen, U Laessoe, S Rasmussen. Incidence and epidemiology of tibial shaft fractures. Injury. 2015; 46(4):746–50.
3. Peltier LF. Fractures: a history and iconography of their treatment. Norman Publishing; 1990.
4. Gustilo RB, JT Anderson. Prevention of infection in the treatment of one thousand and twenty-five open fractures of long bones: retrospective and prospective analyses. J Bone Joint Surg Am. 1976;58(4): 453–8.
5. Cross WW, 3rd, Swiontkowski MF. Treatment principles in the management of open fractures. Indian J Orthop. 2008; 42: 377–86.
6. Lee J. Efficacy of cultures in the management of open fractures. Clin Orthop. 1997; 339: 71–5.
7. Ikem IC, Oginni LM, Bamgboye EA, Ako-Nai AK, Onipede AO. The bacteriology of open fractures in Ile-Ife, Nigeria. Niger J Med. 2004; 13: 359–65.
8. Ojo OD, Oluwadiya KS, Ikem IC, Oginni LM, Ako-Nai AK, Daniel FV. Superficial swab cultures in open fracture management: Insights from a resource-poor setting. J Wound Care. 2010;19: 432–8.

9. Robinson D, On E, Hadas N, Halperin N, Hofman S, Boldur I. Microbiologic flora contaminating open fractures: Its significance in the choice of primary antibiotic agents and the likelihood of deep wound infection. J Orthop Trauma. 1989;3: 283–6.
10. Heitmann C, Patzakis MJ, Tetsworth KD, Levin LS. Musculoskeletal sepsis: Principles of treatment. Instr Course Lect. 2003; 52: 733–43.
11. Seekamp A, Köntopp H, Schandelmaier P, Krettek C, Tscherne H. Bacterial cultures and bacterial infection in open fractures. Eur J Trauma. 2000; 26: 131–8.
12. Faisham WI, Nordin S, Aidura M. Bacteriological study and its role in the management of open tibial fracture. Med J Malaysia. 2001;56:201–6
13. Kreder HJ, Armstrong P. The significance of perioperative cultures in open pediatric lower-extremity fractures. Clin Orthop. 1994; 302: 206–12.
14. D'souza A, Rajagopalan N, Amaravati RS. The use of qualitative cultures for detecting infection in open tibial fractures. J Orthop Surg (Hong Kong) 2008; 16: 175–8.
15. The validity of the mangled extremity severity score in the assessment of upper limb injuries Togawa S., Yamami N., Nakayama H., Mano Y., Ikegami K., and Ozeki S. The Journal of Bone and Joint Surgery. British volume 2005 87-B:11, 1516-1519
16. Jenkins PJ, Keating JF, Simpson AH. Fractures of the tibial shaft. Surgery 2010; 28: 489-93.

17. Wagirayezu, E., Dushimiyimana, J.M.V. and Bonane, A. (2008) Open Fractures I Rwanda: The Kigali Experience. East and Central African Journal of Surgery, 13, 77-84.
18. Paul H. Kim, MD and Seth S. Leopold, MD. Gustilo-Anderson Classification. Clin Orthop Relat Res. 2012 Dec; 470(12): 3624. Published online 2012 Oct 6.
19. Ortho Bullets. https://www.orthobullets.com/trauma/1002/tscherne-classification. [Online] [accessed on 01/12/2022]
20. Rajasekaran, S., Sabapathy, S.R., Dheenadhayalan, J. et al. Ganga hospital open injury score in management of open injuries. Eur J Trauma Emerg Surg 41, 3–15 (2015).
21. Loja MN, Sammann A, DuBose J, Li CS, Liu Y, Savage S, Scalea T, Holcomb JB, Rasmussen TE, Knudson MM, AAST PROOVIT Study Group. The mangled extremity score and amputation: time for a revision. The journal of trauma and acute care surgery. 2017 Mar;82(3):518.
22. Fleming A. ON THE BACTERIOLOGY OF SEPTIC WOUNDS. The Lancet. 1915 Sep 18;186(4803):638–43.
23. Edwards R, Harding KG. Bacteria and wound healing. Curr Opin Infect Dis. 2004 Apr;17(2):91–6.
24. Otchwemah R, Grams V, Tjardes T, Shafizadeh S, Bäthis H, Maegele M, et al. Bacterial contamination of open fractures – pathogens, antibiotic resistances and therapeutic regimes in four hospitals of the trauma network Cologne, Germany. Injury. 2015 Oct 1;46: S104–8.

25. Patzakis MJ, Harvey JP, Ivler D. The role of antibiotics in the management of open fractures. J Bone Joint Surg Am. 1974 Apr;56(3):532–41
26. Glass GE, Barrett SP, Sanderson F, Pearse MF, Nanchahal J. The microbiological basis for a revised antibiotic regimen in high-energy tibial fractures: preventing deep infections by nosocomial organisms. J Plast Reconstr Aesthetic Surg JPRAS. 2011 Mar;64(3):375–80.
27. Laxminarayan R, Duse A, Wattal C, Zaidi AKM, Wertheim HFL, Sumpradit N, et al. Antibiotic resistance-the need for global solutions. Lancet Infect Dis. 2013 Dec;13(12):1057–98.
28. Robinson D, On E, Hadas N, Halperin N, Hofman S, Boldur I. Microbiologic flora contaminating open fractures: its significance in the choice of primary antibiotic agents and the likelihood of deep wound infection. J Orthop Trauma. 1989;3(4):283–6.
29. Alonge TO, Ogunlade SO, Salawu SA, Fashina AN. Microbial isolates in open fractures seen in the accident and emergency unit of a teaching hospital in a developing country. West Afr J Med. 2002 Dec;21(4):302–4.
30. Bergman BR. Antibiotic prophylaxis in open and closed fractures: a controlled clinical trial. Acta Orthop Scand. 1982 Feb;53(1):57–62.
31. Katoch R, Rajagopalan S. Warfare Injuries: History, Triage, Transport and Field Hospital Setup in the Armed Forces. Med J Armed Forces India. 2010 Oct;66(4):304–8.
32. Robertson-Steel I. Evolution of triage systems. Emerg Med J EMJ. 2006 Feb;23(2):154–5

33. Karamanou M, Rosenberg T, Liakakos T, Androutsos G. Baron Dominique-Jean Larrey (1766- 1842): founder of military surgery and trauma care. Chir Buchar Rom 1990. 2011 Feb;106(1):7– 10. 91
34. Bucholz RW. Rockwood and Green's Fractures in Adults: Two Volumes Plus Integrated Content Website (Rockwood, Green, and Wilkins' Fractures). Lippincott Williams & Wilkins; 2012. 2059.
35. Robinson PM, O'Meara MJ. The Thomas splint: ITS ORIGINS AND USE IN TRAUMA. Bone Jt J. 2009 Apr 1; 91–B(4):540–4.
36. Paul GW. The history of external fixation. Clin Podiatr Med Surg. 2003 Jan;20(1):1–8.
37. Uhthoff HK, Poitras P, Backman DS. Internal plate fixation of fractures: short history and recent developments. J Orthop Sci. 2006 Mar;11(2):118–26
38. Cronier P, Pietu G, Dujardin C, Bigorre N, Ducellier F, Gerard R. The concept of locking plates. Orthop Traumatol Surg Res OTSR. 2010 May 4
39. Bhandari M, Guyatt GH, Tornetta P, Swiontkowski MF, Hanson B, Sprague S, et al. Current practice in the intramedullary nailing of tibial shaft fractures: an international survey. J Trauma. 2002 Oct;53(4):725–32.
40. SPRINT Investigators, Bhandari M, Tornetta P, Rampersad S-A, Sprague S, Heels-Ansdell D, et al. (Sample) size matters! An examination of sample size from the SPRINT trial study to prospectively evaluate reamed intramedullary nails in patients with tibial fractures. J Orthop Trauma. 2013 Apr;27(4):183–8.
41. Godina M. Early microsurgical reconstruction of complex trauma of the extremities. Plast Reconstr Surg. 1986 Sep;78(3):285–92.

42. Gopal S, Majumder S, Batchelor AG, Knight SL, De Boer P, Smith RM. Fix and flap: the radical orthopaedic and plastic treatment of severe open fractures of the tibia. J Bone Joint Surg Br. 2000 Sep;82(7):959–66.
43. D'Alleyrand J-CG, Manson TT, Dancy L, Castillo RC, Bertumen JBH, Meskey T, et al. Is time to flap coverage of open tibial fractures an independent predictor of flap-related complications? J Orthop Trauma. 2014 May;28(5):288–93.
44. Trueta J. Treatment of War Wounds and Fractures. Br Med J. 1942 May 16;1(4245):616–7.
45. Kindsfater K, Jonassen EA. Osteomyelitis in grade II and III open tibia fractures with late debridement. J Orthop Trauma. 1995 Apr;9(2):121–7.
46. Kreder HJ, Armstrong P. A review of open tibia fractures in children. J Pediatr Orthop. 1995 Aug;15(4):482–8
47. Orthopaedic Trauma Association: Open Fracture Study G. A new classification scheme for open fractures. Journal of Orthopaedic Trauma. 2010;24(8):457-464
48. Blick SS, Brumback RJ, Poka A, Burgess AR, Ebraheim NA. Compartment syndrome in openTibial fractures. J Bone Joint Surg Am. 1986; 68:1348-53.
49. Tscherne H, Goetzen L, Fractures with soft tissue injuries, Berlin: Springer-Verlag, 1984.
50. Tetanus prophylaxis. Adopted from Rockwood and Green's fractures in adults, Fifth Edition, Vol.1, p 516.
51. Veliskakis KP: Primary internal fixationin open fractures of the tibial shaft: The problem of wound healing. J Bone Joint Surg Br 1959; 41:342-354.

52. Brumback RJ, Jones AL. Interobserver agreement in the classification of open fractures of the tibia. The results of a survey of two hundred and forty-five orthopaedic surgeons. J Bone Joint Surg Am. 1994; 76:1162-6.
53. Johansen K, Daines M, Howey T, Helfet D, Hansen ST Jr. Objective criteria accurately predict amputation following lower extremity trauma. J Trauma 1990; 30:568-72.
54. Slauterbeck JR, Britton C, Moneim MS, Clevenger FW. Mangled extremity severity score: An accurate guide to treatment of the severely injured upper extremity. J Orthop Trauma 1994; 8:282-5.
55. Hansen ST. Overview of the severely traumatized lower limb: reconstruction versus amputation. Clin Orthop 1989;143: 17-19.
56. Lange RH, Bach AW, Hansen ST Jr, Johansen KH. Open tibial fractures with associated vascular injuries: prognosis for limb salvage. J Trauma 1985; 25:203-8.
57. Bosse MJ, McCarthy ML, Jones AL, Webb LX, Sims SH, Sanders RW, et al . The insensate foot following severe lower extremity trauma: An indication for amputation? J Bone Joint Surg Am 2005; 87:2601-8.
58. Taufik A, Wiweko A, Yudhanto D, et al. Bacterial infection and antibiotic resistance pattern in open fracture cases in Indonesia. Annals of Medicine and Surgery (2012). 2022 Apr;76:103510.
59. Naique SB, Pearse M, Nanchahal J. Management of severe open tibial fractures: the need for combined orthopaedic and plastic surgical treatment in specialist centres. J Bone Joint Surg Br. 2006 Mar;88 (3):351-7.

60. Zalavras CG, Patzakis MJ. Open fractures: evaluation management. J Am Acad Orthop Surg. 2003;11(3):212-9.
61. Apurba S Sastry, Sandhya Bhat. Essentials of Medical Microbiology. Third edition. New Delhi: Jaypee Brothers Medical Publishers;2021. Chapter 3.2, Morphology and Physiology of Bacteria; p 15-24.
62. Apurba S Sastry, Sandhya Bhat. Essentials of Medical Microbiology. Third edition. New Delhi: Jaypee Brothers Medical Publishers;2021. Chapter 3.3, Laboratory Diagnosis of Bacterial Infections; p 25-50.
63. Retty AF, Daniel FS and Aice SW. Bailey and Scotts of Diagnostic Microbiology. 14th ed. Press, Houston, Texas. 2017.
64. Kalyani R, Arvind N, Kumar NS, Reddy MM, Dinesh K. Bacterial Colonization of Intensive Care Unit Environment and Healthcare Workers in A Tertiary Care Hospital in Kolar Region, India. J Pure Appl Microbiol. 2021;15(1):402-409.
65. George W, Wood II. Campbell's Operative Orthopaedics 2013 Vol I12th edPAElsevier:2561-2587.
66. M.C.Y. Heng. Wound healing in adult skin: aiming for perfect regeneration Int J Dermatol, 50 (9) (2011), pp. 1058-1066.
67. A. Diwan, K.R. Eberlin, R.M. Smith. The principles and practice of open fracture care, 2018 Chin J Traumatol, 21 (4) (2018), pp. 187-192.
68. C.G. Zalavras. Prevention of Infection in Open Fractures. Infect Dis Clin North Am, 31 (2) (2017), pp. 339-352.

69. H.L. Kakria. Evolution in Fracture Management. Med J Armed Forces India, 61 (4) (2005), pp. 311-312.
70. W.W. Cross, M.F. Swiontkowski.Treatment principles in the management of open fractures. Indian J Orthop, 42 (4) (2008), pp. 377-386.
71. F. Bekara, J. Vitse, S. Fluieraru, R. Masson, A. Runz, V. Georgescu, et al. New techniques for wound management: A systematic review of their role in the management of chronic wounds. Arch Plast Surg, 45 (2) (2018), pp. 102-110.
72. R.J. Fair, Y. Tor.Antibiotics and bacterial resistance in the 21st century.Perspect Med Chem, 6 (2014), pp. 25-64.
73. D.J. Bryson, D.L. Morris, F.S. Shivji, K.R. Rollins, S. Snape, B.J. Ollivere. prophylaxis in orthopaedic surgery: difficult decisions in an era of evolving antibiotic resistance Bone Joint J, 98-B (8) (2016), pp. 1014-1019.
74. C. Zhou, X. Chen, L. Wu, J. Qu. Distribution of drug-resistant bacteria and rational use of clinical antimicrobial agents. Exp Ther Med, 11 (6) (2016), pp. 2229-2232.
75. R.J. Weiss, S.M. Montgomery, A. Ehlin, Z. Al Dabbagh, A. Stark, K.A. Jansson Decreasing incidence of tibial shaft fractures between 1998 and 2004: information based on 10,627 Swedish inpatients Acta Orthop, 79 (4) (2008), pp. 526-533.
76. C.Papakostidis, N.K. Kanakaris, J. Pretel, O. Faour, D.J. Morell, P.V. Giannoudis. Prevalence of complications of open tibial shaft fractures stratified as per the Gustilo-Anderson classification Injury, 42 (12) (2011), pp. 1408-1415.

77. Singh G, Singh M, Dahuja A,Kuar R, Radheshyam, kaur H, Shpra. To study the changing pattern of bacterial flora andtheir sensitivity patterns in grade IIIB open fractures of long bones: a prospective study at a Tertiary Centre in North India. European Journal of Molecular & Clinical Medicine. ISSN 2515-8260 Volume 08, Issue 03, 2021
78. Khatod M, Botte MJ, Hoyt BD,Meyer RS, Smith MJ,Akeson WH. Relationship between delay in treatement and infection. J trauma. 2003;5(55):949-954.
79. Singh J, Rambani R, Hashim Z, Raman R, Sharma HK. The relationship between time to surgical debridement and incidence of infection in grade III open fracture Strat Traum Limb Recon (2012) 7:33– 37.
80. Fernandes MC, Peres LR, Neto AC, José Queiroz Lima Neto, Turíbio FM, Matsumoto MH.Open fractures and the incidence of infection in the surgical debridement 6 hours after trauma acta ortop bras. 2015;23(1):38-42
81. Roth AI, Fry DE, Polk HC. Jr. Infectious morbidity in extremity fractures. J Trauma. 1986;26(8):757-61.
82. Dr. Fred Sitat. Early Bacterial Cultures from Open Fractures -Differences Before and After Debridement. The ANNALS of AFRICAN SURGERY, July 2017 Volume 14 Issue 2
83. Cherian JJ, Lobo JO, Ramesh LJ. A Comparative Study of Bacteriological Culture Results Using Swab and Tissue in Open Fractures: A Pilot Study. *J Orthop Case Rep*. 2019;9(1):33-36.

84. Dellinger EP, Miller SD, Wertz MJ, Grypma M, Droppert B, Anderson PA. Risk of infection after open fracture of the arm or leg. Arch Surg. 1988 ;123(11):1320-27.
85. Lakshminarayana SA, Chavan D, Prakash R, Sangeetha S .Bacteriological Profile of Orthopedic Patients in a Tertiary Care Hospital, Bengaluru. International Journal of Science and Research. 2015;4 (6):1504-08.
86. Johnson EN, Burns TC, Hayda RA, Hospenthal DR, Murray CK. Infectious Complications of Open Type III Tibial Fractures among Combat Casualties Clinical Infectious Diseases 2007; 45:409–15.
87. Agrawal AC, Jain S, Jain RK, Raza HKT. Pathogenic bacteria in an orthopaedic hospital in India. J Infect Developing Countries 2008;2(2):120-2

ANNEXURE

ANNEXURE 1

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

PATIENT INFORMATION SHEET

STUDY TITLE: "A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES"

Study location: R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar.

Details- Patients diagnosed with open fractures of extremities admitted in Orthopaedics ward from OPD and Emergency Medicine Department at R.L.J. HOSPITAL AND RESEARCH CENTRE, attached to SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR

Patients in this study will have to undergo routine Blood Investigations: -CBC, BT, CT, Blood grouping, RBS, RFT,HIV, HBsAg status. A total of 2 culture swabs will be taken. Pre-debridement sample will be taken at the time of presentation and Post-debridement sample will be taken in Operative room.

Please read the following information and discuss with your family members. You can ask any question regarding the study. If you agree to participate in the study we will collect information (as per proforma) from you or a person responsible for you or both. Relevant history will be taken. This information collected will be used publication.

All information collected from you will be kept confidential and will not be disclosed to any outsider. Your identity will not be revealed. This study has been reviewed by the Institutional Ethics Committee and you are free to contact the member of the

Institutional Ethics Committee. There is no compulsion to agree to this study. The care you will get will not change if you don't wish to participate. You are required to sign/ provide thumb impression only if you voluntarily agree to participate in this study.

CONFIDENTIALITY

Your medical information will be kept confidential by the study doctor and staff and will not be made publicly available. Your original records may be reviewed by your doctor or ethics review board. For further information/ clarification please contact

Dr. U. JAGADISH (Post Graduate),

Department of Orthopaedics,

SDUMC, Kolar

Mobile No: 9600222853

ಶ್ರೀದೇವರಾಜ್ ಅರಸ್ ಉನ್ನತ ಶಿಕ್ಷಣ ಮತ್ತು ಸಂಶೋಧನಾ ಸಂಸ್ಥೆ,

ಟಮಕ, ಕೋಲಾರ - 563101.

ತಿಳಿವಳಿಕೆ ಸಮ್ಮತಿ ನಮೂನೆ

ನಾನು, _____ ವಯಸ್ಸಿನ _____, ಅಧ್ಯಯನದ ಉದ್ದೇಶ ಮತ್ತು ಕಾರ್ಯವಿಧಾನದ ಅಪಾಯಗಳು ಮತ್ತು ಹೊಡಕುಗಳ ಬಗ್ಗೆ ನನ್ನ ಸ್ವಂತ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಿದ ನಂತರ, ಯಾವುದೇ ಬಲವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಿ ಅಥವಾ ಗಾಯದ ಮಾದರಿಗಳ ಪೂರ್ವ ವಿಘಟನೆ ಮತ್ತು ಪೋಸ್ಟ್ ಡಿಬ್ರೈಡ್ಮೆಂಟ್ ನನ್ನ ಮೇಲೆ ಪ್ರದರ್ಶನಗೊಳ್ಳಲಿ. ಕಾರ್ಯವಿಧಾನದಲ್ಲಿ ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ. ಕ್ಲಿನಿಕಲ್ ರಿಸರ್ಚ್ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ಅತಿಯಾದ ಮುಕ್ತ ರಚನೆಗಳಲ್ಲಿ ಪೂರ್ವ-ವಿಘಟನೆ ಮತ್ತು ಪೋಸ್ಟ್-ವಿಘಟನೆಯ ಸಂಸ್ಕೃತಿಯ ಪೂರ್ವಭಾವಿ ಹೋಲಿಕೆ ಅಧ್ಯಯನ " ನಡೆಸಲಾಗುತ್ತಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆ ಕೇಳುವ ಅವಕಾಶ ನನಗೆ ಸಿಕ್ಕಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪುತ್ತೇನೆ. ನನ್ನ ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಆಪರೇಟಿವ್ ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ವೈದ್ಯರಿಗೆ / ಸಂಸ್ಥೆಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ.

ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊ ಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಬಳಸಬಹುದು. ಕಾರ್ಯವಿಧಾನ / ಅಧ್ಯಯನದ ಸಮಯದಲ್ಲಿ ಯಾವುದೇ ಅಹಿತಕರ ಪರಿಣಾಮಗಳಿಗೆ ನಾನು ವೈದ್ಯರು / ಸಂಸ್ಥೆ ಇತ್ಯಾದಿಗಳನ್ನು ಹೊಣೆಗಾರರನ್ನಾಗಿ ಮಾಡುವುದಿಲ್ಲ.

ಭಾಗವಹಿಸುವವರಿಗೆ ಈ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ ನಮೂನೆ ಮತ್ತು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯ ನಕಲನ್ನು ಒದಗಿಸಲಾಗಿದೆ.

ಸಹಿ / ಹೆಬ್ಬೆರಳು ಅನಿಸಿಕೆ ಮತ್ತು ರೋಗಿಯ ಹೆಸರು ಸಹಿ ಮತ್ತು ಪಂ. ಅಟೆಂಡರ್

ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ:

ಸಾಕ್ಷಿ:

ಸಹಿ ಮತ್ತು ಸಂಶೋಧನಾ ವ್ಯಕ್ತಿ / ವೈದ್ಯರ ಹೆಸರು:

Annexure 2

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101

INFORMED CONSENT FORM

UHID NO:

TITLE: "A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES"

I, _____ aged _____ ,after being explained in my own vernacular language about the purpose of the study and the risks and complications of the procedure, hereby give my valid written informed consent without any force or Pre debridement and post debridement of wound samples to be performed on me. The nature and risks involved in the procedure have been explained to me to my satisfaction. I have been explained in detail about the Clinical Research on "**A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES**" being conducted. *I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I consent voluntarily to participate as a participant in this research.* I hereby give consent to provide my history, undergo physical examination, undergo the operative procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc.

For academic and scientific purpose the operation / procedure, etc may be video graphed or photographed. All the data may be published or used for any academic purpose. I will not hold the doctors / institute etc responsible for any untoward consequences during the procedure / study.

A copy of this Informed Consent Form and Patient Information Sheet has been provided to the participant.

Signature/Thumb impression & Name of patient Signature & Name of Pt. Attender

Relation with patient:

Witness:

Signature & Name of Research person /doctor:

Annexure 3 Kannada consent

Annexure 4

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH, TAMAKA, KOLAR - 563101.

PROFORMA

Case no:

IP no:

TITLE

"A PROSPECTIVE COMPARATIVE STUDY OF PRE-DEBRIDEMENT AND POST-DEBRIDEMENT CULTURE IN OPEN FRACTURES OF THE EXTREMITIES"

1. BASIC DATA

Name Age/Sex

Address

UHID no:

Date and Time of injury:

Nature of injury: RTA- 2wheeler 3wheeler 4wheeler

PEDESTRIANS- Peds+2W Peds+3W Peds+4W

FALL- Ht. Standing height

Assault

Date and time of admission to hospital: Date of discharge:

Nature of first aid: <6hrs/>6hrs

Tetanus prophylaxis:

Gas gangrene prophylaxis:

Splinting of limb:

Gustilo Anderson type: I II IIIA IIIB IIIC

Date and time from first aid to primary treatment:

Type of treatment Duration OT Blood transfusion

Wounds	Debridement	Fracture	Treatment type
	SSG	External Fixation	
	Flap	Nail	
	Amputation	Plate	
		Screws	
		K wires	
		Others	
Special procedure	Nerve repair		
	Vascular repair		

Date, time and type from primary treatment to secondary treatment Duration OT

Pre debridement samples Post debridement samples

	Microbiology	Culture no.	Organism	Sensitivity
	Pre debridement			
	Post debridement			
	Subsequent cultures			

GENERAL PHYSICAL EXAMINATION:

Vitals: Pulse- B.P-

RR- Temp-

SYSTEMIC EXAMINATION

CVS-

RS-

PS-

CNS-

Pre-existing systemic illness :

Diabetes/Thyroid disorder/ Cervical Spine/ CVS/RS/ CNS/locomotor/ TB/ anaemia/ Hypertension/ malnutrition/others

Local examination:

2. **DIAGNOSIS**:

3. INVESTIGATIONS:

- Blood Investigations:

  -CBC with ESR

  -CRP

  -BT,CT, Blood grouping

  -RBS, RFT

  -HIV, HBsAg status

Annexure 5
Case Photos

Case 1 photos

Figure 1:Pre-Debridement image

Figure 2: Performing debridement in Operation room

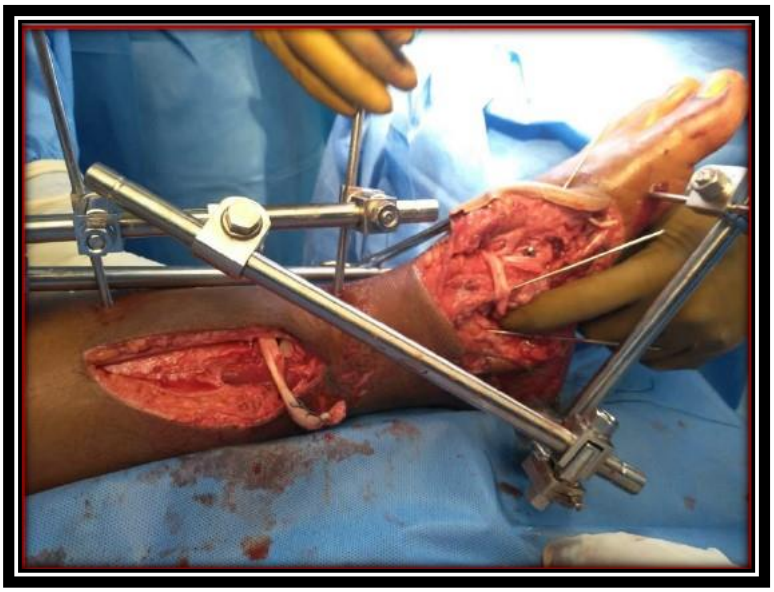

Figure: Post-Debridement image

Case 39 Photos

Figure: Pre-debridement image

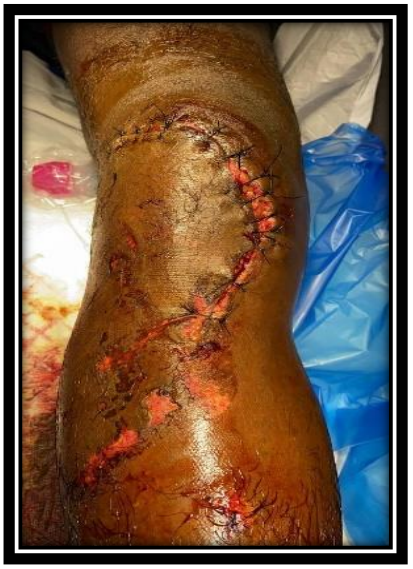

Figure: Post debridement image

Case 6 images

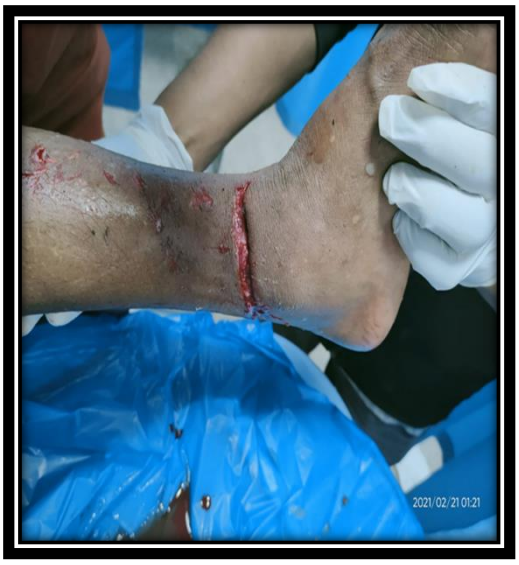

Figure: Pre debridement image

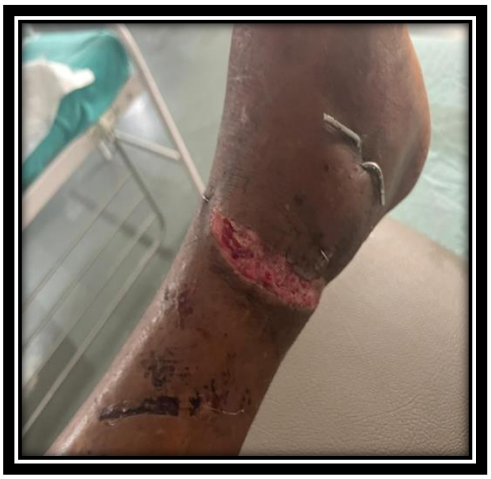

Figure: Post-Debridement image

Case 57 Images

Figure: Case of floating knee; Patient was initially stabilised with external fixator but further underwent below knee amputation due to monophasic vascularity

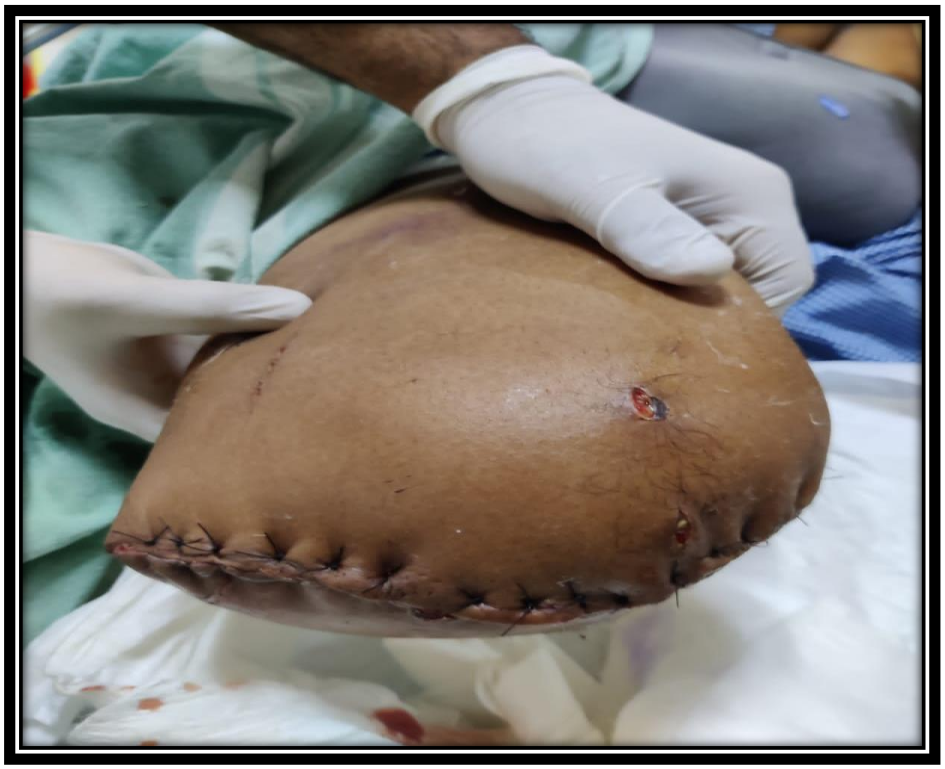

Figure: Post below knee amputation

Case 18 Images

Figure: Open type 3B injury of right humerus fracture; Pre-debridement image

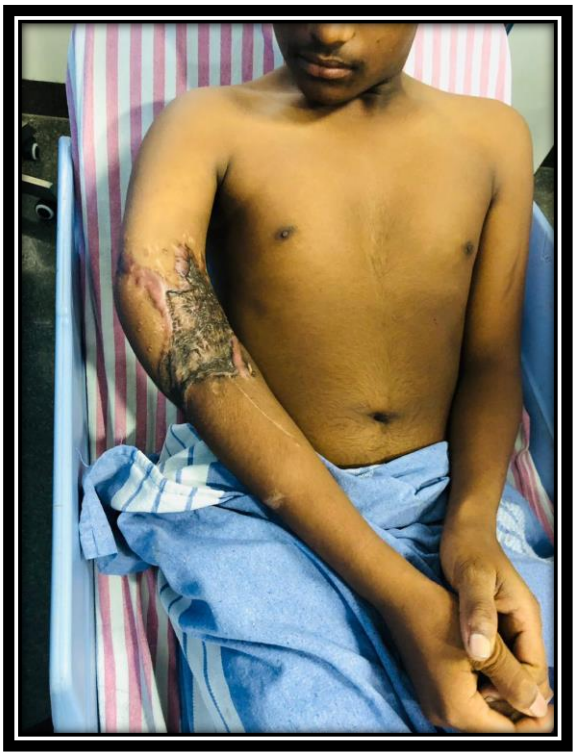

Figure: Post debridement image following which SSG done.

Annexure 6

KEY TO MASTER CHART

M	Male
F	Female
UHID NO.	Unique Hospital Identification
RTA	Road Traffic Accident
S.NO	Serial number
Pre-S	Pre-Debridement culture antibiotic sensitivity
Post-S	Post-Debridement culture antibiotic sensitivity
Subs-S	Subsequent-Debridement culture antibiotic sensitivity

SL. NO	UHID	AGE	SEX	MODE OF INJURY	COMORBIDITIES	SIDE OF INJURY	GUSTILO ANDERSON	LIMB	PRE DEBRIDEMENT	POST DEBRIDEMENT	SUBSEQUENT CULTURES	SUBSEQUENT CULTURE 2		DIAGNOSIS	ASSOCIATED INJURIES	pre	post	subse
1	877491	30	FEMALE	RTA	NIL	RIGHT	3B	LOWER LIMB	PSEUDOMONAS AERUGINOSA, ESCHERICHIA CHOLI	PSEUDOMONAS AERUGINOSA, KLEBSIELLA OXYTOCA	ESCHERICHIA COLI, PSEUDOMONAS AERUGINOSA	PROTEUS MARABILIS, KLEBSIELLA OXYTOCA		OPEN TYPE III B DISPLACED COMMINUTED RIGHT TIBIA FRACTURE	NIL	PIPERACILLIN AND TAZOBACTAM	PIPERACILLIN AND TAZOBACTAM	PIPERACILLIN AND TAZOBACTAM
2	892884	47	MALE	RTA	NIL	RIGHT	2	LOWER LIMB	NO GROWTH	NO GROWTH				OPEN TYPE II COMMINUTED FRACTURE OF RIGHT TIBIA AT DISTAL THIRD REGION WITH SEGMENTAL FIBULA FRACTURE	NIL			
3	897503	50	MALE	RTA	NIL	LEFT	2	LOWER LIMB	ACINETO BACTER	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED SHAFT OF LEFT TIBIA FRACTURE	OPEN 2ND AND 3RD METATARSAL FRACTURE OF LEFT FOOT	LEVOFLOXACIN		
4	900,016	20	MALE	RTA	NIL	RIGHT	2	LOWER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED SHAFT OF RIGHT FEMUR FRACTURE	NIL	PIPERACILLIN AND TAZOBACTAM		
5	913175	45	MALE	RTA	NIL	LEFT	2	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED SHAFT OF LEFT TIBIA FRACTURE	NIL			
6	918364	30	MALE	WORK PLACE INJRY	NIL	RIGHT	2	UPPER LIMB	NO GROWTH	KLEBSIELLA PNEUMONIAE	NIL	NIL		OPEN TYPE II DISPLACED RIGHT HUMERUS SHAFT FRACTURE	NIL		LEVOFLOXACIN	
7	917110	40	MALE	RTA	NIL	RIGHT	3A	LOWER LIMB	ESCHERICHIA CHOLI	NO GROWTH	NIL	NIL		OPEN TYPE III A DISPLACED COMMINUTED SHAFT OF RIGHT TIBIA	NIL	PIPERACILLIN		
8	925862	23	FEMALE	RTA	NIL	LEFT	2	LOWER LIMB	ACINETO BACTER	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED LEFT PROXIMAL TIBIA FRACTURE	NIL	PIPERACILLIN		
9	926646	65	MALE	RTA	DIABETES, HYPERTENSION	RIGHT	3A	LOWER LIMB	COAGULASE NEGATIVE STAPHYLOCOCCI	NO GROWTH	NIL	NIL		OPEN TYPE III A DISPLACED COMMINUTED RIGHT TIBIA FRACTURE	NIL	CIPROFLOXACIN		
10	929799	45	MALE	RTA	DIABETES	LEFT	3A	LOWER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE III A LEFT FEMUR SHAFT FRACTURE	CLOSED 2ND 3RD 4TH LEFT METACARPAL FRACTURE	PIPERACILLIN		
11	934637	17	MALE	RTA	NIL	RIGHT	3A	LOWER LIMB	KLEBSIELLA PNEUMONIAE	NO GROWTH	NIL	NIL		OPEN TYPE IIIA DISPLACED COMMINUTED RIGHT TIBIA SHAFT FRACTURE	NIL	PIPERACILLIN AND TAZOBACTAM		
12	936014	48	MALE	RTA	NIL	RIGHT	NIL	LOWER LIMB	PROVIDENCIA SPECIES, PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN 2ND 3RD 4TH 5TH RIGHT METATARSAL FRACTURE	NIL	PIPERACILLIN AND TAZOBACTAM		
13	929767	74	MALE	RTA	DIABETES, HYPERTENSION	RIGHT	3A	LOWER LIMB	CITROBACTER SPECIES, KLEBSIELLA PNEUMONIAE	NO GROWTH	NIL	NIL		OPEN TYPE III A DISPLACED RIGHT TIBIA FRACTURE	OPEN 2ND 3RD LEFT PHALYNX FRACTURE	MEROPENEM		
14	934334	44	MALE	RTA	NIL	LEFT	2	LOWER LIMB	ENTEROBACTER SPECIES	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT RADIUS SHAFT FRACTURE WITH ULNA SHAFT FRACTURE	NIL	GENTAMYCIN		
15	936669	59	MALE	RTA	DIABETES, HYPERTENSION	LEFT	3B	UPPER LIMB	METHICILLIN RESISTANCE STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE IIIB COMMINUTED LEFT TIBIA FRACTURE	OPEN 2ND 3RD LEFT PHALYNX FRACTURE	TETRACYCLINE		
16	944371	19	MALE	RTA	NIL	RIGHT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I RIGHT FEMUR SHAFT FRACTURE	CLOSED 2ND 3RD RIGHT METATARSAL FRACTURE			
17	942219	30	MALE	WORK PLACE INJRY	NIL	LEFT	3A	UPPER LIMB	NO GROWTH	PSEUDOMONAS AERUGINOSA, ACINETOBACTER SPECIES	NIL	NIL		OPEN TYPE IIIA LEFT HUMERUS SHAFT FRACTURE	NIL		PIPERACILLIN	
18	945840	60	FEMALE	RTA	DIABETES, HYPERTENSION	LEFT	2	UPPER LIMB	KLEBSIELLA PNEUMONIAE	NO GROWTH	NIL	NIL		OPEN TYPE II MIDSHAFT OF LEFT RADIUS FRACTURE	CLOSED 2ND 3RD LEFT METACARPAL FRACTURE	TETRACYCLINE		
19	945853	40	MALE	RTA	NIL	RIGHT	2	UPPER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE II RIGHT RADIUS SHAFT FRACTURE	NIL	PIPERACILLIN		
20	946804	49	MALE	RTA	DIABETES, HYPERTENSION	LEFT	2	LOWER LIMB	ACINETO BACTER SPECIES, COAGULASE NEGATIVE STAPHYLOCOCCUS	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT DISTAL FEMUR FRACTURE	SOFT TISSUE INJURY OVER RIGHT FOREARM	TETRACYCLINE		
21	40522	29	MALE	RTA	NIL	RIGHT	-	LOWER LIMB	ACINETO BACTER SPECIES	NO GROWTH	NIL	NIL		OPEN RIGHT CALCANEUM FRACTURE	NIL	LINEZOLID		
22	46107	35	MALE	RTA	NIL	LEFT	2	UPPER LIMB	COAGULASE NEGATIVE STAPHYLOCOCCI	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT RADIUS SHAFT FRACTURE WITH ULNA FRACTURE	NIL	CLINDAMYCIN		
23	50975	48	MALE	RTA	DIABETES	RIGHT	2	LOWER LIMB	ACINETO BACTER SPECIES	PSEUDOMONAS AERUGINOSA	NIL	NIL		OPEN TYPE II RIGHT PROXIMAL TIBIA FRACTURE	NIL	AMIKACIN		
24	45275	36	MALE	RTA	NIL	LEFT	3A	LOWER LIMB	KLEBSIELLA PNEUMONIAE	NO GROWTH	NIL	NIL		OPEN TYPE III A DISPLACED LEFT TIBIA SHAFT FRACTURE	NIL	AMIKACIN		
25	50756	47	MALE	WORK PLACE INJRY	HYPERTENSION	RIGHT	3B	LOWER LIMB	PSEUDOMONAS SPECIES	PROTEUS VULGARIS	PSEUDOMONAS AERUGINOSA	NIL		OPEN TYPE IIIB DISPLACED COMMINUTED RIGHT TIBIA FRACTURE	NIL	CEFEPIME	AMIKACIN	AMIKACIN
26	51362	50	MALE	RTA	DIABETES, HYPERTENSION	RIGHT	3B	LOWER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE IIIB RIGHT DISTAL TIBIA FRACTURE WITH FIBULA FRACTURE	NIL	AMIKACIN		
27	64117	38	MALE	RTA	NIL	LEFT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED LEFT DISTAL TIBIA FRACTURE WITH FIBULA FRACTURE	NIL			
28	42937	38	MALE	WORK PLACE INJRY	NIL	LEFT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED COMMINUTED LEFT TIBIA SHAFT FRACTURE	NIL			
29	65915	50	MALE	RTA	DIABETES, HYPERTENSION	RIGHT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED RIGHT HUMERUS FRACTURE	CLOSED 2ND 3RD RIGHT METACARPAL FRACTURE			
30	65311	21	FEMALE	WORK PLACE INJRY	NIL	LEFT	1	UPPER LIMB	METHICILLIN RESISTANCE STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT HUMERUS SHAFT FRACTURE	NIL	AMPICILLIN AND SULBACTAM		
31	67496	20	MALE	RTA	NIL	LEFT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED LEFT PROXIMAL HUMERUS FRACTURE WITH INTRAARTICULAR EXTENSION	NIL			
32	49967	62	MALE	RTA	DIABETES, HYPERTENSION	LEFT	2	LOWER LIMB	ACINETO BACTER SPECIES	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT FEMUR FRACTURE	NIL	AMIKACIN		
33	119450	47	MALE	WORK PLACE INJURY	NIL	LEFT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED LEFT TIBIA SHAFT FRACTURE	NIL			
34	125888	17	MALE	RTA	NIL	LEFT	3A	LOWER LIMB	ENTEROBACTER SPECIES	ESCHERICHIA COLI	NIL	NIL		OPEN TYPE IIIA DISPLACED LEFT TIBIA SHAFT FRACTURE	OPEN 2ND 3RD 4TH LEFT METATARSAL FRACTURE, 3RD 4TH PROXIMAL PHALANX FRACTURE	AMIKACIN	AMIKACIN	
35	46954	29	MALE	RTA	NIL	RIGHT	3A	LOWER LIMB	NO GROWTH	PROTEUS VULGARIS	NIL	NIL		OPEN TYPE IIIA RIGHT TIBIA SHAFT FRACTURE	CLOSED RIGHT PATELLA FRACTURE		AMIKACIN	

SL. NO	UHID	AGE	SEX	MODE OF INJURY	COMORBIDITIES	SIDE OF INJURY	GUSTILO ANDERSON	LIMB	PRE DEBRIDEMENT	POST DEBRIDEMENT	SUBSEQUENT CULTURES	SUBSEQUENT CULTURE 2		DIAGNOSIS	ASSOCIATED INJURIES	pre	post	subse
36	125932	15	MALE	RTA	NIL	LEFT	1	UPPER LIMB	STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT HUMERUS SHAFT FRACTUURE	NIL	CHLORAMPHENICOL		
37	126175	71	FEMALE	RTA	DIABETES, HYPERTENSION	LEFT	1	UPPER LIMB	NO GROWTH	KLEBSIELLA PNEUMONIA	NIL	NIL		OPEN TYPE I LEFT RADIUS SHAFT FRACTURE WITH ULNA FRACTURE	NIL		AMIKACIN	
38	56917	21	MALE	RTA	NIL	RIGHT	1	LOWER LIMB	STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE I RIGHT TIBIA SHAFT FRACTURE	NIL	AMOXYCILLIN AND CLAVULUNATE		
39	58625	32	MALE	WORK PLACE INJURY	THYROID DISORDER	LEFT	2	UPPER LIMB	ENTEROCOCCUS	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT DISTAL HUMERUS FRACTURE	NIL	CHLORAMPHENICOL		
40	51420	22	MALE	RTA	THYROID DISORDER	LEFT	1	UPPER LIMB	COAGULASE NEGATIVE STAPHYLOCOCCI	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT DISTAL HUMERUS FRACTURE WITH ULNA SHAFT FRACTURE	NIL	DOXYCYCLINE		
41	23545	29	MALE	RTA	NIL	RIGHT	2	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE II RIGHT TIBIA SHAFT FRACTURE	NIL			
42	63871	16	MALE	RTA	NIL	RIGHT	2	LOWER LIMB	KLEBSIELLA PNEUMONIAE	NO GROWTH	ESCHERICHIA COLI	NIL		OPEN TYPE II RIGHT FEMUR SHAFT FRACTURE WITH CLOSED RIGHT TIBIA FRACTURE	NIL	AMIKACIN		AMIKACIN
43	134481	52	MALE	RTA	DIABETES,HYPER TENSION	RIGHT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I RIGHT TIBIA SHAFT FRACTURE	CLOSED RIGHT DISTAL END RADIUS FRACTURE,RIGHT CLAVICLE FRACTURE			
44	46984	48	MALE	RTA	DIABETES,HYPER TENSION	RIGHT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED RIGHT TIBIA SHAFT FRACTURE	NIL			
45	21334	61	FEMALE	RTA	DIABETES,HYPER TENSION	LEFT	2	LOWER LIMB	STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT PROXIMAL TIBIA FRACTURE	LEFT CLAVICLE FRACTURE	CHLORAMPHENICOL		
46	59462	55	MALE	WORK PLACE INJURY	DIABETES, HYPERTENSION	LEFT	1	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT PROXIMAL TIBIA FRACTURE	LEFT CLAVICLE FRACTURE			
47	125772	20	MALE	RTA	NIL	LEFT	2	LOWER LIMB	ENTEROBACTER SPECIES	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT TIBIA SHAFT FRACTURE	CLOSED LEFT CLAVICLE FRACTURE	AMIKACIN		
48	12851	29	MALE	RTA	NIL	LEFT	2	LOWER LIMB	ACINETOBACTER	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED LEFT FEMUR SHAFT FRACTURE	NIL	LINEZOLID		
49	134481	52	MALE	WORK PLACE INJURY	DIABETES	LEFT	2	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT HUMERUS SHAFT FRACTURE	LEFT CLAVICLE FRACTURE			
50	55954	83	MALE	RTA	DIABETES,HYPER TENSION	RIGHT	2	LOWER LIMB	ESCHERICHIA CHOLI	NO GROWTH	NIL	NIL		OPEN TYPE II RIGHT PROXIMAL TIBIA FRACTURE	NIL	AMIKACIN		
51	67496	22	MALE	WORK PLACE INJURY	NIL	RIGHT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I RIGHT DISTAL HUMERUS FRACTURE WITH ULNA SHAFT FRACTURE	NIL			
52	94852	28	MALE	WORK PLACE INJURY	NIL	LEFT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT PROXIMAL HUMERUS FRACTURE	NIL			
53	80097	16	MALE	RTA	NIL	LEFT	2	UPPER LIMB	STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT HUMERUS SHAFT FRACTURE	NIL	AMOXYCILLIN AND CLAVULUNATE		
54	35306	92	FEMALE	FALL FROM HEIGHT	DIABETES,HYPER TENSION	RIGHT	2	LOWER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE II RIGHT TIBIA SHAFT FRACTURE	NIL			
55	138658	70	MALE	FALL OF HEAVY OBJECT	DIABETES,HYPER TENSION	LEFT	1	LOWER LIMB	KLEBSIELLA PNEUMONIAE	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT TIBIA SHAFT FRACTURE	NIL	AMIKACIN		
56	134332	38	MALE	RTA	NIL	LEFT	3C	LOWER LIMB	ACINETOBACTER	ENTEROBACTER	NIL	NIL		OPEN TYPE IIIC LEFT PROXIMAL TIBIA FRACTURE	CRUSH INJURY OF LEFT FOOT, LEFT CLAVICLE FRACTURE WITH C5C6 BRACHIAL PLEXUS INJURY	AMIKACIN	AMIKACIN	
57	129065	24	FEMALE	RTA	NIL	RIGHT	2	LOWER LIMB	ESCHERICHIA COLI	ESCHERICHIA COLI	NIL	NIL		OPEN TYPE II DISPLACED RIGHT TIBIA FRACTURE	NIL	AMIKACIN	AMIKACIN	
58	70614	30	MALE	RTA	NIL	RIGHT	2	LOWER LIMB	STAPHYLOCOCCUS AUREUS	NO GROWTH	NIL	NIL		OPEN TYPE II DISPLACED RIGHT FEMUR SHAFT FRACTURE	NIL	AMOXYCILLIN AND CLAVULUNATE		
59	78291	23	MALE	RTA	NIL	LEFT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT HUMERUS SHAFT FRACTURE	NIL			
60	101774	60	MALE	RTA	DIABETES,HYPER TENSION	RIGHT	3A	LOWER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE IIIA DISPLACED COMMINUTED RIGHT PROXIMAL TIBIA FRACTURE	NIL	AMIKACIN		
61	35115	42	MALE	RTA	NIL	LEFT	2	LOWER LIMB	PSEUDOMONAS AERUGINOSA	NO GROWTH	NIL	NIL		OPEN TYPE II LEFT TIBIA FRACTURE	NIL	AMIKACIN		
62	110127	40	MALE	RTA	NIL	RIGHT	1	UPPER LIMB	ENTEROBACTER SPECIES	NO GROWTH	NIL	NIL		OPEN TYPE I DISPLACED RIGHT RADIUS SHAFT FRACTURE WITH ULNA FRACTURE	NIL	AMIKACIN		
63	112212	38	FEMALE	RTA	NIL	LEFT	1	UPPER LIMB	NO GROWTH	NO GROWTH	NIL	NIL		OPEN TYPE I LEFT HUMERUS SHAFT FRACTURE	NIL			
64	111348	52	MALE	RTA	DIABETES, HYPERTENSION	RIGHT	2	LOWER LIMB	ACINETOBACTER SPECIES	NO GROWTH	NIL	NIL		OPEN TYPE II RIGHT TIBIA SHAFT FRACTURE WITH FIBULA FRACTURE	NIL	AMIKACIN		