PROSPECTIVE STUDY OF NOSOCOMIAL INFECTION AMONG DERMATOLOGY INPATIENTS IN A TERTIARY CARE CENTRE IN SOUTH INDIA

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A DISSERTATION SUBMITTED IN PARTAIL FULFILLMENT OF THE RULES AND REGULATIONS FOR THE MD BRANCH XX, DERMATOLOGY, VENEREOLOGY AND LEPROSY EXAMINATION OF THE TAMIL NADU DR.M.G.R MEDICAL UNIVERSITY TO BE HELD IN MARCH 2008.

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CONTENTS

INTRODUCTION

Nosocomial infection (NI) is defined as an infection developing in hospitalized patients, which was neither present nor in incubation at the time of their admission. Infections are considered nosocomial if they first appear 48 hours or more after hospital admission or within 10 days after hospital discharge.[1,2]

Nosocomial infections cause substantial morbidity and mortality, prolong the hospital stay of affected patients, and increase direct patient-care costs.[3] NI is among the most difficult problems confronting clinicians who deal with severely ill patients. The incidence of these hospital-acquired infections varies with the size of hospitals, with specialties of wards, and with many other factors such as length of hospital stay, local trends in antibiotic usage, nursing and hygiene conditions, hospital design and geographical distribution of patients at risk.[4]

An average incidence of NI can be estimated at 5-10%, with higher rates in large university hospitals, and reaching up to 28% in the intensive care unit (ICU).[4] The Study on the Efficacy of NI Control (SENIC) in 1975 in USA, found that NI develops in 5%-6% of hospitalized patients.[5] Later in 1984, the National Nosocomial Infection Surveillance (NNIS) in USA, found an overall rate of 3.4 infections per 100 patients discharged and suggested that the true incidence is underestimated.[5]

In general, it is expected that the incidence of NIs in dermatology patients will be low, but information on the occurrence of NIs in dermatology care is very

limited. However, as these infections may lead to a prolonged hospital stay and severe complications, it is useful to provide dermatologists with more detailed data on the incidence on NIs in this setting.[6] The incidence of sepsis in dermatology inpatients in a study in AIIMS, Delhi in India was reported to be 6.6%.[7]

There is a paucity of data on the incidence of NI and of risk factors for acquiring NI among dermatology inpatients and this has prompted this study.

AIMS AND OBJECTIVES

1. To study the incidence of NI among a cohort of dermatology inpatients in a tertiary care centre.

2. To study the pathogens responsible for NI and their antibiotic susceptibility.

3. To study the risk factors for NI in these patients.

REVIEW OF LITERATURE

Health care-associated infections constitute a major challenge of modern medicine and are considered one of the most accurate indicators of the quality of patient care.[8]

The acquisition of nosocomial pathogens depends on a complex interplay of the host, pathogen, and environment. The important host factors found in the development of infection are the underlying medical disorder, immune function, nutrition, age, and genetic factors. Microbial factors include the minimum inoculating dose sufficient to cause infection, virulence, pathogenicity, infectivity, and ability to produce a latent infection. The environment serves as a reservoir where a pathogen maintains its presence and replicates. The reservoirs for Gram-positive bacteria are generally human hosts, whereas Gram-negative bacteria may have a human, an animal or an inanimate reservoir. The source is the place from which the infectious agent passes to the host by either direct or indirect contact.[9]

NI may result from either endogenous flora, reactivation of latent infective agents or exogenous flora.[9] NI can be characterized as sporadic, endemic or epidemic on the basis of past occurrence of that disease in relation to time, place and person. Most NI are endemic, are endogenous in origin and occur in predictable frequencies in a time period. In an epidemic NI there is a significant increase in incidence above the expected.[10] Approximately 10% of hospitalized patients develop infections every year. The rate of developing

nosocomial or hospital acquired infections in developing countries is as high as 25%. It has been estimated that up to one third of these infections are preventable.[2] The incidence of NI is highest in surgical wards and ICUs, and lowest in medical units.[11]

Depending on the site of infection, NI can be classified as infections of the bloodstream, urinary tract, respiratory tract, gastrointestinal tract, burns, surgical site, intravenous catheter related and others.[12]

NI typically affect patients who are immunocompromised because of age, underlying diseases, or medical or surgical treatments.[13] Acute health care facilities serve both as a point of origin and as a reservoir for highly resistant pathogens. This is because the patients admitted to hospitals are subjected to intensive and prolonged antimicrobial use. In addition, failure in infection control practice can result in cross-infection and outbreak of NIs with highly resistant bacterial pathogens such as methicillin-resistant Staphylococcus aureus (MRSA), vancomycin-resistant enterococci (VRE) and multi-resistant Gram-negative bacilli(GNB) as well as resistant fungal infections. Some of these resistant strains have now spread outside hospitals causing infections in the community.[2]

Nosocomial infection adds to functional disability and emotional stress of the patient and may, in some cases, lead to disabling conditions that reduce the quality of life. NI is also one of the leading causes of death. Prolonged stay not only increases direct costs to patients or payers but also indirect costs due to lost work. The direct costs are due to increased use of drugs, the need for isolation, and the use of additional laboratory and other diagnostic studies. NI adds to the

imbalance between resource allocation for primary and secondary health care by diverting scarce funds to the management of potentially preventable conditions.[14]

Surveillance of NI is the foundation for organizing and maintaining an infection control programme. Different methods of surveillance exist:

- Hospital-wide surveillance which provides data on all infection sites and units, establishes baseline rates, identifies risk factors and allows recognition of outbreaks.
- Objective / priority based surveillance focuses on specific problems.
- Targeted surveillance focuses on patients at risk.
- Limited periodic surveillance decreases possibility of missing an outbreak and increases the efficiency of surveillance.[2]

Historical milestones in NI is shown in table 1.[15]

Table 1: HISTORICAL MILESTONES

Name	Relevance	Year
Semmelweiss	Hand-washing practice	1861
Florence Nightingale	Principles of nursing, hospital design and hygiene	1863
Louis Pasteur	Health hygiene	1873
Lister	Antiseptic theory	1874
Gustao Neubar	Introduced use of masks and gowns in surgery	1883
Halsted	Introduced rubber gloves in surgery	1890
Von Bergman	Steam sterilization	1896

PREVALENCE OF NOSOCOMIAL INFECTION

Nosocomial infection is an important focus of infection prevention in all countries, but in developing countries they are a major cause of preventable disease and death.[14] A prevalence survey conducted under the auspices of the WHO in 55 hospitals of 14 countries representing 4 WHO regions (Europe, Eastern Mediterranean, South-East Asia and Western Pacific) showed that an average of 8.7% of hospital patients had NI. The highest frequencies of NI were reported from hospitals in the Eastern Mediterranean and South-East Asia regions, 11.8 and 10.0% respectively, with a prevalence of 7.7 and 9.0% respectively in the European and Western Pacific regions. Nosocomial infection

rates range from as low as 1% in a few countries in Europe and the Americas to more than 40% in parts of Asia, Latin America and sub-Saharan Africa.[16] The studies on the prevalence of NI in developing countries is summarized in table 2.

Table 2: Studies on prevalence of NI in developing countries in different settings

No.	Authors (year)	Setting	(%) NI
1.	Mehta et al (2007)[17]	ICU	4.4
2.	Cherian R et al (1999)[18]	ICU	50.8
3.	Inan et al (2005)[19]	ICU	16.6
4.	Lakshmi KS et al (2006)[20]	PICU	30.17
5.	Khilnani et al (2004)[21]	PICU	16.86
6.	Effird MM et al (2005)[22]	Neonatal ICU	8.4
7.	Malik A et al (2001)[23]	Neonatal unit	26.9
8.	Zacharia A et al (1993)[24]	Medical ward+ICU	9.7
9.	Abdel-Fattah et al (2005)[25]	General ward	48.3
10.	Zhang et al (1991)[26]	Total hospital	13.1
11.	Wu AH et al (2003) [27]	Total hospital	1.3

Annual prevalence surveys are used to measure the burden of hospital acquired infections in many countries. The prevalence per 100 admissions was 9.1 in Greece in 1999, 8.0 in Denmark in 1999, 7.0 in Spain in 1997, 5.1 in Norway in2002 and 4.6 in Slovenia in 2001. In 1995 CDC estimated that 1.9 million hospital associated infections (HAIs) occurred in US hospitals, and in 2002 they estimated 1.7 million HAIs.[28] The prevalence of NI in developed countries is shown in table 3.

Table 3: Reported studies on prevalence of NI in developed countries in different settings

No.	Authors (year)	Setting	% NI
1.	Vincent et al (1995)[29]	ICU	20.6
2.	Pittet et al (1999)[30]	Total hospital	11.6
3.	Ortona et al (1985)[31]	Total hospital	6.75
4.	Lizioli et al (2003)[32]	Total hospital	4.9
5.	Kim et al (2000)[33]	General ward	3.7

Predisposing factors in NI

Patient factors

Various patient factors predisposing to NI include debility, extremes of age, impaired gag reflex, immunosuppressive illness, immobility, dehydration and normal flora.[11]

Surgical and Medical interventions

Various surgical interventions like incisions, intravascular devices, urinary catheterization, prosthetic joints and heart valves provide a protected niche for bacterial growth. Medical interventions such as antibiotic use, immunosuppression, anaesthesia and ventilation, total parenteral nutrition, use of histamine (H_2) receptor blockers may potentiate acquisition of NI.[11]

Hospital environment

Environmental factors such as lack of hygiene, overcrowding, understaffing, cross-infections, environmental organisms, hospital pathogens and duration of hospital stay are responsible for hospital acquired infections.[11]

In the European Prevalence of Infection in Intensive Care (EPIC) study by Vincent LJ et al[29] in ICUs the following factors increased the odds of death: age older than 60 years, wound / blood stream infection, sepsis, pneumonia, urinary tract infection, organ failure, cancer, diabetes and total length of hospital stay. In a study by Dettenkofer M et al[6] in a German university hospital most of the NIs were detected after surgery especially in patients with basal cell carcinoma (BCC), the incidence of surgical site infections (SSI) being 7.6%. Wahie S et al[34] in their study on wound complication rate for dermatology inpatients undergoing diagnostic skin biopsies found wound complications in 29 biopsies, 27(93%) of which were the result of wound infection. Complications occurred significantly more frequently when biopsies were performed below the waist compared with above the waist, in the ward compared with the outpatient operating theatre, in smokers compared with nonsmokers, and in those taking corticosteroids compared with those who were not. Wertheim HFL et al[35] in the study on risk and outcome of nosocomial *S. aureus* bacteraemia in nasal carriers versus non-carriers have shown that nasal carriers have a heightened risk of developing nosocomial *S aureus* bacteraemia. The study by Selwyn S et al[36] has stressed the importance of cross-infection in patients with extensive infected lesions, and communal baths and hands of members of staff, acting as important agents in

the spread of infection. Colsky SA et al[37] in their study on analysis of antibiotic susceptibilities of skin wound flora in hospitalized dermatology patients have shown that increase in oxacillin resistance among S. *aureus* from leg ulcers was due to the chronic nature of the wound and frequent antibiotic exposure. Many of the study patients had long-term treatment, including prior admissions as well as multiple courses of antibiotic therapy in the outpatient setting. Klevens RM et al[38] in their study on invasive MRSA infection have shown that most MRSA were health care-associated, and that incidence was highest among persons 65 years and older and those who had had previous hospitalization within one year. Oie S et al[39] in their study demonstrated MRSA contamination in the dermatological ward over inanimate objects which can pave the way for nosocomial spread to other patients.

Improper antibiotic usage paves the way for development of multi-drug resistant organisms (MDRO) in hospitals like MRSA, VRE, certain GNB, including those producing extended spectrum beta-lactamases (ESBLs) and others that are resistant to multiple classes of antimicrobial agents. Increased lengths of stay, costs, and mortality also have been associated with MDROs.[40]

NOSOCOMIAL INFECTIONS IN DERMATOLOGY

The patients in dermatology ward, with large areas of their skin denuded and thus with severely compromised barrier and immune function of the skin, are especially prone to develop sepsis.[7] Skin lesions form excellent culture media for bacteria and are, therefore, very sensitive indicators of cross-infection.

Furthermore, patients with infected skin lesions are believed to be important dispersers of pathogenic organisms. In a dermatology ward, therefore, conditions should be ideal for studying the mechanism of cross-infection.[36]

The incidence of NI in dermatology patients varies between 2.5% to 62.5%.[6,26] In a prospective survey on NI conducted in Hua Shan hospital, China, Zhang Y et al[26] reported an overall incidence of 13.1% of NI, the incidence being highest in the dermatology ward (19.8%).

In a University hospital in Germany, among 1450 dermatology inpatients surveyed for NIs, 35 patients were identified to have 37 NIs. The overall incidence was 2.5 NIs per 100 patients and the incidence density was 1.9 NIs per 1000 patient days. 21 patients developed SSIs at the rate of 2.1%. The site-specific incidence rates (NI per 100 patients) were 1.4 for SSI's, 0.5 for other soft tissue infections, and 0.07 each for urinary tract and bloodstream infection. *Staphylococcus aureus* (40%) was the commonest organism causing NI.[6]

In a study conducted in dermatology inpatients in UK 1962, 62.5% developed NI mainly with S*.aureus*. Autogenous infection accounted for about one third of the cases. The reservoirs of infection in the wards were the patients with extensive skin lesions. The communal baths and hands of members of staff seemed to be important agents in the spread of infection.[36]

Among 150 patients surveyed during a three month period in a dermatology ward in Delhi, India. *Staphylococcus spp.* was the commonest isolated organism out of which 80% (16/20*)* were MRSA. Ten patients developed sepsis with an incidence of 6.6%. Gram negative isolates included *Acinetobacter*

spp, Pseudomonas aeruginosa and Klebsiella pneumoniae. Three patients died following sepsis.[7]

Nair et al [41], has reported a 3.58% mortality rate due to dermatological diseases. Patients with pemphigus group of diseases were the leading cause of mortality. Most of the patients were elderly (61-70years) and ten patients had more than 70% skin involvement which were risk factors for mortality.

NOSOCOMIAL PATHOGENS

Staphylococcus aureus

Staphylococcus aureus is one of the most common causes of both endemic and epidemic infections acquired in hospitals, which result in substantial morbidity and mortality. MRSA and MSSA are equally pathogenic and are capable of causing the same spectrum of nosocomial infection.[42] MRSA was first isolated in the US in 1968. By the early 1990s, MRSA accounted for 20%-25% of S. *aureus* isolates from hospitalized patients. In NNIS system, among the S. *aureus* isolates from ICUs more than 50% was MRSA in 1999 which rose to 59.5% in 2003.[40]

Prevalence studies

Kaplan et al[43] in their surveillance study of community acquired S. *aureus* in children found that the number of community acquired MRSA (CA-MRSA) isolates and community acquired MSSA (CA-MSSA) isolates increased annually, but the rate of increase was greater for CA-MRSA. An antibiotic surveillance

study by Colsky et al[37] reported that in superficial skin wounds S. *aureus* comprised 77% of isolates and in leg ulcers 43%. Fifty percent of S. *aureus* isolated from leg ulcers were resistant to oxacillin. A study from the bacteraemia reporting system in Britain showed a rise of MRSA infection from 1.8% between 1989 and 1991 to 8.1% by 1994 and to 13.5% in 1995.[44] A similar rise in MRSA bacteremia was seen in children less than 15 years of age in the UK from 0.9% in 1990 to 13% in 2000.[45]

Lin KS et al[46] reported that the proportion of NI due to *S. aureus* had increased from 9.8% in 1985 to 16.7% in 1994. Adwan K et al[47] from Palestine reported a prevalence of 8.7% of MRSA in NIs, among which 39.3% were isolated from skin ulcers. Shigeruko I et al[48] from Japan reported an MRSA prevalence of 35.6% in a hospital, of which 27.8% were cultured from dermatology inpatients. A surveillance program of invasive MRSA from US reported that 58.4% were community-onset infections and 26.6% were hospital-onset infections.[49]

A prospective study of community-acquired primary pyodermas in Mumbai, India showed that S. *aureus* was the predominant pathogen isolated (81.4%). Barring one, all strains were sensitive to methicillin.[50] A prospective study done in Karnataka, India showed that among S*. aureus* infections reported, 18.1% had infection with MRSA. Clindamycin-susceptible MRSA accounted for 61.9% of cases. 46.1% patients were community-acquired MRSA and these were susceptible to multiple antibiotics, as compared to nosocomial isolates.[51] A six month pilot programme on MRSA surveillance in India in 1994 showed a

prevalence of 32% among large hospitals in India.[52] According to the National Staphylococcal Phage Typing Centre, New Delhi, India there is an increase in the occurrence of MRSA from 9.83% in 1992 to 45.44% in 1998.[53] Methicillin resistant S. *aureus* strains were more common in southern India (30.94%) than in the west (20.33%) or north (18.88%).[53] In a study conducted among orthopedic and burns inpatients of a hospital in New Delhi, India in 1998, *S. aureus* was isolated in 41.8%, of which 51.6% were found to be MRSA. 87.3% of isolates were hospital-acquired and all were susceptible to vancomycin.[54] A prevalence study of MRSA from major southern districts of Tamilnadu, India done from 2000-2002 showed that among S. *aureus* isolates 31.1% of clinical samples and 37.9% of carrier samples were found to be MRSA respectively. All the strains were sensitive to vancomycin.[55] Mulla S et al[56] in 2004, from a study done in a tertiary care centre, in Gujarat, India reported an MRSA prevalence of 39.50%.

Outbreak study

Balslev U et al[57] from Denmark in 2000 reported an outbreak of borderline oxacillin-resistant S. *aureus* (BORSA) in a dermatological unit. BORSA was isolated in 37 samples from 11 patients, following which intervention was initiated focusing on infection control. It was observed that the patients with BORSA had more severe skin disease, were more often hospitalized, and had more bed days. Helali NE et al[58] reported an outbreak of staphylococcal scalded skin syndrome (SSSS) in neonates from a maternity unit in France. Over a three-month period, 13 neonates developed SSSS. The probable transmission

occurred from an ancillary nurse who suffered from chronic dermatitis on her hands that favoured S. *aureus* carriage.

An outbreak of MRSA was reported in dermatology indoor patients in 2002, from Mumbai, India. Out of 63 indoor admissions, MRSA isolates were detected in 10 patients, all of whom had erosive or purulent skin lesions. Three of these patients were nasal carriers of MRSA.[59]

Nasal carriage studies

The anterior nares are reservoirs for S.*aureus*. Mucin appears to be the critical surface that is colonized in a process involving interaction between staphylococcal protein and mucin carbohydrate. Three patterns of carriage can be distinguished: Sixty percent of population harbours S. *aureus* intermittently, and the strains change with varying frequency; twenty percent almost always carry a strain and are persistent carriers and another 20% almost never carry S. *aureus.*[42]

In a prospective study of inpatients in Texas, USA in 2002, 21.5% were initially colonized with S. *aureus.* The incidence of subsequent MRSA infection for those initially colonized with MRSA was close to 10 times the incidence for patients colonized with MSSA or not colonized with S. *aureus* at admission.[60] A survey of staphylococcal carriage in the dermatological and burns units in the UK showed that 20% of patients in the dermatology ward carried MRSA at admission compared to 55.6% in patients in the burns unit.[61] Kuehnert MJ et al[62] in a study done in the USA in 2001-2002 reported a national prevalence of S*. aureus* and

MRSA colonization estimates of 32.4% and 0.8% respectively. Saxena S et al[63] in a community prevalence study of MRSA in East Delhi, India in 2002 reported an S.*aureus* nasal carriage of 29.4% with a colonization rate ranging from 10% to more than 40% in a normal adult population. Majumder et al[64] studied the prevalence of MRSA in a referral hospital in Assam. They found a methicillin resistance of 52.9% among S. aureus isolates and 15% among coagulase negative staphylococci.

Coagulase-negative staphylococci

Coagulase-negative staphylococci are among the most commonly isolated bacteria in clinical microbiology laboratories. Such coagulase-negative, novobiocin-susceptible staphylococci as *Staphylococcus epidermidis* have emerged as a major cause of infection, particularly in hospitalized patients with indwelling foreign bodies and in immunocompromised patients.[65]

Enterococcus species

Vancomycin-resistant enterococci (VRE) are formidable organisms renowned for their ability to cause infections with limited treatment options and their potential for transferring resistance genes to other Gram-positive bacteria. They are usually associated with NIs.[66] From 1990 to 1997, the prevalence of VRE in enterococcal isolates from hospitalized patients increased from <1% to approximately 15%. VRE accounted for almost 25% of enterococcus isolates in NNIS ICUs in 1999 (94), and 28.5% in 2003.[40] Bhavnani SM et al[67] in a case

control study found the following factors to be highly associated with VRE bacteremia: positive HIV status and AIDS, drug abuse, prior exposure with parenteral vancomycin, and history of liver transplant. Usually associated with NI, VRE are rarely reported as a cause of community-acquired infection.[66]

Gram negative infection

During the past 20 years, there is an increasing frequency of GNB associated hospital-acquired infection.[68] Gram negative bacilli cause four major types of hospital-acquired infection: pneumonia, surgical site infection (SSI), urinary tract infection (UTI), and bloodstream infection (BSI).[68] Data analysis from the NNIS system from 1986-2003 to determine the most frequent types of hospital-acquired infection in ICUs showed that GNB were associated with 23.8% of BSIs, 65.2% of pneumonia episodes, 33.8% of SSIs, and 71.1% of UTIs. The proportion of ICU pneumonia episodes associated with Acinetobacter species increased from 4% in 1986 to 7.0% in 2003 ($P<.001$). The rate of antimicrobial resistance is especially increasing among Acinetobacter species and P.*aeruginosa*.[68]

Malik A et al[23] in a study on NI in newborns found that the commonest NI was septicaemia which was observed in 45 cases followed by umbilical sepsis in 7.4%. GNB were isolated in nearly 70% of the cases with Klebsiella being the commonest organism causing the infections, accounting for 55.6% of the cases. A prospective survey on NIs in China which included dermatology patients revealed that 66.4% of the nosocomial pathogens were GNBs. They comprised

P.*aeruginosa* (13.3%), K.*pneumoniae* (12.2%), E.*coli* (8.9%) and acinetobacter (7.7%).[26] Dettenkofer M et al[6] in their study on nosocomial infections in dermatology inpatients found that E.*coli* comprised 18% of the isolates among SSIs. Sharma VK et al[7] in their study on sepsis in dermatology wards found 48 bacterial isolates in culture from different sites out of which 28 were GNBs. They comprised acinetobacter in 9 cases, pseudomonas and klebsiella in 6 cases each and E.*coli* and proteus in 7 cases each. These isolates were most sensitive to a combination of piperacillin and tazobactam (100%) followed by cefoperazone-sulbactam (88.9-100%), imipenem (62.5-100%), and meropenem (62.5-83.3%). Amikacin, ciprofloxacin, piperacillin, netilmicin, ceftazidime and ticarcillin-clavulanic acid showed low sensitivity. Mehta et al[69] in their study on bacterial isolates in burn wound infections found that during the period from 2002-2005 pseudomonas species (51.5%) was the commonest pathogen isolated followed by acinetobacter species (14.28%).Pseudomonas species was moderately resistant to piperacillin (41.2%) where as resistance was more marked with antimicrobials like amikacin(85.18%), gentamicin(89.22%), ciprofloxacin(78.81%), carbenecillin (88.26%), tobramycin(87.52%) and ceftazidime(79.09%).

Gram negative bacilli with extended-spectrum beta lactamases (ESBLs), resistant to fluoroquinolones, carbapenems, and aminoglycosides also have increased in prevalence. The resistance rates for K.*pneumoniae* to ceftazidime and other third-generation cephalosporins have varied from 3.6 % (United States) in 1997 to 20.6% (NNIS) ICUs in 2003.[40] Between 1999 and 2003,

Pseudomonas *aeruginosa* resistance to fluoroquinolone antibiotics increased from 23% to 29.5% in NNIS ICUs.[40] Also, a 3-month survey of 15 Brooklyn hospitals in 1999 found that 53% of A. b*aumannii* strains exhibited resistance to carbapenems and 24% of P. *aeruginosa* strains were resistant to imipenem.[40] A retrospective analysis of 5039 pus samples obtained from inpatients at AIIMS, New Delhi in 2002 found that 54.04% of isolates were GNB. Out of them 14.49% were sensitive to all antibiotics. ESBL production was observed in 66.75% of isolates and ranged from 40.6% in Proteus to 76.34% among Klebsiella species.[70]

Viral infection

Any viral infection can spread within hospitals. It can cause viral respiratory infection, outbreaks of diarrhoea, cross infection with blood-borne viruses in both patients and hospital staff.[11] Since identification of nosocomial viral infections depends on both laboratory detection and surveillance intensity, hospitals without diagnostic virology lab support will be unlikely to detect most of these infections.[5]

In dermatology, patients with large areas of skin involvement are prone to develop Kaposi's varicelliform eruption (KVE) a distinct cutaneous eruption caused by herpes simplex virus (HSV) Type I and Type 2 and rarely by coxsackie A16 virus and vaccinia virus over preexisting dermatoses. The most common predisposing condition for KVE is atopic dermatitis, but it has also been

described in various dermatoses with impaired barrier function like Darier's disease, pemphigus and burns. HSV spreads by droplet infection or by direct contact. A mini outbreak of five cases of KVE was reported from the skin ward in Vishakhapatnam, Andhra Pradesh in May 2005. This occurred in a makeshift ward after admission of the first case.[71] There are several reports of HSV out breaks especially in infants under 30 days and patients with burns. Any patient with skin failure has an increased risk as it is easy for entry of HSV.[72] There are several reports of HSV 1 outbreaks with transmission either through patients, hospital personnel or due to hospital environment.[73,74,75]

Spread of blood-borne viruses, particularly hepatitis B and C and HIV, is of greatest concern. Infected patients are not always recognized, and every procedure involving blood (phlebotomy, surgery, dialysis, endoscopy, transfusion) carries a risk of spread of infection to hospital staff or other patients.[11]

Fungal infection

Nosocomial fungal infections are commonly seen in patients with iatrogenic immunosuppression for organ transplantation, and with AIDS, and in oncology wards, high risk nurseries, burn and trauma wards.[76] Some exposures act primarily by inducing immunosuppression (e.g., corticosteroids, chemotherapy, malnutrition, malignancy, and neutropenia). Other exposures primarily provide a route of infection (e.g., extensive burns, indwelling catheter) or a combination of factors. Centre for Disease Control (CDC) has reported a steady increase in the

rate of nosocomial fungal infections from 5.4% in 1980 to 9.9% in 1990.[77] The majority of nosocomial fungal infections are reported to be caused by Candida spp. The data from NNIS during 1980 to 1990 shows that Candida infections accounted for 78.3% of fungal NI, followed by Torulopsis *glabrata* (7.3%), and Aspergillus spp. (1.3%).[70] Newly recognized pathogenic fungi some previously thought to be nonpathogenic, including Malasezzia spp., non-albicans Candida spp., Fusarium spp., trichosporon spp., Mucor and Rhizopus spp., and Alternaria, Bipolaris, and Curvularia spp also have been reported as the cause of Nis.[76]

Only a few reports of incidence of, and spectrum of organisms responsible for candidemia in India are available.[78] A recent study done in Delhi reported 7 cases of candidemia out of 101 BSI. Three (42.8%) were infected with C. *albicans* and the rest with non-albicans species.[79] In a recent 5 year retrospective study of candidemia from AIIMS, Delhi, the majority (80%) of episodes was caused by non-albicans species.[78] In the study by Sahni et al[79] all the patients with candidemia were admitted in the ICUs. Five (71.4%) patients died. Amongst the risk factors, the length of hospitalization, use of broad-spectrum antibiotics, central venous catheters, mechanical ventilation, and total parenteral nutrition were found to be significantly related to acquisition of nosocomial candidemia. Luzzati R et al[80] in a retrospective study have reported an average incidence was 1.14 episodes per 10,000 patient–days per year. Increased age, hospitalization in an intensive care unit, a longer duration of indwelling central lines, and inadequate antifungal therapy were significantly associated with poor outcome.

Archer-Dubon C et al[81] have reported an epidemic outbreak of Malassezia folliculitis in three patients in an intensive care unit.

PREVENTION OF NOSOCOMIAL INFECTION

Infection control is the responsibility of all health care professionals — doctors, nurses, therapists, pharmacists, engineers and others. Hand washing is said to be the single most critical measure in preventing spread of organisms such as MRSA, yet compliance is generally poor.[11] Isolation and barrier nursing are used generally for patients known to be infectious (e.g with tuberculosis, MRSA, or active diarrhoea). Source isolation is done to prevent the transfer of microorganisms from infected patients, who may act as source of infection to staff or other patients. Protective isolation is done for some immunocompromised patients to protect them from infection, rather than to protect their contacts.[2] Adequate bed-spacing, barrier nursing and isolation of suspected cases are mandatory to prevent life-threatening infections such as KVE.[71] Herpes simplex virus transmission is easily prevented by appropriate barrier methods and decontamination of surfaces.[72] Oie S et al[39] studied the contamination of environmental surfaces by S. *aureus* in the dermatological ward of a university hospital. They found 100-10^5 CFU of MRSA or MSSA on items such as immersion bath tub, foot wash bowl, stretcher for immersion bath, and chair for shower. After disinfection, no S. *aureus* was detected on smooth surfaces; however they were detected on porous surfaces made of sponge like material such as the stretcher and the shower chair. In patients with S. *aureus* bacteremia

there is as strong correlation between strains colonizing the nares, strains isolated from foci of infection, and strains isolated from blood, suggesting that S. *aureus* bacteremia may have an endogenous origin. Strategies to interrupt their transmission by elimination of nasal carriage may prevent systemic infections. In several studies nasal treatment with mupirocin led to a reduction by a factor of four in the incidence of S.*aureus* bacteremia per patient year in carriers receiving haemodialysis.[42] Engineering and design considerations are an important part of control of NI. Simple factors such as the availability of sinks for hand washing and over crowding of patients can have a significant effect on the ability of staff to prevent cross infection.[11]

Summary of prevention methods of NI

1. Limiting transmission of organisms between patients in direct patient care through adequate hand washing and glove use, use of clean bed linen and appropriate aseptic practice, isolation strategies, sterilization and disinfection practices.
2. Controlling environmental risks for infection
3. Protecting patients with appropriate use of prophylactic antimicrobials, nutrition, and vaccinations.
4. Limiting the risk of endogenous infections by minimizing invasive procedures.
5. Surveillance of infections, identifying and controlling outbreaks.

6. Prevention of infection in staff members.
7. Enhancing staff patient-care practices, and continuing staff education.[11]

MATERIALS AND METHODS

STUDY DESIGN : A prospective cohort study

STUDY SETTING : The study was conducted in the dermatology inpatient ward of Christian Medical College Vellore, a tertiary care centre, in Tamil Nadu. The dermatology ward included a general ward of 20 beds (a room with 5 beds for females, 2 rooms with 7 beds each for males and a single room), isolation ward and 2 private ward blocks with single and double bed rooms.

STUDY SUBJECTS: The patient population included all patients admitted under the department of Dermatology, Venereology and Leprosy during the study period.

Inclusion criteria:

All inpatients who were admitted with skin diseases to the dermatology ward during the study period and who gave informed consent were included in the study.

Exclusion criteria:

Patients who had been hospitalized 10 days prior to the present episode of hospitalization

Patients hospitalized for < 48 hours

Patients who did not give informed consent to participate in the study.

DURATION OF STUDY: Patients were recruited in the study from 1st November 2006 till 15th September 2007.

METHODOLOGY:

A preliminary study was conducted from July 2006 to October 2006 (four months). A total of 156 inpatients were studied, out of which 9 patients developed NI. During the preliminary study, it was noted that since the growth of bacterial isolates were not quantified, colonization and infection could not be differentiated. Hence it was decided to do four streak method of semi-quantitative culture for the next study period. All inpatients who gave written informed consent (annexure I) were examined by the principal investigator. Demographic details, which included age, gender, occupation, height, weight, body mass index, date of admission and discharge were recorded in a questionnaire (annexure II). A detailed history at admission which included duration of dermatological illness (acute illness was arbitrarily defined as illness <6 weeks and chronic illness as >6 weeks), presence of co-morbid illnesses like diabetes mellitus, essential hypertension, obesity, anemia, HIV infection, history of previous hospitalization and details of previous treatment including intake of antibiotics 2 weeks prior to hospitalization, oral steroid intake (>40mg/day for more than 1 week or >20mg/day for more than 2 weeks), steroid sparing immunosuppressives (methotrexate, azathioprine or cyclophosphamide) were recorded. Clinical parameters assessed at admission included pulse rate, respiratory rate, blood pressure and body temperature. Systemic examination was done in all patients. Type of skin lesions, presentation

of primary and secondary skin lesions and body surface area involvement were noted. If the patient had symptomatic signs of infection at admission, appropriate investigations like Gram's stain from pus, pus culture, urine routine and cultures, chest X ray, blood or sputum cultures were done according to the site of infection. Signs of infection included infection of lesions with either presence of pain or tenderness, localized swelling, surrounding erythema, increased warmth, pus discharge and/or systemic signs of infection like associated fever, tachycardia, tachypnoea or organ dysfunction, Patients were started on empirical antibiotic therapy based on initial Gram's stain of the specimen. The antibiotic was appropriately changed based on the final culture reports. Invasive procedures including intravenous cannulation, skin, lymph node and bone marrow biopsies were recorded. All the patients with diseases which predispose to skin failure were provided strict reverse barrier nursing, dilute potassium permanganate cleansing for skin lesions and alcohol (propanol) based hand disinfectant to prevent transmission of infection while in the ward. The patients were followed up daily after the admission till discharge by the principal investigator. The patients were identified as having NI according to CDC guidelines (annexure III) based on symptoms and signs of infection. The first episode of infection identified 48 hours after admission and not present at admission was diagnosed as NI. The time to onset from day of admission was recorded. Appropriate investigations (total and differential counts, pus Gram's stain and culture, urine routine, urine culture, blood culture, sputum Gram's stain and culture, and chest X ray) were sent, depending on the site of infection.

Cultures were not performed on patients who had no frank pus discharge or cellulitis. If the intravenous site was infected (erythema, edema, tenderness), the intravenous line was disconnected and the catheter tip sent for culture. Once the antibiotic susceptibility pattern was obtained, the antibiotics were continued or changed accordingly. The patients' condition at discharge and the drugs given were recorded. The patients were followed up 1 week after discharge in the dermatology outpatient department.

Method of taking cultures

Pus culture

Pus was collected aseptically using at least 2 swabs in a sterile test tube, from pustules, turbid blisters and/or abscesses. Tissue culture samples were taken from chronic ulcers. Smears were prepared and subjected to Gram's staining. For quantification of colonies, four streak methodology was undertaken.

Pus culture four streak semi-quantification: The wound was cleansed with saline and all debris removed. The pustules/ turbid blisters/ healthy looking granulation tissue was swabbed in a zigzag pattern, gently rotating the tip of the swab. Swabs were transported in culture tubes to the laboratory. The swab was then inoculated on to the primary culture media, blood agar (BA), chocolate agar (CA), MacConkey agar plates (MA) and Thioglycolate broth (TB) in the laboratory and streaked in four quadrants. The results were interpreted as scanty growth (<20 colonies), moderate (20-100 colonies) and heavy growth (>100 colonies).[82] MA and TB plates were incubated at 37º C. BA and CA were incubated in CO_2

atmosphere at 37° C. The plates were read after overnight incubation. Smears were prepared from the different types of colonies. If the isolate was identified as Staphylococcus species, the following tests were done for further identification: slide test for clumping factor, tube-coagulase test and mannitol fermentation test. If gram negative organisms were identified mannitol motility medium, triple sugar iron agar, peptone water for indole test and Simman's citrate agar were used for further identification. Antibiotic sensitivity test was done for all isolates.[83]

Antimicrobial susceptibility was performed on Mueller-Hinton agar by the Kirby-Bauer method of standard disk diffusion method. For gram-positive organisms the antibiotics tested were: Oxacillin (1ug), erythromycin (15ug), gentamicin (10ug), chloramphenicol (30ug), penicillin (10ug), vancomycin (30ug), teicoplanin (30ug), netilmicin (30ug), rifampicin (5ug), linezolid (30ug).

For gram-negative organisms the antibiotics tested were: Ampicillin (10ug), cefuroxime (30ug), ciprofloxacin (5ug), gentamicin (10ug), amox/clavulanic acid (20/10ug), cotrimoxazole (25ug), cefotaxime (30ug), ceftazidime (30ug), amikacin (30ug), nalidixic acid (30ug), nitrofurantoin (300ug), norfloxacin (10ug), imipenem (10ug), meropenem (10ug), cefaperazone-sulbactam (75/30ug), piperacillin-tazobactam (100/10ug) and cefpodoxime (10ug). If the isolate was resistant to the first line panel of antibiotics, then the second line antibiotics were tested at request. The disk strengths used were recommended by the Central Laboratory Standard Institutional guidelines (CLSI).[84]

In polymicrobial isolates, scanty or moderate growth was considered as colonization if it was from open erosion while any bacterial isolate was

considered significant if culture was taken from an intact lesion like a pustule or turbid blister. Growth of coagulase negative staphylococci and diphtheroids were not considered as pathogenic.

Blood culture

In patients in whom septicaemia was suspected, blood culture was done. A blood sample was drawn with full aseptic precaution and received in bottles containing the culture medium. The bottles were incubated in BacT-alert system. If growth was present, it was indicated by an alarm. Smears were done from the colonies, subjected to Gram's stain and cultured in appropriate media for identification.[83]

Urine culture

In patients suspected to have urinary tract infection, a clean-catch mid-stream urine sample or supra-pubic aspirate were obtained in a sterile bottle and transported immediately to the laboratory. It was inoculated into both blood and MacConkey agar plates. According to the number of colonies, growth was quantified as probably significant if colony count was 10^3 - 10^5 CFU/ml and significant if >10^5 CFU/ml .[83]

Faeces culture

In patients with loose stools, a stool sample was collected in a sterile screw-capped bottle. For all watery stool samples a hanging drop preparation was done and examined immediately. Specimens were cultured on plates with blood agar, Desoxycholate citrate agar and MacConkey agar. Biochemical tests were done for further identification.[83]

Viral culture

If a patient was suspected to have herpes infection, a Tzanck smear and herpes culture from the vesicles/erosions were done for identification of the same.

Total hospital nosocomial infection surveillance

Surveillance of NI among the hospital inpatients was done by a nurse who was trained in NI surveillance and was monitored by the Hospital Infection Control Committee (HICC). The nurse visited each ward once a week. A record of the number of patients in the ward at the time of visit was kept. The ward nurse's record and individual patient charts were reviewed to check for patients with fever on that day. A patient who developed fever more than 48 hours after admission, was evaluated for NI. The predisposing factors, site of infection, microbiological and radiological evidence and the clinical course of the patient in the hospital were recorded and categorized into different types of NI.

STATISTICAL ANALYSIS

All the parameters were entered in a data base and analysis was done with SPSS (11) Software Version.

SAMPLE SIZE

Sample size was calculated by using the formula:

$$n = \frac{4pq}{d^2}$$

where p=anticipated proportion

q= (1- p)

d^2= precision (or) width of confidence interval

The number required to estimate an anticipated incidence of 1.1% with 1 unit precision of 95% confidence is 396. Each patient with NI was compared with two randomly selected controls from the study population who did not develop NI. The comparison of the risk factors between the cases and controls was done using t test, Mann-Whitney test and Chi-square test. Kaplan-Meier product limits estimates for survival were used to construct the graph of time to onset of NI. Odds ratio were also calculated. Multivariate logistic regression analysis was done using those variables which were significant in the univariate analysis.

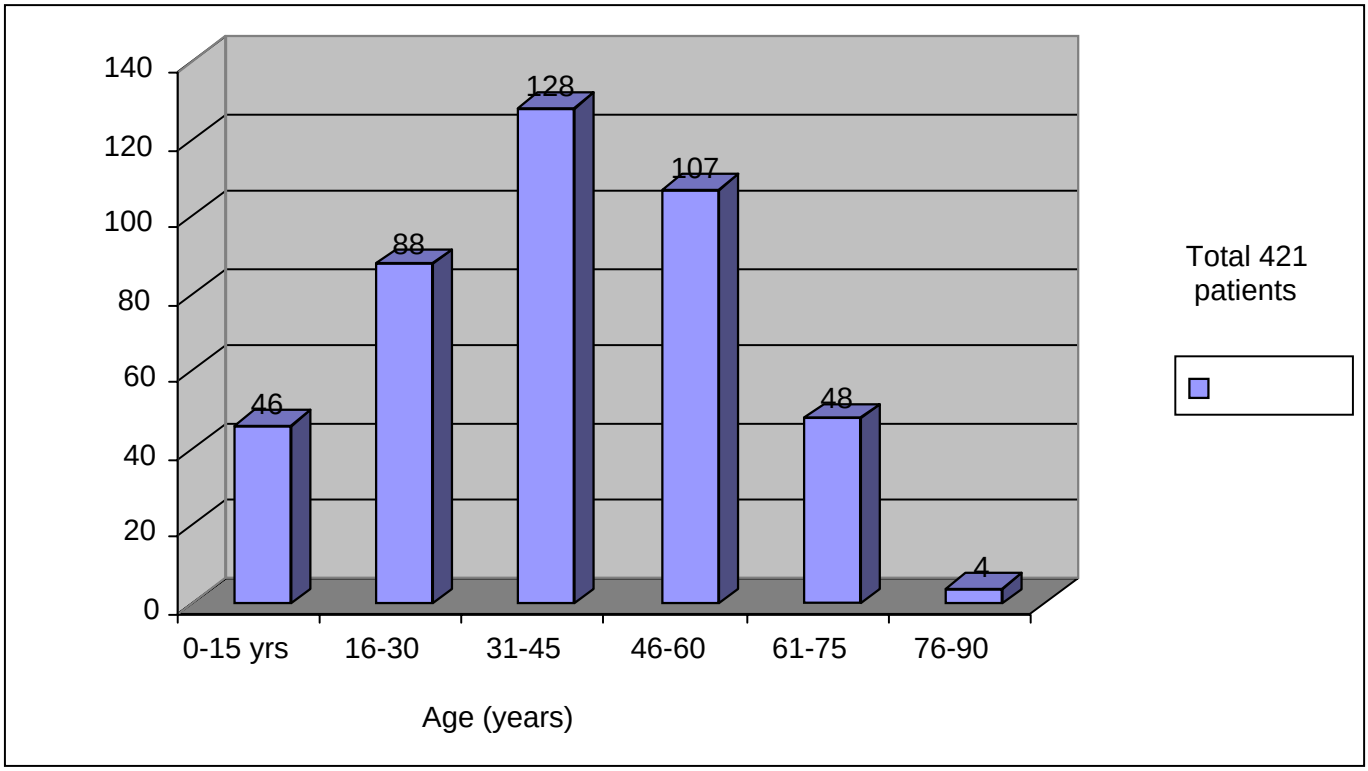
Research Committee approval

This study was approved by the institutional research committee.

RESULTS

Demographic and clinical profile of study patients:

A total of 421 patients were admitted during the study period of which there were 260 males and 161 females. Their age ranged from 4 months to 84 years [mean 39.53 ± SD 18.42] (figure 1).



The details of underlying primary dermatological diagnosis of the study group are given in table 4. The most common dermatological condition was psoriasis which was seen in 71 patients (16.86%) followed by autoimmune-bullous disorders in 50 (11.87%), and Hansen's disease in 43 patients (10.21%). Out of 421, 144 (34.2%) patients had diseases which predisposed to skin failure.

Table 4: Profile of primary dermatological disease

Diagnosis	**No. of Patients (%)**
Psoriasis	71 (16.86)
Autoimmune-bullous disorder	50 (11.87)
Other dermatoses	44 (10.45)
Hansen's disease	43 (10.21)
Allergic contact dermatitis	36 (8.55)
Drug reactions	31 (7.36)
Viral diseases	30 (7.12)
Erythroderma	22 (5.22)
Atopic dermatitis	21 (4.98)
Urticaria	20 (4.75)
Chronic leg ulcer	13 (3.08)
Vasculitis	11 (2.61)
Vitiligo	8 (1.90)
Cellulitis	7 (1.66)
Panniculitis	6 (1.42)
Skin malignancy	4 (0.95)
Connective tissue disorder	4 (0.95)

Co-morbidity:

Among 421 patients, 264 (62.7%) had co-morbidities. The various co-morbid diseases seen in our patients is shown in table 5. Diabetes mellitus and hypertension were present in 15.91% of patients each. 82 patients had more than one co-morbid condition.

Table 5: Total number of patients with co-morbid illness

Co-morbid condition	Number of patients (%)
Type II Diabetes Mellitus	67 (15.91)
Essential Hypertension	67 (15.91)
Anemia (iron deficiency/ chronic disease)	32 (7.60)
Dyslipidemia	19 (4.51)
Obesity	15 (3.56)
COPD	13 (3.09)
Osteoarthritis	12 (2.85)
Hypothyroidism	11 (2.61)
Tuberculosis	10 (2.38)
HIV infection	8 (1.90)
Seizure disorder	6 (1.43)
Osteoporosis	4 (0.95)

INFECTION AT ADMISSION

One hundred and nineteen (28.26%) patients were clinically diagnosed to have infection at admission of which 74 were males (62.18%) and 45 (37.81%) females. Their ages ranged from 1-75 years (mean 38.95 ± SD 21.36). The details of their underlying diseases are given in table 6.

Table 6: Disease profile of patients with infection at admission

Diagnosis	No. patients (%)
Autoimmune-bullous disorder	25 (21.00)
Other dermatoses	20 (16.80)
Psoriasis	20 (16.80)
Atopic dermatitis	10 (8.40)
Allergic contact dermatitis	9 (7.56)
Cellulitis	7 (5.88)
Drug reactions	6 (5.04)
Chronic leg ulcer	6 (5.04)
Erythroderma	5 (4.20)
Viral diseases	4 (3.36)
Vasculitis	4 (3.36)
Hansen's disease	2 (1.68)
Vitiligo	1 (0.84)
Total	**119 (28.26%)**

Seventy (58.83%) out of the 119 patients who were admitted with infection, had appropriate culture and susceptibility testing done as shown in table 7. Cultures could not be sent in 49 patients (41.17%). Nine of these patients had primary pyoderma and 40 had secondary pyoderma.

Table 7: Bacterial isolates in cultures from different sites from patients with infection at admission

Organism	Pus(skin)	Urine	Blood	Stool	Total
Staphylococcus aureus	31		1		32
Beta haemolytic Streptococcus	14				14
Enterococcus	3				3
Escherichia.coli	7	1			8
Pseudomonas aeruginosa	6				6
Klebsiella spp.	4				4
Non fermenting gram negative bacilli (NF GNB)	3				3
Enterobacter spp.	3				3
Proteus mirabilis	3				3
Morganella morgagni	1				1
Shigella sonnei				1	1
Citrobacter freundii	1				1
Total	**76**	**1**	**1**	**1**	**79**

The commonest gram positive isolate from the skin included *S.aureus* in 31 patients (40.78%), followed by beta hemolytic Streptococci in 14 (18.42%). The commonest gram negative bacterial skin isolate was E*. coli* in 7 (9.21%) patients followed by P.*aeruginosa in* 6 (7.89%) patients.

Susceptibility pattern of gram positive organisms

The susceptibility pattern of gram positive organisms isolated from patients with infection at admission is shown in table 8. Out of 32 isolates of *S.aureus,* 9 (28.13%) were MRSA. All isolates of MRSA were susceptible to rifampicin, vancomycin, linezolid and teicoplanin. All the three isolates of

Enterococcus were resistant to erythromycin and 1 (33.33%) of the isolates was also resistant to gentamicin.

Table 8: Susceptibility pattern of Gram positive organisms isolated from patients with infection at admission

Organisms	*S. aureus*				Beta haem Strepto				*Enterococcus*			
Total isolates	32				14				3			
Susceptibility	S	%	R	%	S	%	R	%	S	%	R	%
Gentamicin									2	66.67	1	33.33
Chloramphenicol												
Erythromycin	16	50.00	16	50.00	14	100					3	100
Oxacillin	23	71.88	9	28.13								
Penicillin					14	100						
Ampicillin									3	100		

Susceptibility pattern of gram negative organisms

Of the 6 isolates of P*.aeruginosa* all were susceptible to amikacin and ceftazidime and 2 (33.33%) showed resistance to ciprofloxacin. Of the 3 isolates of NFGNB, 1 (33.33%) was resistant to gentamicin, amikacin, ceftazidime and ciprofloxacin. Five out of 8 (62.5%) isolates of E*..coli* were resistant to co-trimoxazole, 4 (50%) to ciprofloxacin and augmentin and 1 (12.50%) to gentamicin. All the isolates of *Klebseilla spp.* were susceptible to gentamicin, cefuroxime, ciprofloxacin and co-trimoxazole and only one was resistant to augmentin. All the isolates of *Enterobacter spp.* (3), *Citrobacter freundii* (1), *Proteus mirabilis* (3) and *Morganella morgagni* (1) were susceptible to gentamicin.

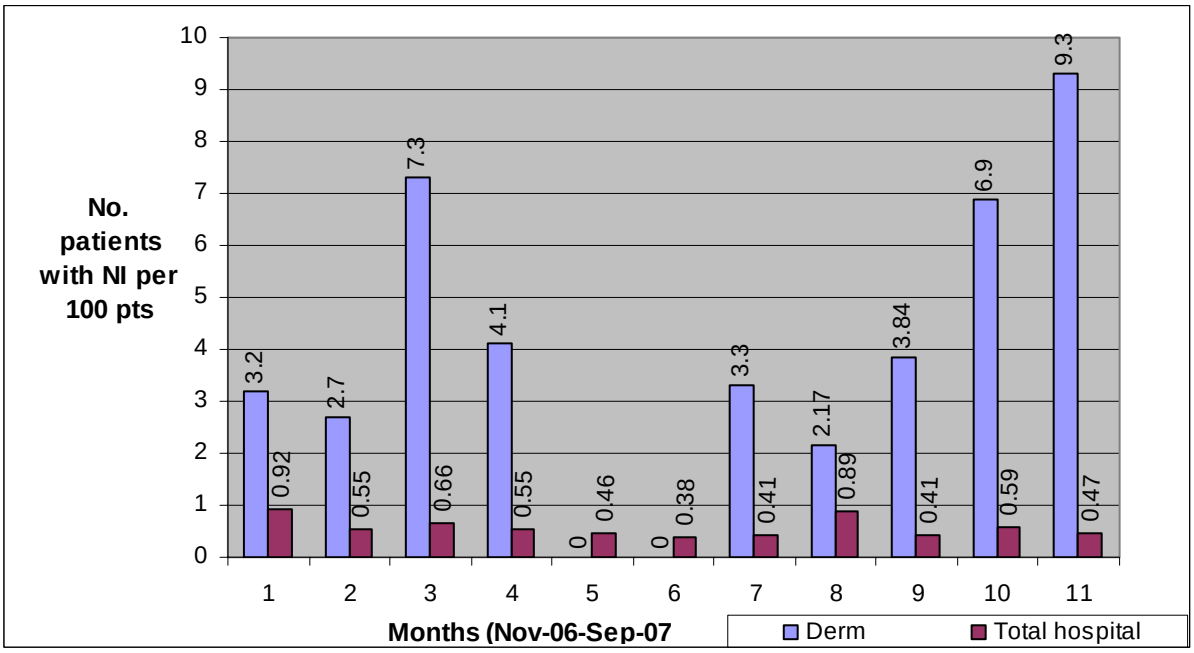
Invasive Procedures

Of the 421 patients, 115 underwent invasive procedures while in the ward.

NOSOCOMIAL INFECTION

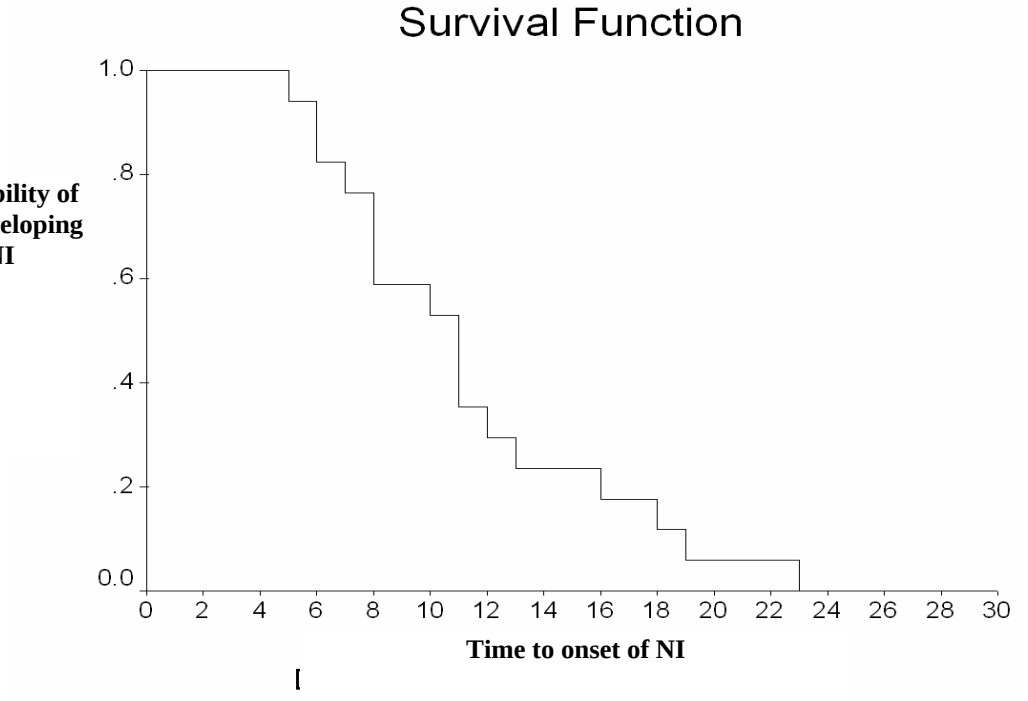
Seventeen of the 421 patients developed NI. The incidence of NI in the dermatology ward was 4.05 infections/100 discharges and NI incidence rate was 6.24 infections/1000 hospital days. The total hospital incidence of NI during the same period was 0.57/100 discharges and the comparison is shown in figure 2.

Fig. 2: Comparison of incidence of NI among dermatology and total hospital inpatients



The median time to onset of NI was 11 days (IQR 8.10, 13.90) as shown in figure 3.

Fig 3: Time to onset of NI



Profile of patients with NI:

Of the 17 patients with NI, 11 (64.7%) were males and 6 (35.30%) females. The age ranged from 20-70 years (mean 42.06 ± SD 14.8), two patients were more than 60 years of age. Five patients were over weight (BMI >25). The body surface area (BSA) involvement varied from 1-100% (median 21, range 10-65) with more than 30% involvement in ten cases. The duration of dermatoses varied from 3 days to 15 years. In acute dermatoses, it varied from 1 week to 4 weeks (n = 3); while in chronic dermatoses, it ranged from 2 months to 15 years (n = 14).

The underlying dermatological disease profile of patients with NI is given in table 9. Nosocomial infections were seen more in patients with autoimmune-bullous disorders (14%), followed by erythroderma (9.09%) and psoriasis (4.22%) respectively.

Table 9: Dermatological diagnosis of patients with NI

Primary dermatological diseases	Total study group (421)	No. patients with NI (17)	(%) of patients
Autoimmune-bullous disorder	50	7	14
Erythroderma	22	2	9.09
Psoriasis	71	3	4.22
Other dermatoses	278	5	1.79

The comorbid conditions seen in these patients were essential hypertension and hypoproteinemia in four patients each, type II diabetes mellitus, chronic kidney disease and anemia (iron deficiency / chronic disease associated) in two patients each and hypothyroidism, HIV infection and ulcerative colitis in one patient each respectively.

Of the 119 patients who were admitted with infections, 10(8.4%) developed an NI.

Risk factors among patients with NI:

There was history of oral antibiotic intake 2 weeks prior to admission in five patients. Eleven patients were on steroids / immunosuppressives agents of whom 2 were on oral steroids, 3 had a combination of oral steroids with steroid sparing immunosuppressants, 4 were on steroid sparing immunosuppressants and 2 were on DCP pulse.

The duration of hospitalization for patients who developed NI was (median 21 days, IQR [13, 25]).

The invasive procedures underwent by the patients with NI are shown in table 10.

Table 10: Invasive procedure done in patients with NI

Procedure	Total No. Patients
Intravenous cannula (peripheral)	9
Skin biopsy	8
Lymph node biopsy	2
Bone marrow biopsy	2
Central line intravenous cannula	1

BACTERIAL PATHOGENS CAUSING NI

A total of 22 bacterial isolates were obtained from different specimens, pus (15), blood (1), urine (1) as shown in table 11. Source of NI in cases included, skin (pustules [11], furuncle [2]), lymph node abscess [n = 2]), blood stream infection [1], urine (supra-pubic aspiration) [1], 5 patients had 2 isolates.

The commonly isolated organism from the skin was *S.aureus* 14/19 (73.68%) followed by beta haemolytic streptococcus (2/19) 10.52%. One isolate each of *E..coli* and *P.aeruginosa* were grown from a single urine culture (supra-pubic aspiration). A solitary blood stream isolate of *E.coli* was seen.

Table 11: Bacterial isolates in cultures from different sites at development of NI

Organism	Pus(skin)	Urine	Blood	Total
Staphylococcus aureus	14			14
Beta haemolytic Streptococcus	2			2
Enterococcus	1			1
Pseudomonas aeruginosa		1		1
Escherichia.coli		1	1	2
Proteus mirabilis	1			1
Non fermenting gram negative bacilli (NF GNB)	1			1
Total	19	2	1	22

Susceptibility pattern:

The susceptibility pattern of gram positive and gram negative organisms causing NI are as shown in table 12 and table 13 respectively. Majority of the isolates of *S..aureus* 13/14 (92.9%) were methicillin-sensitive and only one was methicillin-resistant. The MRSA isolate was susceptible to linezolid, vancomycin, rifampicin and teicoplanin. The isolates of beta haemolytic Streptococci and Enterococcus were susceptible to first line antibiotics.

Table 12: Susceptibility pattern of Gram positive organisms causing NI

Organisms	***S.aureus***				**Beta haemolytic streptococci**				***Enterococci***			
Total no. of isolates	**14**				**2**				**1**			
	S	**%**	**R**	**%**	**S**	**%**	**R**	**%**	**S**	**%**	**R**	**%**
Gentamicin									1	100		
Erythromycin	8	57.1	6	42.9	2	100			1	100		
Oxacillin	13	92.9	1	7.14								
Ampicillin									1	100		
Penicillin					2	100						

Table 13: Susceptibility pattern of Gram negative organisms causing NI

Organisms	*P.aeruginosa*				*E. coli*				*Proteus mirabilis*				NFGNB			
Total no. of isolates	**1**				**2**				**1**				**1**			
	S	**%**	**R**	**%**	**S**	**%**	**R**	**%**	**S**	**%**	**R**	**%**	**S**	**%**	**R**	**%**
Ist line drugs Gentamicin			1	100			2	100			1	100				
Amikacin			1	100	2	100							1	100		
Ceftazidime			1	100			1*	50					1	100		
Ciprofloxacin			1	100			2	100			1	100	1	100		
Co-trimoxazole							1*	50			1	100	1	100		
IInd line drugs																
Imipenem			1	100	2	100										
Meropenem			1	100	1*	50										
Cefaperazone +sulbactum	I	100			1*	50										

Susceptibility not done in 1 isolate.

The isolate of *P.aeruginosa* showed resistance to all antibiotics with intermediate sensitivity to cefaperazone + sulbactam. All the isolates of *E..coli* (2/2,100%) were resistant to gentamicin and ciprofloxacin. *Proteus mirabilis* was resistant to all antibiotics. NFGNB was susceptible to ciprofloxacin and amikacin.

The comparison between cases and controls showing the various demographic profile, disease and other risk factors are shown in table 14A, table 14B and table 14C respectively.

Table 14: Risk factors of NI: cases and control

Table 14 A: Demographic details

	Cases (17)	Control (34)	p value
Age:	42.06 ± 14.8	42.50 ± 18.2	0.931
Sex:			
Male	11 (64.7)	18 (52. 9)	0.424
Female	6 (35.3)	16 (47.1)	
BMI:			
Obese(>25)	5 (29.4)	11 (32.4)	0.831
Non-obese(<24.9)	12 (70.6)	23 (67.6)	
Associated Diabetes mellitus:			
Yes	2 (11.8)	9 (26.5)	0.229
No	15 (88.2)	25 (73.5)	

Table 14 B: Disease profile

	Cases	Control	p value
Primary Dermatological disease: Autoimmune-bullous disorder	7 (41.2%)	6 (17.6%)	
Erythroderma	2 (11.8%)	2 (5.9%)	0.15
Psoriasis	3 (17.6%)	5 (14.7%)	
Others	5 (29.4%)	21 (61.8%)	
Body Surface Area Involvement (BSA%): Median Range	30 (10-65)	13 (5-28.75)	0.086
Duration of illness: (weeks)			
Acute (<6 weeks)	3 (17.6)	7 (20.6)	0.803
Chronic (>6 weeks)	14 (82.4)	27 (79.4)	

Table 14 C: Other risk factors

	Cases	Control	p value
Previous hospitalization:			
Yes	11 (64.7)	9 (26.5)	0.008
No	6 (35.3)	25 (73.5)	
Prior Antibiotic Intake:			
Yes	5 (29.4)	4 (11.8)	0.119
No	12 (70.6)	30 (88.2)	
Previous steroid/immunosuppressive:			
Yes	11 (64.7)	18 (52.9)	0.42
No	6 (35.3)	16 (47.1)	
Infection at admission:			
Yes	10 (58.8)	7 (20.6)	0.006
No	7 (41.2)	27 (79.4)	
Invasive procedure done:			
Yes	15 (88.2)	10 (29.4)	0.000
No	2 (11.8)	24 (70.6)	
Duration of Hospital stay (Days)			
Median IQR	21 (13, 25)	6 (4, 9)	<0.001
Mortality	2	0	-

Table 15: Multivariate logistic regression analysis

	Odds ratio	p value
Duration of hospital stay	(1.51) CI: 1.19-1.92	0.001
Previous hospitalization	(5.09) CI: 1.46-17.83	0.01
Infection at admission	(5.51) CI: 1.29-111.32	0.029
Invasive procedure	(18) CI: 3.08-93.68	0.0000

Multivariate logistic regression was done on the factors found associated with NI in univariate analysis; the odds ratio and p values are as shown in table 15. Duration of hospital stay, previous hospitalization, infection at admission and invasive procedures were found to be associated with NI.

MANAGEMENT OF PATIENTS WITH NI

One patient was treated with parenteral vancomycin for MRSA infection. Six patients were started on empirical oral linezolid therapy for suspected MRSA skin infection which was later changed to oral cloxacillin since the culture grew MSSA. For patients with gram negative infection oral/ i.v ciprofloxacin/ gentamicin/ levofloxacin/ amikacin/ imipenem according to susceptibility pattern were used. Three patients needed to be on artificial ventilation for treatment of NI.

OUTCOME IN PATIENTS WITH NI

Two patients, one with underlying erythroderma and the other with paraneoplastic pemphigus (PNP), developed clinical sepsis. One had bacteremia with Enterococci. The overall mortality rate was 0.48% and mortality among patients with NI was 11.76%.

VIRAL INFECTION

Two patients were clinically diagnosed to have eczema herpeticum while in the ward, Tzanck smear was positive in both and herpes culture was positive in one. Since IgM HSV could not be done these were not included as NI.

OTHER INFECTIONS

One patient developed diarrhoea due to Vibrio cholerae (non 01non 139) probably due to an outside food source.

MRSA SKIN INFECTION

A total of ten patients; Male (n = 6), Female (n= 4) were detected to have MRSA infection during the study period based on pus culture reports. Seven MRSA were isolated in patients who had disease which required hospitalization elsewhere and three were community-acquired. Their age group was (mean 30.8 ± SD15.86 years), body surface involvement (mean 14.3 ± SD 13.37), previous hospitalization within one month (n = 5), prior antibiotic intake within two weeks (n = 7), prior oral steroid (n = 6), immunosuppressant (n = 6), diabetes (n = 2). The primary dermatological diseases of the patients with MRSA is as shown in table 16. The skin lesions at presentations were turbid blisters (n = 4), pustules (n = 2), erosions (n = 2), leg ulcer (n = 1) and discharging sinus (n = 1).

Table 16: Patients with MRSA disease profile

Disease profile	No of patient
Pemphigus	3
Chronic bullous dermatosis of childhood	2
Furunculosis	1
Hansen's disease	2
Allergic contact dermatitis	1
Pyoderma gangrenosum	1
Total	10

Outcome in patients with MRSA infection

Two patients received vancomycin, and the rest oral/ intravenous linezolid. Drugs were given for a total of 14 days. All the patients improved well with resolution of the infection.

DISCUSSION

Nosocomial infections are those acquired in or associated with hospitals. They are also known as hospital-acquired infections or, health care-associated infections. NI are common, and may be serious or fatal.[11] Health care-associated infections constitute a major challenge of modern medicine and are considered one of the most accurate indicators of the quality of patient care.[8] The Study on the Efficacy of NI Control (SENIC) found that a NI develops in 5-6% of hospitalized patients.[5] The overall infection rate (number of hospital-acquired infections per 1,000 patients discharged) was highest in large teaching hospitals and lowest in non-teaching hospitals.[3] Nosocomial infection are an important focus of infection prevention in all countries, but in developing countries they are a major cause of preventable disease and death.[14]

Patients in the dermatology ward, with large areas of their skin denuded, and therefore with severely compromised barrier and immune function of the skin, are susceptible to develop various infections including sepsis.[7] There are only a few studies on the incidence of development of NI among dermatology inpatients and an attempt has been made to study the same in a tertiary care referral centre. During the study period, a total of 421 patients were admitted to the dermatology ward. The primary dermatological diagnoses for which these patients were admitted were psoriasis (16.86%), autoimmune bullous disorders (11.87%), and Hansen's disease (10.21%). Co-morbid diseases were seen in 62.7% of these patients. Of the 421 patients, 28.26% had been diagnosed to

have an infection at admission. Almost 60% of these patients had an appropriate culture done and 62.03% of the isolates were gram positive organisms. Of the isolates, *S.aureus* was the commonest (40.5%), followed by beta haemolytic Streptococcus (17.72%) and *E.coli* (10.12%). 9/32 (28.13%) of the *S.aureus* isolates were MRSA.

During the study period the 421 patients admitted to the dermatology ward were evaluated for incidence of NI. Of the 421 patients, 17 (4.05%) developed NI. A similar study in dermatology patients by Dettenkofer M et al[6] reported an incidence of 2.5%. However, a study conducted in AIIMS, Delhi reported a high incidence of sepsis of 6.6% among dermatology inpatients.[7]

During the study period the comparison of NI between the total hospital patients and the dermatology ward showed a higher incidence among dermatology patients. However the methodology used to detect NI in the total hospital was different since the patients were surveyed only once a week and only those who were febrile were evaluated and hence the incidence of 0.57/100 discharges is probably an underestimation. Two previous studies on incidence of NI, from our institution in the past, showed an incidence of 9.7% in the general medical wards in 1993 and 50.8% in the ICU in 1999. This reveals that NIs are of importance in tertiary care centers and also among dermatology inpatients and surveillance will help to establish the rate of endemic NI.

In our study, the age group of patients who developed NI ranged from 20 to 70 years with a mean age of 42.06 years. Two patients were more than 60 years of age. It has been shown that patients at extremes of age are more prone

to develop NI and have increased mortality.[20,41] The majority of patients with NI were males (64.7%). Five were over weight (BMI>25). Age, gender, BMI or diabetes mellitus were not significantly associated with development of NI in our study.

In our study, the commonest primary dermatological diagnosis of patients who developed NI was autoimmune-bullous disorders. Other studies have reported an increased incidence of sepsis in patients with autoimmune-bullous disorders.[7] The median body surface area involvement (BSA) in patients with NI was 30% with ten patients having more than 30% involvement. It has been shown that extensive skin involvement is a significant factor in patients who develop infection, and they are at high risk of mortality.[7,41] Two patients (2/421) developed eczema herpeticum, their underlying primary disease being pemphigus vulgaris and lepromatous Hansen's disease with necrotizing ENL respectively. Since HSV IgM was not done, they were not included under NI. There is a report of nosocomial outbreak of eczema herpeticum in dermatology ward patients.[71] Eighty two percent of our patients who developed NI had chronic disease. The primary dermatological disease, the duration of illness and body surface area of involvement were not significant risk factors for NI in our study.

In our study, we found that the patients who had infection at admission were more prone to develop NI while in the ward. These patients were on antibiotic treatment after admission to the ward. Acquisition and transmission of antibiotic resistant *S.aureus* in the hospital mainly concerns intermittent and persistent nasal carriers treated with antibiotics.[42] Since the carrier status of our

patients was not determined, the importance of this factor in the development of NI could not be determined. However this merits further study. An increased chance of colonization followed by clinical infection in dermatology and burns patients has been reported.[69] Eleven patients were on systemic steroids/immunosuppressive medications and five patients were on antibiotics prior to hospitalization. Though prior antibiotic and immunosuppressive therapy are regarded as risk factors in the development of NI,[7,11,34,37] this was not found to be significant in our study. Eleven of 17 patients had history of previous hospitalization. This was a significant factor associated with development of NI and has been consistently shown in various studies to promote the development of NI and MRSA.[11,52] The median duration of hospital stay in our patients with NI was 21 days. This was a significant factor among patients with NI as compared to controls and various studies support the evidence that prolonged stay in hospital is a risk factor for development of NI.[7,40] In our study, fifteen patients among the NI group underwent invasive procedures like skin/ lymph node/bone marrow biopsy and nine of them had peripheral intravenous cannulation. Two patients who underwent lymph node biopsy developed abscesses at the local site. Since patients with skin disease may have loss of integrity of skin, this paves the way for colonization with hospital flora and subsequent development of NI. Dettenkofer et al[6] showed that 57% of all NIs documented were SSI. In our study a total of 22 bacterial isolates were obtained at the time of development of NI from these 17 patients. Among the NI, gram positive organisms were the commonest pathogens isolated from the skin lesions, of which *S.aureus*

constituted 73.7% (14/19). Thirteen (92.85%) of these were MSSA and only one was MRSA. In contrast, 9/32 (28.13%) of the *S.aureus* isolates from infected lesions at admission were MRSA. Majority of the isolates causing NI in previous studies on dermatology inpatients was also *S.aureus* 62.5% in the study by Selwyn et al[36] and 41.8% in the study by Sharma et al[7].

E.coli which was isolated from blood culture was resistant to all first line antibiotics and was susceptible only to imipenem. The urinary isolate *P. aeruginosa* was resistant to all first and second line antibiotics and *E.coli* was susceptible only to imipenem. The hospital acquired gram negative bacilli showed resistance to the first line antibiotics as reported by Sharma VK et al[7] in the study of sepsis on dermatology patients.

In this study, 2 of 421 patients who developed sepsis died, mortality being 0.48%. A similar study from Delhi, India on sepsis reported 2% of mortality among dermatology inpatients.[7] So it is important to study the risk factors associated with development of NI among dermatology inpatients, which could help in instituting preventive measures.

CONCLUSIONS

1. The incidence of NI in dermatology inpatient study group was 4.05 / 100 discharges with an incidence rate of 6.24 infections /1000 hospital days.
2. Autoimmune bullous diseases were the commonest dermatological diagnosis among patients who developed NI (7/17, 41%).
3. The types of NI seen were skin infections in 13 patients, SSI in 2 patients, urinary tract and blood stream infection in 1 patient each.
4. The commonest organism causing an NI in our study population was *S. aureus* (63.6%) and among these there was only one isolate of MRSA.
5. Gram negative organisms causing NI were 22.72% of the isolates, and *pseudomonas, E.coli, and P.mirabilis* were resistant to all first line antibiotics.
6. Median time to onset of NI was 11 days (IQR 8.10, 13.90.)
7. Nosocomial infection was significantly associated with the following factors.

   (a) Previous hospitalization (OR5.09, 95% CI 1.46 – 17.83) p value=0.01

   (b) Infection at admission (OR 5.51, 95% CI 1.29 – 111.32) p value=0.029

   (c) Invasive procedures (OR 18, 95% CI 3.08 – 93.68) p value=0.0000
8. Median duration of hospital stay among patients with NI was 21 days (IQR 13, 25) and was significantly associated with NI (OR1.51, 95% CI 1.19 – 1.92) p value=0.001

9. Overall mortality rate among the study population was 0.48%, and mortality among patients with NI was 11.76%.

SUMMARY

A prospective cohort study was done to determine the incidence, the risk factors, and pathogens responsible for development of NI in the dermatology ward of a tertiary care centre from 1st November 2006 to 15th September 2007.

During the study period, a total of 421 patients were admitted to the dermatology ward. The common primary dermatological diagnoses for which these patients were admitted were psoriasis (16.86%), autoimmune-bullous disorders (11.87%), and Hansen's disease (10.21%). Co morbid diseases were seen in 62.7% of these patients. Of the 421 patients, 119 (28.26%) had been diagnosed to have an infection at admission. Almost 60% of these patients had an appropriate culture done and 62.03% of the isolates were gram positive organisms. Of the isolates, *S.aureus* was the commonest (40.5%), followed by Streptococcus (17.72%) and *E.coli* (10.12%). 9/32 (28.13%) of the *S.aureus* isolates were MRSA.

Of the 421 patients studied, 17 patients developed NI with an incidence of 4.05%, and an incidence rate of 6.24 infections/1000 hospital days. Of 119 patients who had infections at admission, 10 developed NI. The mean age of the patients was 42.06 years with a male preponderance (64.7%). The commonest primary underlying dermatological diagnosis in patients with NI was autoimmune bullous-disorders. The median time to onset of NI was 11 days (IQR, 8.1, 13.9). The types of NI seen were skin infections in 13 patients, SSI in 2 patients, urinary tract and blood stream infection in 1 patient each. Eleven of them had previous

hospitalization and were on steroid/immunosuppressive treatment. Fifteen of them had invasive procedures done. The median duration of hospitalization for patients with NI was 21 days (IQR 13, 25). This was significantly associated with NI (OR 1.51, 95% CI 1.19-1.92). The commonest organism causing an NI in our study population was *S. aureus* (63.6%) and among these there was only one isolate of MRSA. Gram negative organisms causing NI were 22.72% of the isolates, and *pseudomonas, E.coli, and P.mirabilis* were resistant to all first line antibiotics. Nosocomial infection was significantly associated with the following factors: Previous history of hospitalization (OR5.09, 95% CI 1.46 – 17.83); infection at admission (OR 5.51, 95% CI 1.29 – 111.32) and invasive procedures (OR 18, 95% CI 3.08 – 93.68). Two patients among 421 died due to sepsis. Overall mortality rate among the study population was 0.48%, and mortality among patients with NI was 11.76%.

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LIMITATIONS

1. Nasal carriage detection could not be done for patients who were admitted due to financial constraints.

2. Serology for IgM antibodies for HSV was not done and so Kaposi's varicelliform eruption could not be classified as NI.

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Annexure I

INFORMED CONSENT DOCUMENT

It has been explained to me that the study involves my examination, the necessity of investigations as well as need for regular follow up. I also understand that the information I divulge is confidential and shall be used for study purposes only. Thereafter I give my informed consent for the same.

Date: Signature of the patient/guardian

CMCH, Vellore.

Annexure II

Proforma:

DERMATOLOGY WARD NOSOCOMIAL INFECTION (NI) SURVEILLANCE

Name Hospital No. Age/Sex

Occupation: Ward: Ht: Wt:

BMI:

DOA: DOD:

No. days hospital stay:

Duration of illness:

Dermatological: Skin lesions: 1. Primary

Non dermatological: 2. Secondary

Body surface Area Involvement:

Temperature at admission : Duration of fever

Associated symptoms:

Previous hospitalization: Yes/ No

Treatment: Previous Medications: Yes/ No

If Yes-date/details: Antibiotics:

Steroids(Topical/Oral)

Immunosuppressive:

Others:

Diagnosis: Dermatological:

Non Dermatological:

Date	PR	RR	BP	Temp	Skin lesions	Systemic exam
1.						
2.						
3.						

Initial Microbiology:

Date	Specimen	Gram stain	Culture/Sensitivity
1.			
2.			
3.			

Treatment:

Antibiotic	date started	last date	reason for stopping
1.			
2.			
3.			

Invasive procedure done: Yes/ No

If Yes- Procedure done:

Date	procedure	result
1.		
2.		
3.		

Suspicion of NI date:

Reason for Diagnosis of NI

1.
2.
3.

Microbiology suspected NI:

Date	specimen	Gram stain	C/S
1.			
2.			
3.			

Antibiotic for NI	date started	last date	reason for stopping
1.			
2.			
3.			

Final outcome:

Condition at discharge: Improved/Statisquo:

Drugs at discharge:

Follow up at 1 week after discharge:

Annexure III

CDC Definition of Nosocomial Infections

INFECTION SITE: Skin

CODE: SST-SKIN

DEFINITION: Skin infections must meet at least one of the following criteria:

Criterion 1: Patient has purulent drainage, pustules, vesicles,or boils.

Criterion 2: Patient has at least *two* of the following signs or symptoms with no other recognized cause: painor tenderness, localized swelling, redness, or heat

and

at least *one* of the following:

a. Organisms cultured from aspirate or drainagefrom affected site; if organisms are normal skin flora (e.g., coagulase negative staphylococci, micrococci, diphtheroids) they must be a pure culture

b. Organisms cultured from blood

c. Positive antigen test performed on infected tissue or blood (e.g., herpes simplex, varicella zoster, *H. influenzae, N. meningitidis*)

d. Multinucleated giant cells seen on microscopic examination of affected tissue

e. Diagnostic single antibody titer (IgM) or fourfold increase in paired sera (IgG) for pathogen

COMMENT:

_ Nosocomial skin infections may be the result of exposure to a variety of procedures performed in the hospital. Superficial incisional infections after surgery are identified separately as SSI-SKIN unless the operative procedure is a CBGB. If the chest incision site after a CBGB becomes infected, the specific site is denoted SKNC; if the donor site becomes infected, the specific site is denoted SKNL. Other skin infections associated with important exposures are identified with their own sites and are listed in the section on reporting instructions.

REPORTING INSTRUCTIONS:

_ Report omphalitis in infants as UMB.

_ Report infections of the circumcision site in newborns as
CIRC.

_ Report pustules in infants as PUST.

_ Report infected decubitus ulcers as DECU.

_ Report infected burns as BURN.

_ Report breast abscesses or mastitis as BRST.

INFECTION SITE: Soft tissue (necrotizing fascitis, infectious
gangrene, necrotizing cellulitis, infectious myositis, lymphadenitis,
or lymphangitis)

CODE: SST-ST

DEFINITION: Soft tissue infections must meet at least one of the following criteria:

Criterion 1: Patient has organisms cultured from tissue or drainage from affected site.

Criterion 2: Patient has purulent drainage at affected site.

Criterion 3: Patient has an abscess or other evidence of infec*Section*

XV: Organization and Implementation 1684 *of Infection Control Programs* tion seen during a surgical operation or histopathologic examination.

Criterion 4: Patient has at least *two* of the following signs of symptoms at the affected site with no other recognized ca use: localized pain or tenderness, redness, swelling, or heat

and

at least *one* of the following:

a. Organisms cultured from blood

b. Positive antigen test performed on blood or urine (e.g., *H. influenzae, S. pneumoniae, N. meningitidis,* group B *Streptococcus, Candida* sp.)

c. Diagnostic single antibody titer (IgM) or fourfold increase in paired sera (IgG) for pathogen

REPORTING INSTRUCTIONS:

_ Report surgical site infections that involve both the skin and deep soft tissue (at or beneath the fascial or muscle layer) as SSI-ST (soft tissue) unless the operative procedure is a CBGB.

For CBGB, if skin and deep soft tissue at the chest incision site become infected, the specific site is STC and if skin and deep soft tissue at the donor site become infected, the specific site is STL.

_ Report infected decubitus ulcers as DECU.

_ Report infection of deep pelvic tissues as OREP.

INFECTION SITE: Decubitus ulcer, including both superficial and deep infections

CODE: SST-DECU

DEFINITION: Decubitus ulcer infections must meet the following criterion:

Patient has at least *two* of the following signs or symptoms with no other recognized cause: redness, tenderness, or swelling of decubitus wound edges

And

at least *one* of the following:

a. Organisms cultured from properly collected fluid or tissue (see later)

b. Organisms cultured from blood

COMMENTS:

_ Purulent drainage alone is not sufficient evidence of an infection.

_ Organisms cultured from the surface of a decubitus ulcer are

not sufficient evidence that the ulcer is infected. A properly collected specimen from a decubitus ulcer involves needle aspiration of fluid or biopsy of tissue from the ulcer margin.

INFECTION SITE: Surgical site infection (deep incisional)

CODE: SSI-[ST (soft tissue)] except following the NNIS operative procedure, CBGB. For CBGB only, if infection is at chest site, use STC (soft tissue-chest) or if at leg (donor) site, use STL (soft tissue-leg)

DEFINITION: A deep incisional SSI must meet the following criteria:

Infection occurs within 30 days after the operative procedure if no implantb is left in place or within 1 year if implant is in place and the infection appears to be related to the operative procedure

and

involves deep soft tissues (e.g., fascial and muscle layers) of the incision

and

patient has at least *one* of the following:

a. Purulent drainage from the deep incision but not from the organ/space component of the surgical site

b. A deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (_38_C) or localized pain or tenderness, *unless* incision is culture-negative c. An abscess or other evidence of infection involving the deep incision is found on direct b A nonhuman-derived implantable foreign body (e.g., prosthetic heart valve, nonhuman vascular graft, mechanical heart, or hip prosthesis) that is permanently placed in a patient during surgery.*Chapter 94: Surveillance of Nosocomial Infections* 1675 examination, during reoperation, or by histopathologic or radiologic examination

d. Diagnosis of a deep incisional SSI by a surgeon or attending physician

REPORTING INSTRUCTIONS:

_ Classify infection that involves *both* superficial and deep incision sites as deep incisional SSI._ Report culture specimen from deep incisions as ID.

INFECTION SITE: Burn

CODE: SST-BURN

DEFINITION: Burn infections must meet one of the following criteria:

Criterion 1: Patient has a change in burn wound appearance or character, such as rapid eschar separation; dark brown, black, or violaceous discoloration of the

char; or edema at wound margin *and* histologic examination of burn biopsy shows invasion of organisms into adjacent viable tissue.

Criterion 2: Patient has a change in burn wound appearance or character, such as rapid eschar separation; dark brown, black, or violaceous discoloration of the

eschar; or edema at wound margin *and* at least *one* of the following:

a. Organisms cultured from blood in the absence of other identifiable infection

b. Isolation of herpes simplex virus, histologic identification of inclusions by light or electron microscopy or visualization of viral particles by electron microscopy in biopsies or lesion scrapings

Criterion 3: Patient with a burn has at least *two* of the following signs or symptoms with no other recognized cause: fever (_38_C) or hypothermia (_36_C), hypotension, oliguria (_20 cm3/hr), hyperglycemia at previously tolerated level of dietary carbohydrate, or mental confusion

and

at least *one* of the following:

a. Histologic examination of burn biopsy shows invasion of organisms into adjacent viable tissue

b. Organisms cultured from blood

c. Isolation of herpes simplex virus, histologic I dentification of inclusions by light or electron microscopy, or visualization of viral particles electron microscopy in biopsies or lesion scrapings

COMMENTS:

_ Purulence alone at the burn wound site is *not* adequate for the diagnosis of burn infection; such purulence may reflect incomplete wound care.

_ Fever alone in a burn patient is *not* adequate for the diagnosis of a burn infection because fever may be the result of tissue trauma or the patient may have an infection at another site.

_ Surgeons in Regional Burn Centers who take care of burn patients exclusively, may require Criterion 1 for diagnosis burn infection.

_ Hospitals with Regional Burn Centers may further divide burn infections into the following: burn wound site, burn graft site, burn donor site, burn donor site-cadaver; the NNIS system, however, will code all of these as BURN.

INFECTION SITE: Clinical sepsis

CODE: BSI-CSEP

DEFINITION: Clinical sepsis must meet at least one of the following criteria:

Criterion 1: Patient has at least *one* of the following clinical signs or symptoms with no other recognized cause:

fever (_38_C), hypotension (systolic pressure _90 mm Hg), or oliguria (_20 cm3/hr)

and

blood culture *not* done or *no* organisms or antigen detected in blood

and

no apparent infection at another site

and

physician institutes treatment for sepsis.

Criterion 2: Patient _1 year of age has at least *one* of the following clinical signs or symptoms with no other recognized cause: fever (_38_C), hypothermia (_37_C), apnea, or bradycardia

and

blood culture *not* done or *no* organisms or antigen detected in blood

and

no apparent infection at another site

and

physician institutes treatment for sepsis.

REPORTING INSTRUCTION:

_ Report culture-positive infections of the bloodstream as BSILCBI.

INFECTION SITE: Symptomatic urinary tract infection

CODE: UTI-SUTI

DEFINITION: A symptomatic urinary tract infection must meet at least one of the following criteria:

Criterion 1: Patient has at least *one* of the following signs or symptoms with no other recognized cause: fever (_38_C), urgency, frequency, dysuria, or suprapubic tenderness

and

patient has a positive urine culture, that is, _105 microorganisms per cm3 of urine with no more than two species of microorganisms.

Criterion 2: Patient has at least *two* of the following signs or *Chapter 94: Surveillance of Nosocomial Infections* 1673 symptoms with no other recognized cause: fever (_38_C), urgency, frequency, dysuria, or suprapubic tenderness

and

at least *one* of the following:

a. Positive dipstick for leukocyte esterase and/or nitrate

b. Pyuria (urine specimen with _10 WBC/mm3 or_3 WBC/high power field of unspun urine)

c. Organisms seen on Gram stain of unspun urine

d. At least *two* urine cultures with repeated isolation of the same uropathogen (gram-negative bacteria or *S. saprophyticus*) with _102 colonies/ mL in nonvoided specimens

e. _105 colonies/mL of a single uropathogen (gram-negative bacteria or *S. saprophyticus*) in a patient being treated with an effective antimicrobial agent for a urinary tract infection

f. Physician diagnosis of a urinary tract infection

g. Physician institutes appropriate therapy for a urinary tract infection

Criterion 3: Patient _1 year of age has at least *one* of the following signs or symptoms with no other recognized cause: fever (_38_C), hypothermia (_37_C), apnea, bradycardia, dysuria, lethargy, or vomiting

and

patient has a positive urine culture, that is, _105 microorganisms per cm3 of urine with no more than two species of microorganisms.

Criterion 4: Patient _1 year of age has at least *one* of the following signs or symptoms with no other recognized

cause: fever (_38_C), hypothermia (_37_C), apnea, bradycardia, dysuria, lethargy, or vomiting

and

at least *one* of the following:

a. Positive dipstick for leukocyte esterase and/or nitrate

b. Pyuria (urine specimen with _10 WBC/mm3 or_3 WBC/high power field of unspun urine)

c. Organisms seen on Gram stain of unspun urine

d. At least *two* urine cultures with repeated isolation of the same uropathogen (gram-negative bacteria or *S. saprophyticus*) with _102 colonies/ mL in nonvoided specimens

e. _105 colonies/mL of a single uropathogen (gram-negative bacteria or *S. saprophyticus*) in a patient being treated with an effective antimicrobial agent for a urinary tract infection

f. Physician diagnosis of a urinary tract infection

g. Physician institutes appropriate therapy for a urinary tract infection

COMMENTS:

_ A positive culture of a urinary catheter tip is *not* an acceptable laboratory test to diagnose a urinary tract infection.

_ Urine cultures must be obtained using appropriate technique, such as clean catch collection or catheterization.

_ In infants, a urine culture should be obtained by bladder catheterization or suprapubic aspiration; a positive urine culture from a bag specimen is unreliable and should be confirmed by a specimen aseptically obtained by catheterization

or suprapubic aspiration.

INFECTION SITE: Asymptomatic bacteriuria

CODE: UTI-ASB

DEFINITION: An asymptomatic bacteriuria must meet at least one of the following criteria:

Criterion 1: Patient has had an indwelling urinary catheter within 7 days before the culture

and

patient has a positive urine culture, that is, _105 microorganisms per cm3 of urine with no more than two species of microorganisms

and

patient has *no* fever (_38_C), urgency, frequency, dysuria, or suprapubic tenderness.

Criterion 2: Patient has *not* had an indwelling urinary catheter within 7 days before the first positive culture

and

patient has had at least *two* positive urine cultures, that is, _105 microorganisms per cm3 of urine with repeated isolation of the same microorganism and no more than two species of microorganisms

and

patient has *no* fever (_38_C), urgency, frequency, dysuria, or suprapubic tenderness.

COMMENTS:

_ A positive culture of a urinary catheter tip is *not* an acceptable laboratory test to diagnose bacteriuria.

_ Urine cultures must be obtained using appropriate technique, such as clean catch collection or catheterization.

APPENDIX A-2. PNEUMONIA ALGORITHMS

Major Site: Pneumonia (PNEU)

Site-Specific Algorithms for Clinically Defined Pneumonia (PNU1)

Radiology Signs/symptoms/laboratory Code

Two or more serial chest radiographs with at least *one*

of the following1,2: New or progressive

and persistent infiltrate

Consolidation

Cavitation

Pneumatoceles, in infants _1 year old

NOTE: In patients without

underlying pulmonary or

cardiac disease (e.g., respiratory distress syndrome, bronchopulmonary dysplasia, pulmonary edema, or chronic obstructive pulmonary disease),

one definitive chest radiograph is acceptable1.

PNU1

FOR ANY PATIENT, at least *one* of the following:

• Fever (_38°C or _100.4°F) with no other recognized cause

• Leukopenia (_4,000 WBC/mm3) *or* leukocytosis (_12,000 WBC/mm3)

• For adults _70 years old, altered mental status with no other recognized cause

and

At least *two* of the following:

• New onset of purulent sputum3, or change in character of sputum4, or increased respiratory secretions, or increased suctioning requirements

• New onset or worsening cough, or dyspnea, or tachypnea5

• Rales6 or bronchial breath sounds

• Worsening gas exchange (e.g., O2 desaturations [e.g., PaO2/FiO2 _240]7, increased oxygen requirements, or increased ventilation demand)

ALTERNATE CRITERIA FOR INFANT _1 YEAR OLD:

Worsening gas exchange (e.g., O2 desaturations, increased oxygen requirements, or increased ventilator demand)

and

at least *three* of the following:

• Temperature instability with no other recognized cause

• Leukopenia (_4,000 WBC/mm3)

or leukocytosis (_15,000 WBC/mm3) and left shift (_10% band forms)

• New onset of purulent sputum3, or change in character of sputum4, or increased respiratory secretions, or increased suctioning requirements

• Apnea, tachypnea5, nasal flaring with retraction of chest wall, or grunting

• Wheezing, rales6, or rhonchi

• Cough

• Bradycardia (_100 beats/min) or tachycardia (_170 beats/min)

ALTERNATE CRITERIA FOR CHILD _1 OR _12 YEARS OLD, at least *three* of the following:

• Fever (_38.4°C or _101.1°F) or hypothermia (_37°C or _97.7°F) with no other recognized cause

• Leukopenia (_4,000 WBC/mm3) *or* leukocytosis (_15,000 WBC/mm3)

• New onset of purulent sputum3, or change in character of sputum4, or increased respiratory secretions, or increased suctioning requirements

• New onset or worsening cough or dyspnea, apnea, or tachypnea5

• Rales6 or bronchial breath sounds

• Worsening gas exchange (e.g., O2 desaturations [e.g., pulse oximetry _94%], increased oxygen requirements, or increased ventilation demand).

Annexure IV GLOSSARY FOR MASTER SHEET

For the columns, code is indicated below the abbrevation

C1	**S.No**	
C2	**Hospt.No**	
C3	**Age (Yrs)**	
C4	**Sex**	
C5	**BMI**	
C6	**DOA**	
C7	**DOD**	
C8	**Dur illness Derm**	
C9	**Diagnosis Dermatological**	1= Psoriasis; 2= Autoimmune-bullous disorder; 3= Other dermatoses; 4= Hansen's disease; 5= Allergic contact dermatitis; 6= Drug reactions; 7= Viral diseases; 8= Erythroderma; 9 = Atopic dermatitis; 10 = Urticaria; 11 = Chronic leg ulcer; 12 = Vasculitis; 13 = Vitiligo; 14 = Cellulitis 15 = Panniculitis; 16 = Skin malignancy; 17 = Connective tissue disorder
C10	**Diagnosis Non Dermatological**	1 = Type II Diabetes Mellitus; 2 = Essential Hypertension; 3 =Anemia (iron deficiency/ chronic disease); 4 = Dyslipidemia 5 = Obesity; 6 = COPD*; 7 = Osteoarthritis; 8 = Hypothyroidism; 9 = Tuberculosis; 10 = HIV infection; 11 =Seizure disorder; 12 = Osteoporosis
C11	**BSA (%)**	
C12	**Previous Hoptalization**	1= Yes; 2 = No
C13	**Previous Antibiotic**	1= Yes; 2 = No
C14	**Previous Steroids**	1A = Prednisolone; 1B = Dexamethasone; 1C = Injectable steroid ; 1D = Topical steroid
C15	**Previous Immunosuppressives**	1A = Methotrexate ; 1B = Azoran; 1C = Mycept ; 1D = Cyclosporin ; 1E = Cyclophosphamide; 1F = DCP pulse 1G = Retinoids (oral)
C16	**Initial Micro (Date)**	
C17	**Specimen**	1 = Pus ; 2 = Blood; 3 = Urine ;4 = Sputum;5 = Stool ;6 = Herpes culture;7 = Tissue culture;8 = Blister fluid;9 = Throat swab;10 = Tsank smear
C18	**Culture sensitivity**	1 = Coag. Neg Staph;2 = MSSA;3 = MRSA;4 = Enterococcus 5 = Enterobacter;6 = Micrococcus;7 = Pseudomonas;8 = Klebsiella; 9 = Proteus;10= β streptococcus;11 = no growth 12 = NFGNB;13 = Diptheria;14 = E.Coli;15 = Herepes growth present;16 = MNG;17 = Shigella;18 Citrobacter
C19	**Treatment Antibiotic**	1 = Cap. Cloxacillin;2 = Inj. Cloxacillin;3 = T.Linezolid 4 = T. Ciprofloxacin;5 = Inj. Ciprofloxcin;6 = Inj. Gentamycin 7 = Inj. Cefataxime;8 = Inj. Vancomycin;9 = Inj. Levofloxacin 10 = T. Levofloxacin;11 = Inj.Cefipime;12 = Inj. Ceftazidime 13 = T. Cefedroxil;14 = Cap. Amoxy;15 = Inj. Linezolid 16 = T. Doxycyclin;17 = T. Acyclovir;18 = Cap. Cephalexin 19 = T.Metronidazole;20 = Inj. Cefazolin;21 = T.Azithromycin 22 = Inj.Amikacin;23 = T.Erythromycin;24 = T.Augmentin 25 = T.Norfloxcin;26 = T.Septran;27 = Fucidin cream; 28 = Muprocin cream
C20	**Procedure**	1 = skin biopsy; 2 = i.v line; 3 = lymph node biopsy; 4 = bone marrow biopsy; 5 = central line; 6 = FNAC; 7 = I&D; 8 = Lumbar puncture
C21	**Micro NI Date**	

C22	**Specimen**	1 = Pus; 2 = Blood; 3 = Urine; 4 = Sputum; 5 = Stool ; 6 = Herpes culture; 7 = Tissue culture; 8 = Blister fluid; 9 = Throat swab; 10 = Tsank smear
C23	**Gram Stain**	1 = GPC; 2 = GNB; 3 = GPB
C24	**C/S**	1 = Coag. Neg Staph;2 = MSSA;3 = MRSA;4 = Enterococcus 5 = Enterobacter;6 = Micrococcus;7 = Pseudomonas;8 = Klebsiella; 9 = Proteus;10= β streptococcus;11 = no growth 12 = NFGNB;13 = Diptheria;14 = E.Coli;15 = Herepes growth present;16 = MNG;17 = Shigella;18 Citrobacter
C25	**Antibiotic for NI**	1 = Cap. Cloxacillin;2 = Inj. Cloxacillin;3 = T.Linezolid 4 = T. Ciprofloxacin;5 = Inj. Ciprofloxcin;6 = Inj. Gentamycin 7 = Inj. Cefataxime;8 = Inj. Vancomycin;9 = Inj. Levofloxacin 10 = T. Levofloxacin;11 = Inj.Cefipime;12 = Inj. Ceftazidime 13 = T. Cefedroxil;14 = Cap. Amoxy;15 = Inj. Linezolid 16 = T. Doxycyclin;17 = T. Acyclovir;18 = Cap. Cephalexin 19 = T.Metronidazole;20 = Inj. Cefazolin;21 = T.Azithromycin 22 = Inj.Amikacin;23 = T.Erythromycin;24 = T.Augmentin 25 = T.Norfloxcin;26 = T.Septran;27 = Fucidin cream; 28 = Muprocin cream
C26	**Date start**	
C27	**Last Date**	
C28	**Reason for Stopping antibiotic**	1 = Complete course; 2 = Empirical; 3 = Coag. Neg staph 4 = Lesion not better; 5 = Angiodema; 6 = difficult i.v access 7 = fever persisting; 8 = continued; 9 = change to oral 10 = better
C29	**Condition at discharge**	1 = improved; 2 = expired; 3 = statisquo
C30	**Follow up at 1 wk**	1 = normal; 2 = new turbid vesicles; 3 = not better; 4 = not known