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Microbial Infections in Patients Suffering from Burn Injuries: A Systematic Review

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Abstract

Polymicrobial infections are common in patients suffering from burn injuries. Hospitalized patients are at a heightened risk of contracting hospital-acquired infections and extended hospital stays raise the possibility of infection with resistant organisms. Pseudomonas aeruginosa, Proteus mirabilis, Acinetobacter baumannii, and Klebsiella pneumoniae are the most often found multidrug-resistant (MDR) Gram-negative bacteria in burn wound infections (BWIs). BWIs caused by Gram positive organism like Staphylococcus and Streptococcus are also prevalent. Fungi-like Candida species appear to occur also. Nonetheless, opportunistic pathogen infection is highly prevalent in burn victims. Variations in geographic location and infection control practices result in variations in the causal agents of BWIs. All things considered, increased serum cytokine levels, systemic immune response and immunosuppression are indicative of burn injuries. Therefore, prompt identification and intervention can quicken the healing of wounds and lower the chance of developing new infections at the site of injury. A multidisciplinary approach from infectious disease experts and burn surgeons is also required to effectively track antibiotic resistance in BWI pathogens, prevent the super-spread of MDR infections and enhance treatment results.

Keywords: *Burn wound infections; Biofilm; Hospital acquired infection; Multidrug resistant; Opportunistic infection.*

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Introduction:

Being the largest anatomical organ in humans, the skin is engaged in several physiological processes including proprioception, thermoregulation, homeostasis maintenance, and defense against environmental threats[1]. Man's physical defense against disease invasion is his skin. Thus, conditions that result in the loss of skin integrity have a number of grave repercussions[1]. A serious global public health concern is burn injuries. The epidermal barrier is damaged, which results in the downregulation of the immune system on both a local and systemic level[1]. Burn wounds thus become the perfect environment for bacteria to grow[1,2]. Burn wound exudates (BWEs), a biological fluid that predominates in the wound, serve as an excellent microenvironment that is optimal for the growth of pathogens[3]. First-degree (superficial) burns damage only the epidermal layer, so they heal rather quickly without scarring[2]. Second-degree (partial-thickness) burns involve the deeper layers of the epidermis and dermis and heal slowly[2]. Third-degree (full-thickness) burns fully destroy the epidermal and dermal layers of the skin and can also cause significant damage to the underlying tissues and bones as well[2]. One extremely prevalent and severe type of trauma is burn. With a crude fatality rate of 5%, it is ranked eighth among all traumatic injuries by the World Health Organization[4]. An estimated 2.65 lakh people die from burn injuries each year worldwide. These situations are more common in undeveloped and underdeveloped nations, where burns that cover more than 40% of the body's surface area have a 100% patient mortality risk[3,5]. Eighty percent of burns happen at home[6]. Children and teenagers are more likely to get burn injuries from domestic sources[6,7]. Southeast Asia leads the globe in deliberate burn injuries, followed by Africa[8]. Asia has the greatest rate of burn injuries worldwide. India is the Asian nation with the greatest number of reported cases of burning self-harm, followed by Bangladesh, Pakistan, and Bhutan. With a burn injury fatality rate of 23.5% annually, Africa has the highest rate[8]. Due to domestic abuse or self-immolation, young women make almost 65% of burn casualties in India[9]. Conversely, children are the most common victims of burn injuries in Africa. Asia and Africa have high population densities, low income rates, low levels of education, and inadequate surveillance systems, all of which contribute to the high incidence of burn injuries[9]. Continents like Europe, North America, South America, and Australia have notably less reported cases of burn injuries[8]. Intentional burning self-harm victims are more common among men in the 40–50 age

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range in Europe[9]. Australia is the country with the greatest annual hospital admissions of burn patients, followed by Asia. When it comes to burn injuries, these developed continents are in a far better position than the underdeveloped and developing nations. Seventy-five percent of burn deaths are the result of polymicrobial infections[2].

Pathogens of Burn Wound Infections:

Microorganisms colonize and grow quickly at the site of injury due to the loss of the skin barrier following burn. The skin barrier otherwise serves as the first line of immune defense for any individua[10,11,12]. Any breach in the skin allows for easy entry and access of the infecting microbe to the inner tissues of the body, thus complicating the etiology[10,11,12]. Hence, it has been observed that microbial infections, especially those caused by multidrug-resistant (MDR)-bacteria, including Pseudomonas and Acinetobacter, are the main cause of increased morbidity and mortality in burn patients[10,11,12]. According to the 2016 National Burn Repository Report, polymicrobial burn wound infections (BWIs) account for seven out of ten of the most common complications in burn patients. UTIs, cellulitis, and pneumonia are the most common BWIs, while respiratory tract infections are the most common[11]. The length of hospital stay following a burn injury is directly correlated with the kinds of bacteria that infect the patients; Staphylococcus aureus is the main cause of infection[13]. Skin and soft tissue infections predominate during the first week of hospitalization, while bloodstream infections, pneumonia, and urinary tract infections typically develop later in the stay[13].

Gram-Positive Bacteria: The most commonly found Gram-positive bacteria in BWI include Staphylococcus species (spp.), Enterococcus spp., and β-hemolytic group A Streptococci (GAS)[10]. Specifically, vancomycin-resistant Enterococci (VRE) and methicillin-resistant Staphylococcus aureus (MRSA) are the pathogens of high concern in patients with severe burns[10,11]. Over recent decades and with the uncontrolled over-the-counter availability of broad-spectrum antibiotics, MRSA has become the most predominant pathogen in the intensive care unit of burn patients[12]. Colonization with any of these bacteria may also lead to biofilm infections, resulting in severe illness and death[12].

Table I. Bacterial pathogens isolated from burn wound infections[13]

Bacterial pathogens	Percentage of occurrence (%)
Staphylococcus aureus	33.85
Pseudomonas aeruginosa	15.38
Acinetobacter baumannii	15.38
Klebsiella pneumoniae	13.85
Escherichia coli	8.46
Proteus mirabilis	4.62
Staphylococcus epidermis	3.85
Pseudomonas putida	3.08
Proteus morganii	0.77
Citrobacter freundii	0.77

One of the most popular therapies for reducing MRSA infection has been vancomycin. However, new antibiotic-resistant strains, such as Vancomycin-intermediate Staphylococcus aureus, have been emerging over the last few years[14]. Novel antimicrobials as daptomycin, tigecycline, quinupristin-dalfopristin, dalbavancin, and linezolid (an oxazolidinone) may offer some relief from this issue[12]. Despite being a Gram-positive bacterium of concern, Enterococcus was luckily not known to be lethal until the advent of VRE[16]. These days, combination therapy is utilized to treat VRE infections, which includes ampicillin and an aminoglycoside[16]. The most common cause of graft failure in burn patients is group B streptococci (Streptococcus agalactiae), which is followed by GAS (Streptococcus pyogenes)[15]. The penicillin class of medicines is effective in eliminating these streptococci[17].

Gram-Negative Bacteria: P. aeruginosa prefers moist conditions, it is not only the primary bacterium that is commonly found in invasive burn wounds[18]. Additionally, sepsis caused by these bacteria results in burn-related death[18]. Infections caused by pseudomonas, especially P. aeruginosa, typically begin as a small, superficial lesion with a characteristic yellow or green color and an unpleasant fruity odor. These infections can progress to an invasive infection known as "ecthyma gangrenosum," which results in blue-purplish "punched-out" lesions in the skin[19]. Sepsis can then be quickly brought on by P. aeruginosa's fast spread into deeper tissues[20]. P. aeruginosa is showing signs of developing drug resistance, hence combination therapy using piperacillin and tazobactam is used. An alternative treatment for MDR-P. aeruginosa is azatreonam[20]. Another Gram-negative bacterium in the list of high- concern microbes in burn patients is A. baumannii. Survivability in both wet and dry conditions, also on both inanimate and animate objects, helps them to achieve this[21]. As a last resort for treating pan-resistant Acinetobacter spp., colisin has been developed[21]. The development of a biofilm in the burn wound microenvironment of a patient is the primary cause of burn treatment regimen failure, and in many complex situations, this can result in mortality[22]. Importantly, the bacterial community enclosed in a polysaccharide matrix biofilm is more tolerant to antibiotics and more resilient to host immune system stresses and disinfection[20].

MDR Bacteria: The Centers for Disease Control and Prevention and the European Centre for Disease Prevention and Control state that there are two types of drug-resistant (DR) bacteria: pan-drug resistant strains, which are resistant to all agents under all antimicrobial categories, and extensively drug-resistant strains, which are resistant to at least one agent in all antimicrobial categories except a few[23,24]. The length of the patient's hospital stay and the intensity and scope of the burn are the two main variables that influence MDR-pathogen attacks[25]. Extended hospital stays elevate the risk of multidrug resistant infections, primarily caused by Gram-negative bacteria (GNB)[25,26]. The use of invasive medical devices such urinary catheters

and prior antibiotic exposure may be the cause of future increases in these BWIs[25]. A research conducted at the Canadian Burn Center, with 125 patients, provided support for this[27]. After 28 days in the hospital, 44% of the bacterial isolates were MDR, up from 6% during the first 7 days[27]. Thus, a significant treatment issue arises from the rise in the prevalence of MDR-GNB during extended hospital stays for burn patients[28]. A. baumannii, P. aeruginosa, Stenotrophomonas maltophilia, and carbapenem-resistant Enterobacteriaceae strains are a few of the MDR-GNB pathogens that are of particular concern. These are thought to be the most prevalent MDR-GNB in BWIs, along with Escherichia coli, Proteus mirabilis, and Klebsiella pneumoniae[28,29]. MRSA and GAS were shown to be prevalent in a study carried out in a burn unit at a tertiary care referral center in North India. MRSA strains were found to be resistant to erythromycin, ciprofloxacin, netilmicin, gentamicin, and cefotaxime[30]. Ninety percent of the bacteria cultivated from the infected burn wounds there showed resistance to amikacin and ceftazidime, making MDR P. aeruginosa one of the most common microbes[30]. These multidrug-resistant bacteria are first identified by examining their physical morphology, Gram-staining features, and biochemical traits[31]. In addition, to look for the zone of growth inhibition, antimicrobial susceptibility tests are performed using a variety of antibiotics, including ceftazidime, ciprofloxacin, gentamicin, trimethoprim-sulfamethoxazole, and others[31]. Here, multi-drug resistance is defined if a pathogen shows resistance to at least one agent in 3 or more antimicrobial classes[32]. Fungal Infections: The second most common microorganism that causes BWI is fungus[33]. Fungi-induced BWIs can be a component of opportunistic infections, rare severe soft tissue infections, fungemia, and mono- or polymicrobial infections[34]. Due to the similarity of these infections' symptoms to those of bacterial infections and the lack of an appropriate mycology laboratory, these infections are frequently misdiagnosed[35]. Only when there is early detection and treatment may an infection become nonfatal due to the extremely high mortality risk of these fungal infections[35,36]. Between 6.3 and 44% of all fungal infections that occur have been reported from various burn hospitals worldwide[35,37-39]. 42% of the BWI infections from a case study involving 220 burn victims were found to be Candida spp.[35,37-39]. One of the main factors contributing to burn victims' morbidity and mortality is invasive Candida infections[37]. Changes in the treatment responses and prevalence of these fungal infections have been noted as a result of the introduction of novel antifungals[40-43]. It has been observed that common anti-mycotic drugs are no longer effective against non-albicans Candida[40-44]. After the second week of their thermal injury, burn victims are typically exposed to these fungal infections[45]. The existence of fungemia, numerous positive cultures, and a deep-seated invasion of healthy skin are the causes of the high death rate[46]. Fungal infections in burn patients are made worse by the patient's age, the extent of the burns, the body surface area (30–60%), full-thickness burns, prolonged hospital stays, prolonged artificial ventilation, inhalational injuries, late surgical excision, artificial dermis, central venous catheters, fungal wound colonization, open dressing, antibiotics (like imipenem, vancomycin, and aminoglycosides), steroid treatment, hyperglycemic episodes, and immunosuppressive disorders[32,40-43,45].The diagnosis techniques used to identify mycoses at the burn site are traditional and primarily organism-specific[40]. In certain situations, a direct tissue biopsy is carried out[40]. However, because fungal cultures grow so quickly, there are instances when it is too late to begin an effective anti-mycotic medication[40]. Samples from burn wounds are taken at appropriate intervals to aid in the detection of fungal infections in the lab[47]. After seven, fourteen, twenty-one, and twenty-eight days, the burned tissue needs to be removed[46]. The purpose of tissue biopsy is to demonstrate fungal wound infections. The following formula is used to analyze tissue-specific biopsy cultures semi-quantitatively:

(CFUs × log reciprocal × 2 =colony count) / Tissue weight (g)[46]

Yeast identity in cultures is assessed using the following methods: tetrazolium reduction test, carbon and nitrogen assimilation tests, characteristic growth on cornmeal agar, and cultural features on HiCrome agar[46]. Lactophenol cotton blue (LPCB) wet mount preparation for conidiogenesis, pattern, and organization is used to identify molds[46]. Slide cultures using potato dextrose agar are used to identify non-sporulating molds[46]. The assays to determine the antifungal susceptibility of yeasts are e-strip or broth micro-dilution utilizing antifungals such as amphotericin B, fluconazole, itraconazole, voriconazole, and caspofungin[47]. Molds' susceptibility to antifungals is determined using an E-strip test employing amphotericin B.[47]. Compared to other Candida species, if Candida albicans is isolated, a lower quantity of nystatin is required for local treatment[35,45,48]. The likelihood of developing fungal infections rises as burn wounds heal longer[44]. Thus, improvements in topical antifungal therapy, the creation of pharmaceutical products to speed up wound healing, and the application of suitable systemic antifungal regimens based on antifungal susceptibility testing all contribute to better treatment outcomes for burn patients who are severely injured and prone to fungal infections[45].

Viral Infections: Burn patients are very susceptible to viral infections[49]. The immunosuppressed state of the patient after an injury triggers the reactivation of latent infection. This becomes the most common cause of viral infection post-injury49. Administration of acyclovir for a minimum of 10 days is the most commonly used antiviral therapy to treat viral infection[49].

Conclusions:

The prevention of burn injuries should be given top priority right now because it is currently a global public health emergency, particularly in developing and impoverished nations. During their hospital stay for treatment, patients

with burn injuries are more vulnerable to a variety of infections, including many multidrug-resistant bacteria, fungi, and viruses. Their weakened immune system responses, improper vascular arrangement in the burn-injured area, and worsening of severe oxidative stress are the main causes of this. Burn patients' vulnerability to deadly virus infections and MDR bacteria is influenced by immunosuppression, length of hospital stay, and geographic location. Reducing microbial transmission and infestation in burn wounds is necessary to increase burn patients' chances of survival. An efficient infection control policy is necessary for this at every level of the healthcare system. The misuse of antibiotics, the provision of a sterile environment, and the use of efficient medical equipment for the effective and critical treatment of patients may all be controlled by burn surgeons and burn care units working together to address the otherwise dire state of burn care worldwide.

References:

1. Mohapatra S, Gupta A, Agrawal K. Bacteriological profiles in burn patients within first twenty-four hours of injury. Int J Med Microbiol Trop Dis. 2016;2:71-4. https://doi.org/10.5958/2455-6807.2016.00008.8

2. Chen YY, Wu PF, Chen CS, Chen IH, Huang WT, Wang FD. Trends in microbial profile of burn patients following an event of dust explosion at a tertiary medical center. BMC Infect Dis. 2020;20 :193. https://doi.org/10.1186/s12879-020-4920-4 PMid:32131752 PMCid:PMC7057658

3. Maslova E, Eisaiankhongi L, Sjöberg F, McCarthy RR. Burns and biofilms: priority pathogens and in vivo models. NPJ Biofilms Microbiomes. 2021;7:73. https://doi.org/10.1038/s41522-021-00243-2 PMid:34504100 PMCid:PMC8429633

4. López-Jácome LE, Chávez-Heres T, Becerra-Lobato N, García-Hernández ML, Vanegas-Rodríguez ES, Colin-Castro CA, et al. Microbiology and infection profile of electric burned patients in a referral burn hospital in Mexico City. J Burn Care Res. 2020;41:390-7. https://doi.org/10.1093/jbcr/irz177 PMid:31711214

5. Forjuoh SN. Burns in low- and middle-income countries: a review of available literature on descriptive epidemiology, risk factors, treatment, and prevention. Burns. 2006;32:529-37. https://doi.org/10.1016/j.burns.2006.04.002 PMid:16777340

6. Peck MD. Epidemiology of burns throughout the world. Part I: distribution and risk factors. Burns. 2011;37:1087-100. https://doi.org/10.1016/j.burns.2011.06.005 PMid:21802856

7. Ho WS, Ying SY. An epidemiological study of 1063 hospitalized burn patients in a tertiary burns centre in Hong Kong. Burns. 2001;27:119-23. https://doi.org/10.1016/S0305-4179(00)00095-4 PMid:11226646

8. Opriessnig E, Luze H, Smolle C, Draschl A, Zrim R, Giretzlehner M, et al. Epidemiology of burn injury and the ideal dressing in global burn care: regional differences explored. Burns. 2023;49:1-14. https://doi.org/10.1016/j.burns.2022.06.018 PMid:35843806

9. Peck MD. Epidemiology of burns throughout the World. Part II: intentional burns in adults. Burns 2012;38:630-7. https://doi.org/10.1016/j.burns.2011.12.028 PMid:22325849

10. Bhardwaj S, Bhatia S, Singh S, Franco F Jr. Growing emergence of drug-resistant Pseudomonas aeruginosa and attenuation of its virulence using quorum sensing inhibitors: a critical review. Iran J Basic Med Sci. 2021;24:699-719.

11. Jeschke MG, Van Baar ME, Choudhry MA, Chung KK, Gibran NS, Logsetty S. Burn injury. Nat Rev Dis Primers. 2020;6:11. https://doi.org/10.1038/s41572-020-0145-5 PMid:32054846 PMCid:PMC7224101

12. Norbury W, Herndon DN, Tanksley J, Jeschke MG, Finnerty CC. Infection in burns. Surg Infect (Larchmt). 2016;17:250-5. https://doi.org/10.1089/sur.2013.134 PMid:26978531 PMCid:PMC4790211

13. El Hamzaoui N, Barguigua A, Larouz S, Maouloua M. Epidemiology of burn wound bacterial infections at a Meknes hospital, Morocco. New Microbes New Infect. 2020;38:100764. https://doi.org/10.1016/j.nmni.2020.100764 PMid:33163199 PMCid:PMC7600360

14. Thabet L, Messadi Aa, Mbarek M, Turki A, Meddeb B, Ben Redjeb S. Surveillance of multidrug resistant bacteria in a Tunisian hospital. Tunis Med. 2008;86:992-5.

15. Thabet L, Turki A, Ben Redjeb S, Messadi A. Bacteriological profile and antibiotic resistance of bacteria isolates in a burn department. Tunis Med. 2008;86:1051-4.

16. Von Baum H, Ober JF, Wendt C, Wenzel RP, Edmond MB. Antibiotic-resistant bloodstream infections in hospitalized patients: specific risk factors in a high-risk population? Infection. 2005;33:320-6. https://doi.org/10.1007/s15010-005-5066-4 PMid:1625886

17. Wilson GR, French GW, Sully L. Loss of split thickness skin grafts due to non-group A beta-haemolytic streptococci. Ann R Coll Surg Engl. 1988;70:217-9.

18. Williams FN, Herndon DN, Hawkins HK, Lee JO, Cox RA, Kulp GA, et al. The leading causes of death after burn injury in a single pediatric burn center. Crit Care. 2009;13:R183. https://doi.org/10.1186/cc8170 PMid:19919684 PMCid:PMC2811947

19. McManus AT, Mason AD, McManus WF, Pruitt BA. Twenty-five year review of Pseudomonas aeruginosa bacteremia in a burn center. Eur J Clin Microbiol. 1985;4:219-23. https://doi.org/10.1007/BF02013601

PMid:3924612
20. Walton MA, Villarreal C, Herndon DN, Heggers JP. The use of aztreonam as an alternate therapy for multi-resistant Pseudomonas aeruginosa. Burns. 1997;23:225-7. https://doi.org/10.1016/S0305-4179(96)00126-X PMid:9232282
21. Corbella X, Montero A, Pujol M, Domínguez MA, Ayats J, Argerich MJ, et al. Emergence and rapid spread of carbapenem resistance during a large and sustained hospital outbreak of multiresistant Acinetobacter baumannii. J Clin Microbiol. 2000;38:4086-95. https://doi.org/10.1128/JCM.38.11.4086-4095.2000 PMid:11060073 PMCid:PMC87546
22. Thomas RE, Thomas BC. Reducing biofilm infections in burn patients' wounds and biofilms on surfaces in hospitals, medical facilities and medical equipment to improve burn care: a systematic review. Int J Environ Res Public Health. 2021;18:13195. https://doi.org/10.3390/ijerph182413195 PMid:34948803 PMCid:PMC8702030
23. Falagas ME, Koletsi PK, Bliziotis IA. The diversity of definitions of multidrug-resistant (MDR) and pandrug-resistant (PDR) Acinetobacter baumannii and Pseudomonas aeruginosa. J Med Microbiol. 2006;55(Pt 12):1619-29. https://doi.org/10.1099/jmm.0.46747-0 PMid:17108263
24. Gupta M, Naik AK, Singh SK. Bacteriological profile and antimicrobial resistance patterns of burn wound infections in a tertiary care hospital. Heliyon. 2019;5:e02956. https://doi.org/10.1016/j.heliyon.2019.e02956 PMid:31886427 PMCid:PMC6921111
25. BioMérieux. Burn patients often have higher rates of multidrug-resistant infections-but there are ways to help [Internet]. BioMérieux; 2023 [cited 2024 May 1].
26. Lachiewicz AM, Hauck CG, Weber DJ, Cairns BA, Van Duin D. Bacterial infections after burn injuries: impact of multidrug resistance. Clin Infect Dis. 2017;65:2130-6. https://doi.org/10.1093/cid/cix682 PMid:29194526 PMCid:PMC5850038
27. Wanis M, Walker SA, Daneman N, Elligsen M, Palmay L, Simor A, et al. Impact of hospital length of stay on the distribution of Gram negative bacteria and likelihood of isolating a resistant organism in a Canadian burn center. Burns. 2016;42:104-11. https://doi.org/10.1016/j.burns.2015.07.010 PMid:26547832
28. Sheridan R, Weber J, Chang P. Multi-drug resistant Gram-negative bacteria colonization and infection in burned children: lessons learned from a 20-year experience. Burns Open. 2018;2:43-6. https://doi.org/10.1016/j.burnso.2017.09.002
29. Rosanova MT, Stamboulian D, Lede R. Risk factors for mortality in burn children. Braz J Infect Dis. 2014;18:144-9. https://doi.org/10.1016/j.bjid.2013.08.004 PMid:24275369 PMCid:PMC9427502
30. Taneja N, Emmanuel R, Chari PS, Sharma M. A prospective study of hospital-acquired infections in burn patients at a tertiary care referral centre in North India. Burns. 2004;30:665-9. https://doi.org/10.1016/j.burns.2004.02.011 PMid:15475139
31. Kabanangi F, Joachim A, Nkuwi EJ, Manyahi J, Moyo S, Majigo M. High level of multidrug-resistant gram-negative pathogens causing burn wound infections in hospitalized children in Dar es Salaam, Tanzania. Int J Microbiol. 2021:6644185. https://doi.org/10.1155/2021/6644185 PMid:34306091 PMCid:PMC8270727
32. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18:268-81. https://doi.org/10.1111/j.1469-0691.2011.03570.x PMid:21793988
33. Gupta N, Haque A, Lattif AA, Narayan RP, Mukhopadhyay G, Prasad R. Epidemiology and molecular typing of Candida isolates from burn patients. Mycopathologia. 2004;158:397-405. https://doi.org/10.1007/s11046-004-1820-x PMid:15630548
34. Horvath EE, Murray CK, Vaughan GM, Chung KK, Hospenthal DR, Wade CE, et al. Fungal wound infection (not colonization) is independently associated with mortality in burn patients. Ann Surg. 2007;245:978-85. https://doi.org/10.1097/01.sla.0000256914.16754.80 PMid:17522525 PMCid:PMC1876957
35. Mousa HA. Fungal infection of burn wounds in patients with open and occlusive treatment methods. East Mediterr Health J. 1999;5:333-6. https://doi.org/10.26719/1999.5.2.333 PMid:10793810
36. Ahmad S, Khan Z, Mustafa AS, Khan ZU. Epidemiology of Candida colonization in an intensive care unit of a teaching hospital in Kuwait. Med Mycol. 2003;41:487-93. https://doi.org/10.1080/1369378031000147458 PMid:14725322
37. Murray CK, Loo FL, Hospenthal DR, Cancio LC, Jones JA, Kim SH, et al. Incidence of systemic fungal infection and related mortality following severe burns. Burns. 2008;34:1108-12. https://doi.org/10.1016/j.burns.2008.04.007 PMid:18691821
38. Becker WK, Cioffi WG, McManus AT, Kim SH, McManus WF, Mason AD, et al. Fungal burn wound infection: a 10-year experience. Arch Surg. 1991;126:44-8. https://doi.org/10.1001/archsurg.1991.01410250048008 PMid:1985634
39. Ballard J, Edelman L, Saffle J, Sheridan R, Kagan R, Bracco D, et al. Positive fungal cultures in burn patients: a multicenter review. J Burn Care Res. 2008;29:213-21.

https://doi.org/10.1097/BCR.0b013e31815f6ecb
PMid:18182925
40. Greenhalgh DG, Saffle JR, Holmes JH, Gamelli RL, Palmieri TL, Horton JW, et al. American Burn Association consensus conference to define sepsis and infection in burns. J Burn Care Res. 2007;28:776-90.
https://doi.org/10.1097/BCR.0b013e3181599bc9
PMid:17925660
41. Mathew BP, Nath M. Recent approaches to antifungal therapy for invasive mycoses. ChemMedChem. 2009;4:310-23.
https://doi.org/10.1002/cmdc.200800353
PMid:19170067
42. Dries DJ. Management of burn injuries: recent developments in resuscitation, infection control and outcomes research. Scand J Trauma Resusc Emerg Med. 2009;17:14.
https://doi.org/10.1186/1757-7241-17-14
PMid:19284591 PMCid:PMC2666628
43. Struck MF. Infection control in burn patients: are fungal infections underestimated? Scand J Trauma Resusc Emerg Med. 2009;17:51-6.
https://doi.org/10.1186/1757-7241-17-51
PMid:19818134 PMCid:PMC2763002
44. De Macedo JL, Santos JB. Bacterial and fungal colonization of burn wounds. Mem Inst Oswaldo Cruz. 2005;100:535-9.
https://doi.org/10.1590/S0074-02762005000500014
PMid:16184232
45. Church D, Elsayed S, Reid O, Winston B, Lindsay R. Burn wound infections. Clin Microbiol Rev. 2006;19:403-34.
https://doi.org/10.1128/CMR.19.2.403-434.2006
PMid:16614255 PMCid:PMC1471990
46. Capoor MR, Sarabahi S, Tiwari VK, Narayanan RP. Fungal infections in burns: diagnosis and management. Indian J Plast Surg. 2010;43(Suppl):S37-42.
https://doi.org/10.4103/0970-0358.70718
PMid:21321655 PMCid:PMC3038393
47. National Committee for Clinical Laboratory Standards. Reference method for broth dilution antifungal susceptibility testing of yeasts; approved standard. Second edition. NCCLS document M27-A2 [ISBN 1-56238-469-4]. National Committee for Clinical Laboratory Standards. 2002;[cited 2024 May 1].
48. Mousa HA. Aerobic, anaerobic and fungal burn wound infections. J Hosp Infect. 1997;37:317-23.
https://doi.org/10.1016/S0195-6701(97)90148-1
PMid:9457609
49. Baj J, Korona-Głowniak I, Buszewicz G, Forma A, Sitarz M, Teresiński G. Viral infections in burn patients: a state-of-the-art review. Viruses. 2020;12:1315.
https://doi.org/10.3390/v12111315
PMid:33213058 PMCid:PMC7698518