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RESEARCH ARTICLE

CLINICAL SIGNIFICANCE AND IMPACT OF FUNGAL AND BACTERIAL INFECTIONS IN HUMAN HEALTH

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Abstract

Our study aims to explore the origins of infectious diseases and the responses of the human body to invading pathogens. Despite the development of antibiotics and vaccines, the threat of infection remains ever-present. Continuously emerging pathogens and antibiotic-resistant strains pose challenges to effective treatment, while diseases such as AIDS and rabies remain incurable. While infectious diseases have been recognized for millennia, comprehensive understanding of their causes has only emerged in the last century. We focus on elucidating the diverse array of disease-causing organisms and the underlying mechanisms that drive clinically apparent infections. This knowledge is essential for individuals and serves as a fundamental requirement for the diagnosis, treatment, and prevention of infectious diseases.

Introduction:-

Human skin is perhaps the greatest organ in the body, representing around 10% of complete body weight. For an average human, it weighs generally 5kg and covers a surface space of 2 sq. mt. Skin comprises of Epidermis, dermis, and hypodermis[1]. The epidermis is the top layer of the skin, and it is composed of keratinocytes, which are quickly separating undifferentiated organisms that help produce epidermal cells[2]. The dermis, the subsequent layer, is comprised of collagen strands in a gel-like state, just as fibroblasts. It supports the limiting of the epidermis, permitting it to adjust to the state of the body. The subcutaneous layer, which is the most profound layer of the skin, is for the most part comprised of greasy tissue[3]. The outside skin layer's essential occupation is to give a solid obstruction that secures the whole body. It fills in as the underlying line of protection[4]. It secures against radiation, infection, gases, poisons, and an assortment of other destructive substances. Skin conditions contribute 1.79% of the worldwide weight of infection around the world[5]. American Academy of Dermatology Association reports that 1 out of 4 individuals in the United States have a skin infection. Skin is suspectable to different contaminations brought about by various microorganisms like microscopic organisms, growths, protozoa, infections. Around 0.15% skin illnesses are brought about by organisms[6].

Fungus

A fungus is a primitive organism. There are millions of species of fungi. They can be plant on the soil, on shops, on shells around the house, and on your skin. Mycosis, or fungal infection, is a complaint caused by fungus[7]. Different kinds are generally classified as superficial, subcutaneous, or systemic, depending on where area of the body is

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tormented. Tinea pedis, tinea corporis, tinea corporis, tinea Eumycetoma and chromoblastomycosis are two subcutaneous kinds that affect apkins in and under the skin. Cryptococcosis, histoplasmosis, pneumocystis pneumonia, aspergillosis, and Mucor mycosis are exemplifications of systemic fungal conditions. A fungal infection within or beneath the skin can beget a bulge and skin changes[8]. People with a weakened vulnerable immune system are more susceptible to contract it. This includes persons who have HIV/ AIDS and those who are entering specifics like steroids or cancer curatives. Provocations, moulds, and fungi that can survive as both a mould and a incentive are among the fungi that beget ails in humans. In certain cases, defiled towel must be surgically removed. Fungal infections are current and have a global distribution, impacting about one billion individualities each time. In 2020, an estimated1.7 million people will die from fungal illness. Several conditions are overlooked, including sporotrichosis, chromoblastomycosis, and mycetoma[9].

Aspergillus Niger-

Aspergillus Niger is a fungus that belongs to the rubric Aspergillus. It's one of the most current species in the rubric Aspergillus[10]. It's a common food adulterant that produces a sickness known as" black earth"on colorful fruits and vegetables similar as grapes, apricots, onions, and peanuts. It's plant in soil and is constantly recorded from interior situations, where its black colonies are incorrect for Stachybotrys (species of which have also been called" black earth"). Some strains ofA. Niger have been claimed to induce strong mycotoxins nominated ochratoxins; still, other sources dispute this assertion, suggesting that the fungus species was misapplied[11]. Aspergillus Niger is classified as part of the Aspergillus subgenus Circumdati, section Nigri.A. tubingensis,A. foetidus,A. carbonarius, andA. awamori are among the 15 black-spored species in the Nigri section that may be incorrect withA. Niger. Samson et. al. reported a number of morphologically analogous taxa in 2004. The ATCC 16404 Aspergillus Niger strain was reclassified as Aspergillus brasiliensis in 2007. This needed an update to the United States Pharmacopoeia and the European Pharmacopoeia, both of which are extensively used in the pharmaceutical sector[12].

Implications-

Onions and cosmetic shops are infected with Aspergillus Niger, which produces sooty earth.A. Niger infection of onion seedlings can come systemic, appearing only when the conditions are right.A. Niger produces a frequent onion postharvest complaint in which black conidia can be seen between the bulb's scales[13]. Peanuts and grapes are also affected by the fungus.Aspergillus Niger is less likely than other Aspergillus species to beget mortal complaint. Humans may come bad in exceedingly rare cases; still, this is due to a deadly lung illness known as aspergillosis. Horticultural workers who gobble peat dust, which can be high in Aspergillus spores, are particularly susceptible to Aspergillosis. The fungus has also been discovered in ancient Egyptian grave corpses, which may be breathed whendisturbed.Otomycosis (fungal observance infections) caused byA. Niger can beget discomfort, temporary hail loss, and, in extreme cases,damage to the observance conduit and tympanic membrane[14].

The artificial turmoil ofA. Niger produces a variety of precious enzymes. For illustration,A. Niger glucoamylase (P69328) is used to make high-fructose sludge saccharinity, while pectinases (GH28) are employed in the clarifying of cider and wine[15]. Beano and other flatulence- reducing products contain nascence-galactosidase (GH27), an enzyme that breaks down certain complex carbohydrates. In the biotechnology sector,A. Niger is also used to make glamorous isotope-containing performances of natural macromolecules for NMR disquisition. Due to its strong affinity for-D-glucose, Aspergillus Niger is also grown for the birth of the enzyme glucose oxidase (P13006), which is employed in the creation of glucose biosensors.Gold, tableware, bobby, iron, and zinc cyano- essence complexes were plant in Aspergillus Niger growing in gold-mining results. The fungus also aids in the dissolution of heavy essence sulphides[16].A. Niger has also been plant to be able of resolving acid mine drainage by bobby and manganese biosorption.A. Niger that has been alkali-treated binds to tableware to the tune of 10 of its dry weight. Gray biosorption occurs by stoichiometric exchange with the sorbent's Ca (II) and Mg (II).

Mucor

Mucor is a microbiological rubric comprising about 40 mould species belonging to the Mucoraceae family[17]. Species can be plant in soil, digestive systems, factory shells, certain crapola, similar as Tomme de Savoie, decaying vegetable waste, and iron oxide residue left before after the biosorption process. Colonies of this fungus are typically white to faceless or slate in color, and they grow snappily. On culture media, colonies can develop to be several centimeters altitudinous. Due to the growth of spores, aged colonies turn slate to brown in color. Mucor spores, also known as sporangiospores, develop apical, globular sporangia that are supported and raised by a column- shaped columella. They can be simple or branching. The form and insertion of the columella, as well as the lack of stolons and rhizoids, distinguish Mucor species from

moulds of the rubrics Absidia, Rhizomucor, and Rhizopus. Chlamydospores are produced by several Mucor species. Earth is formed bynon-septate hyphae raying at large angles (> 90 °) to induce irregularnon-septate hyphae[18].

Reproduction-

Asexual reduplication is used by Mucor mucedo (rubric species). The conformation of standing hyphal sporangiophores. The sporangiophore's tip expands, forming a globose sporangium with uninucleate, haploid sporangiospores. The columella is a sporangiophore outgrowth that protrudes into the sporangium. The sporangium walls are fluently damaged, releasing the spores, which germinate snappily and produce new mycelium on suitable shells. Compatible strains produce gametangia, which are small, technical hyphae that arise during sexual reduplication. A thick-walled, globular zygosporangium formed when two complimentary gametangia unite[19].

Clinical Significance-

Due to their incapability to develop in heated conditions close to 37 degrees, utmost species of'Mucorare unfit to infect humans and endothermic creatures. Zygomycosis is an opportunistic and constantly gormandize spreading necrotizing infection caused by thermotolerant organisms like Mucor indicus. Fungi warrant chlorophyll and are classified as saprophytes. That is, they get their food by breaking downnon-living organic stuff. It's plant in saprotrophs and is constantly linked to fungus (similar as Mucor) and soil bacteria.Cellulitis is a skin illness that affects the soft apkins beneath the face. It occurs when bacteria enter a skin break and spreads. Cellulitis accounts for0.04 of total skin conditions[20].

Bacteria-

Bacteria are wide, generally free-living brutes that are constantly made up of just one naturalcell. They make up a broad group of prokaryotic organisms[21]. Bacteria play an important part inseveral stages of the nutrient cycle, similar as the obsession of nitrogen from the atmosphere.The corruption of dead cores is part of the nutrition cycle, and microorganisms areresponsible for the corruption stage.

Pathogen:

Beneficial commensals, which live on the skin and mucous membranes, and saprophytes,which live mostly in the soil and decomposing debris, are constantly present in the humanbody[22]. The nutrients in blood and tissue fluids are adequate to support the development ofmany bacteria. The body possesses defense systems that allow it to withstand microbialinvasion of its tissues and provide it with natural immunity or intrinsic resistance to numerousmicrobes.Each pathogen species has a distinct range of interactions with its human hosts. Skininfections, pneumonia, meningitis, and sepsis, a systemic inflammatory reaction resulting inshock, massive vasodilation, and death, can all be caused by bacteria such as Staphylococcusor Streptococcus. These organisms, on the other hand, are part of the natural human flora andcan be found on the skin or in the nose without causing any sickness[23].

Serratiamarcescens:

Serratia is a Yersiniaceae genus of Gram-negative, facultatively anaerobic rod-shaped bacteria[24]. S. marcescens, S. aquatilis, S. entomophila, S. ficaria, S. fonticola, S. grimesii, S. liquefaciens, S. microhaemolytica, S. myotis, S. nematodiphila, S. odoriferae, S. oryzae, S. proteamaculans, S. quinivorans, S. qui They may be found in water, soil, plants, and animals and are normally 1–5 m long. They do not form spores and can be found in water, soil, plants, and animals. S. marcescens is the most common, accounting for around half of all strains discovered.

Serratia aquatilis is a new Serratia species discovered in drinking water[24].

Cellulitis is a skin illness that affects the soft tissues beneath the surface. It occurs when bacteria enter a skin break and spreads[25]. Cellulitis accounts for 0.04% of total skin diseases. Serratia marcescens is a gram-negative motile facultative anaerobic bacillus from the Enterobacteriaceae family that has been linked to septic shock deaths. Individuals in immunocompromised states, such as diabetics and severe cirrhosis patients, as well as immunocompetent populations, have been found to be affected by this pathogen[26]. Pneumonia, urinary tract infection, bacteremia, biliary tract infection, wound infection, meningitis, and endocarditis are among illnesses caused by S. marcescens. Cellulitis and necrotizing fasciitis (NF) are rare symptoms, although they can lead to severe sepsis and multiorgan failure. We present a case of cellulitis, bacteremia, septic shock, and multi-organ failure caused by an unusual cause[27].

Opportunistic human pathogen:

S. marcescens, an opportunistic human disease, is considered to be spread by non-infected persons via hand-to-hand transfer, hospital equipment, and hospital employees. Serratia species prefer to invade the respiratory and urinary tracts in adults in hospitals rather than the gastrointestinal system. Serratia infection causes around 2% of nosocomial infections in the bloodstream, lower respiratory tract, urinary tract, surgical wounds, skin and soft tissues, and other conditions that are typically caused by other bacteria. In pediatric wards, outbreaks of S. marcescens meningitis, wound infections, and arthritis have occurred[28].

It is known to cause hospital and community acquired infections such as bacteremia, pneumonia, endocarditis, meningitis, and septic arthritis, but it is a rare cause of cellulitis or NF, especially in immunocompromised patients.Because the bacteria may be found in fresh, stagnant water, soil, and animals, community-acquired illnesses have been documented.Serratia bacteremia is caused by a community-acquired infection in around half of the cases (47 percent). Colonization can occur after earlier hospitalizations or operations, as our patient experienced after weekly paracentesis for end-stage liver illness and a recent catheterization for moderate aortic stenosis[29].

Pathogenesis:

It is known to create the red pigment prodigiosin. The development of serralysin (a protease enzyme), hemolysin Sh1A (a pore-forming exotoxin), and the formation of biofilm are all thought to have a role in pathogenesis. According to a mouse model, cell-mediated immunity in the spleen aids in the clearance of Serratia marcescens, hence splenectomized individuals are at a higher risk of severe soft tissue infection[30].

Treatment:

Renal illness, chemotherapy, immunosuppressants, steroid, and liver disease are all linked to severe septic shock and significant mortality in immunocompromised people[31]. Pain, erythema, and edoema often appear within 24 hours of direct tissue damage in immunocompromised persons, and can show as painful nodules, solitary plaques, ulcer, cell ulitis, abscess, granulomatous ulcer, pyoderma gangrenosum, and NF in immunocompromised individuals. Due to re sistant bacteria, highrisk individuals might acquire severe bacteremia and septic shock quickly.Oral therapy with amoxicalvulinic acid is preferable, but intravenous therapy with -lactamase inhibitors (ampicillin-sulbactam/piperacillin-tazobactam) is preferred. Serratia marcescens, like other Enterobacteriaceae, has inherent resistance to penicillin, macrolide, first-generation cephalosporin, linezolid, and rifampin. Resistance to penicillins, cephalosporins, and carbapenems can be chromosomal or plasmid-mediated, with either AmpC—lactamase, extended-spectrum-lactamase, or carbapenemase being produced[32].

Conclusion:-

The importance of microbial interactions in health and disease has been increasingly recognised in recent years, and as a result, many researchers have abandoned the historical separation of pro- and eukaryotes and begun investigating co-colonization and co-infections across kingdom boundaries.These interactions are intricate, and the type of contact that happens is frequently determined by a variety of pathogen,environmentaland host factors.Furthermore, understanding the pathways by which competing microorganisms counteract one another could lead to novel treatment options for serious human infections, which is now more than ever a critical need.

References:-

1. Odds FC, Brown AJ, Gow NA. Antifungal agents: mechanisms of action. Trends in microbiology. 2003 Jun 1;11(6):272-9.
2. Casadevall A, Pirofski LA. Host-pathogen interactions: redefining the basic concepts of virulence and pathogenicity. Infection and immunity. 1999 Jun 1;67(8):3703-13.
3. Brown GD, Denning DW, Levitz SM. Tackling human fungal infections. Science. 2012 May 25;336(6082):647-7.
4. Spellberg B, Edwards Jr J, Ibrahim A. Novel perspectives on mucormycosis: pathophysiology, presentation, and management. Clinical microbiology reviews. 2005 Apr;18(3):556-69.
5. Weinstein RA, Gaynes R, Edwards Jr JR. Overview of nosocomial infections caused by gram-negative bacilli. Clinical Infectious Diseases. 2005 Oct 1;41(6):848-54.
6. Bassetti M, Righi E, Carnelutti A, Graziano E, Russo A, Multidrug-resistant Klebsiella pneumoniae: challenges for treatment, prevention and infection control. Expert review of anti-infective therapy. 2018 May 4;16(10):749-61.

7. Palacios-Baena ZR, Oteo J, Conejo C, Larrosa MN, Bou G, Fernández-Martínez M, Pascual A, Martínez-Martínez L, González-López JJ, Rodríguez-Baño J, Spanish Group for the Study of Nosocomial Infections (GEIH), and Spanish Society of Clinical Microbiology and Infectious Diseases (SEIMC). Comprehensive clinical and epidemiological assessment of colonisation and infection due to carbapenemase-producing Enterobacteriaceae in Spain. Journal of Infection. 2016 Mar 1;72(3):152-60.
8. Lautenbach E, Patel JB, Bilker WB, Edelstein PH, Fishman NO. Extended-spectrum β-lactamase–producing Escherichia coli and Klebsiella pneumoniae: risk factors for infection and impact of resistance on outcomes. Clinical Infectious Diseases. 2001 Sep 1;32(8):1162-71.
9. Cornaglia G, Rossolini GM. New β-lactamases: a paradigm for the investigation of genomic plasticity. Clinical microbiology and infection. 2008 Nov;14(s6):80-4.
10. Tacconelli E, Carrara E, Savoldi A, Harbarth S, Mendelson M, Monnet DL, Pulcini C, Kahlmeter G, Kluytmans J, Carmeli Y, Ouellette M. Discovery, research, and development of new antibiotics: the WHO priority list of antibiotic-resistant bacteria and tuberculosis. The Lancet Infectious Diseases. 2018 Mar 1;18(3):318-27.
11. Perfect JR, Casadevall A. Cryptococcosis. Infectious disease clinics of North America. 2002 Sep 1;16(3):837-74.
12. Pfaller MA, Diekema DJ. Epidemiology of invasive candidiasis: a persistent public health problem. Clinical microbiology reviews. 2007 Jan 1;20(1):133-63.
13. Spellberg B, Edwards Jr J, Ibrahim A. Novel perspectives on mucormycosis: pathophysiology, presentation, and management. Clinical microbiology reviews. 2005 Apr;18(3):556-69.
14. Diekema DJ, Pfaller MA. Nosocomial candidemia: an ounce of prevention is better than a pound of cure. Infection Control & Hospital Epidemiology. 2004 Jun;25(6):624-6.
15. Tischler AD, Camilli A. Cyclic diguanylate (c-di-GMP) regulates Vibrio cholerae biofilm formation. Molecular microbiology. 2004 May;53(3):857-69.
16. Díaz-Horta O, Abreu MT. Bacterial overgrowth syndrome. Current gastroenterology reports. 2004 Aug;6(4):307-12.
17. Singh AK, Prakash P, Achra A, Singh A. Bacterial biofilms: Recent advances in their control and its impact on ocular therapeutics. Journal of Ocular Pharmacology and Therapeutics. 2020 Jul 1;36(6):379-92.
18. Sebghati TS, Engle JT, Goldman WE. Intracellular parasitism by Histoplasma capsulatum: fungal virulence and calcium dependence. Science. 2000 Sep 22;290(5498):1368-72.
19. Fisher MC, Henk DA, Briggs CJ, Brownstein JS, Madoff LC, McCraw SL, Gurr SJ. Emerging fungal threats to animal, plant and ecosystem health. Nature. 2012 Apr;484(7393):186-94.
20. Marques MB. Histoplasmosis. Seminars in respiratory and critical care medicine. 2020 Feb;41(01):013-8.
21. Alfano JR, Collmer A. Bacterial pathogens in plants: life up against the wall. The Plant Cell. 1996 Oct;8(10):1683.
22. Macpherson AJ, Harris NL. Interactions between commensal intestinal bacteria and the immune system. Nature Reviews Immunology. 2004 Jun;4(6):478-85.
23. Guzman-Cottrill JA, Cheesebrough B, Nadel S, Goldstein B. 11 The Systemic Inflammatory Response Syndrome (SIRS), Sepsis, and Septic Shock. pediatrics. 2005;6:2-8.
24. Wang, Juan, et al. "Serratia microhaemolytica sp. nov., isolated from an artificial lake in Southern China." Antonie van Leeuwenhoek 112.10 (2019): 1447-1456.
25. Mahlen SD. Serratia infections: from military experiments to current practice. Clinical microbiology reviews. 2011 Oct;24(4):755-91.
26. Steyn L. The basics of bacterial skin infections. SA Pharmacist's Assistant. 2020 Sep 1;20(3):21-3.
27. Puvanendran R, Huey JC, Pasupathy S. Necrotizing fasciitis. Canadian Family Physician. 2009 Oct 1;55(10):981-7.
28. Chamilos G, Lionakis MS, Kontoyiannis DP. Call for action: invasive fungal infections associated with ibrutinib and other small molecule kinase inhibitors targeting immune signaling pathways. Clinical Infectious Diseases. 2018 Jan 18;66(1):140-8.
29. Kauffman CA, Pappas PG, Sobel JD, Dismukes WE. Essentials of Clinical Mycology, 2nd ed. Clinical Infectious Diseases. 2005 Dec 15;41(12):1781-2.
30. Haug A, Hostmark AT, Harstad OM. Bovine milk in human nutrition–a review. Lipids in health and disease. 2007 Dec;6(1):25.
31. Groll AH, Shah PM, Mentzel C, Schneider M, Just-Nuebling G, Huebner K. Trends in the postmortem epidemiology of invasive fungal infections at a university hospital. Journal of Infection. 1996 Nov 1;33(3):23-32.

32. Philippon A, Arlet G, Lagrange PH. Origin and impact of plasmid-mediated extended-spectrum beta-lactamases. European Journal of Clinical Microbiology and Infectious Diseases. 1994 Jan;13(1):S17-29.