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A Review of Antimicrobial Therapy for Human Pythiosis

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Introduction

Pythium insidiosum causes human pythiosis with a poor prognosis and significant mortality. The in vitro and in vivo efficacy of antimicrobials against P. insidiosum is inconsistent. Although antimicrobials are clinically useful, without surgery and immunotherapy, they are unlikely to achieve therapeutic success. As a result, new therapeutic options are required. The rationale for antimicrobial therapy, minimum inhibitory concentrations, and efficacy of antibacterial and antifungal agents against P.insidiosum are discussed in this non-exhaustive review.This review expands on the immunomodulatory effects of antimicrobials, which can improve the immune response to infections. The use of antimicrobial combination therapy for the pharmacotherapeutic management of human pythiosis is supported by current data. Furthermore, the success or failure of antimicrobial treatment in human pythiosis may be dependent on drug immunomodulatory effects.

Description

Human pythiosis is a highly contagious disease with a high morbidity and mortality rate. Pythium insidiosum is a fungus-like aquatic oomycete microorganism that causes pythiosis. The motile flagellate zoospore is important in the initiation of an infection. P. insidiosum zoospores adhere to skin cuts or wounds and encyst on the injured tissue's surface. The encysted spore forms a germination tube (hypha) that uses chemotaxis to locate and infiltrate human blood vessels. The risk of pythiosis is higher in tropical and subtropical areas, such as Southeast Asia, eastern coastal Australia, and South America.

Because of the difficulty in diagnosing the infection and the lack of effective therapeutic agents against this disease, human pythiosis has a poor prognosis. Human pythiosis clinical features are classified into four types: Arteritis, thrombosis, gangrene, aneurysm, or limb claudication are symptoms of vascular pythiosis; corneal ulcers, decreased visual acuity, conjunctival redness, eyelid swelling, or multiple, linear, tentacle-like infiltrates and dot-like or pinhead-shaped infiltrates in the surrounding cornea are symptoms of cutaneous and subcutaneous pythio Because P. insidiosum has a higher affinity for iron, risk factors for vascular pythiosis include thalassemia, hemoglobinopathy, paroxysmal nocturnal hemoglobinuria, aplastic anaemia, and leukaemia.

When P. insidiosum infection is diagnosed, treatment options include surgery, pharmacotherapy, and immunotherapy. The mainstay treatment for human pythiosis is surgical intervention, but this significantly increases the financial burden on patients, as well as postsurgical complications and uncontrolled infection. Immunotherapy, in which antigens of P. insidiosum from in vitro cultures are injected into the patient, is a promising approach for treating human pythiosis. The mechanism underlying P.insidiosum antigen immunotherapy in human pythiosis includes a shift in the host's immune response from T helper-2 to T helper-1, with the Th1 response producing higher levels of interferon- and interleukin 2. Even though Th2 to Th1 switching can imply a good prognosis in PIA-treated patients, the efficacy of P. insidiosum antigen as a vaccine is inconclusive.

Antimicrobial therapy should produce a clinical response by eradicating the invading microorganism while minimising cost, side effects, and antimicrobial resistance. Both the pharmacokinetic and pharmacodynamic properties of the drug(s) must be considered when selecting appropriate antimicrobial therapy to ensure that effective agents are administered in sufficient doses for therapeutic success. Identifying potential antimicrobial targets for species such as P. insidiosum is critical for pythiosis management. The microbial cell wall is an important target for antimicrobials, and P. insidiosum's cell wall is primarily composed of -glucan and cellulose. However, the cell wall of P. insidiosum reduces drug molecule penetration and prevents drug access to targets within the cell wall.

The innate immune system defends the host against toxins and infectious agents such as bacteria, fungi, viruses, and parasites through phagocytosis and intracellular killing, recruitment of other inflammatory cells, and antigen presentation. Physical and anatomical barriers, effector cells, antimicrobial peptides, soluble mediators, and cell receptors comprise the innate immune system. Pathogens, on the other hand, can bypass the early innate immune mechanisms. In these cases, modifying the function of immune cells may result in the elimination of the pathogenic intruder. Surprisingly, host immunity is frequently overlooked in the pathogen clearance process. Infections, a favourable innate immune response can significantly reduce the need for more prolonged antimicrobial therapy.

Allylamines, such as terbinafine, work primarily by inhibiting the enzyme squalene monooxygenase. As a result, these drugs prevent fungal ergosterol synthesis. Azoles, including miconazole, ketoconazole, fluconazole, itraconazole, posaconazole, and voriconazole, have antifungal activity in fungi by inhibiting 14-lanosterol demethylase, a key enzyme in ergosterol biosynthesis. When monocytes, macrophages, and neutrophils are combined with azoles, their microbiocidal activity against intracellular Candida albicans appears to be enhanced. Terbinafine, on the other hand, has been shown to stimulate pro-inflammatory cytokines [1-5].

Conclusion

With evidence of the efficacy of some antimicrobials in the treatment of human pythiosis, we propose using new drug delivery systems to deliver the drug to the target site in the body while minimising off-target drug accumulation. To improve targeting and specificity at infected sites, antibiotics can be reformulated using nanotechnology-derived delivery systems. Because of genetic differences, not all people with pythiosis have the same therapeutic responses to antimicrobials.As a result, it is critical to incorporate the pharmacogenomics assay into clinics in order to personalise antimicrobial treatment in pythiosis.

The evidence supports using the antimicrobials discussed in our article as a new therapeutic option in the treatment of human pythiosis. Tetracyclines, macrolides, oxazolidinones, lincosamides, streptogramins, phenicols,

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aminoglycosides, polyenes, allylamines, azoles, and echinocandins inhibited the growth of P. insidiosum in vitro, which has implications for future research on their use in the management of human pythiosis. However, because of the side effects and threat of antimicrobial resistance, prolonged use of antimicrobials and treatment with antimicrobials is not warranted. A practical pharmacological intervention guideline for human pythiosis is still needed to help practitioners and patients make decisions, reduce treatment costs, and improve patient outcomes.

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