Case Report

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Metadichol® and MRSA Infections: A Case Study

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ABSTRACT

Metadichol® is a Nano emulsion of long-chain alcohols called as Policosanol and is present in foods such as rice, sugar cane, wheat, and peanuts [1]. Metadichol® acts on Nuclear Vitamin D receptors (VDR) that are present in cells throughout the body to stimulate the immune system and inhibit a variety of disease processes, resulting from viral, bacterial and parasitic infections. Infectious agents can cause disease by avoiding normal host defense mechanisms or by subverting them to promote their replication. They do so by blocking VDR receptor that is responsible for innate immunity, and this suppression of the immune response contributes to persistent infections.

We present a case study of a patient who had acquired MRSA infections and how Metadichol® by its actions on the VDR has resolved the problem of this deadly disease without any side effects. Metadichol® is safe because it consists of natural components of conventional foods and has no known adverse side effects. Its constituents are present in many foods that we consume every day.

Metadichol® has the potential to serve as a novel, antibacterial treatment for MRSA and other skin infections that confront the number one public health threat in the world today.

Keywords

VDR, Vitamin D, Metadichol®, Innate immunity, MRSA, Inverse agonist, Protean agonist, Nano emulsion, Lipid alcohols.

Introduction

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a growing problem in shared facilities such as hospitals, healthcare facilities, and nursing homes. Studies indicate that the incidence of MRSA in the past few years has extensively increased worldwide. Globally, 2 billion people are estimated to carry some form of *S. aureus*; of these, up to 53 million (2.7% of carriers) are thought to carry MRSA.

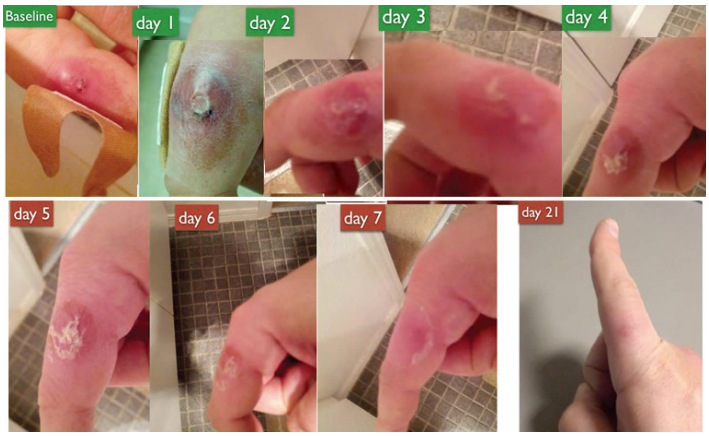
Infections caused by drug-resistant pathogens are one of the biggest health problems the world faces today. Bacteria and other pathogens have always evolved to resist the new drugs that modern medicine uses to combat them. But in recent years the rise in drug resistance has been a particular worry, especially the emergence of antibiotic-resistant superbugs. Unless action is taken to address this huge global issue, our conservative estimate is that it will cost the world an additional 10 million lives a year by 2050, more than the number of people currently dying from cancer annually. It will also have an increasing cost of 100 trillion USD, more than one and a half times the annual world GDP today, or roughly the equivalent of losing the UK economy from global output every year [2]. The Centers for Disease Control and Prevention (CDC) estimated that about 1.7 million nosocomial infections occurred in the United States in 2002, with 99,000 associated deaths [3,4]. The estimated incidence is 4.5 nosocomial infections per 100 admissions, with direct costs (at 2004 prices) ranging from $10,500 per case (for bloodstream, urinary tract, or respiratory infections in immunocompetent patients) to $111,000 per case for antibiotic-resistant infections in the blood in patients with transplants. With these numbers, conservative estimates of the total direct costs of nosocomial infections are above $17 billion, in U.S alone and about $75 billion worldwide.

MRSA multi-drug resistant bacterial pathogens are causing serious community and hospital-acquired infections much as skin and soft tissue infections, bone, joint and implant infections, ventilator-associated pneumonia, and sepsis. MRSA can be transmitted from person to person via skin or the sharing of contaminated objects [5].

The current treatment approach is to modify and do some minor tweaks of existing antibiotics which already have infectious agents that have developed resistance. There is a need for new antibacterial molecules that can treat infections caused by MRSA [6].

Case Presentation

A male patient in his early 40's diagnosed with a CA-MRSA (community associated methicillin-resistant *Staphylococcus aureus*) finger infection. The patient was treated by applying Metadichol 5mg dose twice a day topically on the infected area for a week. He reported that the pain eased after two days and had a complete clearance of infection seen after a week (Figure 1). He continued to use it topically on the wound for an additional two weeks, and the wound cleared up completely.



Discussion

Approximately 2.5 billion people in the world are continuously colonized with *Staphylococcus aureus*, and the rest are hosts for intermittent colonization [7]. *S. aureus* typically resides in the nose but is also found on the skin and in the gastrointestinal tract. Although its presence in humans does not lead to disease, the risk is higher in those who are carriers of *S.aureus*. Skin and soft-tissue infections are the in this population and can lead to more severe diseases like sepsis [8]. Also, *S. aureus* can cause pneumonia, osteomyelitis, infectious arthritis, abscesses in many organ tissues and infections of surgical wounds or prosthetic materials. A key feature of *S. aureus* disease is its recurrence, which occurs for 8-33% of the cases [9]. Prior infection disease does not protect against subsequent *S. aureus* infection. People with an elevated risk for staphylococcal infection are low-birth-weight infants, children, the elderly and patients with indwelling catheters, endotracheal intubation, medical implantation of foreign bodies, trauma, surgical procedures, hemodialysis, diabetes or immunosuppressive or cancer therapy.

Vitamin D plays a significant role in mediating immune function via pathways, by production of release of antimicrobial peptides in the skin. MRSA-infected patients had significantly lower serum vitamin D levels than non-MRSA infected patients [10]. The study showed that people with 25, Hydroxy vitamin D levels above 30 ng/ml were 50% less likely to be *S. aureus* carriers. They suggest that vitamin D can up-regulate the antibacterial immune response and thereby prevent *S. aureus* colonization and carriage, and to develop other diseases. Their findings suggest that vitamin D supplementation may reduce the incidence of methicillin sensitive and methicillin resistant *staphylococcal aureus* infections.

The host immune system has developed many mechanisms to neutralize and remove pathogenic bacteria. In turn, bacteria have evolved mechanisms to alter and evade the host immune response [11]. Slowing the innate immune defenses by down-regulating the VDR is a likely mechanism used in the host defense against pathogens [12-14]. For example, *Mycobacterium tuberculosis*, *Mycobacterium Leprae*, and *Aspergillus Fumigate* are found to down-regulate VDR activity. This down regulation allows intracellular bacteria to persist in the cytoplasm of nucleated cells and increases susceptibility to other diseases [15].

Bacteria can also exploit endogenous signaling cascades to initiate airway inflammation. *Staphylococci* recognize TNF-a receptor 1 (TNFR1) and activate TNF signaling in airway cells leading to IL-8 induction. *Staphylococcal* protein, a signaling through TNFR1, plays a central role in the pathogenesis of *S. aureus* pneumonia [16].

The likely mechanism of how Metadichol works is in its action through the vitamin D receptor. We have shown that Metadichol binds to the VDR and acts as an inverse but more likely as a protean agonist [17]. It could competitively displace the bacteria and restore normal VDR transcription activity of producing antimicrobial peptides against the pathogens [18]. This action of Metadichol enhances innate immunity responses in patients and allows them to overcome the infection. Also, Metadichol is a TNF-alpha inhibitor, and it prevents the pathogenesis of *S. Aureus* as described above (1).

The treatment of infections with antibiotics has become increasingly difficult owing to the widespread occurrence of strains that are resistant to multiple antibiotics, MRSA strains that have also become resistant to vancomycin, the last drug to which the organism had been uniformly sensitive, raises the stakes in overcoming this disease. Metadichol ® by activating the VDR and enhancing innate immune response also reduces inflammation by modulating TNF. Thus, it serves as a key that fits many locks. Metadichol fulfills the need that many disease states require action through multiple pathways to be efficacious. Metadichol is a product made from agricultural waste and is a renewable resource. It has the potential to serve as an anti-bacterial molecule with a broad spectrum of activity, particularly given that its constituents (long-chain lipid alcohols) are present in foods commonly consumed on a daily basis and that it has demonstrated no toxicity at doses of up to 5000 mg/kg [19-21].

Conclusion

A key feature of MRSA disease is in its recurrence. Prior exposure to disease does not elicit protection against subsequent infection. In our case study, it has been four years since the patient's MRSA recovery, and he has not had a recurrence.

Therefore, Metadichol could be a novel OTC supplement and serves as a cheaper and also as a far more efficient [22,23] substitute to prescription drugs, which have been largely ineffective in infectious diseases and have many side effects that add to health care costs. Given its overall safety it is ready for large scale testing in areas where infectious diseases are rampant.

References

1. P.R.Raghavan US patents; 8,722,093 (2014) and 9,006,292 (2015).
2. http://amr-review.org/publications
3. Klevens RM, Edwards JR, Richards CL, et al. Estimating health care-associated infections and deaths in U.S. hospitals, 2002. Public Health Rep. 2007; 122: 160-166.
4. Lindsay J, Holden M. Staphylococcus aureus: superbug, super genome? Trends in Microbiology. 2004; 18: 378-385.
5. Holcomb HG, Durbin KJ, Cho M, et al. Methicillin-resistant Staphylococcus aureus as a threat to public health: a cellular approach. Georgetown Univ J Health Sci. 2008; 5: 2.
6. Braine T. Race against time to develop new antibiotics. Bull World Health Organ. 2011; 89: 88-89.
7. van Belkum A, Melles DC, Nouwen J, et al. Co-evolutionary aspects of human colonisation and infection by Staphylococcus aureus. Infect Genet Evol. 2009; 9: 32-47.
8. David MZ, Daum RS. Community-associated methicillin-resistant Staphylococcus aureus: epidemiology and clinical consequences of an emerging epidemic. Clin Microbiol Rev. 2010; 23: 616-687.
9. Kallen AJ, Mu Y, Bulens S, et al. Health care-associated invasive MRSA infections, 2005-2008. JAMA. 2010; 304: 641-648.
10. Matheson EM, Mainous AG 3rd, Hueston WJ, et al. Vitamin D and methicillin-resistant Staphylococcus aureus nasal carriage. Scand J Infect Dis. 2010; 42: 455-460.
11. Dermine JF, Desjardins M. Survival of intracellular pathogens within macrophages. Protop. 1999; 210: 11-24.
12. Xu Y, Xie J, Li Y, et al. Using a cDNA microarray to study cellular gene expression altered by Mycobacterium tuberculosis. Chin Med J. 2003; 116: 1070-1073;
13. Liu PT, Wheelwright M, Teles R, et al. MicroRNA-21 targets the vitamin D-dependent antimicrobial pathway in leprosy. Nat Med. 2012; 18: 267-273.
14. Coughlan CA, Chotirmall SH, Renwick J, et al. The effect of Aspergillus fumigatus infection on vitamin D receptor expression in cystic fibrosis. Am J Respir Crit Care Med. 2012; 186: 999-1007.
15. Marks R, Allegrante JP. Comorbid disease profiles of adults with end-stage hip osteoarthritis. Med Sci Monit. 2002; 8: CR305-309.
16. Gomez MI, Lee A, Reddy B, et al. Staphylococcus aureus protein A induces airway epithelial inflammatory responses by activating TNFR1. Nat Med. 2004; 10: 842-848.
17. Neubig RR. Missing Links: Mechanisms of Protean Agonism, Mol Pharmaco. 2007; l71: 200-1202.
18. Mangin M, Sinha R, Fincher K. Inflammation and vitamin D: the infection connection. Inflamm Res. 2014; 63: 803-819
19. Alemán CL, Más R, Hernández, et al. A 12-month study of policosanol oral toxicity in Sprague Dawley rats. Toxicol Lett. 1994; 70: 77-87.
20. Alemán, CL, Más Ferreiro. Carcinogenicity of policosanol in Sprague-Dawley rats: A 24-month study. Teratog Carcinog Mutagen. 1994; 14: 239-249.
21. Aleman, CL, Puig MN, EIN, EC, et al. Carcinogenicity of policosanol in mice: An 18-month study. Food Chem Toxicol. 1995; 33: 573-578.
22. P.R.Raghavan. In vitro Inhibition of Zika Virus by Metadichol®. A Novel Nano Emulsion Lipid, J Immunol Tech Infect Dis. 2016; 5: 4.
23. P. R. Raghavan: Inhibition of Dengue and other enveloped viruses by Metadichol®, a Novel Nano emulsion Lipid, Journal of the of Science of Healing Outcomes: 2016; Vol 8, No 31,19-25.