

Metadichol[®] and MRSA Infections: A Case Report

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Abstract

Metadichol[®] [1] is a Nanoemulsion of long-chain alcohols called as Policosanol and is present in foods such as rice, sugar cane, wheat, and peanuts. Metadichol[®] acts on Nuclear Vitamin D receptors (VDR) that have a ubiquitous presence in cells and tissues of 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 leads 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.

Keywords: VDR; Vitamin D; Metadichol[®]; Innate immunity; MRSA; Inverse agonist; Protean agonist; Nanoemulsion; Lipid alcohols

Introduction

Methicillin-resistant *Staphylococcus aureus* (MRSA) is a major problem hospitals, healthcare facilities, Globally, two billion people are estimated to carry some form of *S. aureus*, of these, up to 60 million (approximately 3 % of carriers) are thought to carry MRSA [2].

Bacteria and other pathogens fight back by acquiring resistance to drugs rapidly. This resistance has led to the emergence of antibiotic-resistant superbugs. It is projected that ten million lives a year will be lost by 2050. 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. It has been estimated that in USA about 1.7 million nosocomial infections occurred in 2002, with 99,000 associated deaths [3,4]. The 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 immune-competent patients) to \$111,000 per case for antibiotic-resistant infections in the blood in patients with transplants. Total direct costs of nosocomial infections are \$17 billion, in U.S and about \$75 billion worldwide.

MRSA multi-drug resistant bacterial pathogens are causing serious community and hospital-acquired infections much of it 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 research in search of new agents is to modify and do some minor tweaks of existing antibiotics which already have infectious agents that have developed resistance to it. There is a need for new antibacterial molecules that can treat infections caused by MRSA [6].

Case Presentation

Patient Male early 40's was diagnosed with a CA-MRSA (community associated methicillin-resistant *Staphylococcus aureus*) finger infection. A culture from the infection was sent for pathology analysis, but the patient began use of Metadichol at a dosage of 5 mg twice day topically on the wound while waiting for the lab result. The result confirmed the diagnosis as CA-MRSA, and an oral antibiotic (clindamycin, generic for Cleocin) was prescribed. The patient did not use the antibiotic but instead continued with its use topically for infection as well as pain (as an alternative to hydrocodone).

He reported that the pain eased after two days and had a complete clearance of infection seen after a week (Figure 1). He continued use topically on the wound for an additional 6 weeks, and the injury cleared up completely. He did not use the antibiotics, and after four years he is disease free.

Discussion

Staphylococcus 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 common in this population and can lead to more severe diseases like sepsis [7,8]. 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 to *S. aureus* does not protect against subsequent infection. People with an elevated risk for staphylococcal infection are low-birth-weight infants, children, the elderly and patients with indwelling catheters, medical implantation of devices, hemodialysis, diabetes or undergoing immunosuppressive or cancer therapy.



Figure 1: A complete clearance of infection seen after a week with Metadichol.

Vitamin D plays a significant role in mediating immune function by up-regulating the antibacterial immune response and thereby preventing *S. aureus* colonization. MRSA-infected patients have lower serum vitamin D levels than non-MRSA infected patients [10]. People with 25-hydroxy vitamin D levels above 30 ng/ml were 50% less likely to be *S. aureus* carriers. It is likely that vitamin D supplementation may reduce the incidence of methicillin sensitive and methicillin resistant *Staphylococcal aureus* infections.

The Immune systems have developed mechanisms to neutralize and remove pathogenic bacteria. In turn, bacteria have evolved mechanisms to alter and evade the host immune response [11]. Pathogens slow the innate immune defenses by down-regulating the VDR [12-14], for example *Mycobacterium tuberculosis*, *Mycobacterium Leprae*, and *Aspergillus Fumigate* down-regulate VDR activity. This action allows intracellular bacteria to persist in the cytoplasm of nucleated cells and increases susceptibility to other diseases [15]. Bacteria also exploit signaling cascades to initiate airway inflammation. Staphylococcal protein signaling through TNFR1, plays a central role in the pathogenesis of *S. aureus* [16].

The likely mechanism of how Metadichol works is by acting on 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]. Metadichol is a TNF-alpha inhibitor, and prevents the pathogenesis of *S. aureus* through the TNFR1 signaling pathway [1].

MRSA strains that have become resistant to vancomycin, the last drug to which the organism had been uniformly sensitive, raises the stakes in combating this disease. Metadichol by activating the VDR inhibiting TNF alpha, serves as a key that fits many locks and works through multiple pathways. It fulfills the need that many disease states require action through multiple pathways to be efficacious. Metadichol has the potential to serve as an anti-bacterial molecule with a broad spectrum of activity, particularly given that it has demonstrated no toxicity at doses of up to 5000 mg/kg [19-21].

Conclusion

Metadichol could be a novel OTC supplement and efficient substitute to prescription drugs, which have been largely ineffective in infectious diseases [22,23] 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.

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