

A Case of MRSA Controlled: Predisposing Factors and Immune Stimulation

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Abstract

Most treatments for methicillin-resistant *Staphylococcus aureus* (MRSA) focus on agents to eliminate the bacterium. Since MRSA infection is not universal, susceptibility factors are possible. Immune resistance could be lowered in such individuals; therefore, locating immune-inhibiting or immune-enhancing factors might decrease susceptibility. Such seemed to be the case in a 48-year-old female who presented with recurring MRSA despite multiple rounds of a variety of antibiotics. When the patient encountered an intensely stressful situation an outbreak of MRSA occurred. The patient had additional underlying health issues that suppressed her immune system and made her more susceptible to stress. Gluten allergy and hypothyroidism were discovered and alleviated but did not end the MRSA outbreaks. Implementation of a popular treatment from the 1930s, intravenous dilute hydrochloric acid (for immune stimulation), prevented most MRSA outbreaks when administered frequently. This case provides anecdotal support for the proposition that immune enhancement is a viable approach to forestall or clear recurring MRSA. (*Altern Med Rev* 2010;15(2):147-151)

Introduction

Staphylococcus aureus is a gram-positive, aerobic bacterium that can live on human skin and in the anterior nares.^{1,2} The axilla, groin, and gastrointestinal tract can also become inhabited. There is some protection in being a *S. aureus* carrier. In a study of staph carriers versus non-carriers, when infection by staph occurred the non-carriers had a significantly higher risk of death from bacteremia. However, the carriers were three times more likely to develop nosocomial-related bacteremia than non-carriers.³ Pathogenic staph can cause skin infections, most commonly abscesses and boils. If bacteremia occurs, pneumonia, endocarditis, osteomyelitis, toxic shock syndrome, gastroenteritis, or scalded skin syndrome can result. *S. aureus* can produce penicillinase, which inactivates the

beta-lactam category of antibiotics (penicillins, cephalosporins, carbapenems, and penems).² However, for methicillin-sensitive *S. aureus*, the first line of therapy is usually a penicillin category of antibiotic.^{4,5}

A review of the protective mechanisms ensuring survival of *S. aureus* was published by Gordon.¹ Briefly, *S. aureus* has structural protection from surface proteins (allowing adherence to host cells), biofilm formation, small colony variants, and microcapsule formation. Surface protein A is able to bind to the Fc portion of immunoglobulin, preventing potential opsonization. *S. aureus* is able to evade the immune system by hiding inside human cells. *S. aureus* also secretes chemotaxis-inhibiting protein, which interferes with neutrophil extravasation and movement to the infection site. The bacteria release leukocides that cause pore formation resulting in leukocyte death (Figure 1).

Methicillin-resistant *Staphylococcus aureus* (MRSA) is categorized into “hospital-acquired” (HA-MRSA) and “community-acquired” (CA-MRSA). CA-MRSA is more sensitive to a wider range of drugs than HA-MRSA.² Furthermore, Panton-Valentine leukocidin (PVL) gene has been found in CA-MRSA, but not HA-MRSA.¹ The PVL gene is specific to human neutrophils (does not affect mouse or monkey neutrophils), causing a pore-forming toxin that rapidly kills neutrophils.⁶

Localized MRSA is usually treated first with surgical drainage and, if necessary, with oral vancomycin.^{2,7} Available antibiotics for MRSA bacteremia include intravenous vancomycin, daptomycin, clindamycin, linezolid, quinupristin/dalfopristin, tigecycline, and oral trimethoprim-sulfamethoxazole.⁸

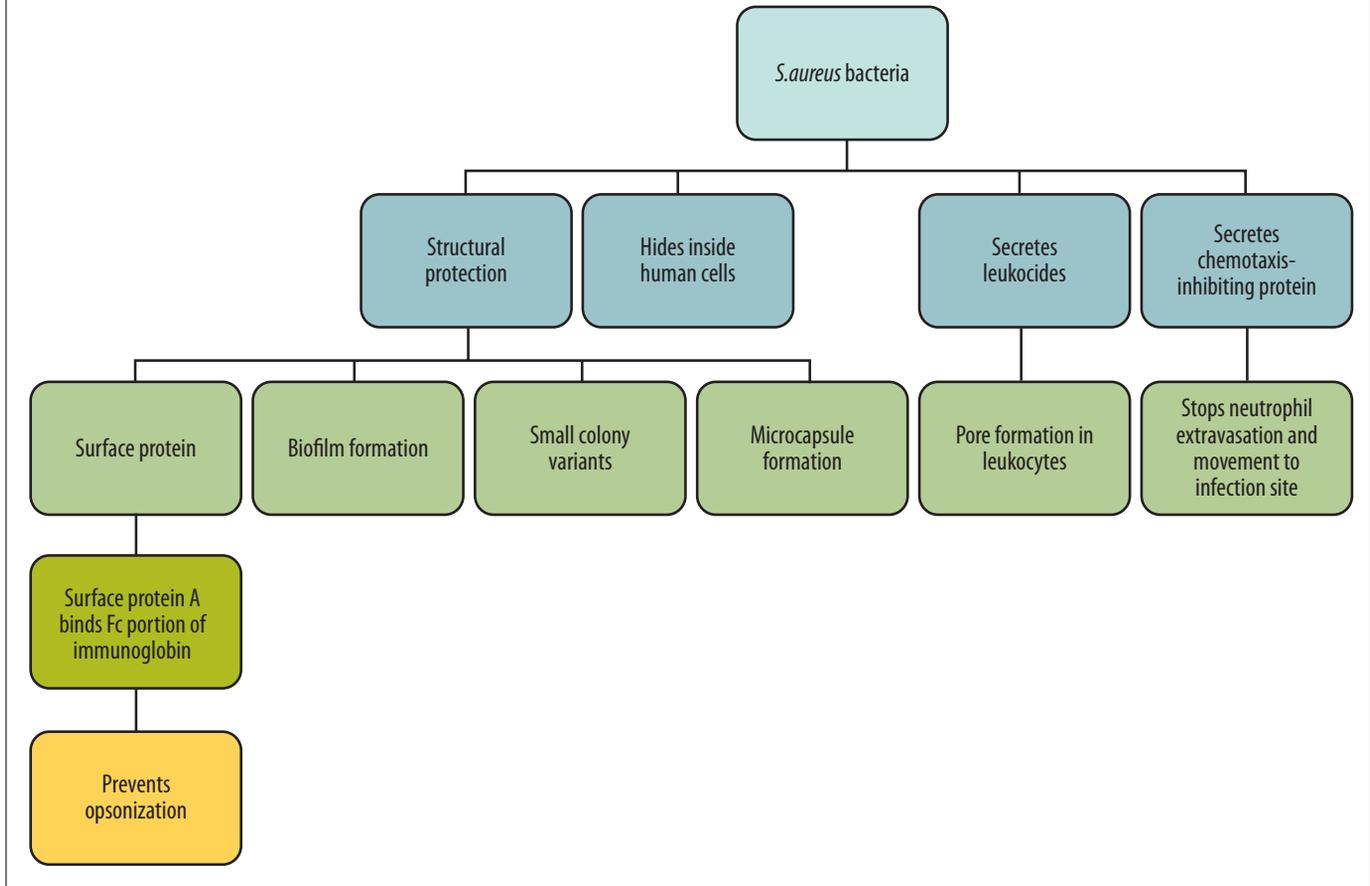
Although death can occur from MRSA, there is usually complete recovery. In 2005 the U.S. Centers for Disease Control and Prevention estimated there were 94,360 cases of invasive MRSA, with approximately 18,650 associated hospital deaths.⁹

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Figure 1. Protective Mechanisms of *Staphylococcus aureus*¹



Most of these infections were HA-MRSA, which appears to indicate the bacteria is either more resilient and/or the patient is more susceptible because of the underlying reason(s) for hospitalization. Because CA-MRSA can affect seemingly healthy people without concomitant disease, it would appear there is silent susceptibility occurring within this “healthy” group.

The capacity to acquire MRSA infection is widespread. Since MRSA does not cause infection in a large proportion of the population, individual predisposing factors may increase susceptibility, and discovery of these factors may provide additional treatment approaches to antibiotics. Several cases have brought the authors to this conclusion. The case outlined below describes characteristic predisposing factors and the benefit of immune-stimulating therapy in a woman with recurring episodes of MRSA.

Case Report

In October 2007, a slender 48-year-old female presented with recurring MRSA infections, appearing as skin boils on the buttocks, making it difficult for her to sit. She reported that one emergency room (ER) physician described a grapefruit-size boil. Personal history revealed a series of periodic ER visits between January and May 2005 that required antibiotics for MRSA infection. There would be a brief period without infection; then recurrences required antibiotic treatment almost every two weeks. The patient became alarmed as the MRSA became unresponsive to more specific antibiotics. Previous lab results showed bone density in the “very osteopenic range” and fasting triglycerides of 37 mg/dL (range less than 150 mg/dL, average 95-100). Physical exam revealed extremely dry lower leg skin and low oral (97.8°F) and basal (97.3°F, average) temperatures.

Initial treatment included 720 mg allicin, a reputed MRSA antibacterial,^{10,11} three times daily and probiotics because of multiple rounds of antibiotics. Laboratory tests revealed:

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- ◆ Cortisol and DHEA were below the 24-hour urine reference ranges
- ◆ Thyroid-stimulating hormone (TSH), T₄, and T₃ were in range, but reverse T₃ (rT₃) was elevated at 23.3 (range 6.7-21.8 ng/dL)
- ◆ Antibodies to dairy and gluten (combined IgG and IgE) were increased
- ◆ Fasting triglycerides of 53 mg/dL
- ◆ Fasting BUN of 7 mg/dL
- ◆ WBC 2.8 (range minimum 4.0 K/μL), RBC 4.27 (normal range 3.80-5.80 M/μL), absolute neutrophils 1.3 (range minimum 2.0 K/μL), absolute lymphocytes 1.10 (range minimum 1.00 K/μL)

At the second visit the patient was placed on cortisol (2.5 mg three times daily), DHEA (escalation to 25 mg daily), and T₃ (gradual escalation of triiodothyronine to 37.5 mcg daily as determined by testing and body temperature). The patient was counseled to strictly avoid dairy and gluten. Vitamin, mineral, and mixed amino acid supplements were recommended because of malabsorption from gluten allergy. (A publication in preparation by the authors discusses the use of lower values of BUN and fasting triglycerides as indicators of malabsorption.) Before the second office visit, the patient required one trip to the ER for MRSA.

By the mid-December visit the patient had required three ER trips due to MRSA outbreak, regardless of allicin intake and “perfect adherence” to a dairy- and gluten-free diet. A fasting laboratory profile revealed BUN of 9 mg/dL, triglycerides of 44 mg/dL, WBC at 2.8 K/μL, and absolute neutrophils at 1.3 K/μL. Indication of malabsorption continued even after elimination of dietary allergens for approximately six-weeks.

After another ER visit in January 2008, the patient began to receive intravenous injections of dilute hydrochloric acid (HCl) twice weekly (5 cc 1:500 HCl and 5 cc saline, slow push). This treatment ended ER visits for an extended period (the immune-enhancing effects of IV dilute HCl are discussed below).

The May 2008 laboratory profile disclosed BUN of 11 mg/dL, triglycerides of 41 mg/dL, RBC at 4.0 M/μL, WBC at 2.5 K/μL, absolute neutrophils at 1.3 K/μL, and absolute lymphocytes at 0.8 K/μL. These results continued to indicate intestinal malabsorption of nutrients and pointed to probable refractory celiac disease. (This problem remains unsolved in medicine with relatively few research publications on the topic; most just

decrying the lack of a solution. Hopefully the recognition of the frequent occurrence of celiac disease will bring more attention to the problem).

From January to mid-July 2008 no MRSA outbreaks occurred. In July 2008 the patient received at least one dilute HCl injection per week. At the August 4, 2008 visit, the patient reported an HCl treatment was missed and a MRSA outbreak had occurred at the end of July. She requested an increase of IV frequency to twice weekly. August laboratory results showed improved WBC count (3.7 K/μL), RBC (4.3 M/μL), absolute neutrophils (2.3 K/μL), and absolute lymphocytes (0.9 K/μL).

From late August through October 2008 communication with the patient was by telephone, since the dilute HCl treatments were performed by the clinic’s nursing staff. The patient was conscious of the health benefits of avoiding gluten. It was suggested that her husband and three dogs be cultured for MRSA (since pets can serve as a reservoir¹²⁻¹⁴), but the suggestion was declined due to cost. In November 2008, the patient discontinued IV dilute HCl treatment due to expense.

In March 2009, a telephone consultation revealed the patient had separated from her husband in September 2008, a topic little discussed previously. There were multiple outbreaks of MRSA from November 2008 through March 2009, with Zyvox used as treatment. The patient recognized that outbreaks only appeared with great stress, such as visits to a lawyer, a court appearance, or seeing her former husband with his girlfriend. (This supports the idea that stress can significantly decrease immune function.)¹⁵⁻¹⁷ Table 1 summarizes the course of treatment for this patient.

IV Dilute Hydrochloric Acid

IV dilute hydrochloric acid became popular among a small number of physicians in the 1930s (the pre-antibiotic era). Two books and a composite of publications from the journal, *The Medical World*, demonstrate extensive use at that time. The latter publication is now available on-line without charge.¹⁸⁻²⁰ Many successful cases with IV dilute HCl have been reported for a wide variety of conditions, including iritis, sinusitis, wound infection, bronchial infection, *Staphylococcus aureus* and *Streptococcus* infections, hepatitis with jaundice, tuberculosis, kidney infection, pneumonia, malaria, elephantiasis, leprosy, and cerebrospinal meningitis. One reported case showed a WBC increase of 2,500 K/μL one hour after IV injection of 1:1,000 HCl.

Contraindications for IV dilute HCl include most enclosed infections, such as tooth abscess or infected appendix. This is apparently due to a dramatic WBC increase in the enclosed space with increased pressure. Caution is advised with sinus infection, middle ear infection, and raised WBC in myelogenous leukemia. In the 1930s, a temporary low-grade fever was considered a side effect of IV dilute HCl and thought to be a positive sign. However, when given in high concentration (1:200), a high fever did result. From author experience of stinging at a 1:500 concentration, a 1:200 concentration would seem likely to cause vascular irritation.

Unpublished research by the principal author presented at a medical meeting²¹ shows increased WBC between three and six hours after slow-push injection. In some cases of bacterial infection, the neutrophils increased. In one case of apparent viral infection, lymphocytes increased. The elevation of WBC count by one injection lasted three to four days. Although the mechanism of action for IV dilute HCl is unclear, the small amount of acid is not enough to surpass the buffer capacity of blood. The zone of high acidity at the injection site may somehow influence the activation of white blood cells.

Conclusion

Most treatments for MRSA focus on agents to eliminate the bacterium. The case discussed implies that susceptibility to MRSA may have predisposing factors. Immune resistance could be lowered in such individuals, and locating immune-inhibiting or immune-enhancing factors might improve susceptibility. In the case presented above, factors for lowered immunity were discovered and corrected (low thyroid effect, low adrenal hormones, and gluten and dairy allergy), although the contribution to the immune system was not successful in eliminating MRSA recurrence. With the amount of marital stress and likely refractory celiac disease, even IV dilute HCl did not completely forestall MRSA outbreaks; without the IV dilute HCl, outbreaks occurred much more often.

Table 1. Patient Timeline

Noteworthy Dates	Labs	Other
Initial Visit October 2007	“Very osteopenic” Fasting triglycerides 37 mg/dL Oral temperature 97.8°F Basal temperature 97.3°F	Repetitive MRSA outbreaks every two weeks requiring ER visits
October 2007	Low cortisol and DHEA Normal TSH, T4, T3, elevated rT3 Elevated dairy and gluten antibodies Fasting triglycerides 53 mg/dL Fasting BUN 7 mg/dL RBC 4.27 M/μL WBC 2.8 K/μL Absolute neutrophils 1.3 K/μL Absolute lymphocytes 1.10 K/μL	MRSA outbreak
November and December 2007	Fasting triglycerides 44 mg/dL Fasting BUN 9 mg/dL WBC 2.8 K/μL Absolute neutrophils 1.3 K/μL	Three MRSA outbreaks
January 2008		MRSA outbreak – Start I.V. dilute HCl twice weekly
May 2008	Fasting triglycerides 41 mg/dL Fasting BUN 11 mg/dL RBC 4.0 M/μL WBC 2.5 K/μL Absolute neutrophils 1.3 K/μL Absolute lymphocytes 0.8 K/μL	No MRSA outbreaks since beginning I.V. dilute HCl
July 2008		One dilute HCl treatment per week MRSA outbreak
August 2008	RBC 4.3 M/μL WBC 3.7 K/μL Absolute neutrophils 2.3 K/μL Absolute lymphocytes 0.9 K/μL	Dilute HCl treatments twice weekly
September 2008		MRSA outbreak Separation from husband
November 2008		Discontinue dilute HCl treatments
November 2008 to March 2010 (at time of this writing)		Multiple MRSA outbreaks treated with Zyvox

This case provides anecdotal support for the proposition that immune enhancement is a viable approach to forestall or clear recurring MRSA. The authors are exploring other agents and points of view for this condition.

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