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# Clinical manifestations in children with vancomycin-tolerant methicillin-resistant *Staphylococcus aureus* (MRSA-TV) infection, compared with vancomycin-sensitive methicillin-resistant *S. aureus* (MRSA-SV) infection

## Abstract

**Introduction:** The clinical relevance of vancomycin-tolerant methicillin-resistant *Staphylococcus aureus* (MRSA-TV) strains remains a subject of debate. Although some studies in adults have associated this phenotype with treatment failure and adverse outcomes, evidence in the pediatric population is limited. The objective was to compare the clinical characteristics, risk factors, and outcomes of children with MRSA-VT infection versus those with vancomycin-susceptible MRSA (MRSA-VS) infection.

**Materials and methods:** An observational, cross-sectional, and comparative study was conducted in patients aged 0 to 16 years with MRSA infection treated at a tertiary-level pediatric hospital between 2005 and 2009. Demographic variables, comorbidities, the presence of invasive therapy, surgical history, disease severity, length of hospital stay, treatment failure, complications, and mortality were analyzed. Odds ratios (OR) with 95% confidence intervals were calculated.

**Results:** Over a four-year period, 54 patients with MRSA infection were identified; 22 were excluded due to incomplete information, leaving 32 cases for analysis. No differences were observed between the groups regarding age, sex, disease severity, or length of hospital stay. Most patients had prolonged hospitalization (>7 days), comorbidities, and use of invasive devices. The presence of invasive therapy (OR 0.5; 95% CI 0.04–5.15), surgical history (OR 0.81; 95% CI 0.67–0.99), immunosuppression (OR 1.33; 95% CI 0.26–6.65), and renal failure (OR 1.08; 95% CI 0.21–5.51) was not associated with vancomycin tolerance. No differences were found in treatment failure (OR 0.44; 95% CI 0.08–2.20), complications (OR 0.5; 95% CI 0.04–5.15), or mortality (OR 0.23; 95% CI 0.04–1.37). Although four of seven deaths occurred in the MRSA-TV group, the difference was not statistically significant.

**Conclusions:** In this pediatric cohort, MRSA-TV infections showed no differences in clinical outcomes, complications, or mortality compared to MRSA-SV infections. Prospective studies with larger sample sizes are needed to define the clinical impact of vancomycin tolerance in the pediatric population.

**Keywords:** methicillin-resistant *staphylococcus aureus* (MRSA), vancomycin tolerance, microbial drug resistance, child, bacteremia, treatment outcome

Volume 16 Issue 2 - 2026

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**Received:** June 3, 2026 | **Published:** June 17, 2026

## Introduction

*Staphylococcus aureus* infection is a major cause of illness and death, accounting for nearly one-fifth of bacteremia cases in the United States.[1] In 1997, Hiramatsu et al.,[2] reported the first strain of methicillin-resistant *S. aureus* (MRSA) that exhibited reduced susceptibility to vancomycin, and subsequently, strains exhibiting "heteroresistance" to vancomycin were isolated. Currently, clinical laboratories do not test for heteroresistance for several reasons: its study is complex, standardized methods do not exist, and perhaps, most importantly, the clinical significance of this phenotype remains unknown. There are no studies demonstrating that patients with infections caused by these strains differ from patients with similar infections caused by MRSA strains that are homogeneously susceptible to vancomycin. Furthermore, many aspects of heteroresistance remain to be defined. First, there is no standardized definition, and researchers define it using various criteria. Second, the clinical significance of heteroresistance is unclear, as there is evidence supporting the hypothesis that heteroresistant strains have a higher risk of developing homogeneous intermediate resistance compared to susceptible strains, while other studies suggest an association between infection with VISA (homogeneous intermediate resistance) strains and an adverse outcome. Third, few studies have analyzed whether there is a relationship between the presence of risk factors for h-VISA (intermediate heteroresistance) infection and whether such infection is associated with a worse prognosis; Similarly, studies that have analyzed risk factors associated with h-VISA infection have been conducted on small samples, have inadequate generalizability, or use a selection control method that does not allow for comparison between patients infected with h-VISA and those infected with susceptible strains.[1] The presence of comorbidities, disease severity, surgical history, and the use of invasive therapy have been shown to be strong predictors of mortality in reports involving adults infected

with h-VISA strains; however, the studies remain small, and there are no reports in children in the literature.[3–6]

## Background

Currently, *S. aureus* remains one of the most important and versatile pathogens in humans, playing a predominant role in skin and soft tissue infections, osteomyelitis, pneumonia, endocarditis, and bacteremia, the treatment of which is becoming increasingly complex due to the microorganism's great capacity to develop antibiotic resistance through a wide range of mechanisms, including enzyme production, the synthesis of molecular targets with low affinity for antimicrobials, mutations or enzymatic modification of proteins or nucleic acids, the generation of persistent, slow-growing forms with defects in electron transport, and alterations in cell wall synthesis or structure.[7,8]

The treatment of staphylococcal infections has evolved since the dawn of the antibiotic era as a result of the emergence of resistance and the development of new antibacterial agents. Glycopeptides have been the mainstay of treatment for methicillin-resistant *Staphylococcus aureus* (MRSA) infections since the introduction of these antibiotics in 1958. Nearly a decade later, in 1997, the first strain with a minimum inhibitory concentration (MIC) for vancomycin of 8 mcg/mL was reported in Japan, and the first isolate with an MIC >32 mcg/mL was recorded in the United States in mid-2002.[9] Today, there are various studies observing the emergence of vancomycin-intermediate (VISA) and vancomycin-resistant (VRSA) strains, in addition to MRSA. There are also strains of *S. aureus* with heterogeneous vancomycin resistance (hVISA); these have an MIC considered susceptible (< or equal to 2 mcg/mL), but include a subgroup of bacterial populations that express a resistance phenotype. This heteroresistance represents the first step toward vancomycin-resistant (VRSA). Infections caused by h-VISA are associated with treatment failure and prolonged or persistent bacteremia in some studies, but not in others. Although h-VISA strains have been identified in various parts of the world, little is known regarding their prevalence and epidemiology, as the most appropriate method for laboratory identification of h-VISA remains uncertain. Currently, the "gold standard" for identifying h-VISA is the detection of PAP-AUC (*population analysis profiling—area under the curve*); however, this method is labor-intensive and is not routinely performed in microbiology laboratories.

Furthermore, it has been observed that in patients colonized by h-VISA strains, a subsequent infection may occur. It has also been reported that in patients colonized by MRSA, there is a 35% risk of developing invasive disease within the first year. Additionally, family members of MRSA-carrying patients may be at risk of becoming colonized themselves. Subsequent infection may also occur in patients colonized with h-VISA strains.

Local variations of MRSA strains exist in every country. This resistance pattern differs between community-acquired and hospital-acquired strains, as hospital-acquired strains exhibit greater antibiotic resistance in an effort to survive in that environment. These mechanisms are constantly evolving, and they not only develop resistance to penicillins and cephalosporins but also to a wide range of antibiotics commonly used in the hospital setting, not to mention that MRSA is associated with multidrug resistance, an aggressive course, and high mortality and morbidity rates, in both community-acquired and nosocomial strains. Today, as a result of antibiotic pressure in hospitals, new strains with higher resistance rates are emerging and replacing previous strains. While methicillin resistance in *S. aureus* is lower in countries such as Norway (1%), Sweden (1%), Denmark (2%), and Canada (5–10%), in the United States it is 25–50%, and in Portugal and Italy 43–58%. Many authors have reported a substantial increase in MRSA in recent years, and its significant impact on many aspects of patient care and infection control. Policies regarding antibiotic use must be updated regularly, as must the monitoring of antibiotic prescribing and consumption. That is why monitoring new MRSA strains, their prevalence, and resistance patterns is essential for infection control, and resistance patterns must be periodically evaluated both clinically and microbiologically in order to appropriately modify infection control and treatment measures and anticipate the rise in resistance. It is also important to remember that these microorganisms are capable of transferring resistance genes, and that if such resistance to vancomycin were to develop, not only *S. aureus* but also other microorganisms within the hospital setting—such as enterococci—would be involved.[10]

There are few studies in Latin America that address the impact of MRSA or VISA infections, the high morbidity and mortality rates, the costs,[11] and the significant impact this has as a public health problem. Timely detection and appropriate treatment are some of the measures to contain this problem.[12] Likewise, epidemiological and clinical monitoring of this microorganism is essential to prevent its spread, the emergence of resistance, and to limit the socioeconomic repercussions that this entails.

## Problem statement

To date, there is a lack of knowledge regarding the clinical impact that infections caused by MRSA-TV may have.

## Research question

Are there clinical differences between infections caused by MRSA-TV compared to MRSA-SV infections in children?

## Justification

Currently, *S. aureus* remains one of the most significant and versatile pathogens affecting humans, playing a major role in skin and soft tissue infections, pneumonia, and bacteremia. Over the past decade, its treatment has become increasingly complex due to the microorganism's high capacity to develop antibiotic resistance. This decrease in bactericidal activity results in the persistence or recurrence of infection, with a consequent increase in length of hospital stay and morbidity and mortality. The differences in clinical presentation between an infection caused by MRSA-TV and one caused by MRSA-SV are unknown, as is whether these differences are reflected in associated in-hospital morbidity and mortality.

## Objectives

### Primary objective

To compare the clinical characteristics of children treated at the Federico Gómez Children's Hospital of Mexico (HIMFG) during the 2005–2009 period with MRSA-TV infection against cases of MRSA-SV infection.

### Secondary objectives

(i) To describe the number of cases treated at the HIMFG during the 2005–2009 period with positive cultures for MRSA-SV and MRSA-TV.

(ii) To describe and compare the clinical characteristics of patients treated during the study period with positive cultures for MRSA-SV and MRSA-TV.

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DOI: 10.15406/jpnc.2026.16.00625

(iii) To describe and compare the complications and comorbidities presented by patients with positive cultures for MRSA-SV and MRSA-TV.

(iv) To describe the antibiotic treatment used, its duration, and the doses administered to patients with positive cultures for MRSA-SV and MRSA-TV.

(v) To describe and compare the causes of mortality in patients with positive cultures for MRSA-SV and MRSA-TV.

**Hypothesis**

The clinical manifestations of patients with MRSA-TV differ from those of patients with MRSA-SV

## Materials and methods

Methodological design: comparative cross-sectional observational study.

### Study population

Patients with MRSA infection.

### Inclusion criteria

Patients at HIMFG aged 0 to 16 years who tested positive in a clinically significant culture (peripheral blood culture, central line culture, pleural fluid, bronchial aspirate, cellulitis, peritoneal fluid, cerebrospinal fluid, catheter tip) for MRSA during the 2005–2009 period.

### Exclusion criteria

(i) Patients who do not belong to the HIMFG population.

(ii) HIMFG patients outside the 2005–2009 period.

(iii) Patients with no MRSA growth in a culture.

### General description of the study

From a sample of 58 cases with documented MRSA infection based on a positive culture (-SV or –TV) between 2005 and 2009, vancomycin-sensitive and vancomycin-tolerant strains were identified according to a previously described methodology. Two mortality scales were applied to the selected subjects upon study enrollment and during the presence of infection to assess whether there was a relationship between a higher score and the presence of vancomycin tolerance. Additionally, various baseline patient conditions during hospitalization and the presence of infection were considered to determine whether the fact that the infection was secondary to a vancomycin-tolerant strain influenced the subject's clinical course compared to a vancomycin-sensitive strain. Among these factors evaluated were sex, prolonged hospital stay (>7 days), the presence of invasive therapy, underlying disease, prior surgical event, changes in treatment when vancomycin was used (the latter considered a failure of vancomycin treatment), the presence of infections, and/or death.

### Statistical analysis plan

Descriptive statistics were used, including measures of central tendency and dispersion. Odds ratios were calculated with their 95% confidence intervals. A *p-value* was considered statistically significant if it was less than 0.05. The SPSS 20 program for Windows was used.

### Description of variables

**Independent variables**

| Variable | Conceptual definition | Operational definition | Type of variable | Measurement scale | Unit of measurement |
|---|---|---|---|---|---|
| Age | Time elapsed since an individual's birth | The patient's age in years from birth | Discrete quantitative | Years | Numeral (0, 1, 2…) |
| Sex | A characteristic by which organisms can be classified according to their reproductive functions | Patient's gender | Qualitative | Nominal | Male=0<br>Female=1 |
| Length of hospital stay | Time elapsed since hospital admission | Time from admission to discharge or death of the patient | Discrete quantitative | Days | Numeral (0, 1, 2…) |
| Medical history | Patient medical history | The patient's chronic condition(s) at hospital admission | Qualitative | Nominal | Renal failure=1<br>Immunosuppression=2<br>Malnutrition=3<br>Previous surgery=4<br>Invasive therapy* = 5<br>Other**=6 |
| Source of infection | Origin, source, root, and cause of something | Onset of mrsa infection (TV or SV) | Qualitative | Nominal | Peripheral blood culture = 1<br>Central blood culture = 2<br>Cellulitis = 3<br>Intestine = 4<br>Bronchial aspirate = 5<br>Pleural fluid = 3 |

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DOI: 10.15406/jpnc.2026.16.00625

 

| | | | | | |
|---|---|---|---|---|---|
| Antibiotic | A chemical substance produced by a living organism or manufactured synthetically, capable of inhibiting the growth of certain pathogenic microorganisms through its bacteriostatic action, or causing their death through its bactericidal action. | Chemical substance used to treat infections caused by MRSA, TV, and SV | Qualitative | Nominal | Ampicillin=1<br>Amikacin=2<br>Amoxicillin/clavulanic acid=3 amphotericin b=4<br>Cefepime=5<br>Cefotaxime=6<br>Cefuroxime=7<br>Ceftriaxone=8<br>Ceftazidime=9<br>Clindamycin=10<br>Dicloxacillin=11<br>Fluconazole=12<br>Imipenem=13<br>Meropenem=14<br>Metronidazole=15<br>Rifampicin=16<br>Piperacillin/tazobactam=17<br>Teicoplanin=18<br>Vancomycin=19 |

Invasive therapy: Mahurkar catheter, Tenckhoff catheter, Niagara catheter.

**Dependent variables**

| Variable | Conceptual definition | Operational definition | Type of variable | Measurement scale | Unit of measurement |
|---|---|---|---|---|---|
| PRISM[13] | Pediatric risk of mortality | Scale designed to calculate the risk of mortality in children in the intensive care unit | Nominal | Ordinal | Low mortality score <8=0<br>High mortality score >8=1 |
| Complications | Difficulty or entanglement arising from the concurrence and interplay of various factors; the action and effect of complicating | Refers to any unsuccessful secondary process in the management of the disease | Nominal | Qualitative | Abscess=1<br>Endocarditis=2<br>Sepsis = 3<br>Renal failure=4<br>Respiratory failure=5<br>Surgery=6<br>Pleural effusion=7 |
| Death within 28 days | Death | Life expectancy of 28 days following confirmation of MRSA TV or MRSA SV infection | Nominal | Qualitative | Yes=1<br>No=0 |

## Results

Over a 4-year period, 54 patients diagnosed with MRSA infection were identified, of whom 22 cases were excluded due to missing information in the medical records.

### Clinical characteristics

The clinical characteristics of MRSA-TV compared with MRSA-SV are shown in Table 1. Demographic factors (sex and age), length of hospital stay, and disease severity at admission and at the onset of infection showed no differences between the two groups. In all cases, the hospital stay exceeded 7 days, consistent with the fact that MRSA infection, regardless of vancomycin susceptibility, is associated with a prolonged hospital stay. On the other hand, the mortality scale used to assess risk at the onset of infection—PRISM (Pediatric Risk of Mortality)—coincided in only one of the cases with MRSA-TV infection with a high mortality index and the patient's death.

**Table 1** Clinical characteristics

| | Vancomycin-tolerant | Vancomycin-sensitive |
|---|---|---|
| | **n=10** | **n=22** |
| **Sex** | | |
| Males, n (%) | 4 (40) | 13 (59.09) |
| Age (months), mean (± SD) | 68.3 (67.44) | 49.67 (62.2) |
| Years | 5.6 (5.6) | 4.1 (5.1) |
| **LHD >7 (days)** | | |
| <20 | 1 (10) | 4 (18.1) |
| >20 | 3 (30) | 12 (54.5) |
| >60 | 3 (30) | 2 (9) |
| >150 | 3 (30) | 4 (18.1) |
| PRISM* | 1 (10) | 0 |

*The PRISM (Pediatric Risk Index Score Mortality) mortality scale was used, with a mortality rate of >50% considered significant.

** LHD: length of hospital stay

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### Risk factors for MRSA-TV infection

In most of the cases analyzed, comorbidities were present, such as invasive therapy (central venous catheter, ventriculo-peritoneal shunt, gastrostomy, peritoneal dialysis catheter, orotracheal intubation, pleural or silo drain; OR 0.5; 95% CI 0.049–5.15; $p$ = 1), surgery (all cases had at least one prior surgical history before the infection (OR 0.81; 95% CI 0.67–0.99; p=0.2), immunosuppression (OR 1.33; 95% CI 0.26–6.65; p=1), or renal failure (OR 1.08; 95% CI 0.21–5.51; p=1), without being directly associated with the presence of vancomycin tolerance (Table 2).

**Table 2** Risk factors for MRSA infection – TV

| Risk factor | MRSA-TV | MRSA-SV | OR 95% CI | *p-value* |
|---|---|---|---|---|
| | n=10 | n= 22 | | |
| Invasive therapy, n (%) | 9 (90) | 18 (81.8) | 0.5 (0.049–5.15) | 1 |
| Surgery, n (%) | 10 (100) | 18 (81.8) | 0.81 (0.67 – 0.99) | 0.2 |
| Immunosuppression, n (%) | 3 (30) | 8 (36.3) | 1.33 (0.26 – 6.65) | 1 |
| Renal failure, n (%) | 3 (30) | 7 (31.8) | 1.08 (0.21 – 5.51) | 1 |

MRSA-TV: Methicillin-resistant, vancomycin-tolerant *Staphylococcus aureus*. MRSA-TV: Methicillin-resistant, vancomycin-sensitive *Staphylococcus aureus*.

### Clinical outcome

On the other hand, treatment failure was defined as the need to change the antimicrobial agent due to sluggish response despite appropriate management in terms of dose, duration, and spectrum; however, no significant differences were found between the two groups (MR 0.44; 95% CI 0.08–2.20; p=0.4). No higher number of complications (abscess, septic shock, bacteremia, pleural effusion, respiratory failure, neuroinfection; OR 0.5; 95% CI 0.04–5.15; p=1) nor higher mortality in patients with MRSA-TV compared to the MRSA-SV group (OR 0.23; 95% CI 0.04–1.37; p=0.1). Although bacteremia was observed in most cases, it was not associated with vancomycin tolerance. Of the seven deaths, four occurred in the MRSA-TV group; however, no significant difference was observed ($p < 0.05$) (Table 3).[14]

**Table 3** Clinical outcomes

| Outcome | MRSA-TV | MRSA-SV | ICRM (95%) | *p-value* |
|---|---|---|---|---|
| | n=10 | n= 22 | | |
| Treatment failure, n (%) | 4 (40) | 5 (22.7) | 0.44 (0.08–2.20) | 0.4 |
| Complications, n (%) | 9 (90) | 18 (81.8) | 0.5 (0.04 – 5.15) | 1 |
| Death, n (%) | 4 (40) | 3 (13.6) | 0.23 (0.04 – 1.37) | 0.1 |

MRSA-TV: Methicillin-resistant, vancomycin-tolerant *Staphylococcus aureus*. MRSA-TV: Methicillin-resistant, vancomycin-susceptible *Staphylococcus aureus*.

## Discussion

Several studies (often retrospective, involving few patients, and mostly adults) have been published on the clinical significance of heteroresistant *S. aureus* strains to glycopeptides, some of which demonstrate that these strains are associated with treatment failure. This observation is difficult to evaluate, both due to the lack of standardization of microbiological methods for defining this phenotype and because other studies do not reach the same conclusion.[15] Since there are no reports in the literature showing the clinical differences between a group infected with an MRSA-TV strain and a group infected with a susceptible strain, this study sought to compare the clinical course, outcome, and mortality between a group of 10 children infected with MRSA-TV strains and a group of 22 children with infections caused by susceptible strains, in order to determine whether the clinical presentation differed between the two groups. Although this study has the aforementioned limitations, the results suggest that vancomycin resistance does not confer clinical manifestations different from those caused by a susceptible strain in pediatric patients.

Since vancomycin resistance is due to the insertion of the *vanA* gene from vancomycin-resistant enterococci,[16] it cannot be ruled out that the expression of its virulence may also differ. Pozzi et al.,[17] demonstrated that the acquisition of the *mecA* gene, in addition to conferring methicillin resistance, also adds other virulence mechanisms such as the production of enzymes and toxins, the ability to form biofilms, and the capacity to evade h immunity. Neoh et al.,[18] observed that, in patients with h-VISA infection, what initially behaved as a common MRSA infection soon developed into VISA and led to treatment failure. They also noted that although all the strains studied had MICs between 1–2 mg/L and were therefore considered susceptible to vancomycin, the clinical course of the patients differed. The less susceptible the MRSA was to vancomycin, the longer it took for the fever to subside, indicating that varying levels of vancomycin susceptibility in MRSA play a critical role in the clinical outcome of bacteremias; therefore, identifying these cases would be preferable for initiating treatment of these infections.[18] In contrast, Schwaber et al.,[1] found no clinical differences between bacteremia caused by susceptible strains and those with probable heteroresistance; however, they propose that although a higher number of deaths or longer hospital stays are not observed, a longer duration of bacteremia and the presence of recurrent or non-eradicated and indolent infections[1] after treatment cannot be ruled out.

This study has several limitations. The analyzed data are retrospective, and it is difficult to determine whether the growth in cultures is secondary to infection or to colonization by the microorganism. Furthermore, the lack of statistical significance may be due to the small sample size resulting from missing data in the studied records; therefore, the results obtained could show significant differences if a larger population were analyzed. On the other hand, the presence of infection in conjunction with the use of broad-spectrum antibiotics other than vancomycin may have altered the clinical course of the disease and, consequently, the observed characteristics.

In summary, in this study we observed that most cases of MRSA SV and TV infection are associated with the presence of comorbidities, invasive therapy, prolonged hospital stay, and the presence of bacteremia.[16] The included patients showed no significant differences in clinical course between a vancomycin-tolerant strain and a susceptible one. However, studies with larger cohorts and longer follow-up periods are needed to validate the findings.

## Conclusion

This study shows no differences in clinical presentation and mortality between the two groups. Although this finding is not confirmed in the literature, further studies are needed to support our findings.

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## Study limitations

i. Lack of information in the medical record.

ii. Lack of positive cultures reported in the medical records.

iii. Absence of a severity and mortality measurement scale outside the intensive care unit.

## Acknowledgements

None.

## Funding

None.

## Conflicts of interest

The authors declare that there are no conflicts of interest.

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