RESEARCH PAPER

Inflammatory myocardial and pericardial syndrome secondary to community acquired MRSA infection in an immunocompetent child- a case report

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Abstract

Background: Myocarditis and pericarditis are inflammatory diseases of the myocardium and pericardium, respectively, with potential overlap. European society of cardiology has published clinical practice guidelines-2025 covering the whole spectrum of these diseases for which an umbrella term Inflammatory myopericardial syndrome (IMPS) is coined [1]. Its causes can be viral, bacterial, or autoimmune. While there are several reports describing staphylococcal purulent pericarditis in the pediatric literature, only a few identify community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) as the culprit in children [2,3]. Methicillin-resistant Staphylococcus aureus (MRSA) is a significant cause of sepsis in pediatric patients, leading to severe complications such as infective endocarditis and purulent pericarditis. **Case Presentation**: We present a case of a previously healthy 10-year-old girl who developed CA- MRSA positive sepsis with severe pericardial effusion, myocarditis followed by endocarditis, and multi-organ dysfunction. Despite broad-spectrum antimicrobial therapy, pericardiocentesis, intrapericardial fibrinolysis, and pericardial window surgery, she succumbed to refractory septic shock and cardiac failure. **Conclusions**: This case underscores the aggressive nature of MRSA infections, their multisystem complications, antibiotic resistance, and the importance of early recognition and multidisciplinary management including timely serial 2 d echo and cardiac interventions.

Keywords: Inflammatory myocardial and pericardial syndrome (IMPS); CA-MRSA; Purulent Pericarditis; Myocarditis, antibiotic resistance, intrapericardial fibrinolysis, pericardiocentesis, pericardial window

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1. Introduction

Methicillin-resistant Staphylococcus aureus (MRSA) is a leading cause of hospital-acquired and community-acquired infections, contributing to severe sepsis, infective endocarditis, and pericardial effusion in pediatric patients. The incidence of MRSA sepsis has been increasing, with complications such as infective endocarditis, pericardial effusion, and septic shock, often leading to poor outcomes【4】. Early recognition and appropriate antimicrobial therapy are crucial in reducing morbidity and mortality【5】. This case highlights a rare and severe manifestation of MRSA sepsis, emphasizing the challenges in diagnosis and management.

2. Case Presentation

10 yrs old girl, born of non-consanguineous marriage, 1st by birth order presented with fever, vomiting, lower limb pain since 8 days, vesicular rash on lower and upper limb noticed since 4 days (figure 1), increasing drowsiness and respiratory distress since 1 day. On admission she had tachypnea, tachycardia, hypotension, delayed CRT, pericardial rub on auscultation, use of accessory muscles of respiration, fever associated irrelevant talk, bilateral lower limb swelling, power was 3/5 in upper limbs and 2/5 in lower limbs. She was started on non-invasive positive pressure ventilation, broad spectrum antibiotics, antiviral and adrenaline infusion for cardiogenic shock. Her initial blood investigations revealed no significant abnormality except high CRP od 59 mg/dL . tropical fever panel including Rickettsial antibodies, ANA, CSF routine, CSF PCR were negative. 2

D echo was done in view of audible pericardial rub, cardiomegaly on x ray (figure 2), hypotension, tachycardia- showed ejection fraction 60% with mild non-tappable pericardial effusion. Fever spikes persisted and next day noradrenaline and dobutamine was added due to septic shock. Sensorium improved in 2 days. Respiratory distress also reduced and respiratory support was weaned. Lower limb doppler was done to rule out deep venous thrombosis in the setting of cardiomyopathy as she had lower limb swelling and pain, doppler was normal. Macrophage activation syndrome was suspected due to multisystem involvement and negative tropical infection tests, but inflammatory markers like ferritin and LDH were mildly raised. After 4 days of admission, blood culture that was sent on admission, came positive for MRSA and vancomycin was started according to sensitivity pattern. Ionotropic support was weaned off and she was continued on IV antibiotics with diuretics and fluid restriction for treating pericardial effusion. Her NT pro BNP was 3963 pg/ml, CRP was 180 mg/dl. 2 D echo was repeated after 4 days due to persistent tachycardia, pericardial rub, increased respiratory distress needing NIPPV support again and found to have increased large pericardial effusion leading to cardiac tamponade, EF 50%. Pericardiocentesis was done by subxiphoid approach, 300 ml frank pus drained (figure 3). Respiratory distress reduced after that and she was on O2 by nasal prongs. Serial 2D echo were done daily to look for relapse of pericardial effusion, showed nontappable trace effusion. Her LFTs and RFTs were marginally affected during this flared MRSA sepsis, which resolved in next few days. High grade fever spikes persisted. Pericardial fluid showed more than 50,000 pus cells, 85% polymorphs. Fluid CBNAAT was negative. Pericardial fluid culture was also positive for MRSA

Repeat 2 D echo 3 days after pericardiocentesis showed mildly dilated RA/RV, EF 35%, moderate pericardial effusion (nontappble) with microfibrinous strands in effusion, and echogenic structure arising from interatrial septum reaching till tricuspid valve (figure 4). She developed spontaneous right sided pneumothorax, for which ICD was inserted (figure 5). Gentamycin and rifampicin were added to battle advancing MSRA leading to endocarditis. Due to increasing pericardial effusion despite IV antibiotic cover, 2nd time pericardiocentesis done with pigtail catheter in situ (figure 6), 500 ml pus drained. Serial pus drainage done through pigtail catheter at 24 hrs interval daily. 55 ml purulent pericardial fluid tapping done next day. 50 ml on day 3 of pigtail. Repeat blood culture after 14 days of vancomycin again grew MRSA. Vancomycin trough levels were sent which came to be 9.3 mcg/dl (therapeutic range 15-20 mcg/dl) [6] Hence vancomycin was stopped and patient was started on daptomycin and ceftarolin. 3 sets of separate blood cultures were sent to establish diagnosis of infective endocarditis out of which 2 came positive for MRSA. Repeat pericardial fluid culture also came positive for MRSA. Repeat blood culture grew carbapenemaze resistent acinetobactor boumani for which polymixin B and sulbactam was added. Indomethacin trial given for 2 days to reduce pericardial effusion [7] . Fever spikes were multiple in a day, high grade, with CRP more than 200 mg/dl. Serial 2 D echo showed thick fibrinous strand with minimal effusion over posterior wall. Trial of intrapericardial fibrinolysis with TPA (alteplase) 10 mg in 20 ml NS was given [8]. NT pro BNP repeated and came 14314 pg/dl. After 1st alteplase trial, 110 ml fluid was drained. 75 ml fluids drained on second day before giving second trial of alteplase and 55 ml after alteplase. She was still having fever spikes. After 15 days of pitail in situ and continuous high volume of daily pus drainage, CVTS team was involved and planned pericardial window. (Cumulative pericardial fluid aspirated in the whole course =1734 ml)

Pericardial window operation done in which dense pleural adhesions separated, 100-150 ml serosanguinous fluid with pus flakes drained out. No. 24 drain placed in pericardial window and No. 28 in ICD placed in left pleural cavity (figure 7). She remained hypotensive despite giving ionotropes support with adrenaline, noradrenaline, vasopressing and milrinone. Heart contractility was very poor. In view of anuria for 4 hrs, peritonial dialysis was done. But due to poor myocardial contractility and persistent hypotension, Ef 20% on POD-2 she succumbed to death by multiorgan dysfunction syndrome and cardiac arrest.

Fig. 1: Vesicular rash on left hand.

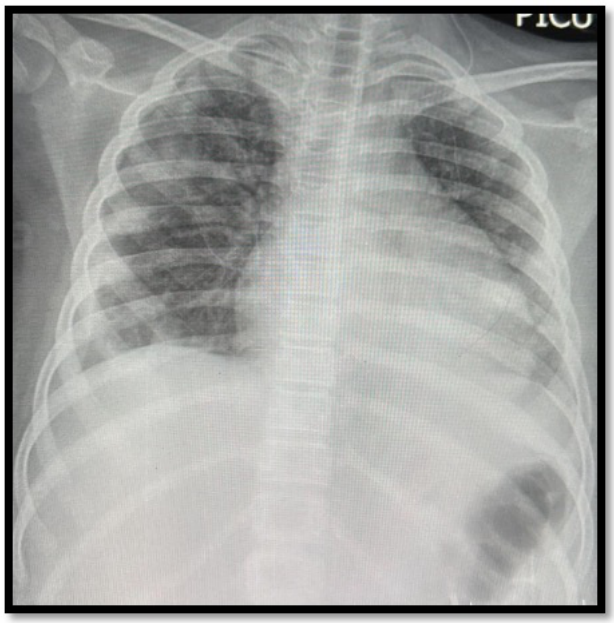


Fig. 2: Cardiomegaly on X-ray chest on admission – Cardiac index 0.6

Fig. 3: Post pericardiocentesis -drainage of 300cc pus

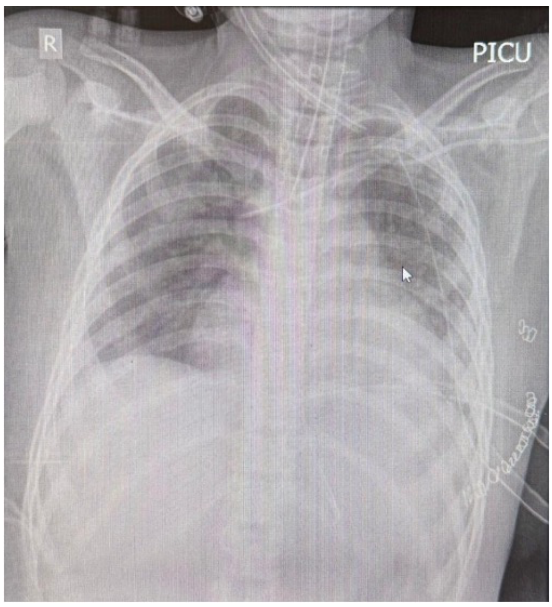
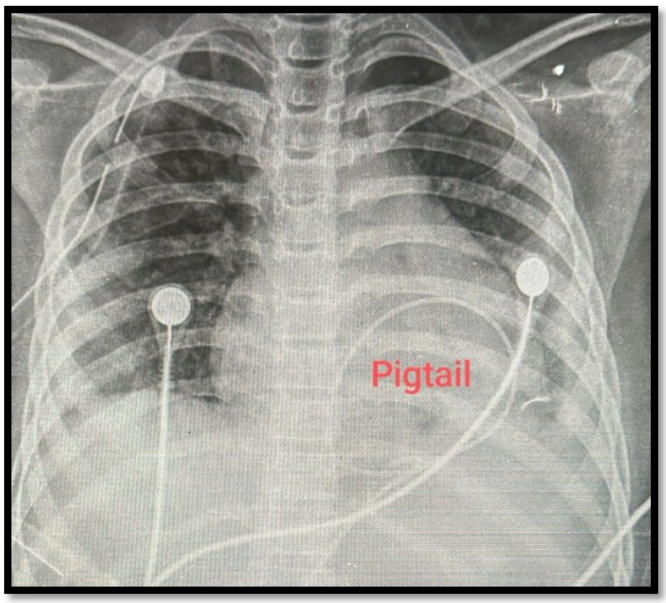


Fig.4: Echogenic structure (vegetation) arising from interatrial septum into RA



3. Discussion

Methicillin-resistant Staphylococcus aureus (MRSA) is a major cause of bloodstream infections, with significant morbidity and mortality in pediatric intensive care settings.

Vancomycin treatment failure: Vancomycin remains the first-line treatment for MRSA infections, but treatment failures are common, leading to the use of daptomycin or linezolid in refractory cases【9】Based on adult studies and the Infectious Diseases Society of America (IDSA) guidelines, the recommended vancomycin trough levels for severe MRSA infections are 15 to 20 µg/mL [10]. However, reaching this trough in children often requires very high doses; our patient did not reach target levels with 25 mg/kg/dose after 14 days of vancomycin therapy. Linezolid or clindamycin were chosen in combination as therapeutic agents because of their bacteriostatic activity. With the challenge of attaining therapeutic troughs and potential for renal toxicity with vancomycin, alternative therapies for MRSA are necessary in children. In our case, daptomycin was added to increase confidence in antimicrobial coverage, while vancomycin troughs remained below recommended levels. Although there are limited reports of daptomycin use in infants, a dose of 6 mg/kg daily was chosen

based on one pharmacokinetic study of 20 infants, demonstrating more rapid drug clearance in infants compared with adult [11] .

In children, daptomycin is generally not considered as the first-line therapy due to limited data on efficacy, pharmacokinetics, and adverse events. However, in the era of MRSA, there is a renewed interest in daptomycin as an alternative to vancomycin. One prior case study reports effective treatment of MRSA endocarditis with daptomycin in a child [12], but there are no similar reports of daptomycin use in children for pericarditis. A wide gap remains between antibiotic regimens available to adults and children, demonstrating a need for further studies in the pediatric population.

3.2 Surgical management of purulent pericarditis: The surgical interventions for our patient involved a multi-disciplinary team, including pediatric cardiology, pediatric cardiothoracic surgery, and pediatric surgery. The approach to draining the effusion was surgical following pericardiocentesis due to persistent pericardial drainage despite appropriate antibiotic therapy and pericardial pigtail drainage for 14 days and formation of fibrinous strands . MRSA-related infective endocarditis often leads to thrombotic complications. Yamaguchi et al. (2021) found that intracardiac vegetations are present in up to 40% of MRSA-IE cases, increasing the risk of embolic events【13】. Our patient developed intracardiac vegetation, emphasizing the need for early identification of embolic risks. Despite aggressive interventions, mortality remains high in MRSA-induced purulent pericarditis and endocarditis. However, our patient's deteriorating cardiac function and persistent bacteremia despite prolonged therapy highlight the limitations of current treatment strategies.

3.3 Inflammatory myocarditis and pericarditis syndrome: Our patient had developed myocarditis and pericarditis as proven by 2 D echo showing decreased ejection fraction, high NT pro BNP levels, and pericardial fluid culture positive for MRSA. Treatment of IMPS includes cause specific intervention like appropriate antibiotic therapy in our case and use of anti-inflammatory medicines like cholchicin, indomethacin, steroids [14] . We used indomethacin but still our patient developed constricitive pericarditis. Steroids were not started due to high bacterial load in our patient. IMPS needs detailed cardiac studies like cardiac MRI to assess the extent of myocarditis and pericarditis [14] but our patient was hemodynamically unstable to perform a cardiac MRI.

4. Conclusions

MRSA sepsis is a highly aggressive infection in pediatric patients, particularly when complicated by infective endocarditis, myocarditis and purulent pericarditis. This case demonstrates the rapid progression, multi-organ involvement, and high mortality associated with MRSA sepsis despite aggressive antimicrobial therapy and surgical interventions. Early recognition, escalation of therapy, and a multidisciplinary approach are critical for improving outcomes.

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