ISSN 0971-9032
Curr Pediatr Res 2025; 29 (02): 1-9 www.currentpediatrics.com

Multifocal Necrotizing Fasciitis Due to Methicillin-Resistant *Staphylococcus aureus* Complicated by Tracheal Stenosis in a 10-Year-Old Boy: A Case Report

Alharbi Abdullah*, Alalem Hala

[1]Department of Pediatric Intensive Care, King Abdullah Specialized Children Hospital, National Guard Hospital, Riyadh , Saudia Arabia

[2]Department of Pediatric Intensive Care, King Abdullah Internatnational Medical Research Center, King Saud Bin Abdulaziz University for Health Sciences Riyadh, Saudi Arabia

Received: *19 March, 2025, Manuscript No. AAJCP-25-162683;* **Editor assigned:** *21 March, 2025, Pre QC No. AAJCP-25-162683 (PQ);* **Reviewed:** *04 April, 2025, QC No. AAJCP-25-162683;* **Revised:** *11 April, 2025, Manuscript No. AAJCP-25-162683 (R);* **Published:** *21 April, 2025, DOI:10.35841/0971-9032.29.02.2358-2362*

Abstract

Necrotizing fasciitis (NF), a rapidly progressive soft tissue infection with high mortality, is increasingly caused by Methicillin-resistant Staphylococcus aureus (MRSA) strains.

We report a unique and complex case of a 10-year-old Syrian boy with no prior medical history who developed multifocal necrotizing fasciitis caused by MRSA. The fulminant course was complicated by septic shock, multiple organ dysfunction syndrome (MODS), and ultimately, life-threatening tracheal stenosis secondary to bacterial tracheitis leading to recurrent cardiac arrest. Despite extensive surgical debridement, broad-spectrum antibiotics, and mechanical ventilation, the patient developed tracheal pseudo-membrane obstruction requiring multiple bronchoscopies and ultimately a tracheostomy for airway security. Fortunately, with proper management, he achieved clinical stability and was discharged home with a tracheostomy.

This case emphasizes the aggressive nature of MRSA infection-induced NF, the potential for unexpected complications, and the importance of a multidisciplinary approach in patient management to identify and address such complications and ensure optimal outcomes.

Keywords: Multifocal Necrotizing Fasciitis (MRSA); Septic shock; Tracheal stenosis; Pediatric case

Accepted on 04th April; 2025

Introduction

Necrotizing Fasciitis (NF) is a rare but life-threatening soft tissue infection, that presents a significant challenge due to its rapid tissue destruction, demanding treatment regimen, and high mortality rate [1]. Staphylococcus aureus (*S.aureus*) is the most prevalent pathogen, with an increasing proportion attributed to Methicillin-resistant S. aureus (MRSA) strains [2].

Historically, NF has been primarily associated with immunocompromised adults or individuals with pre-existing medical conditions such as diabetes mellitus, peripheral vascular disease, or chronic wounds [3,4]. Pediatric cases of NF, specifically those caused by MRSA, are less frequent compared to adults [5]. However, recent studies suggest a concerning shift in the epidemiology of this devastating infection, with reports of a growing number of pediatric necrotizing fasciitis cases caused by MRSA in previously healthy children, highlighting the evolving landscape of this potentially devastating infection [6-8].

The complications associated with NF extend far beyond the initial infection and can be devastating. The rapid tissue destruction characteristic of NF can lead to extensive skin and muscle loss, often requiring radical surgical debridement. This can result in significant functional impairment and the need for reconstructive surgery [9]. Furthermore, the systemic inflammatory response triggered by NF can progress to Multi-Organ Dysfunction Syndrome (MODS), septic shock, and even death [9]. Additionally, NF can lead to long-term sequelae such as scarring and contractures as well as potential airway compromise, impacting a patient's quality of life [10].

To our knowledge, there is no case report on tracheal stenosis as a complication of MRSA-induced NF in children. Here, we report a unique case of a previously healthy child with MRSA necrotizing fasciitis complicated by severe, near-fatal tracheal stenosis. By sharing this case, we aim to contribute to the evolving understanding of pediatric MRSA-induced NF, emphasize the aggressive nature of this infection, and underscore the importance of early recognition and comprehensive management to mitigate devastating sequelae.

Case Report

A 10-year-old Syrian boy with no prior medical or surgical history presented to the emergency department at King Abdullah Specialized Children's Hospital (KASCH) in Riyadh, Saudi Arabia, with a 3-day history of multiple, progressively painful, and enlarging swellings on his extremities, trunk, and forehead accompanied by a rash over his body. He was accompanied by his mother, who reported that he had also developed a high-grade fever, difficulty walking, and fatigue.

He had received amoxicillin-clavulanic acid for 5 days without improvement.

There was no history of recent trauma, insect bites, malignancy, immune deficiencies, rheumatologic conditions, or similar illnesses in the patient or his family and he had no known allergies. The family immigrated from Syria as refugees and resided in a crowded flat with three siblings. The patient was not vaccinated and did not attend school.

Clinical findings

On examination, the patient appeared ill and in significant pain. Vital signs were notable for tachycardia (heart rate 133 beats per minute) and a moderate-grade fever (temperature 38.5°C). Respiratory rate was elevated (43 breaths per minute) but with normal oxygen saturation (98% on room air) and clear breath sounds bilaterally. Peripheral pulses were weak in the lower extremities compared to the upper extremities, with cold extremities. He was dehydrated with delayed skin turgor. Neurological examination revealed an alert and oriented child with no cranial nerve deficits.

Musculoskeletal examination identified multiple well-demarcated, erythematous, exquisitely tender, indurated, and swollen lesions involving the left leg (largest and most prominent), bilateral thighs, right gluteal area, left upper limb (around the elbow), right upper limb, and forehead (Figures 1-6).

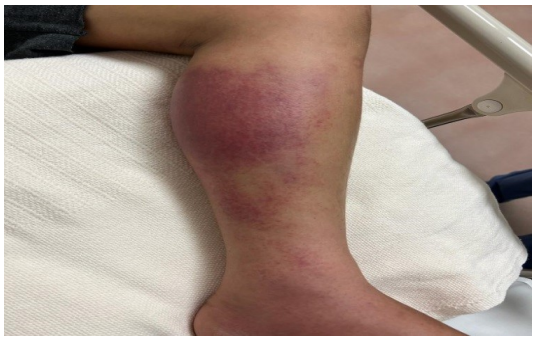

Figure 1: The left leg shows the largest and most prominent lesion with erythema, induration, and swelling. Crepitus was present upon palpation.

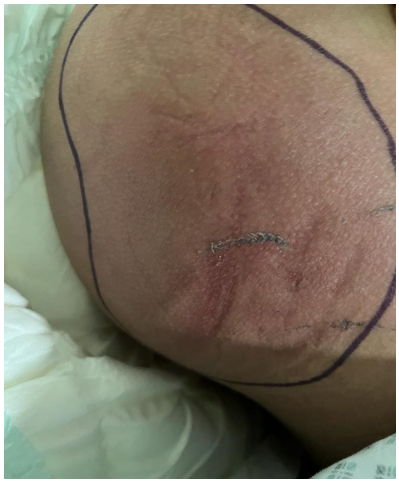

Figure 2: The proximal right thigh demonstrates a well-demarcated, erythematous, tender upon palpation and indurated lesion.

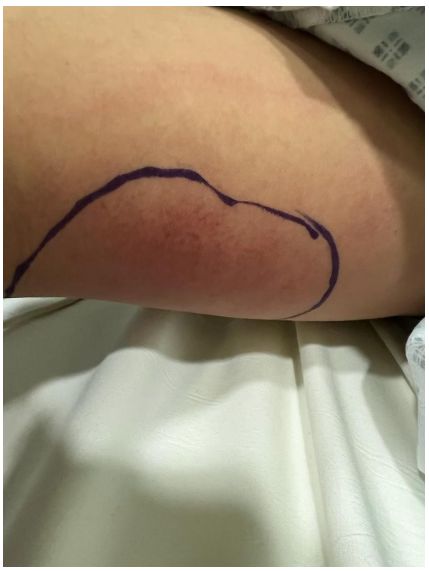

Figure 3: Right gluteal area, illustrating a well-demarcated, erythematous, tender upon palpation, and indurated lesion.

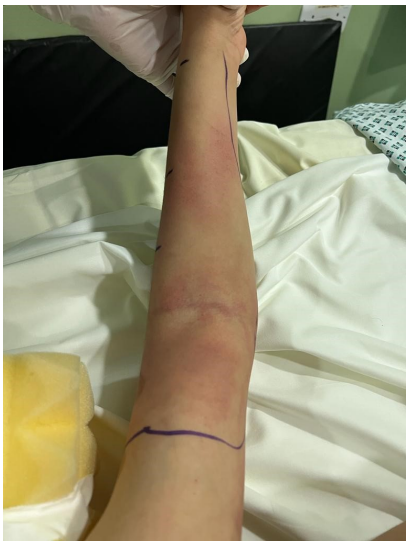

Figure 4: The left upper limb and periarticular region of the elbow show a well-demarcated, erythematous, tender upon palpation, indurated, and swollen lesion.

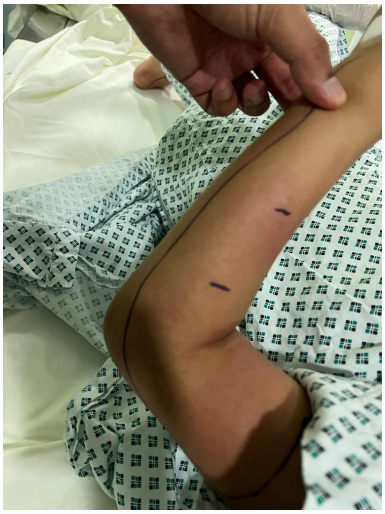

Figure 5: Right upper limb, demonstrating a well-demarcated, erythematous, tender upon palpation, and indurated lesion.

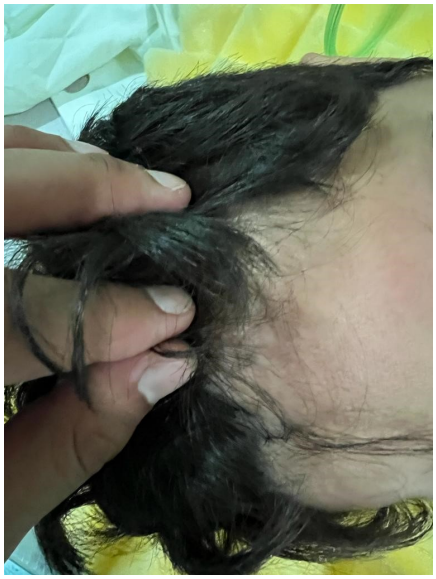

Figure 6: Forehead, illustrating a well-demarcated, erythematous, tender upon palpation, and indurated lesion.

Timeline

The patient's clinical course is summarized in Table 1.

Diagnostic Assessment

Laboratory evaluation demonstrated a constellation of abnormalities suggestive of an inflammatory state (Table 2). A mildly low white blood cell count (4.74 x 10^9/L) was accompanied by significantly elevated inflammatory markers including procalcitonin (47 ng/mL; erythrocyte sedimentation rate (25 mm/hr; C-reactive protein (174 mg/L; and ferritin (1268 ng/mL; all pointing towards a potential infectious process. Markedly elevated cardiac enzymes (creatine kinase-MB 48.7 U/L, total creatine kinase 1830 U/L, and lactate dehydrogenase 714 U/L) suggested possible muscle damage. Additionally, mildly low albumin (22 g/L) with elevated Alkaline Phosphatase (ALP; aspartate aminotransferase (AST; Gamma-Glutamyl Transferase (GGT; and total bilirubin (T BIL) indicated potential liver dysfunction. X-rays of the extremities were obtained (Figure 7).

Stage	Date (Hospital Day)	Event (s)	Intervention (s)	Outcome
Diagnosis & Source Control	05-06-2023 (Admission)	Multifocal Necrotizing Fasciitis Confirmed	Multiple Debridement Surgeries (By Plastic & Pediatric Surgery),	Extensive tissue removal, hemodynamic instability persists (Day 2)
			Broad-spectrum therapy started	
	07-06-2023 (Day 2)	Positive Cultures for MRSA	IV Antibiotics Adjusted (Continued linezolid, started vancomycin & daptomycin, discontinued clindamycin & piperacillin/tazobactam, added gentamycin & IVIG (2nd dose)).	Source control yet to be achieved (Day 5-7)
		Worsening clinical presentation (increased redness, fever)	Analgesics and Sedation.	
			Vasopressor support needed	
			Surgery Repeated	
	12-06-2023	Pan-CT Scan Reveals Abscesses & Lung Cavitations	Antibiotic regimen optimized: Vancomycin, ceftaroline, ceftazidime, Discontinued gentamycin, linezolid and daptomycin	Improved hemodynamic stability requiring lower vasopressor support, and achieving acceptable levels of sedation and analgesia
	13-06-2023 (Day 8-9)	Respiratory culture showed Pseudomonas aeruginosa		
		Whole body MRI confirms left distal femur osteomyelitis		
	15-06-2023 to 14-07-2023 (Day 10-39)	Prolonged ICU hospitalization	Multiple surgical interventions for wound management and closure (16 debridements, 2 closure procedures)	Patient improved, achieved hemodynamic stability without vasopressors, and was extubated to high-flow nasal cannula then to room air.
		Negative repeated cultures	Continue a 6-week course of vancomycin and a 7-day course of ceftazidime for VAP Pseudomonas aeruginosa as per pediatric infectious disease team recommendation	Source control achieved.
Airway Compromise	15-07-2023 (Day 40)	Sudden Cardiac Arrest	Prolonged CPR (26 min)	ROSC achieved, Intervention restores airway patency, requires permanent solution
		Bronchoscopy reveals airway obstruction	BAL	

	16-07-2023 (Day 41)	Brain and Chest CT Scans	Brain unremarkable, chest CT shows septic emboli	Multisystem involvement identified
			Brain MRI (mild global brain volume loss	
Stenosis Management	20-07-2023 (Day 45)	Tracheal Stenosis Identified	Tracheal Surgery (Pseudo-membrane Removal) & Biopsy	Airway obstruction addressed, biopsy shows acute inflammation (no malignancy), continued antibiotics (MRSA & pseudomonas coverage)
	03-08-2023 (Day 59)	Recurrent Tracheal Stenosis (Stridor)	Debridement (Collapsing Pseudo-membrane)	
			Adjusted antibiotics (Ceftazidime/avibactam for pseudomonas) and continue vancomycin for a total of 8 weeks	
		Multiple Dilation Procedures for Stenosis	DLB + Dilation (ENT Surgery)	Ongoing efforts to address persistent stenosis
		Workup for Underlying Cause	Rheumatology & Immunodeficiency Workup	Negative results
Definitive Airway & Recovery	30-10-2023	Tracheostomy Placement	Permanent airway secured	Discharged home with regular ENT follow-up and rehabilitation program (PT/OT)
	To 22-11-2023	Patient stable and lab results within normal range		

Note: MRSA: Methicillin-resistant Staphylococcus aureus; CT: Computed Tomography;DLB: Direct Laryngoscopy & Bronchoscopy; ENT: Ear, Nose, and Throat specialist; ICU: Intensive Care Unit; IV: Intravenous; MRI: Magnetic Resonance Imaging; VAP: Ventilator-Associated Pneumonia; CPR: Cardiopulmonary Resuscitation; BAL: Bronchoalveolar Lavage; ROSC: Return of Spontaneous Circulation; OT: Occupational Therapy; Pan-CT: Panoramic Computed Tomography; PT: Physical Therapy

Table 1: Timeline of Clinical Course.

Category	**Test**	**Result**	**Units**
CBC with differential	WBC	4.74	
	Hgb	111	
	PLT	50	
Inflammatory Markers	Procalcitonin (PCT)	47	
	ESR	25	mm/hr
	CRP	174	mg/L
	Ferritin	1268	ng/mL
Cardiac Enzymes	CK-MB	48.7	U/L
	CK	1830	U/L
	LDH	714	U/L
Coagulation Profile	PT	14.1	sec
	INR	1.27	ratio
	PTT	36.4	sec
Electrolytes	Sodium	117	mmol/L
	Potassium	4.5	mmol/L
	Chloride	86	mmol/L
	Calcium	1.78	mmol/L
	Adjusted Ca	2.14	mmol/L
	Magnesium	0.86	mmol/L
	Phosphorus	1.24	mmol/L

Liver Function Test	Glu	5.2	mmol/L
	ALP	141	U/L
	AST	169	U/L
	ALT	66	U/L
	Albumen	22	g/L
	GGT	70.8	U/L
	Total Protein	44	g/L
	T BIL	19.6	μmol/L
Lipids	Triglyceride	1.41	mmol/L
	Cholesterol	2.1	mmol/L
Renal Profile	Creatinine	64	umol /L
	BUN	13	Mmol/L
Note: WBC: White Blood Cell Count; Hgb: Hemoglobin; PLT: Platelet Count; PCT: Procalcitonin; ESR: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein; CK-MB: Creatine Kinase-MB; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; PT: Prothrombin Time; INR: International Normalized Ratio; PTT: Partial Thromboplastin Time; ALP: Alkaline Phosphatase; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; GGT: Gamma-Glutamyl Transferase; T BIL: Total Bilirubin; BUN: Blood Urea Nitrogen			

Table 2: Laboratory Findings on Admission.

Category	**Test**	**Result**	**Units**
CBC with differential	WBC	16.2	
	Hgb	88	
	PLT	235	
Inflammatory Markers	Procalcitonin (PCT)	0.37	
	ESR	16	mm/hr
	CRP	15	mg/L
	Lactate	0.9	mmol/L
Coagulation Profile	PT	14.9	sec
	INR	1.39	ratio
	PTT	30	sec
Electrolytes	Sodium	141	mmol/L
	Potassium	4.2	mmol/L
	Chloride	103	mmol/L
	Calcium	1.9	mmol/L
	Adjusted Ca	2.2	mmol/L
	Magnesium	0.87	mmol/L
	Phosphorus	1.42	mmol/L
Liver Function Test	Glu	6.3	mmol/L
	Albumen	25	g/L

Renal Profile	Creatinine	36	umol /L
	BUN	6.7	Mmol/L

Note: WBC: White Blood Cell Count; Hgb: Hemoglobin; PLT: Platelet Count; PCT: Procalcitonin; ESR: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein; CK-MB: Creatine Kinase-MB; CK: Creatine Kinase; LDH: Lactate Dehydrogenase; PT: Prothrombin Time; INR: International Normalized Ratio; PTT: Partial Thromboplastin Time; ALP: Alkaline Phosphatase; AST: Aspartate Aminotransferase; ALT: Alanine Aminotransferase; GGT: Gamma-Glutamyl Transferase; T BIL: Total Bilirubin; BUN: Blood Urea Nitrogen

Table 3: Laboratory Findings on Admission.

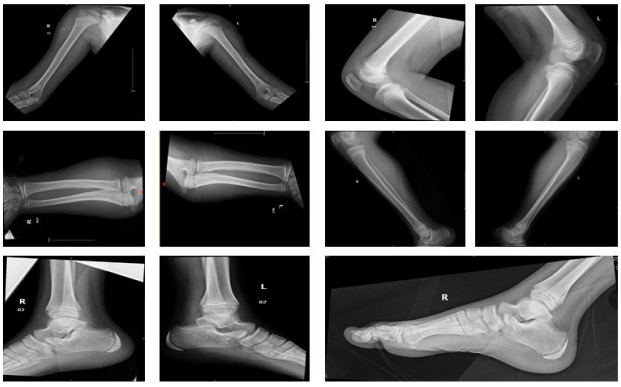

Figure 7: Initial X-rays of Extremities.

Diagnosis and therapeutic interventions

Based on the clinical presentation, laboratory findings, and imaging, a presumptive diagnosis of multifocal necrotizing fasciitis was made and the patient was emergently taken to the operating room within 24 hours of admission for extensive surgical debridement of all necrotic lesions. Intraoperatively, significant purulent discharge was encountered in all affected areas. Tissue samples were obtained for culture and histopathological analysis. Broad-spectrum intravenous antibiotic therapy was initiated empirically, covering MRSA, with piperacillin-tazobactam, clindamycin, and linezolid.

Follow-up and outcomes

Following surgery, the patient developed septic shock requiring vasopressor support and mechanical ventilation. Blood cultures subsequently grew MRSA, prompting adjustments to the antibiotic regimen. Repeat imaging studies and further workup revealed bilateral pulmonary cavitary lesions concerning septic emboli and left distal femur osteomyelitis. A pan-CT scan of the abdomen and pelvis demonstrated multiple intramuscular abscesses. The patient underwent additional surgical debridements of the identified abscesses.

The initial histopathological analysis of fascia from the left thigh revealed viable soft tissue, while fascia from the right forearm demonstrated necrotic skeletal muscle with bacterial colonies, confirming the diagnosis of MRSA necrotizing fasciitis.

Over the ensuing month, the patient underwent multiple (eight) additional surgical procedures for ongoing wound debridement by the plastic surgery team (Figure 8). Two subsequent procedures involved wound irrigation and closure using the vessel loop technique. The pediatric infectious disease team recommended a 6-week course of vancomycin and a 7-day course of ceftazidime for resolving tracheitis.

During this period, the patient was successfully weaned from sedation and mechanical ventilation. He progressed to high-flow nasal cannula oxygen therapy, then regular nasal cannula, and ultimately weaned to room air. An extended-length right internal jugular central venous catheter was replaced with a peripherally inserted central catheter (PICC) for prolonged antibiotic administration.

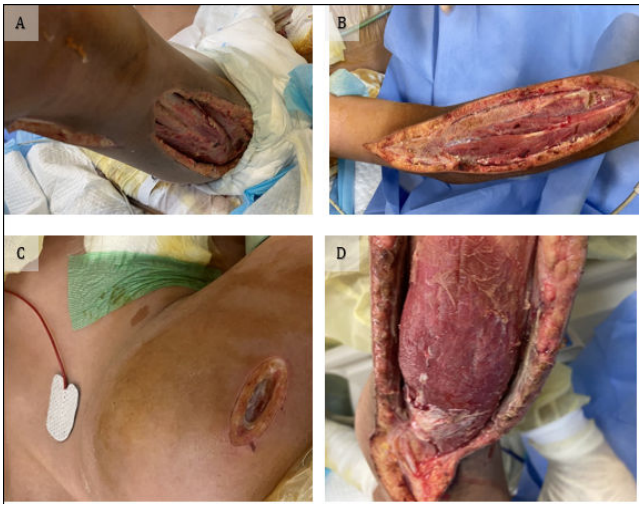

Figure 8: Post-operative wound appearances following incision and drainage (I&D) procedures. A: Gluteal wound after I&D; B: Right arm wound after I&D; C: Left arm wound after I&D; Panel D: Left lower leg wound after I&D.

Following a month of hospitalization, On the 40th hospital day, the patient experienced sudden cardiac arrest requiring prolonged cardiopulmonary resuscitation. Subsequent bronchoscopy revealed severe inflammation with granulation tissue obstructing nearly 70% of the endotracheal tube lumen, consistent with bacterial tracheitis (Figure 9). This critical airway compromise was attributed to the prolonged intubation and presumed ascending bacterial colonization.

Cultures of the tracheal pseudo-membrane and BAL confirmed pseudomonas aeruginosa growth, sensitive to ceftazidime and ciprofloxacin, and resistance to carbapenems. The patient's antibiotic regimen was adjusted accordingly, with ceftazidime continued for presumed ongoing tracheitis and vancomycin. The Tracheal mucosa biopsy histopathology showed acute inflammation and ulceration. A tracheal stent was placed bronchoscopically to secure the airway. The patient's condition stabilized, and he was successfully extubated a few days later.

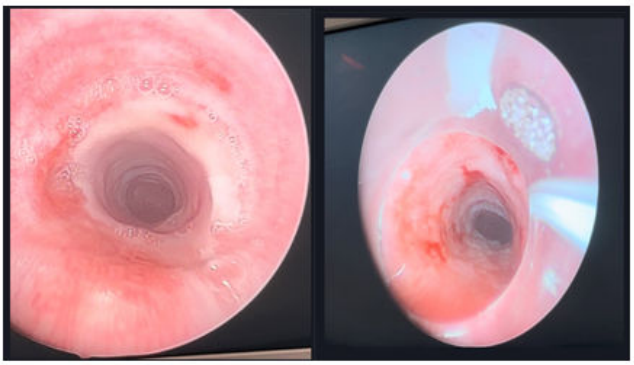

Figure 9: Bronchoscopy image demonstrating severe inflammation.

The patient made significant progress in rehabilitation with physical and occupational therapy. He received nutritional support, and his wounds progressively healed. However, on the 59th hospital day, he developed another 2 episodes of cardiac arrest, briefly lasting 2 and 5 minutes respectively, with similar clinical presentation and presumed airway compromise. Urgent bronchoscopy revealed a partially sloughed segment of the pseudo-membrane obstructing the airway, necessitating debridement, multiple DLBs, and dilation of the tracheal stenosis (Figure 10). The patient's clinical course was further complicated by the development of keloid scars at the surgical sites.

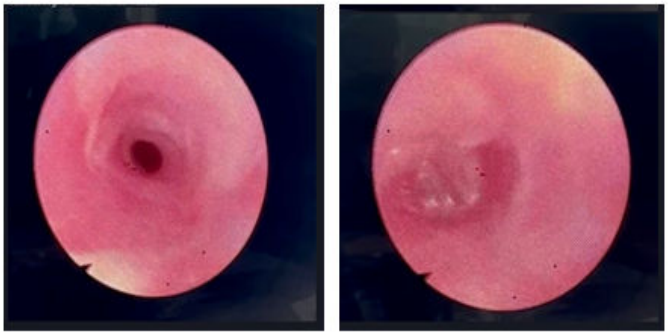

Figure 10: View of the trachea obtained during DLB, demonstrating a pseudo-membrane originating from the sixth tracheal ring, causing moderate to severe airway obstruction. Debridement performed.

Given this and his history of recurrent infections, a workup for rheumatologic and immunologic etiologies was undertaken. Extensive investigations, including rheumatologic workup (negative for proteinase 3 and myeloperoxidase- antineutrophil cytoplasmic antibody),Ophthalmology (Ruled out Uveitis & Episcleritis) and immunological workup (including normal immunoglobulin levels, flow cytometry , Immune deficiency Panel , and negative burst test), whole exome sequencing failed to identify a specific underlying condition.

Multiple multidisciplinary team meetings involving general pediatrics, pediatric intensive care, otolaryngology, and pulmonology specialists were conducted. Given the recurrent and life-threatening airway compromise secondary to the severe tracheal stenosis, a surgical tracheostomy with an extra-long tracheal tube to bypass the stenotic segment was deemed necessary.

The patient successfully underwent the tracheostomy procedure. Following surgery, his condition remained stable without further episodes of stridor or respiratory distress. He was eventually transferred to the general ward and subsequently discharged home with a tracheostomy in place and a plan for regular follow-up with otolaryngology, and physical/occupational therapy. Repeat lab and echocardiogram prior to discharge demonstrated normal findings.

Discussion

This case report presents a complex and multifaceted clinical scenario of multifocal NF in a previously healthy child, complicated by the development of life-threatening tracheal stenosis. The case highlights the aggressive nature of MRSA-induced NF, emphasizing the critical need for early recognition and aggressive management to prevent devastating sequelae.

The occurrence of tracheal stenosis as a late complication of NF is an exceptionally rare and challenging clinical entity. This case underscores the importance of vigilant airway monitoring in patients recovering from NF, as well as the need for prompt and decisive intervention when airway compromise is identified. The multidisciplinary approach employed in this case, involving surgical, infectious disease, critical care, and pulmonary teams, was instrumental in managing this complex clinical trajectory.

While this case report provides valuable insights into the clinical presentation, diagnostic challenges, and therapeutic approaches to NF, it is essential to acknowledge its limitations. As a single case report, the generalizability of findings to the broader pediatric population is restricted. Prospective studies with larger sample sizes are warranted to establish the incidence, risk factors, and prognostic indicators associated with NF and its complications.

The etiology of the patient's keloid scar formation and susceptibility to recurrent infections remains unclear despite extensive investigations. While a specific underlying immunologic or rheumatologic condition could not be identified, further evaluation may be warranted in the future.

Despite these limitations, this case report contributes significantly to the existing literature by emphasizing the potential severity and complexity of pediatric NF. The detailed description of the patient's clinical course, diagnostic workup, and therapeutic interventions offers valuable insights for clinicians managing similar cases. By highlighting the challenges and successes encountered in this case, we aim to stimulate further research and improve patient outcomes.

Conclusion

This case report underscores the critical importance of early recognition, aggressive management, and a multidisciplinary approach in the care of pediatric patients with NF. The development of tracheal stenosis as a late complication

emphasizes the need for ongoing vigilance and prompt intervention to prevent fatal outcomes. Further research is essential to elucidate the pathophysiology, identify prognostic factors, and develop novel therapeutic strategies for this devastating condition.

References

1. Goh T, Goh LG, Ang CH, et al. Early diagnosis of necrotizing fasciitis. Br J Surg 2014;101(1): 119-125.
2. Chambers HF, DeLeo FR. Waves of resistance: *Staphylococcus aureus* in the antibiotic era. Nat Rev Microbiol 2009; 7:629-641.
3. Olsen RJ, Burns KM, Chen L, et al. Severe necrotizing fasciitis in a human immunodeficiency virus-positive patient caused by methicillin-resistant *Staphylococcus aureus*. J Clin Microbiol. 2008;46(3):1144-1147.
4. Miller LG, Perdreau RF, Rieg G, et al. Necrotizing fasciitis caused by community-associated methicillin-resistant *Staphylococcus aureus* in Los Angeles. N Engl J Med. 2005; 352(14):1445-1453.
5. Szilagyi J, Kuester V, Reznicek J. Pediatric necrotizing fasciitis: Current concept review. J Pediatr Orthop Soc North Am 2023; 5(4).
6. Yamamoto T, Nishiyama A, Takano T, et al. Community-acquired methicillin-resistant *Staphylococcus aureus*: Community transmission, pathogenesis, and drug resistance. J Infect Chemother 2010; 16(4):225-54.
7. Khalid M, Junejo S, Mir F. Invasive community acquired methicillin-resistant *Staphylococcal aureus* (CA-MRSA) infections in children. J Coll Physicians Surg Pak 2018; 28(9):S174-S177.
8. Tancevski A, Bono K, Willis L, et al. Necrotizing fasciitis in a pediatric orthopedic population. Orthopedics 2013; 36(6):e741-e745.
9. Necrotizing fasciitis (flesh-eating disease). Centers for Disease Control and Prevention 2023.
10. Jain A, Meher R, Sharma R. MRSA causing necrotizing fasciitis in 18-month-old boy. Ear Nose Throat J 2023;102(1):NP3-NP4.

Corresponding Author:

Alharbi Abdullah

Department of Pediatric intensive care;

King Abdullah specialized Children hospital, National Guard Hospital

Riyadh, Saudia Arabia

Email: AlharbIAb91@mngha.med.sa