Cureus
Part of SPRINGER NATURE

Open Access Case Report

Severe Respiratory Syncytial Virus Bronchiolitis Complicated by Bilateral Massive Pleural Effusions in a Previously Healthy Infant: A Case Report

Fadl Abdulaal [1], Dima Bani Issa [2], Dua' N. Samara [3], Loay Abdulsamad [4], Fadi Toonsi [5]

1. Department of Neonatology, Dr Sulaiman Al Habib Medical Group (HMG), Jeddah, SAU 2. Pediatric Intensive Care Unit, Dr Sulaiman Al Habib Medical Group (HMG), Jeddah, SAU 3. Department of Pediatric Pulmonology, University of Colorado Anschutz Medical Campus, Aurora, USA 4. Pediatric Intensive Care Unit, King Faisal Specialist Hospital and Research Centre, Jeddah, SAU 5. Department of Radiology, King Abdulaziz University, Jeddah, SAU

Corresponding author: Fadl Abdulaal, fmt681@gmail.com

Review began 03/09/2026
Review ended 04/26/2026
Published 05/03/2026

© Copyright 2026
Abdulaal et al. This is an open access article distributed under the terms of the Creative Commons Attribution License CC-BY 4.0., which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

DOI: 10.7759/cureus.108167

Abstract

Respiratory syncytial virus (RSV) bronchiolitis is one of the leading causes of hospitalization in infants, typically presenting with mild-to-moderate respiratory distress. Severe complications such as apnea, respiratory failure, and pneumothorax are known, but massive pleural effusions remain an exceptionally rare manifestation.

We report a case of an eight-week-old previously healthy Saudi female infant presenting with severe RSV bronchiolitis. The patient developed progressive respiratory failure requiring mechanical ventilation, re-intubation, and circulatory support. Notably, she developed bilateral massive pleural effusions necessitating urgent bilateral pigtail catheter insertion. Pleural fluid was sterile and transudative in nature. The infant showed significant clinical improvement following drainage and supportive care, with eventual full recovery.

This case highlights an unusual but severe complication of RSV bronchiolitis. It underscores the need for clinicians to consider pleural effusion in infants with RSV who deteriorate despite supportive measures, even in the absence of bacterial infection.

Categories: Pediatrics, Infectious Disease, Pulmonology
Keywords: bronchiolitis, mechanical ventilation, pleural effusion, respiratory failure, rsv

Introduction

Respiratory syncytial virus (RSV) bronchiolitis is one of the leading causes of hospitalization in infants, accounting for a substantial proportion of acute lower respiratory tract infections and an estimated 3.6 million RSV-associated hospitalizations in children under five years of age worldwide each year [1]. Most infants experience a self-limited course, although severe complications such as respiratory failure, apnea, and the need for escalating respiratory support are well described, particularly in those with underlying comorbidities [2,3].

RSV bronchiolitis primarily affects the small airways, and significant pleural involvement is not a typical feature of viral lower respiratory tract infections. Pleural effusion in this context is therefore considered uncommon, with only a limited number of cases reported in the literature. Reports of large, bilateral pleural effusions in the absence of bacterial superinfection are particularly rare [4-6].

We report a case of an otherwise healthy infant with severe RSV bronchiolitis who developed large bilateral pleural effusions without evidence of bacterial infection. This case highlights an unusual and poorly characterized complication and aims to contribute to the understanding of potential inflammatory and mechanical mechanisms in viral-induced pleural effusions.

Case Presentation

An eight-week-old Saudi female infant, who was born at term (birth weight ~2.5kg) and had an unremarkable neonatal course, initially presented with a two-week history of rhinorrhea and mild cough. She remained well until one day before her first presentation, when she developed noisy breath sounds and increased work of breathing, characterized by rapid, labored breathing, perioral cyanosis, and hypoactivity; notably, she was afebrile.

On admission, the infant appeared sick and was in moderate-to-severe respiratory distress. Vital signs were as follows: heart rate 154bpm, respiratory rate 68 breaths/min, blood pressure 97/55mmHg, and oxygen

saturation of 98% on HFNC at 8L/min with an FiO_2 of 50%. She was alert (GCS 15/15) with an open, soft, and flat anterior fontanelle and preserved tone. Cardiovascular examination revealed tachycardia with normal heart sounds and no murmurs, while pulmonary examination demonstrated tachypnea with subcostal retractions, audible wheezing, and bilateral crepitations with decreased air entry. Abdominal and skin examinations were unremarkable aside from mild mottling. Initial labs were significant for mild leukocytosis WBC 15.3×10^3/µL, with 25% neutrophils and 58.9% lymphocytes, and elevated CRP 10.5 mg/L (Table *1*). A chest radiograph was obtained on admission (Figure *1A*).

Variable	Day 1	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 14	Reference range
WBCs (× 10³/μL)	15.3	8.2	9.3	6.4	13.8	12.9	8.1	14.4	6-18
Neutrophils%	25.8	31	65.5	50	31.9	32.6	27.7	23	25-60
Lymphocytes%	58.9	45	16.9	34	56.2	57.8	59	67	20-70
Hb (g/dl)	10.9	10.1	10.7	11.2	11.3	10.3	8.9	9.9	12.5-20.5
Plt (× 10³/μL)	505	366	537	370	385	349	403	881	150-450
BUN	3.4	1.1	3.9	2.2	2.7	2.7	1.3	2.9	1.2-6
Creatinine (μmol/L)	27.6	25.3	43.6	33.9	33.5	27.7	21.2	21.9	15-37.1
CRP (mg/L)	10.5	12.4	9.6	4	5.6	29.8	28.8	1.8	0-5
Procalcitonin (ng/mL)	1.32	0.21	2.5	-	1.06	0.40	-	-	> 0.1 ng/ml, no systemic inflammatory response 0.1 – 0.49, minor or no significant inflammatory response. 0.5 – 1.99, moderate risk for progression to severe systemic infection. 2.0 – 9.99, severe systemic inflammatory response, most likely due to sepsis, unless other causes are known. ≥ 10.0, high likelihood of severe sepsis or septic shock.
pH	-	-	7.1	-	-	-	-	-	7.32-7.42
pCO2 (mmHg)	-	-	81	-	-	-	-	-	38-52
HCO3 (mmol/L)	-	-	20	-	-	-	-	-	20-26
D-dimer (ng/mL)	-	-	947	-	-	-	-	-	< 500
Serum LDH (U/l)	-	-	193	-	-	-	-	-	180-360
Pleural Protein (g/dL)	-	-	0.37	-	-	-	-	-	< 2
Pleural LDH (IU/L)	-	-	9.42	-	-	-	-	-	
Pleural RBCs (cells/μL)	-	-	100	-	-	-	-	-	
Pleural WBCs (cells/μL)	-	-	3.75	-	-	-	-	-	
ALT (IU/L)	-	-	36	40	-	-	42	49	5-55
AST (IU/L)	-	-	43	53	-	-	51	61	5-34
Albumin (g/l)	-	-	33	29	-	-	26	33	38-54
Total protein (g/l)	-	-	50	45	-	-	41	50	44-76

TABLE 1: Laboratory findings during admission

WBCs: white blood cells; Hb: hemoglobin; Plt: platelets; BUN: blood urea nitrogen; CRP: C-reactive protein; LDH: lactate dehydrogenase; RBCs: red blood cells; ALT: alanine aminotransferase; AST: aspartate aminotransferase

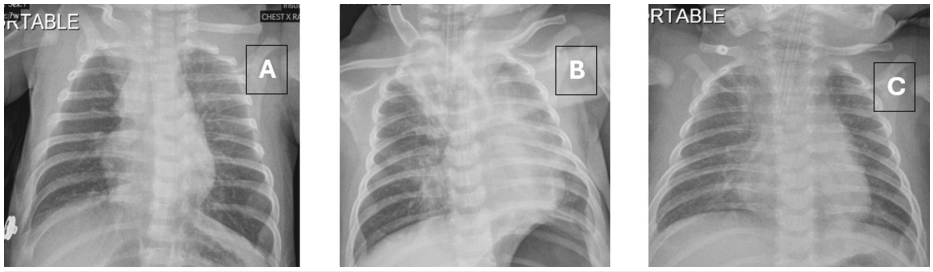


FIGURE 1: A: At admission, showing bilateral hyper-inflated lungs and hyper-accentuated broncho-vascular markings, consistent with acute bronchiolitis. B: After the first intubation, showing right upper lobe collapse and left-sided opacities with appropriate endotracheal tube position. C: Before the first extubation, showing clear lung fields with satisfactory lung inflation and no consolidation or effusion.

The infant was initially managed on HFNC with flow of 8L/min; however, despite acceptable saturations, her respiratory distress persisted. She soon developed apnea with desaturations. Nebulized bronchodilators, chest physiotherapy, and airway suctioning were administered, and respiratory support was escalated to nCPAP (initial PEEP 7cm H_2O, increased to 8cm H_2O) as the FiO_2 requirement rose to 80%. A dexmedetomidine infusion was initiated to achieve sedation. Due to persistent severe distress, rising pCO_2, and high oxygen requirements, the patient was intubated. Post-intubation chest X-ray (Figure *1B*) confirmed proper ETT position, right upper lobe collapse, and left-sided opacities. Sedation was maintained with fentanyl and midazolam, and dexmedetomidine was discontinued. Blood, tracheal aspirate, and urine cultures were obtained. A respiratory viral panel by polymerase chain reaction (PCR) was positive for RSV.

Few hours later, the patient developed hypotension managed with three 0.9% sodium chloride solution boluses (15ml/kg each) and a transient adrenaline infusion. Midazolam was held, and a low-dose ketamine infusion was started. The epinephrine infusion was discontinued a few hours later. Over the next several days, ventilator settings were gradually weaned in accordance with serial VBGs. All cultures returned negative.

After five days of mechanical ventilation (Day 6 of admission), a chest X-ray was obtained (Figure *1C*), then the infant was extubated to HFNC (8L/min, FiO_2 60%) with stable saturation and equal bilateral air entry.

However, six hours post-extubation, she acutely redeveloped marked respiratory distress with bilateral wheezing and prolonged expiratory phases. Despite intensive bronchodilator therapy and non-invasive ventilation, her respiratory failure recurred, necessitating re-intubation and CPAP/BiPAP trials with mild sedation; the patient's distress persisted. VBG: pH 7.1, pCO_2 81 mmHg, HCO_3^- 20 mmol/L. So, she required urgent re-intubation and mechanical ventilation with high settings.

Approximately 18 hours after re-intubation, the infant experienced recurrent bradycardia and desaturation episodes requiring bag-mask ventilation. Signs of poor perfusion and anuria (no urine output for 6 hours) were noted. Fluid resuscitation was provided with three 0.9% sodium chloride solution boluses (20 ml/kg, then 15 ml/kg twice), and a continuous epinephrine infusion was initiated. Repeat cultures (blood, urine, and tracheal aspirate) were obtained.

In the setting of ongoing clinical instability and concern for evolving sepsis despite initially negative microbiological results, empirical antimicrobial therapy was initiated with intravenous meropenem (30 mg/kg/dose every eight hours) in combination with vancomycin (15 mg/kg/dose every six hours), while a repeat respiratory viral panel by PCR remained positive for RSV.

A chest radiograph at this juncture revealed bilateral lung collapse with significant pleural effusions (Figure *2A*). An urgent echocardiogram demonstrated mild left ventricular hypertrophy with preserved systolic function, no pulmonary hypertension, and a small pericardial effusion. Bedside chest ultrasonography confirmed bilateral large-volume pleural effusions with internal echoes. An abdominal ultrasound revealed gallbladder wall thickening with edema and pericholecystic fluid, minimal free fluid in the right iliac fossa, and bilateral inguinal lymphadenopathy with preserved morphology.

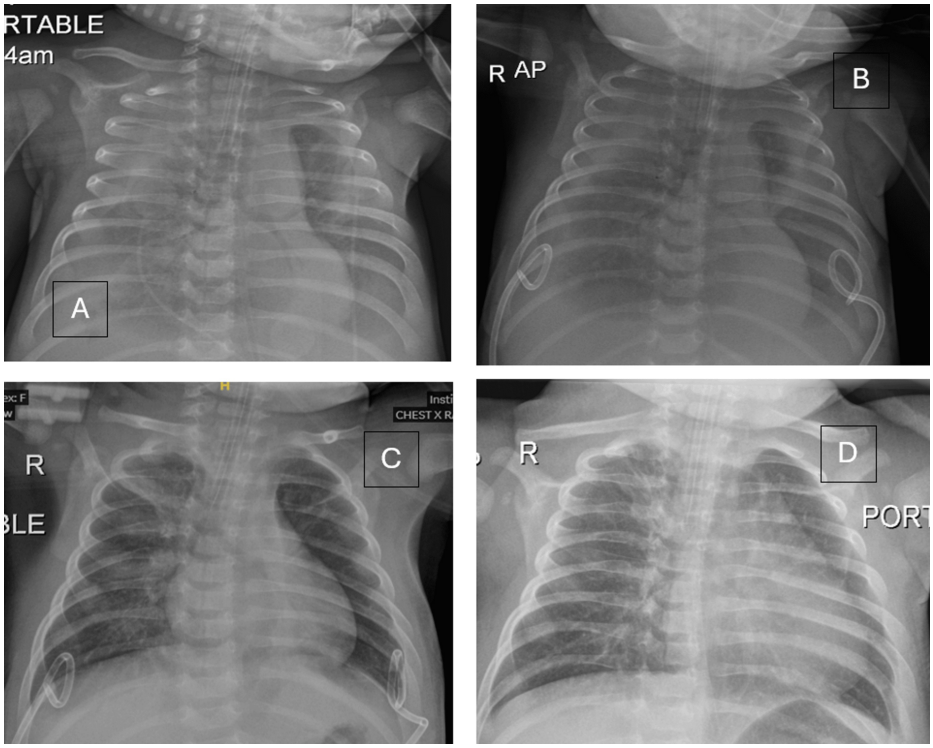


FIGURE 2: A: Chest radiograph demonstrating marked bilateral pleural effusions with blunting of both costophrenic angles and diffuse lung opacities, worse on the right. B: After insertion of bilateral pigtail catheters, showing early regression of pleural fluid but persistent reduced lung volumes and underlying collapse/consolidation. C: Before removal of pigtails, showing good lung inflation with clear costophrenic angles, indicating near-complete resolution of effusions. D: After removal of pigtails, demonstrating maintained clear costophrenic angles bilaterally and improved aeration.

At the time of effusion detection, creatinine had risen from 25.3 to 43.6 µmol/L, with D-dimer 947 ng/mL and procalcitonin 2.5 ng/mL, prompting concern for evolving multi-organ dysfunction and justifying escalation of invasive drainage. Ultrasound-guided bilateral pigtail catheters were inserted at the bedside (Figure *2B*). To minimize the risk of re-expansion pulmonary edema, pleural fluid was aspirated in aliquots of 10 ml/kg from each side hourly over three hours, after which both pigtail catheters were connected to an underwater seal. The total drainage was 230ml on the right and 200ml on the left (Figure *2C*). Following chest tube placement, the epinephrine infusion was discontinued, and urine output improved with additional intravenous fluids and diuretics (furosemide). Pleural fluid analysis revealed a clear, colorless fluid with a white blood cell count of 3.75 cells/µL, red blood cell count of 100 cells/µL, lactate dehydrogenase (LDH) of 9.42 IU/L, and protein of 0.37 g/dL, with corresponding serum protein of 50 g/L and LDH of 193 IU/L. The pleural fluid protein-to-serum protein ratio, pleural fluid LDH-to-serum LDH ratio, and absolute pleural LDH level were all well below the exudative thresholds defined by Light's criteria, confirming a transudative effusion.

Cultures of pleural fluid, as well as repeat blood, urine, and tracheal aspirate cultures, remained negative. Vancomycin was discontinued after three days, and Meropenem was continued for a total of seven days. Chest tubes were removed after three days (Figure *2D*), and serum creatinine returned to baseline over the next 48 hours.

She was gradually weaned off ventilatory support based on serial VBGs and clinical tolerance (extubated again on day 11 of admission) and transitioned to room air over the following several days (Figure *3*). Table *1* summarizes the laboratory findings during the course of admission, and Figure *4* summarizes key clinical events as a timeline.

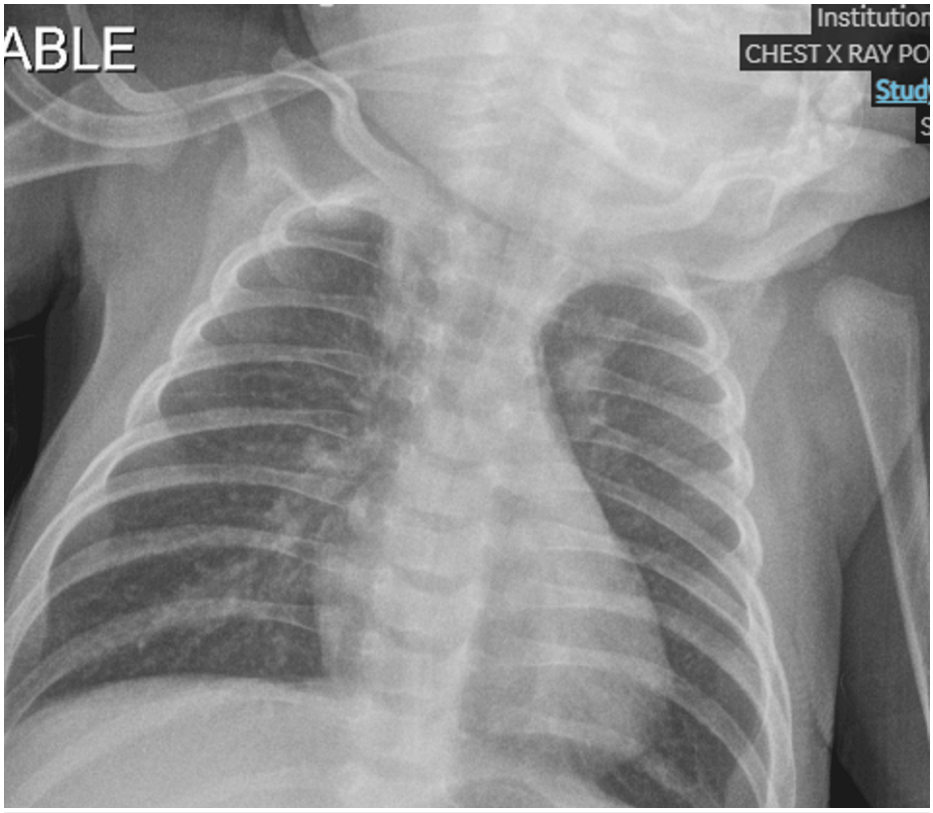


FIGURE 3: Chest radiograph after the second extubation showing well-expanded lungs without pleural effusion or new consolidation.

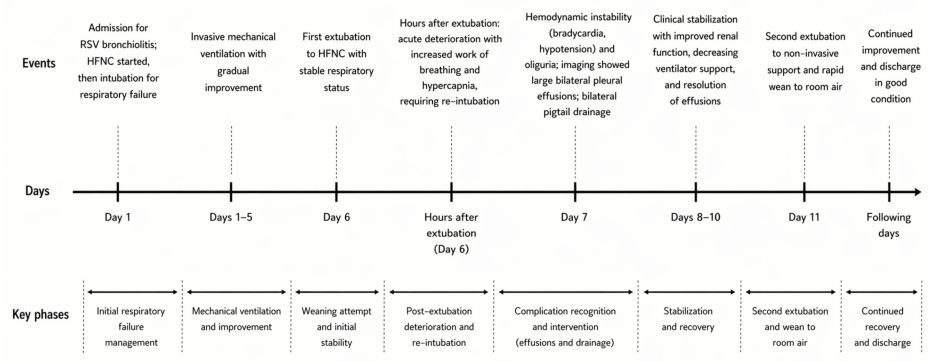


FIGURE 4: Clinical timeline of the infant with severe RSV bronchiolitis, highlighting key respiratory and hemodynamic events, onset of large bilateral pleural effusions, and timing of bilateral pigtail drainage and recovery.

RSV: Respiratory syncytial virus

Discussion

This case represents a previously healthy eight-week-old infant who developed severe RSV bronchiolitis complicated by bilateral massive pleural effusions, an exceptionally rare manifestation in pediatric viral respiratory infections. The clinical course was characterized by progressive respiratory failure, cardiovascular instability, and eventual recovery following bilateral pleural drainage and intensive supportive care. The absence of bacterial pathogens in all cultures, normal echocardiography, and persistent

RSV positivity strongly suggest a direct viral etiology for the pleural effusions.

Pleural effusion is a well-recognized complication of bacterial pneumonia, heart failure, and systemic diseases; however, it remains a rare complication of viral bronchiolitis, particularly in previously healthy infants [4,6]. There are scattered reports indicating that RSV alone can induce severe complications, including pneumothorax and multi-organ failure, suggesting that RSV pathophysiology is broader and potentially more severe than previously appreciated, even in the absence of comorbidities [7,8], which is more consistent with the case of our patient.

The precise pathophysiological mechanisms underlying the development of pleural effusions in viral bronchiolitis remain incompletely understood; however, several plausible hypotheses have been proposed. Increasing evidence supports a cytokine-mediated inflammatory pathway [5]. RSV infection induces a robust proinflammatory response characterized by elevated levels of key cytokines, including interleukin (IL)-1β, IL-6, IL-8, tumor necrosis factor-α (TNF-α), and interferon-γ (IFN-γ) [9, 10]. Studies in RSV-infected infants demonstrate significantly increased concentrations of these mediators in respiratory secretions compared to healthy controls and an association with poor prognosis [11,12]. Animal studies of viral pericarditis have demonstrated that inflammatory injury accompanied by markedly elevated IL-1β and IL-6 in serosal fluid correlates with increased serosal permeability [13]. Given RSV's documented ability to upregulate these same mediators, it is plausible that RSV-associated pleural effusions arise through similar cytokine-driven mechanisms that increase capillary permeability in both visceral and parietal pleura [14]. Further studies are needed to evaluate cytokine levels in pleural fluid in cases of pleural effusion associated with viral bronchiolitis.

In our patient, the development of a transudative pleural effusion, rather than the exudative pattern typically expected with viral inflammation, likely reflects multifactorial pathophysiologic processes observed in critically ill, mechanically ventilated infants with RSV bronchiolitis [14,15]. Aggressive fluid resuscitation to manage hypotension, compounded by elevated central venous pressure from mechanical ventilation, increases the hydrostatic pressure within the systemic capillaries of the parietal pleura, promoting fluid movement into the pleural space. The use of high PEEP further contributed by elevating pleural pressure, reducing lymphatic drainage efficiency, and compressing the thoracic duct, all of which diminish fluid clearance from the pleural space. Additionally, the presence of lung collapse and atelectasis on imaging may have enhanced localized negative pleural pressure, facilitating further fluid accumulation via the “trapped lung” effect, commonly seen in mechanically ventilated patients with poor lung compliance [15,16]. Mechanical ventilation can be challenging in patients with heterogeneous lung disease like severe RSV bronchiolitis [17]. The critical opening pressure is elevated in a non-uniform manner, making optimal PEEP selection challenging and potentially contributing to regional lung collapse and pleural complications [18].

The transudative nature of the effusion in our patient would suggest that in critically ill infants with RSV and mechanical ventilation, systemic and mechanical factors, rather than local inflammatory responses, may dominate in pleural effusion formation. Pleural fluid analyses are still lacking to confirm the protein content in the literature, and all reported cases have relied on ultrasound findings without studying pleural fluid characteristics directly [5].

A landmark study of 69 infants with acute bronchiolitis revealed pleural effusion in 49.2% of cases, detected via thoracic ultrasonography, regardless of disease severity. These effusions, typically small and asymptomatic, showed no significant correlation with clinical outcomes or recovery timelines. The study population included both non-ventilated patients and those with severe bronchiolitis, though mechanical ventilation status was not explicitly analyzed as an independent variable. The high prevalence suggests that pleural effusion constitutes a common but underrecognized feature of viral bronchiolitis pathophysiology. The presence of pleural effusion was significantly more common in patients with underlying risk factors. These findings suggest that while pleural effusion may occur more often than expected in bronchiolitis, clinically significant or symptomatic effusions remain rare and may be limited to those with severe disease or predisposing conditions [5].

The application of lung ultrasonography in viral bronchiolitis has emerged as a revolutionary diagnostic and prognostic method, with rising data supporting its integration into clinical practice while arguing certain limits [19,20]. Given the low overall incidence of clinically significant pleural effusion in RSV bronchiolitis, routine imaging for all patients may not be justified. However, in cases where patients show signs of severe disease or deteriorating cardiopulmonary status, targeted screening using modalities such as chest ultrasound could be beneficial. Current clinical guidelines do not universally recommend such screening, reflecting a gap in our understanding of which patients may benefit most from early detection and intervention. Further research is warranted to determine whether a more proactive imaging strategy could improve outcomes in severe cases of RSV bronchiolitis.

Conclusions

Our case highlights that severe RSV bronchiolitis in previously healthy infants can be complicated by massive bilateral pleural effusions with a transudative profile, likely driven by hemodynamic stress, fluid

resuscitation, and positive-pressure ventilation rather than primary viral pleuritis. This underscores the importance of considering pleural fluid accumulation as a potential contributor to cardiorespiratory instability in critically ill, ventilated infants with RSV, and of integrating pleural-effusion assessment into the management of such patients. Further case series and mechanistic studies are needed to clarify the incidence, triggers, and optimal management of pleural effusions in mechanically ventilated children with RSV bronchiolitis, which may ultimately refine ventilator strategies, fluid management, and monitoring protocols in this vulnerable population.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Fadl Abdulaal, Dima Bani Issa, Dua' N. Samara, Loay Abdulsamad, Fadi Toonsi

Acquisition, analysis, or interpretation of data: Fadl Abdulaal, Dima Bani Issa, Dua' N. Samara

Drafting of the manuscript: Fadl Abdulaal, Dima Bani Issa, Dua' N. Samara

Critical review of the manuscript for important intellectual content: Fadl Abdulaal, Dima Bani Issa, Dua' N. Samara, Loay Abdulsamad, Fadi Toonsi

Supervision: Fadl Abdulaal, Dima Bani Issa, Loay Abdulsamad, Fadi Toonsi

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

References

1. Li Y, Wang X, Blau DM, et al.: Global, regional, and national disease burden estimates of acute lower respiratory infections due to respiratory syncytial virus in children younger than 5 years in 2019: a systematic analysis. Lancet. 2022, 399:2047-64.
2. Kaler J, Hussain A, Patel K, Hernandez T, Ray S: Respiratory syncytial virus: a comprehensive review of transmission, pathophysiology, and manifestation. Cureus. 2023, 15:e36342. 10.7759/cureus.36342
3. Sommer C, Resch B, Simões EA: Risk factors for severe respiratory syncytial virus lower respiratory tract infection. Open Microbiol J. 2011, 5:144-54. 10.2174/1874285801105010144
4. Nestor J, Huggins T, Kummerfeldt C, DiVietro M, Walters K, Sahn S: Viral diseases affecting the pleura. J Clin Virol. 2013, 58:367-73. 10.1016/j.jcv.2013.06.013
5. Yilmaz AE, Koktener A, Celik N, Akca H, Bilici M, Mete E: Frequency of pleural effusion in acute bronchiolitis and its effect on prognosis. Multidiscip Respir Med. 2011, 6:371-6. 10.1186/2049-6958-6-6-371
6. Nascimento-Carvalho CM, Oliveira JR, Cardoso MR, et al.: Respiratory viral infections among children with community-acquired pneumonia and pleural effusion. Scand J Infect Dis. 2013, 45:478-83. 10.3109/00365548.2012.754106
7. Gkentzi D, Dimitriou G, Karatza A: Non-pulmonary manifestations of respiratory syncytial virus infection. J Thorac Dis. 2018, 10:S3815-8. 10.21037/jtd.2018.10.38
8. Mishra R, Rup AR, Jain MK, Sahoo B, Patnaik S: A rare case of respiratory syncytial virus bronchiolitis with spontaneous bilateral pneumothorax in an infant. J Integrative Med Res. 2024, 2:245-7. 10.4103/jimr.jimr_46_24
9. Russell CD, Unger SA, Walton M, Schwarze J: The human immune response to respiratory syncytial virus infection. Clin Microbiol Rev. 2017, 30:481-502. 10.1128/CMR.00090-16
10. Vázquez Y, González L, Noguera L, González PA, Riedel CA, Bertrand P, Bueno SM: Cytokines in the respiratory airway as biomarkers of severity and prognosis for respiratory syncytial virus infection: an update. Front Immunol. 2019, 10:1154. 10.3389/fimmu.2019.01154
11. Bennett BL, Garofalo RP, Cron SG, Hosakote YM, Atmar RL, Macias CG, Piedra PA: Immunopathogenesis of respiratory syncytial virus bronchiolitis. J Infect Dis. 2007, 195:1532-40. 10.1086/515575
12. Moreno-Solís G, Torres-Borrego J, de la Torre-Aguilar MJ, Fernández-Gutiérrez F, Llorente-Cantarero FJ, Pérez-Navero JL: Analysis of the local and systemic inflammatory response in hospitalized infants with respiratory syncitial virus bronchiolitis. Allergol Immunopathol (Madr). 2015, 43:264-71. 10.1016/j.aller.2014.02.002
13. Nakayama Y, Kishimoto C, Shioji K, Sasayama S: Significance of pericardial cytokines in giant cell myocarditis in rats--pathological comparison to viral myocarditis in mice. Jpn Circ J. 2000, 64:977-81.

10.1253/jcj.64.977

14. Ferreiro L, Toubes ME, Suárez-Antelo J, Rodríguez-Núñez N, Valdés L: Clinical overview of the physiology and pathophysiology of pleural fluid movement: a narrative review. ERJ Open Res. 2024, 10:10.1183/23120541.00050-2024
15. Bediwy AS, Al-Biltagi M, Saeed NK, Bediwy HA, Elbeltagi R: Pleural effusion in critically ill patients and intensive care setting. World J Clin Cases. 2023, 11:989-99. 10.12998/wjcc.v11.i5.989
16. Malbrain ML, Pelosi P, De Laet I, Lattuada M, Hedenstierna G: Lymphatic drainage between thorax and abdomen: please take good care of this well-performing machinery.... Acta Clin Belg. 2007, 62:152-61. 10.1179/acb.2007.62.s1.020
17. Pickles RJ, DeVincenzo JP: Respiratory syncytial virus (RSV) and its propensity for causing bronchiolitis. J Pathol. 2015, 235:266-76. 10.1002/path.4462
18. Sood S, Ganatra HA, Perez Marques F, Langner TR: Complications during mechanical ventilation-a pediatric intensive care perspective. Front Med (Lausanne). 2023, 10:1016316. 10.3389/fmed.2023.1016316
19. Basile V, Di Mauro A, Scalini E, et al.: Lung ultrasound: a useful tool in diagnosis and management of bronchiolitis. BMC Pediatr. 2015, 15:63. 10.1186/s12887-015-0380-1
20. Özkaya AK, Yilmaz HL, Kendir ÖT, Gökay SS, Eyüboğlu İ: Lung ultrasound findings and bronchiolitis ultrasound score for predicting hospital admission in children with acute bronchiolitis. Pediatr Emerg Care. 2020, 36:e135-42. 10.1097/PEC.0000000000001705