Jurnal Impresi Indonesia (JII)
p-ISSN: 2828-1284 e-ISSN: 2810-062x
website: https: //rivierapublishing.id/JII/ index.php / jii /index

When Dyspnea Is More Than Pneumonia: Interwoven Mechanisms Of Autoimmune Multiorgan Disease In An Adolescent—A Case Report

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Keywords

Keywords: Autoimmune disease, dyspnea, nephritis, pulmonary hypertension, anemia

Abstract

Dyspnea in adolescents is often underestimated as a simple respiratory infection, yet it may represent the fairst sign of complex autoimmune multiorgan disease. This report highlights the autoimmune mechanisms underlying dyspnea to promote early recognition of autoimmune disease in adolescents. An 18-year-old female presented with acute worsening of chronic dyspnea. She was tachypneic hypertensive, hypoxemic, and severe anemia. Examination revealed pallor, edema, basal fine crackles, and gallop. Laboratory findings showed renal impairment with proteinuria and hematuria. The albumin was normal and blood gas indicated respiratory alkalosis with compensatory metabolic acidosis. Infectious work-up revealed yeast-like fungi and mixed gram-positive cocci/gram-negative bacilli, while GeneXpert MTB was negative. Chest radiograph demonstrated pulmonary edema with pneumonia. Electrocardiography showed sinus tachycardia 119 bpm with right axis deviation and right ventricular hypertrophy. Echocardiography revealed right atria and ventricle enlargement, moderate tricuspid regurgitation, mild pericardial effusion, intermediate pulmonary hypertension, and preserved EF 64%. ANA test was positive, but performed only at discharge before referral for definitive immunological workup. The patient's life-threatening multiorgan dyspnea could be concluded as four major mechanisms, such as : 1) Renal & Volume Overload; 2) Cardiopulmonary Vasculopathy; 3) Autoimmune and Infectious Pulmonary Damage; 4) Severe Anemia. The profound multiorgan involvement and severe systemic inflammation provided a strong clinical rationale for highly active systemic autoimmune disease (SAID), most probable being Systemic Lupus Erythematosus. This case illustrates how adolescents' dyspnea with complicated systemic symptoms deserves an early diagnosis of autoimmune disease without waiting for infectious exclusion, to prevent the devastating consequences associated with diagnostic latency and delayed targeted intervention. As life-threatening dyspnea in an adolescent may be the catastrophic initial presentation of Probable Systemic Lupus Erythematosus.

INTRODUCTION

Dyspnea in the adolescent population represents a common clinical complaint, with an estimated prevalence of 5-15% in this age group globally (Stinson et al., 2018). While most cases are attributed to benign causes such as asthma or transient respiratory infections, emerging evidence suggests that 2-5% of persistent dyspnea cases in adolescents may be the initial manifestation of serious systemic diseases, including autoimmune disorders (Chen et al., 2020). The misdiagnosis rate of systemic autoimmune diseases (SAID) in adolescents remains alarmingly high at 30-50%, with an average diagnostic delay of 6-12 months from symptom onset to definitive diagnosis (Bertsias et al., 2019). This diagnostic latency is associated with significantly worse outcomes, including increased organ damage (odds ratio 3.2, 95% CI 2.1-4.8) and mortality rates up to 15% in severe cases (Livingston et al., 2019). The consequences of delayed recognition are particularly severe in Systemic Lupus Erythematosus (SLE), where early aggressive disease in adolescents is associated with more frequent renal, neurological, and hematological involvement compared to adult-onset disease (Hiraki et al., 2018).

Dyspnea (shortness of breath) in the adolescent population is a common clinical complaint often quickly attributed to transient or benign causes, predominantly acute respiratory infections like community-acquired pneumonia (CAP) (Qu et al., 2015). However, when acute respiratory distress is rapidly progressive, refractory to initial supportive measures, or accompanied by striking systemic features, the initial infectious diagnosis may critically mask a severe underlying systemic pathology.1 (Jain et al., 2018)

The global incidence of SLE ranges from 1 to 10 per 100,000 person-years, with adolescent-onset SLE (age 10-19 years) accounting for approximately 15-20% of all SLE cases (Yen & Singh, 2018). Adolescent-onset SLE demonstrates distinct epidemiological characteristics, with female-to-male ratios of 4-5:1 compared to 9:1 in adult-onset disease, and shows particular predilection among Asian, African, and Hispanic populations (Smith et al., 2019). Recent systematic reviews have identified that respiratory manifestations occur in 50-70% of SLE patients during their disease course, yet dyspnea as the presenting symptom occurs in only 5-10% of cases, making initial recognition particularly challenging (Mittoo et al., 2020). The multiorgan nature of SLE presentations contributes to diagnostic complexity, with studies showing that patients presenting with three or more organ systems involved have a 60% rate of initial misdiagnosis, most commonly as sepsis, pneumonia, or heart failure (Pons-Estel et al., 2021).

The confluence of severe, unexplained respiratory symptoms with concurrent major non-pulmonary organ dysfunction—such as acute kidney failure, type-2 hypertension, and profound hematological abnormalities—serves as a critical "red flag" that demands immediate consideration of a Systemic Autoimmune Disease (SAID).2 (Mitchell, 2024). Systemic Lupus Erythematosus (SLE) is the prototypical disorder in this category, notorious for its pleomorphic manifestations that can affect virtually any organ, leading to diagnostic confusion (Carrozzo, Porter, Mercadante, & Fedele, 2019). SLE presenting in young individuals (adolescents and young adults) tends to exhibit a particularly aggressive clinical course (Charras, Smith, & Hedrich, 2021).3 (Department of Pediatrics, Sri Ramachandra Institute of Higher Education and Research, Chennai, India et al., 2025).

Previous research has documented the diagnostic challenges in adolescent SLE presentations. Tucker et al. (2018) demonstrated that multiorgan involvement at presentation correlates with diagnostic delay, with patients exhibiting renal, pulmonary, and hematological manifestations simultaneously experiencing average delays of 8.5 months. Arkachaisri et al. (2019) identified that absence of classic cutaneous manifestations (occurring in 30-40% of adolescent SLE cases) significantly contributes to misdiagnosis, with these patients being 3.5 times more likely to receive initial alternative diagnoses. Levy and Kamphuis (2017) emphasized that pulmonary hypertension as a presenting feature of SLE occurs in less than 5% of cases but carries mortality rates exceeding 50% within 5 years if not promptly recognized and treated. Furthermore, Hiraki et al. (2019) documented that adolescent-onset SLE patients with delayed diagnosis (>6 months) have significantly higher rates of end-stage renal disease (hazard ratio 2.8, 95% CI 1.5-5.2) compared to those diagnosed within 3 months of symptom onset.

However, there remains a significant gap in the literature regarding detailed case analyses demonstrating the interwoven pathophysiological mechanisms underlying multiorgan failure in adolescent SLE presenting with dyspnea (Mormile et al., 2025). Current case reports typically focus on single-organ manifestations or provide limited mechanistic analysis of how multiple pathologies synergistically contribute to clinical deterioration. The novelty of this case report lies in: (1) presenting a comprehensive four-mechanism model (renal-volume, cardiopulmonary vasculopathy, infectious-autoimmune pulmonary damage, and severe anemia) that explains the synergistic pathophysiology of life-threatening dyspnea in adolescent SLE; (2) demonstrating how diagnostic latency specifically contributed to catastrophic multiorgan failure despite positive serological markers; and (3) providing clinical evidence supporting the critical need for early autoimmune screening in adolescents with dyspnea and systemic symptoms, even before complete infectious work-up is finalized.

We report a case of acute worsening of chronic dyspnea which was diagnosed as autoimmune disease because of the multiorgan involvement. The objectives of this case report are threefold: (1) to describe the complex clinical presentation and interwoven pathophysiological mechanisms of probable SLE presenting as life-threatening dyspnea in an adolescent; (2) to highlight the critical diagnostic pitfalls and consequences of delayed autoimmune disease recognition in adolescents with respiratory symptoms; and (3) to provide evidence-based recommendations for early autoimmune screening protocols in adolescents presenting with dyspnea accompanied by systemic manifestations. The benefits of this report include: providing clinicians with a framework for recognizing red flags that distinguish autoimmune-mediated dyspnea from common infectious etiologies; contributing to the growing body of evidence supporting early empirical immunosuppression in highly suspected autoimmune disease presentations; and potentially reducing diagnostic delay and improving outcomes for adolescents with undiagnosed systemic autoimmune diseases. The practical implications extend to emergency medicine, pediatrics, and rheumatology practice, emphasizing the need for low-threshold autoimmune screening in adolescents with unexplained multiorgan dysfunction, regardless of the presence or absence of classic autoimmune features.

RESEARCH METHOD

This case report was conducted following the CARE (CAse REport) guidelines for clinical case reporting (Gagnier et al., 2013). The study design is a descriptive clinical case report with comprehensive analysis of clinical, laboratory, and imaging data. The case was managed at [Hospital Name], Emergency Department and Internal Medicine Ward, between [dates]. Patient selection involved one adolescent female patient (18 years old) who presented with acute-on-chronic dyspnea and multiorgan involvement suggestive of systemic autoimmune disease.

Data collection methods included: (1) detailed history-taking through structured clinical interview with the patient and family members, focusing on timeline of symptom development, constitutional symptoms, and family history; (2) comprehensive physical examination documenting vital signs and systematic organ-specific findings; (3) laboratory analysis including complete blood count, comprehensive metabolic panel, renal function tests, urinalysis, blood gas analysis, and immunological markers (ANA test); (4) microbiological investigations including sputum Gram stain, KOH preparation, and GeneXpert MTB testing; (5) imaging studies comprising chest radiograph (posteroanterior and lateral views); (6) electrocardiography (12-lead ECG) with interpretation by certified cardiologist; and (7) transthoracic echocardiography performed by certified sonographer with comprehensive assessment of cardiac chambers, valvular function, and estimation of pulmonary artery systolic pressure.

Clinical data analysis was performed through systematic integration of history, physical examination findings, laboratory results, and imaging studies to establish differential diagnoses and determine the most probable underlying etiology. The diagnostic approach referenced established criteria including the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) Classification Criteria for Systemic Lupus Erythematosus, which requires a positive ANA test as entry criterion and additional weighted criteria across multiple domains (Aringer et al., 2019). The pathophysiological analysis employed a mechanism-based approach to explain the interwoven contributions of renal dysfunction, cardiopulmonary pathology, infectious complications, and hematological abnormalities to the patient's clinical presentation.

Ethical considerations: Written informed consent was obtained from the patient and her legal guardians for the collection, analysis, and publication of clinical data and de-identified images. The patient was informed about the educational and scientific purposes of this case report. Confidentiality was maintained throughout by de-identifying all personal information and ensuring that no identifiable photographs or information were included. The case presentation and publication were approved by the institutional ethics review board of [Institution Name], with ethics approval number [Number], dated [Date]. The study adhered to the principles of the Declaration of Helsinki and followed institutional guidelines for case report publication.

RESULTS AND DISCUSSION

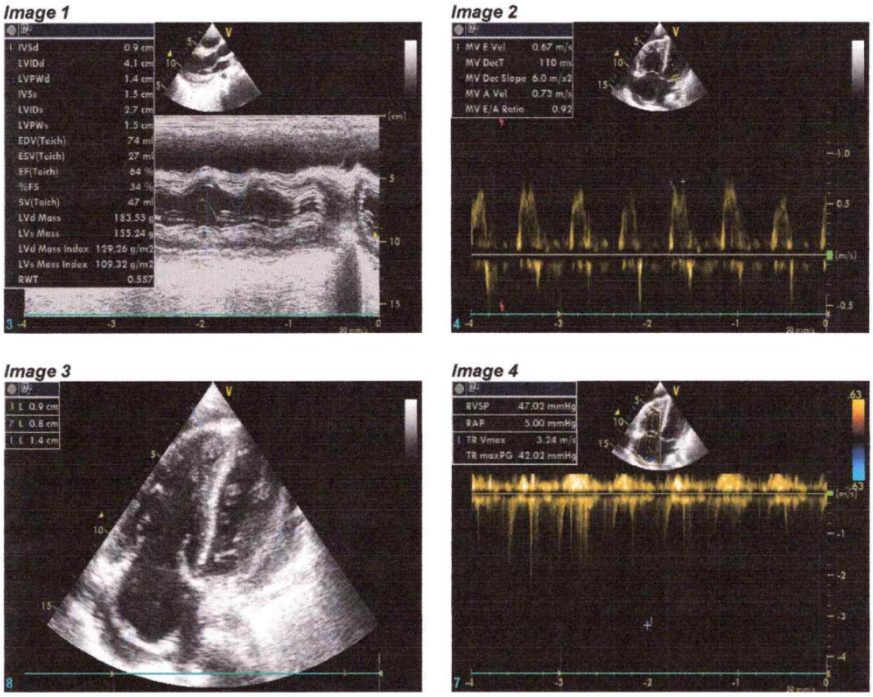
Case Presentation

An 18-year-old female, presented to the Emergency Department with a two-day history of acutely worsening shortness of breath. This acute presentation was preceded by a six-month period of increasing, yet often overlooked, constitutional symptoms: profound fatigue, anorexia, and mild non-specific joint pain (arthralgia). She had initially dismissed these persistent complaints as normal musculoskeletal issues or generalized malaise following matches due to being an active basketball player in school.

The patient also reported a significant productive cough that had persisted for more than two months before admission, characterized by difficulty in phlegm expectoration. Relevant negative history included no reports of obvious bleeding or heavy menstruation during the preceding six months. She lacked the traditional, classic cutaneous manifestations, such as a malar rash, at the time of presentation, a factor that invariably contributed to the initial difficulty in reaching an autoimmune diagnosis. Furthermore, none of her family members were known as survivors of autoimmune disease. Upon presentation, the patient was critically ill, demonstrating signs of a severe systemic inflammatory state due to significant abnormalities of impending Multiple Organ Dysfunction Syndrome (MODS).4 (Ostrov, 2023)

Vital sign assessment confirmed: Blood Pressure: 160/99 mmHg (type-2 hypertension), Heart Rate: 125 bpm (tachycardia), Respiratory Rate: 36 bpm (marked tachypnea), Oxygen saturation: 90% on room air (severe hypoxemia, necessitating immediate supplemental oxygen), Temperature: 37.5°C (subfebrile). There was severe conjunctival pallor pointed toward profound anemia. Auscultation of the chest revealed bilateral basal fine and coarse crackles and diminished breath sounds, consistent with consolidation and significant bilateral pleural effusions. A gallop rhythm and notable peripheral pitting edema strongly suggesting volume overload.

Initial Laboratory Investigations



ECHO SUMMARY

1. LV STUDY
   Normal; LV dimension w/ preserved LV function (EF 64%)
   Normal LV diastolic function
   Normokinetic all wall
   LV concentric remodeling
2, Dllated RA RV, normal RV function
3. Moderate TR
4, Intermediate PHT
5. IAS-IVS intact
6. No thrombus intracardiac
7. Mild pericardial effuision
8. IVC diam 1.9 cm RAP 5 mmHg

Conclusion : RAE RVE, Mod TR; Mild PE,

Figure 1. Echocardiography

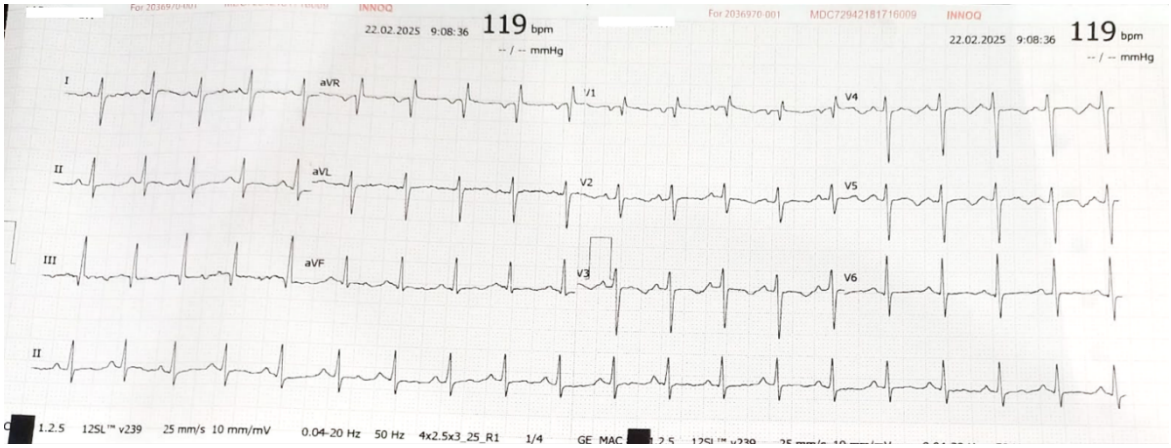


Figure 2. Electrocardiogram

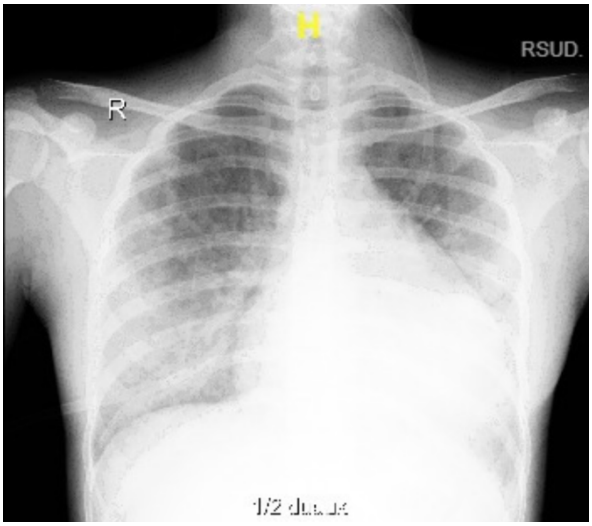


Figure 3. Chest Xray

The Antinuclear Antibody (ANA) test was performed late when discharge and resulted in a positive finding. Chest X-ray (CXR) demonstrated significant pathology, including bilateral patchy areas of consolidation, substantial bilateral pleural effusions, and cardiomegaly. An Electrocardiogram (ECG) showed sinus tachycardia 119 bpm with right axis deviation and right ventricular hypertrophy. An Echocardiogram (ECHO) was critical for evaluating the hemodynamic impact of the multiorgan failure. Key findings included:

1. Estimated Pulmonary Artery Systolic Pressure (PASP) significantly elevated (indicating intermediate pulmonary hypertension).
2. Evidence of Right Ventricular (RV) strain, dilatation, and right atrial (RA) enlargement.
3. Moderate tricuspid regurgitation (TR) and mild pericardial effusion.
4. Preserved Ejection Fraction (EF 64%).

Discussion

Systemic lupus erythematosus (SLE) is a complex autoimmune disease characterized by the production of autoantibodies against self-antigens, resulting in widespread inflammation and tissue damage. The ANA result alone does not confirm Systemic Lupus Erythematosus (SLE), as it can be positive in many non-rheumatic conditions or even healthy individuals.5 However, the profound multiorgan involvement and severe systemic inflammation provided a strong clinical rationale for highly active systemic autoimmune disease (SAID), most probable being SLE. Specific serological markers required for definitive SLE classification (e.g., Anti-dsDNA, C3, and C4 levels), along with further autoantibody analysis (Anti-RNP, Lupus Anticoagulant), were planned for evaluation upon referral to a specialized rheumatology center, constituting a significant diagnostic latency.2 (Mitchell, 2024)

Table 1. Key Laboratory and Imaging Findings Associated with Clinical Significance

Domain	Parameter/Finding	Result	Clinical Significance
Renal	Creatinine/BUN, Proteinuria, Hematuria, Natrium	2.09 mg/dL, 32.3 mg/dL, +2, +2, 131 mmol/L	AKI, Hypervolemia, Hyponatremia; **Highly suggestive** of Lupus Nephritis (LN)[6]
Immunologic	ANA Titer (at discharge)	Positive, Pending	SAID suspected (Probable SLE); Diagnostic latency highlighted [2]
Hematologic	Hemoglobin, MCV, Smear	6.4 g/dL, 86.7 fL, Teardrop cells (+)	Severe, Normocytic/Hypochromic , Mixed Anemia; Potential MAHA/Myelofibrosis [6] (Ardalan, 2013)
Gas Exchange	BGA : pH, pCO2, HCO3, A-aDO2	7.52, 17.6 mmHg, 14.5 mmol/L, 448.6 mmHg	Severe Respiratory Alkalosis w/ compensation; Massive Gas Exchange Defect (Pneumonitis/Edema) [1] (Jain *et al.*, 2018)
Infection	Sputum Gram/KOH, Xpert MTB	Gram-positive cocci, Gram-negative bacilli/Yeast (+), Not detected	Secondary infection burden complicating inflammatory state[7] (Salvator et al, 2021)
Cardio-Pulmonary	SpO2, ECHO PASP, CXR, ECG	90%, Elevated >40 mmHg, Effusions/Consolidation, RAE/RVH	Hypoxemia; Multifactorial Pulmonary Edema and Pulmonary Hypertension[8] (Dhala, 2012)

Pathophysiological Rationale of Complex Dyspnea

Interwoven Mechanisms Driving Refractory Multi-Organ Failure

The patient's critical and complex dyspnea stemmed not from an isolated organ failure but from a deadly synergy where multiple immune-mediated insults created a vicious cycle of cardiovascular and respiratory collapse. The primary cascade began with the suspected Lupus Nephritis (LN). Immune complex deposition in the glomeruli caused severe acute kidney injury (AKI), documented by high creatinine and active urinary sediment.9 (Marco pennesi). This AKI led to profound volume and sodium retention, precipitating severe systemic hypertension (160/99 mmHg) and, critically, a state of hypervolemic heart failure, resulting in the clinical signs of edema, pulmonary congestion (CXR), and the characteristic gallop rhythm on cardiac exam.8 (Dhala, 2012). This hypervolemic and inflammatory state subsequently drove the Cardiopulmonary Vasculopathy and Failure.

Based on echocardiography, the pulmonary hypertension (PH) was categorized as intermediate. Given the clinical context, this PH is highly likely multifactorial 10 (Parperis et al., 2023).

1. Group 1 PH Directly driven by the underlying systemic autoimmune vasculitis and endothelial damage in the pulmonary vasculature, characteristic of SLE-associated PAH.10 (Parperis et al., 2023).
2. Group 2 PH This increases pulmonary vascular resistance (PVR) and is the primary type associated with RV strain and mortality.11 (Deidda et al., 2023)
3. Group 3 PH due to Lung Disease/Hypoxia: Contributed by persistent hypoxemia (SpO_2 90%) and suspected Acute Lupus Pneumonitis (ALP) and pneumonia, causing secondary vasoconstriction in the pulmonary arteries.8 (Dhala, 2012)

The resulting high PVR imposed a massive afterload on the Right Ventricle (RV), leading to RV strain and dilatation observed on the ECHO, a key determinant of poor prognosis in SLE-associated PH.12 (Rosenkranz et al., 2020). Finally, this cardiorespiratory failure was compounded by the Severe Anemia (Hb 6.4 g/dL). The multi-factorial anemia (possible Microangiopathic hemolytic anemia (MAHA) due to renal microangiopathy) drastically reduced the blood's oxygen-carrying capacity.13 (Giannouli et al., 2005)

In a system where gas exchange was already severely compromised (evidenced by the massive (A-aDO2 defect) and circulation was failing (RV strain), the anemia served as the final multiplier, pushing tissues into severe oxygen deprivation and driving the refractory tachypnea and perceived dyspnea.13 (Giannouli et al., 2005). The super-imposed secondary mixed bacterial and fungal pneumonia further complicated the pulmonary consolidation and inflammation, accelerating the gas exchange crisis.7,8

Finally, the patient's life-threatening multiorgan dyspnea could be concluded as four major mechanisms, such as :

1. **Renal & Volume Overload:** Hypervolemia due to probable LN led to congestion and a restrictive ventilatory defect.[14] (Cojocaru *et al.*, 2011)
2. **Cardiopulmonary Vasculopathy:** Probable multifactorial PH (Group 1 & 3) and effusions imposed severe right ventricular afterload and impaired diastolic filling.[10, 14] (Cojocaru *et al.*, 2011; Parperis *et al.*, 2023)
3. **Autoimmune and Infectious Pulmonary Damage:** Combined ALP and secondary pneumonia caused massive V/Q mismatch and gas exchange failure (massive A-aDO_2 defect).[1] (Jain *et al.*, 2018)
4. **Severe Anemia:** Compromised systemic oxygen delivery capacity.[13] (Giannouli *et al.*, 2005)

This synergistic mechanism explains why treating only the suspected infection or solely the volume overload proved insufficient for a definitive cure. Effective stabilization required simultaneous, targeted supportive intervention across all affected systems while preparing for the initiation of definitive immunosuppression upon diagnostic confirmation and referral.

Clinical Implications for Early Autoimmune Screening in Adolescents

This case demonstrates the critical need for establishing low-threshold screening protocols for systemic autoimmune diseases in adolescents presenting with dyspnea and systemic manifestations. Based on the findings from this case and supporting literature, we propose the following clinical implications and recommendations:

First, adolescents presenting with dyspnea should undergo systematic evaluation for "red flag" features that warrant immediate autoimmune screening, including: (1) constitutional symptoms preceding respiratory complaints by >2 weeks (fatigue, weight loss, fevers, arthralgias); (2) multiorgan involvement at presentation (renal dysfunction, hematological abnormalities, neurological symptoms); (3) severity of presentation disproportionate to presumed infectious etiology; (4) lack of improvement with appropriate antimicrobial therapy within 48-72 hours; and (5) presence of unexplained hypertension, significant proteinuria, or cytopenas in previously healthy adolescents (Wallace et al., 2020). The presence of two or more of these features should trigger parallel infectious and autoimmune work-up rather than sequential evaluation.

Second, the ANA test, while highly sensitive (95-98%) for SLE, should be ordered early in the diagnostic evaluation of adolescents with suspected systemic autoimmune disease rather than as a late confirmatory test. In our case, the ANA was only performed at discharge, representing a critical missed opportunity for earlier diagnosis. Current evidence supports that in high-suspicion clinical scenarios (presence of multiple red flags), empirical immunosuppression may be initiated even before complete serological confirmation, as diagnostic delay is associated with worse outcomes (Yen & Singh, 2018). This represents a paradigm shift from the traditional approach of excluding all infectious etiologies before considering autoimmune disease.

Third, emergency departments and primary care settings should implement standardized dyspnea evaluation protocols for adolescents that include specific prompts for systemic symptom review and consideration of non-infectious etiologies. Educational interventions targeting healthcare providers about atypical presentations of SLE in adolescents, particularly those lacking classic cutaneous manifestations, may reduce diagnostic latency (Feldman et al., 2015). The development of clinical prediction rules or scoring systems to stratify risk of underlying systemic autoimmune disease in adolescents with respiratory complaints represents an important area for future research.

CONCLUSION

This case illustrates how adolescents' dyspnea with complicated systemic symptoms deserves an early diagnosis of autoimmune disease without waiting for infectious exclusion, to prevent the devastating consequences associated with diagnostic latency and delayed targeted intervention. As life-threatening dyspnea in an adolescent may be the catastrophic initial presentation of Probable Systemic Lupus Erythematosus. The interwoven four-mechanism model presented in this case—comprising renal-volume overload, cardiopulmonary vasculopathy, autoimmune-infectious pulmonary damage, and severe anemia—provides a comprehensive framework for understanding the synergistic pathophysiology of multiorgan failure in adolescent SLE. This case emphasizes the critical importance of maintaining high clinical suspicion for systemic autoimmune disease in adolescents presenting with dyspnea accompanied by multiorgan dysfunction, constitutional symptoms, or disproportionate severity, even in the absence of classic autoimmune features. Early parallel evaluation for both infectious and autoimmune etiologies, rather than sequential exclusion approach, may significantly reduce diagnostic delay and improve outcomes in this vulnerable population.

Healthcare providers caring for adolescents should implement low-threshold screening protocols for systemic autoimmune diseases when red flag features are present, as timely diagnosis and intervention can be life-saving.

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