Case Reports and Reviews

Case Report

Acute Respiratory Distress Syndrome Caused by Group A Streptococcus: A Case Report and Literature Review

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Abstract

Background: *Group A Streptococcus (GAS) can occasionally cause severe, life- threatening infections. We present a case in which a GAS infection led to acute respiratory distress syndrome (ARDS).*

Case Presentation: *A 43-year-old man was admitted to the hospital with a sore throat, hoarseness, and flu-like symptoms. He subsequently developed a productive cough with purulent green sputum. Laboratory investigations revealed acute kidney injury (AKI) and elevated inflammatory markers, including a raised white blood cell (WBC) count and C-reactive protein (CRP). Initially managed for pneumonia and AKI, further testing identified GAS in a sputum sample, although blood cultures remained negative. He required oxygen therapy, intravenous antibiotics, and haemodialysis. As his respiratory status rapidly deteriorated with worsening sepsis, a chest X-ray confirmed ARDS, necessitating mechanical ventilation. Given the severity of his condition, intravenous immunoglobulin (IVIG) was administered, leading to a favourable response.*

Conclusion: *ARDS should be considered in cases of septic shock to prevent fatal outcomes, as delayed recognition contributes to high mortality rates. Early diagnosis and aggressive management—including fluid resuscitation, appropriate antibiotic therapy, IVIG, and infection control—can significantly improve survival.*

Case Report

Infectious causes, such as bacterial pneumonia and non-infectious conditions, can trigger acute respiratory distress syndrome (ARDS). Severely affected patients display similar clinical and histopathologic features, suggesting a shared form of immune response in ARDS [1]. The disease is caused by elements from both infections and damaged cells, which vary in size and type. At the molecular level, these molecules exhibit diverse sizes. Furthermore, biological properties, categorizing them into proteinaceous and non-proteinaceous substances [2]. The severity of ARDS is determined by the presence of causative substances and the related immune reactions, as well as the duration and repertory of specific immune cells involved in controlling these substances [3].

Group A Streptococcal (GAS) infections can lead to severe conditions like streptococcal toxic shock syndrome (STSS), which involves failure of several organs. Despite its seemingly harmless appearance, GAS can quickly result in lethal necrotizing infections. A variety of illness, including pharyngitis, cellulitis, rheumatic fever, post-streptococcal glomerulonephritis, and streptococcal toxic shock syndrome (STSS), can be brought on by the Gram-positive bacteria Group A Streptococcus (GAS) [4]. STSS is marked by low blood pressure multiple organ failure due to a systemic inflammatory response syndrome (SIRS) believed to be brought on by a severe inflammatory response brought on by the endotoxins and superantigens that GAS releases [5]. Acute respiratory distress syndrome (ARDS) occurs in over 50% of STSS cases and should be considered when there is bilateral lung involvement. ventilation strategies to protect the lungs can reduce mortality in these patients [6].

Case Presentation

A 43-year-old male presented to the Emergency Department with a sore throat, hoarse voice, and flu-like symptoms. He subsequently developed a productive cough with greenish purulent sputum, followed by worsening shortness of breath. Previously healthy, the patient had no significant medical history, was a non-smoker, and consumed alcohol occasionally. There was no history of drug use or allergies prior to symptom onset.

Biochemical Analysis

Initial Laboratory tests showed an elevated white blood cell (WBC) count of 29.8 cells/μL, indicating severe inflammation or infection, which remained high but normalized to

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10.2-10.3 as the patient recovered. Haemoglobin (Hb) levels were initially normal at 135 g/L but dropped to 87 g/L, suggesting anaemia likely due to inflammation, blood loss, or nutritional deficits, but improved during recovery. The platelet count increased from 377 x 10^9/L, indicating reactive thrombocytosis due to the acute inflammatory process. Urea levels rose from 21.9 mmol/L to

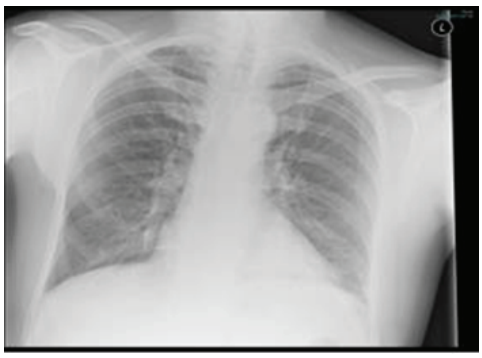
40.0 mmol/L, and creatinine peaked at 727 µmol/L, reflecting acute kidney dysfunction, but both gradually subsided with recovery. Serum Immunoglobulin IgG levels remained low throughout.

Abdominal ultrasound

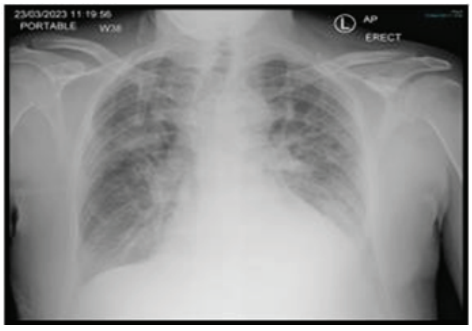
The abdominal ultrasound showed enlarged kidneys without hydro-nephrosis or focal masses. While the imaging could not confirm pyelonephritis, the findings suggested a possible renal parenchymal abnormality, potentially due to Inflammation or infiltration. Despite no evidence of hydronephrosis or stones, pyelonephritis was the likely explanation of AKI, especially given the ongoing GAS infection, which is well known to cause kidney inflammation even without clear ultrasound signs.

Clinical summary

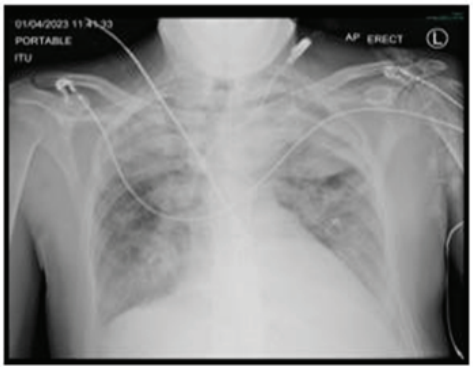
The patient's assessment with clinical severity index showed a CURB-65 score of 3, AKI score of 2, and NEWS (National Early Warning Score) score of 7, receiving initial treatment with intravenous fluids, oxygen, and antibiotics. The patient remained clinically stable with unchanged biochemical marker values for the first two days. On day 3, renal function began to deteriorate, and by day 5, myelocytes were detected on the blood film, indicating significant infection-related bone marrow stress. The patient's condition declined to further acute respiratory distress by day 6, necessitating dialysis. Despite gradual improvement in inflammatory markers, oxygen saturation worsened by day 10, and the chest X-ray (CXR) showed features consistent with ARDS. The patient was transferred to the intensive care unit (ICU), requiring invasive mechanical ventilation and inotropic infusion support. Following intravenous immunoglobulin administration, a beta-D-glucan titre of 378 pg/mL was noted, leading to antifungal treatment with caspofungin (70 mg/day for 5 days, then 50 mg/day for 7 days) and voriconazole (100 mg/day for 10 days). The X ray changes during admission.



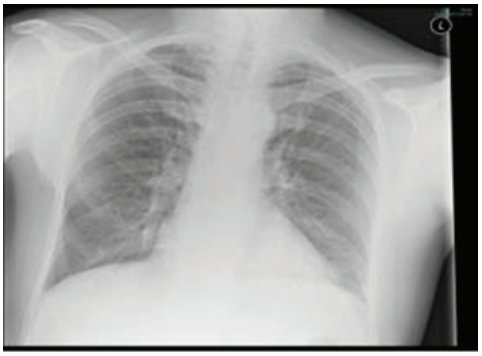
Day 1, Thinly dispersed marking through with pulmonary shadowing in both lower zone



Day 8, Widespread opacity in both lungs predominantly in upper lobe. Collapse consolidation left lower lobe. Low volume bilateral pleural effusion



Day 10, Bilateral pulmonary shadowing affecting all zones but upper zone predominance. Bilateral pleural effusion and bibasal atelectasis



1 Month after discharge showing marked improvement lung field

Clinical Discussion

Group A Streptococcal (GAS) infections present in various forms, including pharyngitis, myositis, and soft tissue infections, as well as more severe conditions like bacteraemia, necrotizing fasciitis, and toxic shock syndrome (STSS) [7, 8]. According to the Centers for Disease Control and Prevention (CDC), STSS is defined by hypotension, a positive GAS culture, and involvement of at least two organ systems, such as the kidneys, liver, lungs, skin, or muscle [8]. Organ involvement alone qualifies, without the requirement for organ failure, allowing for the inclusion of conditions like necrotizing fasciitis and desquamating rash, which often develop later in the illness.

GAS can cause asymptomatic infections, pharyngitis, scarlet fever, and severe invasive infections, triggering immune sequelae. GAS also affects innate and adaptive immune responses to infection. In 2022, WHO reported a 28% increase in invasive GAS (IGAS) in children under 15 in England, sometimes presenting unusually, such as with pulmonary emphysema [9].

Multiple organ dysfunction syndrome (MODS) is a hallmark of severe sepsis, characterized by impaired organ function that cannot be maintained without medical intervention [10]. GAS primarily causes pharyngitis but can also colonize the female genital tract, although this is rare [11]. GAS is arecognized cause of postpartum endometritis and may be transmitted through sexual contact [12-15].

In a study of ICU patients with Group A Streptococcus (GAS) infections, it was discovered that 34% of the patients had acute respiratory distress syndrome, 55% had acute kidney injury, of which 21% required renal replacement therapy, 64% had hepatic dysfunction, and 69% had coagulopathy [16]. Strep-EURO's global epidemiological investigation conducted in Europe in 2003 and 2004 found that the 7-day death rate for severe GAS sepsis was 19%. The occurrence of ARDS and necrotizing fasciitis was significantly linked to elevated mortality rates, with 7-day mortality rates of 44% and 32%, respectively [19]. The significance of drainage in reducing the bacterial load has been emphasized in STSS [20].

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Clinical trials for ARDS management have shown that low inspiratory pressures and tidal volume improve survival, while prone positioning reduces mortality. Severe STSS cases have been successfully managed with extracorporeal membrane oxygenation (ECMO), although it was not required due to the patient's positive response to less intensive treatment [19].

Empirical antimicrobial regimens typically cover GAS, which remains susceptible to penicillin [20].The Infectious Diseases Society of America recommendations propose penicillin and Clindamycin as the main therapy for acute respiratory distress syndrome (ARDS). The guidelines recommend combining penicillin and Clindamycin for GAS necrotizing fasciitis [21]. However, Clindamycin is effectively recommended for GAS necrotizing fasciitis due to its antitoxin effects [22], Linezolid is equally effective in inhibiting toxin production and is a viable alternative for invasive GAS infections in the event of clindamycin resistance [23].

Intravenous immunoglobulin (IVIG) therapy has been shown to reduce mortality in patients with GAS-related STSS and necrotizing fasciitis. A multicenter prospective study demonstrated that the absence of intravenous immunoglobulin (IVIG) injection was linked to a higher risk of death after 90 days in cases of Group A Streptococcus (GAS)-caused necrotizing soft tissue infections [24]. Intravenous immunoglobulin (IVIG) significantly decreased mortality rates in patients with streptococcal toxic shock syndrome (STSS) receiving clindamycin treatment, according to a meta-analysis. Mortality dropped from 33.7% to 15.7%31 when used IVIG [25].

Immunoglobulins are glycoproteins secreted by differentiated B cells responsible for producing antibodies and consequently acting against the pathogen. Studies indicate that IVIG therapy significantly improves survival rates when combined with Clindamycin. In patients with severe organ dysfunction, IVIG can be life-saving, with some studies showing a marked decrease in mortality without affecting ICU stay length [24, 26].

Conclusion

This case highlights the importance of early recognition and decisive management of ARDS, particularly by promptly identifying early warning signs and initiating treatment without delay. Haemodynamically unstable patients, especially those with sepsis, require immediate multi-organ supportive therapy in a critical care environment.

Our patient, who developed ARDS, AKI, and sepsis following a GAS infection, initially presented with flu-like symptoms. Rapid intervention with oxygen therapy, intravenous antibiotics, haemodialysis, mechanical ventilation, and intravenous immunoglobulin (IVIG) was key in achieving a positive outcome. Given the high mortality risk associated with ARDS, early diagnosis and prompt management incorporating infection control, appropriate antimicrobial therapy, immunoglobulin therapy and organ support with a multi-disciplinary approach was vital in achieving positive outcomes.

References

1. Wong JJM, Leong JY, Lee JH, Albani S, Yeo JG. Insights into the immuno-pathogenesis of acute respiratory distress syndrome. Ann Transl Med. 2019;7(19):502. doi:10.21037/atm.2019.09.42
2. Yang CY, Li WC, Lee YZ, et al. New insights into the immune molecular regulation of the pathogenesis of acute respiratory distress syndrome. Int J Mol Sci. 2018;19(2):588. doi:10.3390/ijms19020588
3. Lee KY. Pneumonia, acute respiratory distress syndrome, and early immune-modulator therapy. Int J Mol Sci. 2017;18(2):388. doi:10.3390/ijms18020388
4. Hand RM, Snelling TL, Carapetis JR. Group A Streptococcus. In: Ryan ET, Hill DR, Solomon T, Aronson NE, Endy TP, eds. Hunter's Tropical Medicine and Emerging Infectious Diseases. 10th ed. Elsevier; 2020:429-438. doi:10.1016/B978-0-323-55512-8.00040-5
5. Schmitz M, Rouzé A, Luyt CE, et al. Streptococcal toxic shock syndrome in the intensive care unit. Ann Intensive Care. 2018;8(1):84. doi:10.1186/s13613-018-0431-2
6. Fan E, Brodie D, Slutsky AS. Acute respiratory distress syndrome: advances in diagnosis and treatment. JAMA. 2018;319(7):698-710. doi:10.1001/jama.2017.21907
7. de Fays C, Pilette C, Detry B, et al. Secretory immunoglobulin A immunity in chronic obstructive respiratory diseases. Cells. 2022;11(8):1324. doi:10.3390/cells11081324
8. Waddington CS, Snelling TL, Carapetis JR. Management of invasive group A streptococcal infections. J Infect. 2014;69(Suppl 1):S63-S69. doi:10.1016/j.jinf.2014.07.021
9. Prince S, Naresh K, Tulasi R. Case report on paediatric nephrotic syndrome. EJIFCC. 2020;31(2):164-169.
10. Bray S, Morgan J. Two cases of group A streptococcal vulvovaginitis in premenopausal adults in a sexual health setting. Sex Health. 2006;3(3):187-188. doi:10.1071/SH06015
11. Mead PB, Winn WC. Vaginal-rectal colonization with group A streptococci in late pregnancy. Infect Dis Obstet Gynecol. 2000;8(5-6):217-219. doi:10.1155/S1064744900000340
12. Gisser JM, Marchaim D, Biederman R, Kaye KS. Invasive group A streptococcus associated with an intrauterine device and oral sex. Sex Transm Dis. 2002;29(8):483-485. doi:10.1097/00007435-

200208000-00014

13. Lamagni TL, Efstratiou A, Vuopio-Varkila J, Jasir A, Schalén C; Strep-EURO Project Participants. The epidemiology of severe Streptococcus pyogenes associated disease in Europe. Euro Surveill. 2005;10(9):179-184. doi:10.2807/esm.10.09.00577-en
14. Guérin C, Reignier J, Richard JC, et al. Prone positioning in severe acute respiratory distress syndrome. N Engl J Med. 2013;368(23):2159-2168. doi:10.1056/NEJMoa1214103
15. Mehta C, Vijayan VK, Mittal A. Overdiagnosis of asthma and its relationship to body mass index. Chest. 2006;130(4):97S.
16. Mehta S, Narula S, Ghosh S, et al. Morbidity and mortality of patients with invasive group A streptococcal infections admitted to the ICU. Chest. 2006;130(6):1679-1686. doi:10.1378/chest.130.6.1679
17. Wood TF, Potter MA, Jonasson O. Streptococcal toxic shock-like syndrome: the importance of surgical intervention. Ann Surg. 1993;217(2):109-114. doi:10.1097/00000658-199302000-00001
18. Iwasaki N, Terauchi T, Okamoto M, et al. Venoarterial extracorporeal membrane oxygenation for group B streptococcal toxic shock syndrome: a case report and literature review. Medicine (Baltimore). 2023;102(37):e34680. doi:10.1097/MD.0000000000034680
19. Snyder A, Schmalzle SA. Spontaneous Streptococcus pyogenes pelvic inflammatory disease: Case report and review of the literature. IDCases. 2020;20:e00785. doi:10.1016/j.idcr.2020.e00785
20. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft-tissue infections. Clin Infect Dis. 2005;41(10):1373-1406. doi:10.1086/497143
21. Andreoni F, Zürcher C, Tarnutzer A, Schuepbach RA, Schäfer M, Zinkernagel AS. Clindamycin affects Group A Streptococcus virulence factors and improves clinical outcome. J Infect Dis. 2017;215(2):269-277. doi:10.1093/infdis/jiw536
22. Heil EL, Johnson JK, Johnson TM, et al. Comparison of adjuvant clindamycin vs linezolid for severe invasive group A Streptococcus skin and soft tissue infections. Open Forum Infect Dis. 2023;10(12):ofad588. doi:10.1093/ofid/ofad588
23. Raithatha AH, Bryden DC. Use of intravenous immunoglobulin therapy in the treatment of septic shock, in particular severe invasive group A streptococcal disease. Indian J Crit Care Med. 2012;16(1):37-40. doi:10.4103/0972-5229.94424
24. Parks T, Wilson C, Curtis N, Norrby-Teglund A, Sriskandan S. Polyspecific intravenous immunoglobulin in clindamycin-treated patients with streptococcal toxic shock syndrome: A systematic review and meta-analysis. Clin Infect Dis. 2018;67(9):1434-1440. doi:10.1093/cid/ciy294
25. Parks T, Barrett L, Jones N. Invasive streptococcal disease: a review for clinicians. Br Med Bull. 2015;115(1):77-89. doi:10.1093/bmb/ldv027