

Original Research

Exploring phenotypic patterns of cytokine storm in pediatric patients with severe viral infections and multisystem dysfunction: a case series illustrating successful novel therapeutic strategies

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Received (first version): 14-Apr-2026

Accepted: 03-Jun-2026

Published online: 05-Aug-2026

Abstract

Background: Cytokine storm syndrome (CSS) in pediatric patients with severe viral infections is a life-threatening hyper-inflammatory response leading to multi-organ dysfunction. Despite growing recognition, limited data exists on its clinical phenotype and therapeutic strategies in children. **Objective:** To describe the phenotypic features, diagnostic findings, and outcomes of pediatric patients with viral-induced CSS treated at a tertiary care hospital using advanced immunomodulatory therapies. **Methods:** We conducted a retrospective analysis of eleven previously healthy children admitted to Al Jalila Children's Hospital from October 2022 to October 2024. Demographics, viral etiology, biomarkers, imaging, and treatment responses were evaluated. **Results:** Patients presented with systemic inflammation, respiratory distress, and frequent cardiovascular involvement. All patients received intravenous immunoglobulin; six required corticosteroids, and four received biologic agents including anakinra, tocilizumab, or ruxolitinib. All patients showed clinical resolution without long-term complications. **Conclusion:** Early identification of CSS and timely implementation of targeted immunomodulatory therapy in pediatric patients with severe viral infections may significantly improve outcomes. These findings highlight the need for diagnostic vigilance and a multidisciplinary treatment approach in managing pediatric CSS.

Keywords: cytokine storm; pediatric; viral infections

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INTRODUCTION

Cytokine storm syndrome (CSS), a cascade of inflammatory reactions that create extensive tissue damage and multi-organ dysfunction, is becoming more widely identified in

circumstances of severe pediatric viral infections. A excessive level of pro-inflammatory cytokines, which sets off a series of inflammatory reactions that cause severe tissue damage and multi-organ failure, is what defines CSS¹. The COVID-19 pandemic has recently brought CSS in pediatric patients back into the spotlight due to research showing that a subset of children infected with SARS-CoV-2 develop a hyper-inflammatory syndrome known as multisystem inflammatory syndrome in children (MIS-C), which shares many characteristics with CSS²⁻⁵. Because their health can rapidly deteriorate, it is crucial to identify CSS in young patients with viral infections. Therefore, early recognition of this hyperinflammatory syndrome is crucial for guiding efficient timely immunomodulatory therapy.

However, there is still no precise medical characterization of the CSS caused by viral illnesses in pediatric kids, in particular when it comes to immunomodulatory treatment. This retrospective analysis of case series intends to analyze the clinical features, lab abnormalities, therapeutic management and responses, and phenotypic patterns of CSS among the included cases.

METHODOLOGY

Study design and setting

This study was a retrospective case series that was undertaken at Al Jalila Children's Hospital (AJCH), a tertiary referral hospital in the United Arab Emirates throughout two years from October/2022 to 2024.



Inclusion and exclusion criteria

All admitted children aged from 0-12 years with proven viral infection and evidence of hyperinflammatory state that is consistent with diagnosis of CSS defined by rise in inflammatory markers and indicators of organ involvement. Whereas, all children with autoimmune disease, immunodeficiency, hereditary hyperinflammatory syndromes, or malignant disorders were excluded. Additionally, all cases with inadequate or missing medical records or laboratory data were not included.

Data collection

Demographic data such as age and gender were gathered from medical records. To describe phenotypic trends that might help in diagnostic and treatment decisions in similar scenarios, we carefully reviewed a variety of clinical data. The information gathered included viral test results to confirm the existence of specific infections in addition to a thorough immunoglobulin profile (IgG, IgM, and IgA) and lymphocyte subsets to assess the condition of the immune response.

Inflammatory markers, including procalcitonin (PCT), ferritin, fibrinogen, D-dimer, lactate dehydrogenase (LDH), coagulation profile, cardiac enzymes, C-reactive protein (CRP), brain natriuretic peptide (BNP), liver, and renal functions, were evaluated to assess the degree of the inflammatory response. Chest X-rays and echocardiograms (ECHO) were used to evaluate pulmonary involvement and any associated cardiovascular problems, both of which are common in juvenile CSS.

Therapeutic options included the use of antivirals when needed, immunomodulatory medications (such as corticosteroids, interleukin-6 inhibitors, and other biologics), and supportive care methods like mechanical breathing and inotropic support. These treatments were assessed for their ability to lower inflammation, alleviate respiratory and cardiac problems, and ultimately improve patient outcomes. Cases of CSS were defined by elevated pro-inflammatory cytokines and clinical evidence of multiple organ involvement with abnormalities in test markers. At least two elevated biomarkers and indications of organ failure were present in every patient that was considered.

Intravenous immunoglobulins (IVIG), corticosteroids, and a variety of biological drugs, including JAK1/2 inhibitors, IL-1 receptor antagonists, and IL-6 receptor antagonists, were documented as therapeutic interventions. Anticoagulants and supportive care were also provided for the included individuals.

Clinical improvement without long term complication was the primary outcome of this research study. Whereas time of admission and normalization of laboratory biomarkers

were the secondary outcomes. This study used anonymized data from a retrospective chart review. According to institutional rules, consent was already waived.

RESULTS

Demographic and clinical characteristic of the included patients

Eleven cases—six females and five males—were hospitalized to AJCH during a two-year period with viral infections that were worsened by severe cytokine storms. The patients had previously been healthy. The age range was two weeks to nine years, with over half (n=6, 55.5%) being under two years old. All included patients presented with significant manifestations compatible with diagnosis of CSS.

Among the eleven children who were diagnosed with viral-induced CSS, six patients (54.4%) necessitating admission to the critical care unit with significant respiratory, circulatory, and systemic inflammatory response.

Polymerase chain reaction (PCR), a highly sensitive and specific technique that enabled precise identification of the infecting pathogens, was used to identify viruses. Precise pathogen identification was made possible by this method, guaranteeing suitable antiviral treatment and isolation measures for viral infections.

A dengue virus infection was identified in two patients, both of whom had severe systemic inflammatory response such as multi-organ failure and noticeable hemodynamic instability. Three patients had adenovirus infection, and all three had serious respiratory symptoms, such as acute complicated pneumonia, necessitating mechanical ventilation.

In one case, influenza B was found, and in another, parainfluenza virus, one patient who had influenza A and subsequently developed pneumonia secondary to *Streptococcus pneumoniae*, which probably made the inflammatory response worse and made managing him more difficult, two more cases had Respiratory syncytial virus (RSV), and lastly one case had Human metapneumo virus (HMP) infection Table 1.

The occurrence of cytokine storm in each of the eleven patients was evaluated using biomarker analysis in addition to viral testing. The diagnosis of cytokine storm syndrome was supported by elevated levels of important inflammatory biomarkers, including procalcitonin, ferritin, interleukin-6 (IL-6), and C-reactive protein (CRP), which were all consistent with a hyper-inflammatory response. Together with the clinical presentation, these biomarkers were essential in helping to direct each patient's treatment and management.

Notably eight children had abnormal echocardiogram (72.7%), four of whom were admitted to pediatric ICU and subsequently improved. Seven out of eleven (63.6%) developed pulmonary complications including necrotizing pneumonia in one case these



Table 1. Demographic and clinical characteristic of the included patients

Characteristic	N (%)
Age (median) range	2 (11 days-9 years)
Gender (male)	5 (45.5)
Diagnosis on admission	
RSV with coronary dilatation	1 (9.1)
Dengue	2 (18.2)
Adenovirus/RSV rhino	2 (18.2)
Parainfluenza/rhino	1 (9.1)
Human metapneumovirus	1 (9.1)
Adenovirus pneumonia	1 (9.1)
RSV pneumonia	1 (9.1)
Influenza A with strep pneumonia	1 (9.1)
Influenza B with bilateral pneumonia	1 (9.1)

were abnormalities which suggested cytokine-mediated cardiac involvement, included reduced left ventricular ejection fraction, pericardial effusion, and myocardial dysfunction features. These results highlight how the cytokine storm in pediatric patients is systemic, often affecting the cardiovascular and pulmonary systems. Table 2 shows a more thorough summary of the Laboratory characteristics, biomarker analysis, and imaging findings of the included patients

Treatment of the included children & outcomes

All eleven pediatric patients with viral-induced CSS received IVIG as part of their treatment. The immunomodulatory qualities of IVIG, a commonly used therapeutic drug, aid in regulating the hyperactive immune response linked to cytokine storms. The purpose of IVIG in this group was to provide passive immunity and lessen the inflammatory cascade, both of which are essential in situations involving severe viral infections and cytokine dysregulation.

Due to the severity of the inflammatory response observed in some patients, corticosteroid treatment was initiated. In total, six patients received corticosteroids along side IVIG. Corticosteroids are frequently used to reduce inflammation and inhibit the overactive immune response. In order to successfully alleviate the negative consequences of the cytokine storm and stop more tissue damage, corticosteroids can decrease the production of pro-inflammatory cytokines.

Among the six patients who received steroids, two patients even after receiving steroids continued to deteriorate clinically and in laboratory markers so were given biologic treatments, demonstrating the increasing significance of targeted immunotherapies

in the management of cytokine storms. The interleukin-1 (IL-1) receptor antagonist Anakinra. By inhibiting the actions of IL-1, a crucial mediator in the cytokine storm pathway, anakinra has been demonstrated to lower inflammation. Anakinra helps to restore immunological homeostasis and lessen the harm brought on by excessive inflammation by blocking this pro-inflammatory cytokine. Additionally, among the two patients who received anakinra one improved but other one had to be given Janus kinase (JAK) inhibitor (Ruxolitinib), another biologic drug employed in this cohort. JAK1 and JAK2, which are implicated in the signaling pathways of multiple cytokines, including those that cause the inflammatory response observed in cytokine storm, are inhibited by ruxolitinib. Ruxolitinib may be able to lessen cytokine-driven inflammation and the symptoms of cytokine storm syndrome by specifically targeting this pathway.

One patient who didn't improve with steroids received tocilizumab, an antagonist of the interleukin-6 (IL-6) receptor. Tocilizumab effectively inhibits the function of IL-6, which is a key factor in triggering the inflammatory response in cytokine storm. This reduces inflammation and enhances clinical results. Tocilizumab's use in this group was justified by its capacity to target a crucial cytokine implicated in immunological dysregulation brought on by viruses.

Five patients received aspirin as part of their anticoagulant treatment and one received clopidogrel as he was Glucose 6 phosphate dehydrogenase deficient. Because the cytokine storm can cause a procoagulant condition, aspirin is frequently used to prevent thromboembolic events. Aspirin helped lower the risk of vascular problems, which are frequently observed in cases of severe viral infections and cytokine-induced inflammation, by preventing platelet aggregation and lowering the chance of clot formation.

All patients including the six patients those were admitted to PICU exhibited notable clinical improvement after these treatment procedures. After obtaining the proper follow-up care, they were later sent home in stable health. The multidisciplinary treatment approach was successful in handling these difficult patients, as evidenced by the follow-up examinations that showed no long-term effects or sequelae associated with the initial viral infection or the cytokine storm.

Table 3 Shows extensive information on the individual interventions utilized, patient responses to the treatment techniques and clinical outcomes for each patient.

DISCUSSION

The recent cases of viral-induced cytokine storm syndrome (CSS) at Al Jalila Children's Hospital reveal a number of critical clinical, laboratory findings and demographic aspects in the management of severe viral infections in children. Notably, the age distribution of the patients, with more than half (55.5%) under the age of two, illustrates the higher vulnerability of younger children to severe immunological responses, as previously observed as detailed previously⁶. Viral infections such as influenza virus, respiratory

Table 2. Laboratory characteristics, biomarker analysis, and imaging findings of the included patients	
Laboratory, biomarker analysis	N (%)
WBC (high level)	4 (36.4)
Absolute Neutrophil Count (high level)	1 (9.1)
Platelets count (high)	1 (9.1)
Platelets count (low)	2 (18.2)
CRP (high)	7 (63.6)
Procalcitonin	10 (90.9)
Erythrocyte sedimentation rates (high)	7 (63.6)
Ferritin (high)	11 (100)
Fibrinogen (high)	10 (90.9)
Pro BNP	5 (45.5)
IL 1 (high)*	1 (9.1)
IL 6**	2 (18.2)
COVID antibodies (positive)	9 (81.8)
ECHO (abnormal)	6 (54.5)
Blood culture (Streptococcus pneumonia)	1 (9.1)
Urine culture (Achromobacter dentrificans)	1 (9.1)
Respiratory culture (Enterobacter cloacae complex)	1 (9.1)
Chest X ray (abnormal findings)	6 (54.5)
***ECG (abnormal)	8 (72.7)

*Done for one patient
echocardiogram

** Done for 4 patient

syncytial virus (RSV), adenovirus, and dengue virus were prevalent among these patients, consistent with previous research indicating that viral infections can cause severe inflammatory responses, particularly in immunologically immature populations referred by other researchers⁷⁻¹⁰.

The prevalence of complex co-infections, such as rhinovirus and adenovirus in one patient, highlights the difficulty of managing multi-pathogen infections, which aggravate the cytokine response and complicate treatment options^{4,11-13}.

Furthermore, examples of dengue virus infection resulting in multi-organ failure and hemodynamic instability demonstrate the systemic impact of certain viral infections, as well as the vital necessity for immediate, aggressive care as per other researchers¹⁴. The necessity for mechanical ventilation in patients with severe adenovirus-related respiratory symptoms highlights the importance of early detection and intensive respiratory assistance in treating severe viral infections in children¹⁵.

These findings advance our understanding of the intricacies of viral infections in young children, emphasizing the importance of prompt diagnosis and multidisciplinary therapy in improving patient outcomes.

The numerous viral pathogens found in these pediatric patients highlight the complexities of handling viral-induced CSS, especially when co-infections are present. The detection of respiratory pathogens such as human metapneumovirus (hMPV), influenza B, parainfluenza virus, and influenza A, as well as Streptococcus pneumoniae co-infection, emphasizes the importance of viral-bacterial interactions in complicating clinical outcomes¹⁶⁻¹⁹.

Notably, the presence of bacterial superinfection caused by Streptococcus pneumoniae in influenza A resulted in more severe clinical development, a condition well described in respiratory illnesses²⁰⁻²¹. The use of polymerase chain reaction (PCR) technology proved crucial in precisely identifying viral diseases, allowing for targeted antiviral therapies and infection control measures. This emphasizes the significance of molecular diagnostics in determining optimal therapeutic strategies²². Biomarker analysis validated the diagnosis of cytokine storm, with high levels of procalcitonin, ferritin, interleukin-6 (IL-6), and C-reactive protein (CRP), all of which indicate a hyper-inflammatory state²³.

These biomarkers, combined with clinical presentations, were critical in guiding treatment methods, proving inflammation's significance in CSS pathogenesis. The echocardiography findings of left ventricular failure, pericardial effusion, and myocardial dysfunction in four out of the six patients admitted to PICU highlight the systemic nature of the cytokine storm, with the cardiovascular system being especially vulnerable²⁴. These findings highlight the necessity of monitoring several organ systems, especially the heart and lungs, in critically unwell young patients with viral infections. Finally, the use of precise viral diagnoses, biomarker profiling, and imaging proved critical in controlling these patients and improving patient outcomes.

In the current study at AJCH, treatment of pediatric patients with viral-induced CSS using a combination of immunomodulatory medications was aimed at reducing the hyper-inflammatory response. All eleven patients received IVIG, a well-known therapy for immunological dysregulation during cytokine storms. Previous research has extensively documented IVIG's potential to modify immune function by providing passive immunity and reducing excessive inflammation^{5,23}. Corticosteroids were also used to minimize the inflammatory cascade, with their ability to suppress the generation of pro-inflammatory cytokines supporting their utility in preventing tissue damage in severe viral infections²⁵.

The use of biologic medications in four individuals represents a significant advancement in the management of cytokine storm. Anakinra, an interleukin-1 receptor antagonist, was utilized in two patients to reduce inflammation, an approach that has shown promise in situations with overactive immune responses²⁶. The use of ruxolitinib, a Janus kinase (JAK) inhibitor, in another patient demonstrates the growing significance of targeted immunotherapies in treating cytokine storms by selectively blocking cytokine signaling pathways involved in inflammation²⁷. Tocilizumab, an IL-6 receptor antagonist, was also employed, taking advantage of its capacity to block IL-6, a critical mediator in cytokine-driven inflammation, an approach that has gained popularity in the treatment of



Table 3. Therapeutic management and outcomes of the included patients	
Therapy	N (%)
IVIG	11 (100)
Steroids	6 (54.5)
Anakinra	2 (18.2)
Acetyl salicylic acid	5 (45.5)
Tocilizumab	1 (9.1)
Clopedogril	1 (9.1)
Ruxolitinib	1 (9.1)
PICU Admission	6 (54.5)
Oxygen required	5 (45.5)
Outcome*	
Good & discharged	11 (100)
candidate for heart, lung transplant	1 (9.1)
Hospital stay (mean & range) days	24.2 (3-81)*Good clinical outcomes based on biomarker normalization which was correlated with clinical recovery

*Good clinical outcomes based on biomarker normalization which was correlated with clinical recovery inflammatory disorders such as cytokine storms²⁸.

Furthermore, using aspirin as primary anticoagulant in five patients was critical in minimizing thromboembolic consequences, as cytokine storms are frequently associated with a procoagulant condition⁴. Surprisingly, all

eleven patients showed significant clinical improvement, with no long-term consequences from treatment. This accomplishment emphasizes the value of a multidisciplinary approach that includes immunotherapy, supportive care, and anticoagulation in the treatment of severe pediatric viral infections accompanied by cytokine storms.

Limitations

This study is constrained by its retrospective, single-center design and limited sample size. The absence of standardized cytokine profiling and lack of long-term biomarker follow-up may impact the generalizability of the results. Future prospective, multicenter studies are needed to confirm the therapeutic efficacy and to establish optimal timing and selection of interventions.

CONCLUSION

These findings offer valuable insights into the management of pediatric cytokine storm syndrome, highlighting the potential benefits of individualized, immune-targeted therapies; such as IVIG, corticosteroids, and biologic agents in children with severe viral infections. The complete recovery of all patients without long-term complications emphasizes the promise of early, targeted intervention in enhancing both survival and functional outcomes.

AUTHOR CONTRIBUTION

All authors made substantial contributions to conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting the article or revising it critically for important intellectual content; agreed to submit to the current journal; gave final approval of the version to be published; and

References

1. Abuhammour W, Yavuz L, Jain R, Abu Hammour K, Al-Hammouri GF, El Naofal M, Halabi N, Yaslam S, Ramaswamy S, Taylor A, Wafadari D, Alsarhan A, Khansaheb H, Deesi ZO, Varghese RM, Uddin M, Al Suwaidi H, Al-Hammadi S, Alkhaja A, AlDabal LM, Loney T, Nowotny N, Al Khayat A, Alsheikh-Ali A, Abou Tayoun A. Genetic and Clinical Characteristics of Patients in the Middle East With Multisystem Inflammatory Syndrome in Children. JAMA Netw Open. 2022 May 2;5(5):e2214985. doi: 10.1001/jamanetworkopen.2022.14985. PMID: 35639375; PMCID: PMC9157271

2. Consiglio CR, Cotugno N, Sardh F, Pou C, Amodio D, Rodriguez L, Tan Z, Zicari S, Ruggiero A, Pascucci GR, Santilli V, Campbell T, Bryceson Y, Eriksson D, Wang J, Marchesi A, Lakshmikanth T, Campana A, Villani A, Rossi P; CACTUS Study Team; Landegren N, Palma P, Brodin P. The Immunology of Multisystem Inflammatory Syndrome in Children with COVID-19. Cell. 2020 Nov 12;183(4):968-981.e7. doi: 10.1016/j.cell.2020.09.016. Epub 2020 Sep 6. PMID: 32966765; PMCID: PMC7474869.

3. Abuhammour W and Dawoud T. "What Can We Learn From Kawasaki Disease To Treat COVID-19 Patients?". EC Paediatrics 9.6 (2020): 27-28.

4. Tang XD, Ji TT, Dong JR, Feng H, Chen FQ, Chen X, Zhao HY, Chen DK, Ma WT. Pathogenesis and Treatment of Cytokine Storm Induced by Infectious Diseases. Int J Mol Sci. 2021 Nov 30;22(23):13009. doi: 10.3390/ijms222313009. PMID: 34884813; PMCID: PMC8658039.

5. Abu Hammour K, Abu Farha R, Manaseer Q, Dawoud T, Abu Hammour W. Is COVID-19 multisystem inflammatory syndrome a new variant of Kawasaki Disease? Arch Rheumatol. 2021 Dec 24;37(2):230-244. doi: 10.46497/ArchRheumatol.2022.9086. PMID: 36017200; PMCID: PMC9377180.

6. Kajon AE. Adenovirus infections: new insights for the clinical laboratory. J Clin Microbiol. 2024 Sep 11;62(9):e0083622. doi: 10.1128/jcm.00836-22. Epub 2024 Aug 27. PMID: 39189703; PMCID: PMC11389149.

7. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, Wang B, Xiang H, Cheng Z, Xiong Y, Zhao Y, Li Y, Wang X, Peng Z. Clinical Characteristics of 138 Hospitalized Patients with 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. JAMA. 2020 Mar 17;323(11):1061-1069. doi: 10.1001/jama.2020.1585. Erratum in: JAMA. 2021 Mar 16;325(11):1113. doi: 10.1001/jama.2021.2336. PMID: 32031570; PMCID: PMC7042881.



8. Baumeister SHC, Mohan GS, Elhaddad A, Lehmann L. Cytokine Release Syndrome and Associated Acute Toxicities in Pediatric Patients Undergoing Immune Effector Cell Therapy or Hematopoietic Cell Transplantation. *Front Oncol.* 2022 Mar 24;12:841117. doi: 10.3389/fonc.2022.841117. PMID: 35402259; PMCID: PMC8989409.
9. Jarczак D, Nierhaus A. Cytokine Storm-Definition, Causes, and Implications. *Int J Mol Sci.* 2022 Oct 3;23(19):11740. doi: 10.3390/ijms231911740. PMID: 36233040; PMCID: PMC9570384.
10. Liu BM, Martins TB, Peterson LK, Hill HR. Clinical significance of measuring serum cytokine levels as inflammatory biomarkers in adult and pediatric COVID-19 cases: A review. *Cytokine.* 2021 Jun;142:155478. doi: 10.1016/j.cyto.2021.155478. Epub 2021 Feb 23. PMID: 33667962; PMCID: PMC7901304.
11. Ye Q, Wang B, Mao J. The pathogenesis and treatment of the 'Cytokine Storm' in COVID-19. *J Infect.* 2020 Jun;80(6):607-613. doi: 10.1016/j.jinf.2020.03.037. Epub 2020 Apr 10. PMID: 32283152; PMCID: PMC7194613.
12. Yaremkevych, R., Bodnarchuk-Sokhatska, N., Sydorov, S., Kyivska, Y. & Gangkui, H. (2025). The Impact of Cytokine Storms on Severe Infectious Diseases Progression: A Narrative Review. *Journal of Pioneering Medical Sciences*, 14(4), 89-97.
13. Butts RJ, Boyle GJ, Deshpande SR, Gambetta K, Knecht KR, Prada-Ruiz CA, Richmond ME, West SC, Lal AK. Characteristics of Clinically Diagnosed Pediatric Myocarditis in a Contemporary Multi-Center Cohort. *Pediatr Cardiol.* 2017 Aug;38(6):1175-1182. doi: 10.1007/s00246-017-1638-1. Epub 2017 May 23. PMID: 28536746.
14. Chaturvedi UC, Agarwal R, Elbishbishi EA, Mustafa AS. Cytokine cascade in dengue hemorrhagic fever: implications for pathogenesis. *FEMS Immunol Med Microbiol.* 2000 Jul;28(3):183-8. doi: 10.1111/j.1574-695X.2000.tb01474.x. PMID: 10865168.
15. Wu Z, Zhang R, Liu D, Liu X, Zhang J, Zhang Z, Chen S, He W, Li Y, Xu Y, Liu X. Acute Respiratory Distress Syndrome Caused by Human Adenovirus in Adults: A Prospective Observational Study in Guangdong, China. *Front Med (Lausanne).* 2022 Jan 27;8:791163. doi: 10.3389/fmed.2021.791163. PMID: 35155471; PMCID: PMC8829445.
16. Mirkarimi M, Alisamir M, Saraf S, Heidari S, Barouti S, Mohammadi S. Clinical and Epidemiological Determinants of Lower Respiratory Tract Infections in Hospitalized Pediatric Patients. *Int J Pediatr.* 2020 Nov 17;2020:8844420. doi: 10.1155/2020/8844420. PMID: 33281906; PMCID: PMC7688345.
17. Moscovitch DP, Averbuch D, Kerem E, Cohen-Cymberknob M, Berkun Y, Brooks R, Reiff S, Meir MB, Wolf D, Breuer O. Pediatric respiratory admissions and related viral infections during the COVID-19 pandemic. *Pediatr Pulmonol.* 2023 Jul;58(7):2076-2084. doi: 10.1002/ppul.26434. Epub 2023 Apr 25. PMID: 37097057.
18. Singh, Ajay & Datta, Priyanka & Sethi, Sameer & Singla, Karan. (2020). Hemophagocytic lymphohistiocytosis: a rare and fatal manifestation of dengue fever. *International Journal of Research in Medical Sciences*. 8. 10.18203/2320-6012.ijrms20200553.
19. Hay KA, Hanafi LA, Li D, Gust J, Liles WC, Wurfel MM, López JA, Chen J, Chung D, Harju-Baker S, Cherian S, Chen X, Riddell SR, Maloney DG, Turtle CJ. Kinetics and biomarkers of severe cytokine release syndrome after CD19 chimeric antigen receptor-modified T-cell therapy. *Blood.* 2017 Nov 23;130(21):2295-2306. doi: 10.1182/blood-2017-06-793141. Epub 2017 Sep 18. PMID: 28924019; PMCID: PMC5701525.
20. Annamalai A, Le Souëf P. Viral-Bacterial Interactions in Childhood Respiratory Tract Infections. *Viral Infections in Children, Volume I.* 2017 May 5:193-214. doi: 10.1007/978-3-319-54033-7_8. PMCID: PMC7122469.
21. Białka S, Zieliński M, Latos M, Skurzyńska M, Żak M, Palaczyński P, Skoczyński S. Severe Bacterial Superinfection of Influenza Pneumonia in Immunocompetent Young Patients: Case Reports. *J Clin Med.* 2024 Sep 24;13(19):5665. doi: 10.3390/jcm13195665. PMID: 39407724; PMCID: PMC11476596.
22. Bustin SA, Benes V, Nolan T, Pfaffl MW. Quantitative real-time RT-PCR--a perspective. *J Mol Endocrinol.* 2005 Jun;34(3):597-601. doi: 10.1677/jme.1.01755. PMID: 15956331.
23. Mehta P, Cron RQ, Hartwell J, Manson JJ, Tattersall RS. Silencing the cytokine storm: the use of intravenous anakinra in haemophagocytic lymphohistiocytosis or macrophage activation syndrome. *Lancet Rheumatol.* 2020 Jun;2(6):e358-e367. doi: 10.1016/S2665-9913(20)30096-5. Epub 2020 May 4. PMID: 32373790; PMCID: PMC7198216.
24. Diorio C, Teachey DT, Canna SW. Cytokine Storm Syndromes in Pediatric Patients. *J Allergy Clin Immunol Pract.* 2023 Jun;11(6):1636-1644. doi: 10.1016/j.jaip.2023.03.033. Epub 2023 Mar 27. PMID: 36990432.
25. Langarizadeh MA, Ranjbar Tavakoli M, Abiri A, Ghasempour A, Rezaei M, Ameri A. A review on function and side effects of systemic corticosteroids used in high-grade COVID-19 to prevent cytokine storms. *EXCLI J.* 2021 Feb 15;20:339-365. doi: 10.17179/excli2020-3196. PMID: 33746666; PMCID: PMC7975631.
26. Rajasekaran S, Kruse K, Kovey K, Davis AT, Hassan NE, Ndika AN, Zuiderveen S, Birmingham J. Therapeutic role of anakinra, an interleukin-1 receptor antagonist, in the management of secondary hemophagocytic lymphohistiocytosis/sepsis/multiple organ dysfunction/macrophage activating syndrome in critically ill children*. *Pediatr Crit Care Med.* 2014 Jun;15(5):401-8. doi: 10.1097/PCC.0000000000000078. PMID: 24583503; PMCID: PMC7122718.
27. Keenan C, Albeituni S, Nichols KE, Hines M. JAK Inhibitors in Cytokine Storm Syndromes. *Adv Exp Med Biol.* 2024;1448:583-600. doi: 10.1007/978-3-031-59815-9_39. PMID: 39117841.
28. Price CC, Altice FL, Shyr Y, Koff A, Pischel L, Goshua G, Azar MM, Mcmanus D, Chen SC, Gleeson SE, Britto CJ, Azmy V, Kaman K, Gaston DC, Davis M, Burrello T, Harris Z, Villanueva MS, Aoun-Barakat L, Kang I, Seropian S, Chupp G, Bucala R, Kaminski N, Lee AI, LoRusso PM, Topal JE, Dela Cruz C, Malinis M. Tocilizumab Treatment for Cytokine Release Syndrome in Hospitalized Patients With Coronavirus Disease 2019: Survival and Clinical Outcomes. *Chest.* 2020.