Journal of Community Hospital Internal Medicine Perspectives

Volume 12 | Issue 6 Article 13

2022

A Young Adult With Multisystem Inflammatory Syndrome in Adults With Concomitant Mycoplasma Infection: A Case Report

Punya Hari Dahal
Department of Hospital Medicine, Faith Regional Health Services, Norfolk, Nebraska, USA., punyahari@gmail.com

Ojbindra KC
Department of Hospital Medicine, Faith Regional Health Services, Norfolk, Nebraska, USA.

Manisha Koirala
Department of Hospital Medicine, Faith Regional Health Services, Norfolk, Nebraska, USA.

Afua Duker Ntem-Mensah
Department of Infectious Disease, Faith Regional Health Services, Norfolk, Nebraska, USA.

Recommended Citation

Dahal, Punya Hari; KC, Ojbindra; Koirala, Manisha; and Ntem-Mensah, Afua Duker (2022) "A Young Adult With Multisystem Inflammatory Syndrome in Adults With Concomitant Mycoplasma Infection: A Case Report," *Journal of Community Hospital Internal Medicine Perspectives*: Vol. 12: Iss. 6, Article 13.
DOI: 10.55729/2000-9666.1132
Available at: https://scholarlycommons.gbmc.org/jchimp/vol12/iss6/13

A Young Adult With Multisystem Inflammatory Syndrome in Adults With Concomitant Mycoplasma Infection: A Case Report

Punya H. Dahal [a,*], Ojbindra KC [a], Manisha Koirala [a], Afua D. Ntem-Mensah [b]

[a] Department of Hospital Medicine, Faith Regional Health Services, Norfolk, NE, USA
[b] Department of Infectious Disease, Faith Regional Health Services, Norfolk, NE, USA

Abstract

Several cases of Multisystem Inflammatory Syndrome in Adults (MIS-A) have been reported in adults since June 2020 after COVID-19 was first reported in December 2019. It was initially reported in children as MIS-C with Kawasaki-like disease, but a similar condition has been well recognized in adults. Although Mycoplasma co-infection has been reported with COVID-19, to our knowledge, concomitant *Mycoplasma pneumoniae* infection has not been reported together with MIS-A. We present a case of MIS-A with concomitant *M. pneumoniae* infection. It is unclear if concomitant Mycoplasma infection resulted in increased severity of the patient's illness or if it resulted in inciting the immune response in our patient who had recently recovered from COVID-19 infection. This case highlights the need to diagnose a patient with a typical presentation of MIS-A and any concomitant infection or illnesses.

Keywords: Multisystem inflammatory disorder, MIS-A, Mycoplasma, Infection, COVID-19, Coronavirus, Kawasaki-like disease

1. Introduction

COVID 19 illness has resulted in a global pandemic causing more than 380 million cases globally and more than 75 million cases in the US alone.[1] Multisystem inflammatory syndrome in children (MIS-C) is a rare but severe condition in children and adolescents infected with SARS-CoV-2.[2] Since June 2020, there have been several reports of a similar multisystem inflammatory syndrome in adults (MIS-A).[3–9] We did literature review for cases of MIS-A and concomitant Mycoplasma infection in databases such as PubMed, Google Scholar and Cochrane review for cases written in English language using terms "MIS-A″, "COVID-19" and "Mycoplasma". We were unable to find any reported cases. Here, we present a case of MIS-A with concomitant *Mycoplasma pneumoniae* infection.

2. Case presentation

A healthy 22-year-old Hispanic male with no significant past medical history presented to the emergency room (ER) with fever, neck pain, redness of eyes, and abdominal pain of two days. He was diagnosed with COVID-19 infection five weeks prior to the presentation. After the complete resolution of his symptoms and two negative COVID-19 polymerase chain reaction (PCR) tests, he had returned to his work.

On presentation to ER, the vitals were blood pressure of 114/65 mmHg, heart rate of 96 beats per minute, temperature of 100.8 Fahrenheit, respiratory rate of 16 and oxygen saturation of 100%.

Received 22 July 2022; revised 22 September 2022; accepted 26 September 2022.
Available online 7 November 2022

* Corresponding author at: Faith regional health services, Hospital Medicine Department, 1400 Amberwood Dr apt 8, 2700 W Norfolk Ave, Norfolk, NE 68701, USA.
E-mail addresses: punyahari@gmail.com (P.H. Dahal), ojbindra@gmail.com (O. KC), mkoirala@frhs.org (M. Koirala), anmensah@frhs.org (A.D. Ntem-Mensah).

https://doi.org/10.55729/2000-9666.1132

He was found to have non-purulent conjunctivitis, cervical lymphadenopathy, thrombocytopenia, elevated bilirubin, mildly elevated liver function tests (LFTs). The Mononucleosis screen test (Monospot) for heterophile antibodies was negative. He was prescribed azithromycin and prednisone and was discharged from ER.

The next day, he presented back to the ER with persistent fevers, neck pain, trismus, generalized body ache, abdominal pain, and redness of eyes. He was febrile with a temperature of 103.7 Fahrenheit, tachycardic with a heart rate of 125 beats per minute, his respiratory rate was 18 and blood pressure was 100/50 mmHg. On physical examination, he had cervical lymphadenopathy and non-purulent conjunctivitis. The rest of the physical examination was unremarkable. The laboratory workups revealed lymphopenia, thrombocytopenia, hyponatremia, elevated bilirubin, and mildly elevated LFTs. His COVID-19 PCR was positive in the respiratory viral panel; however, the nasal SARS-CoV-2 antigen test was negative. The rest of the laboratory workup was unremarkable, as shown in Table 1. Additional workup including mononucleosis screen, *Group A Streptococcus,* HIV 1 and 2 Antigen, antibody tests, and hepatitis panel were negative. CT neck revealed cervical lymphadenopathy (Fig. 1). Chest X-ray was unremarkable. US abdomen revealed increased echogenicity along portal veins, gallbladder sludge, normal caliber hepatic ducts, negative Murphy's sign. He was continued on prednisone and azithromycin and admitted to the medical floor.

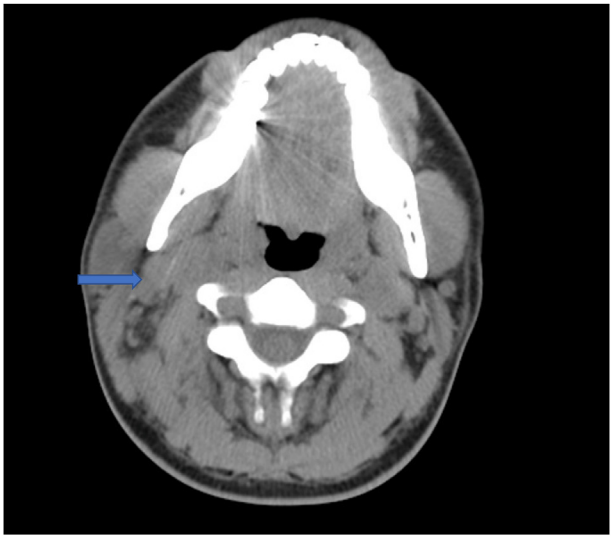

Fig. 1. Level II cervical lymphadenopathy in CT soft tissue neck.

On day 2, his fever persisted, and he developed hypoxia requiring 1−2 Liter/min oxygen via nasal cannula. He also developed hypotension and required intravenous fluids and subsequently norepinephrine to maintain the blood pressure. His hypoxia gradually worsened, requiring 12 L/min oxygen via a hi-flow nasal cannula. Repeat laboratory workups revealed persistent thrombocytopenia, elevated LFTs, elevated inflammatory markers-elevated d dimer, CRP, ferritin, and procalcitonin (Table 1). Blood cultures showed no growth. Peripheral smear showed normochromic

Table 1. The laboratory workup during hospitalization.

	Ref range	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
WBC count	4−10 × 1000/cmm	7.7	7.6	9.6	9.4	9.2	10.1	11.5
Neutrophil count	1.6−8 × 1000/cmm	6.8	6.7	8.4	7.8	6.8	6.5	7.1
Lymphocyte count	0.8−4.5 × 1000/cmm	0.4	0.7	0.9	1.3	1.5	2.5	3.4
Hemoglobin	13.5−17 g/dL	14.1	12.8	12.5	13.4	12.9	13.7	13.3
Platelet count	150−450 × 1000/cmm	73	58	82	121	117	144	186
Sodium	135-146 mEq/L	130	136	138	136	135	136	136
Potassium	3.5−5.3 mEq/L	3.8	4.2	4.2	4.0	4.2	4.1	3.8
Calcium	8.5−10.4 mg/dL	9.3	7.8	8.1	8.2	7.8	7.6	7.8
Blood Urea Nitrogen	7−25 mg/dL	16	20	21	29	27	25	22
Creatinine	0.7−1.3 mg/dL	1.3	1.2	0.8	0.9	0.9	0.9	0.8
Total bilirubin	0.1−1.5 mg/dL	5.8	6.2	2.9	1.8	1.8	1.4	1.3
Aspartate aminotransferase	2−50 U/L	59	63	125	79	113	66	55
Alanine aminotransferase	2−60 U/L	87	81	133	141	169	160	155
Alkaline phosphatase	20−125 U/L	134	118	106	94	87	109	108
International normalized ratio	0.9−1.1	N/C*	1.41	1.24	N/C*	N/C*	N/C*	N/C*
C-Reactive protein	<0.8 mg/dL	N/C*	23.7	24.9	13.5	7.2	N/C*	2.9
D-dimer	<230 ng/mL	N/C*	2241	1376	841	821	N/C*	N/C*
Ferritin	20−345 ng/mL	N/C*	1547	1970	1993	N/C*	N/C*	933
Procalcitonin	<0.1 ng/mL	N/C*	5.91	N/C*	1.47	0.78	0.42	0.25
Troponin, hs	<53 ng/L	N/C*	169	110	N/C*	N/C*	N/C*	N/C*
Brain natriuretic peptide	0−100 pg/mL	N/C*	1072	N/C*	N/C*	N/C*	N/C*	N/C*
Lactic acid	0.5−2 mmol/L	1.3	N/C*	N/C*	N/C*	N/C*	N/C*	N/C*

N/C*- Not Checked.

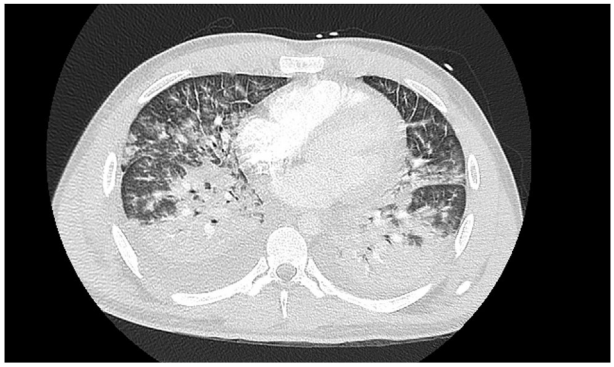

Fig. 2. CT chest with alveolar and interstitial edema, bilateral posterior lower lobe consolidations, bilateral pleural effusions.

normocytic anemia, absolute lymphopenia, and thrombocytopenia without evidence of micro-angiopathic hemolytic anemia. The *M. pneumoniae* IgM was noted to be positive. Due to worsening hypoxia, a CT angiogram of the chest was done, which revealed pulmonary vascular congestion with alveolar and interstitial edema, bilateral posterior lower lobe consolidations, moderate right and small left pleural effusions but no evidence of pulmonary embolism (Fig. 2). Bilateral lower and upper extremities duplex was negative. He developed atrial fibrillation with a rapid ventricular response (Fig. 3 and converted to sinus rhythm after receiving intravenous metoprolol. He was started on broad-spectrum antibiotics piperacillin-tazobactam and vancomycin.

On day 3, his condition continued to worsen with persistent fever, tachycardia, hypoxia, hypotension requiring norepinephrine, cervical lymphadenopathy, trismus, non-purulent conjunctivitis, lymphopenia, thrombocytopenia, elevated cardiac biomarkers including troponin, brain natriuretic peptide, elevated inflammatory markers including CRP, D-dimer, ferritin, procalcitonin. These findings were concerning for MIS-A, and intravenous immunoglobulin (IVIG) 25 g was administered. He was continued on vancomycin, piperacillin-tazobactam, azithromycin, and steroids. Vancomycin was later discontinued after MRSA nasal screen was negative.

On day 4, the patient had a significant improvement; he was weaned off norepinephrine, and oxygen was weaned off to room air as well. He had improvement in his white blood cell counts, lymphocyte counts, inflammatory markers and platelet counts along with bilirubin (see Fig.4, Fig. 5). He continued to improve significantly over the next three days and was discharged on day 7. He was discharged on a tapering dose of dexamethasone to be completed over the next two weeks. On post-discharge follow-up in two weeks, he had complete resolution of his symptoms.

3. Discussion

Center of Disease Control (CDC) defines MIS-A as a patient aged ≥21 years hospitalized for ≥24 h, or with an illness resulting in death, who meets the following clinical and laboratory criteria. The patient should not have a more likely alternative diagnosis for the illness (e.g., bacterial sepsis, exacerbation of a chronic medical condition).[10]

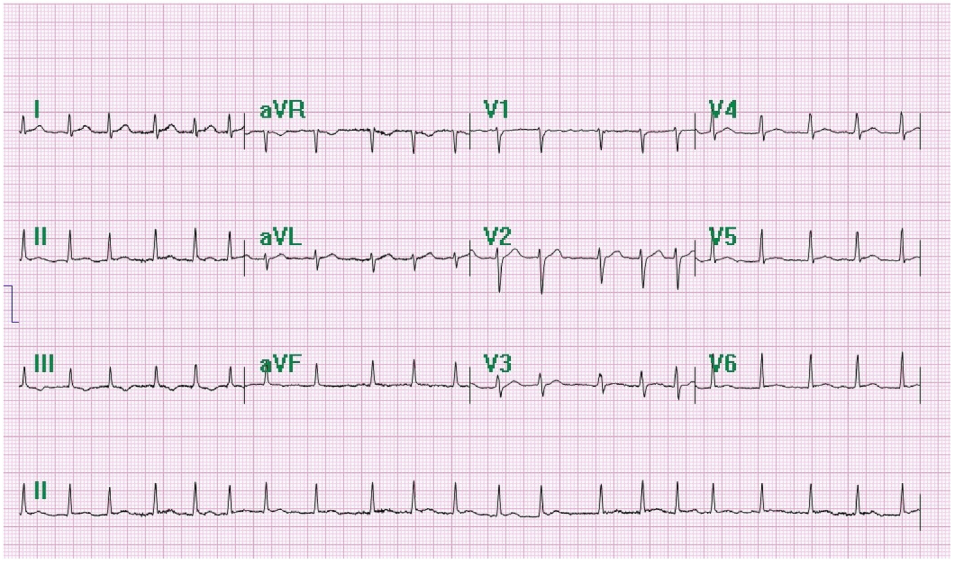

Fig. 3. EKG with atrial fibrillation and rapid ventricular response.

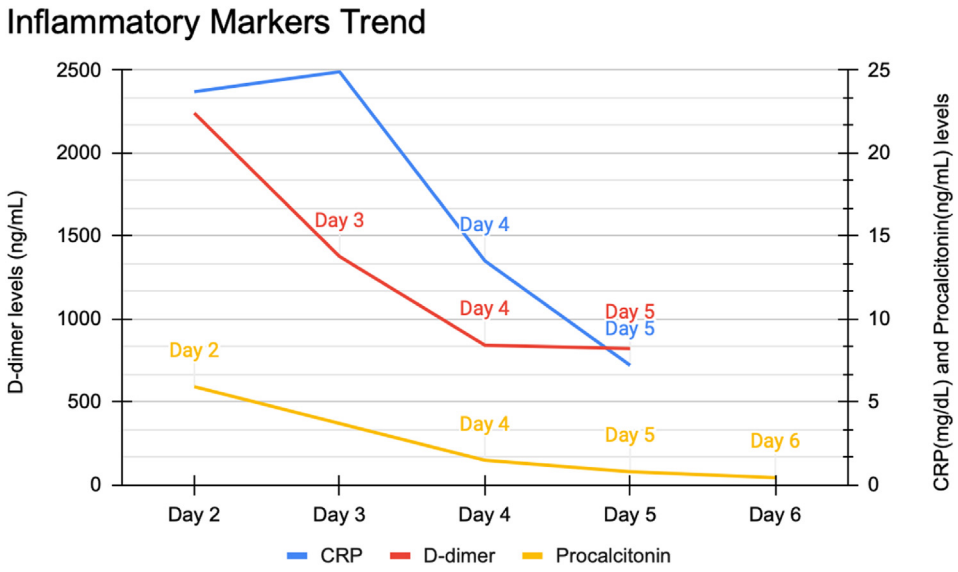

Fig. 4. The trend of inflammatory markers during hospitalization.

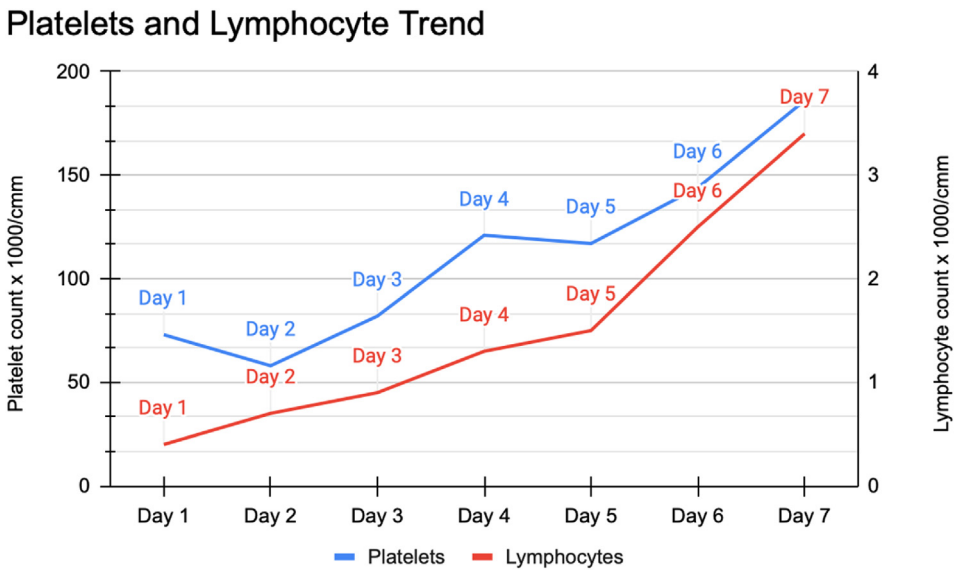

Fig. 5. The trend of platelets and lymphocyte counts during hospitalization.

3.1. Clinical criteria

- Subjective fever or documented fever (≥38.0 C) for ≥24 h prior to hospitalization or within the first 3 days of hospitalization and at least 3 of the following clinical criteria occurring prior to hospitalization or within the first 3 days of hospitalization. At least 1 must be a primary clinical criterion.
  - o Primary clinical criteria
    1. Severe cardiac illness-Includes myocarditis, pericarditis, coronary artery dilatation/aneurysm, or new-onset right or left ventricular dysfunction (LVEF<50%), 2nd/3rd degree A-V block, or ventricular tachycardia
    2. Rash and non-purulent conjunctivitis
- Secondary clinical criteria
  1. New-onset neurologic signs and symptoms-Includes encephalopathy in a patient without prior cognitive impairment, seizures, meningeal signs, or peripheral neuropathy (including Guillain-Barré syndrome)
  2. Shock or hypotension not attributable to medical therapy (e.g., sedation, renal replacement therapy)
  3. Abdominal pain, vomiting, or diarrhea

4. Thrombocytopenia (platelet count <150,000/microliter)

3.2. Laboratory evidence

- The presence of laboratory evidence of inflammation and SARS-CoV-2 infection.
  - o Elevated levels of at least 2 of the following: CRP, ferritin, IL-6, erythrocyte sedimentation rate, procalcitonin
  - o A positive SARS-CoV-2 test for current or recent infection by RT-PCR, serology, or antigen detection

Our patient met criteria for MIS-A with documented fever, cardiac illness with myocarditis and atrial fibrillation with a rapid ventricular response, non-purulent conjunctivitis, hypotension/shock not attributable to medical therapy requiring norepinephrine, abdominal pain, thrombocytopenia, elevated inflammatory markers including CRP, ferritin and procalcitonin and evidence of recent COVID-19 infection around five weeks prior to presentation. Other possible causes for this type of presentation were ruled out with diligent workup and testing. Additional workup for bacterial superimposition was done and our patient was found to have a concomitant *M. pneumoniae* infection. Antibiotic therapy with macrolides, fluoroquinolones, or tetracyclines is the mainstay of treatment for Mycoplasma pneumonia.[11] Current data from CDC suggest that the prevalence of macrolide resistance in *M. pneumoniae* may be around 10% in the United States, with regional variability.[11] Hence our patient was treated with azithromycin.

Although Mycoplasma co-infection has been reported with COVID-19, to our knowledge, concomitant *M. pneumoniae* infection has not been reported together with MIS-A.[12] However, Mycoplasma co-infection has been reported together with Multisystem Inflammatory Syndrome in Children (MIS-C).[13] It has been shown that *M. pneumoniae* co-infection in pediatric patients with MIS-C may contribute to a more severe clinical course.[13] It is unclear whether Mycoplasma infection incited an immune response or if concomitant Mycoplasma infection caused the symptoms and clinical picture to be more severe in our patient. Further studies are needed to understand more about MIS-A and any potential inciting factors, including infection, that could result in the multisystem inflammatory response in a patient with recent COVID-19 infection with subsequent recovery. It is prudent to diagnose concomitant illnesses or infections in a patient with MIS-A and treat them appropriately.

4. Conclusion

It is important to diagnose MIS-A in a patient with a delayed immunologic response to SARS-CoV-2 infection in adults with hyperinflammation. The treatment mainly includes corticosteroids and IVIG, along with supportive management. It is also important to diagnose any concomitant infection or illnesses and treat them accordingly. More studies are needed to understand more about this phenomenon and understand if there are any factors such as an infection or illnesses inciting an immune response resulting in multisystem inflammatory disorder.

Conflict of interest

The authors declare that we have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

The authors have received no funding and have no multiplicity of interests to disclose.

References

1. Centers for disease Control and prevention coronavirus-19 (COVID-19) tracker. Center for disease Control and prevention website. Updated February 1, 2022. https://covid.cdc.gov/covid-data-tracker/#datatracker-home. Accessed February 1, 2022
2. Multisystem inflammatory syndrome in children (MIS-C) associated with coronavirus disease 2019 (COVID-19). Center for disease Control and prevention website. Updated on May 14, 2020. https://emergency.cdc.gov/han/2020/han00432.asp. Accessed February 1, 2022
3. Morris SB, Schwartz NG, Patel P, et al. Case series of multisystem inflammatory syndrome in adults associated with SARS-CoV-2 infection—United Kingdom and United States, March–August 2020. *MMWR Morb Mortal Wkly Rep*. 2020;69(40):1450. https://doi.org/10.15585/mmwr.mm6940e1external icon.
4. Sokolovsky S, Soni P, Hoffman T, Kahn P, Scheers-Masters J. COVID-19 associated Kawasaki-like multisystem inflammatory disease in an adult. *Am J Emerg Med*. 2021;39:253-e1. https://doi.org/10.1016/j.ajem.2020.06.053.
5. Shaigany S, Gnirke M, Guttmann A, et al. An adult with Kawasaki-like multisystem inflammatory syndrome associated with COVID-19. *Lancet*. 2020;396(10246):e8-10. https://doi.org/10.1016/S0140-6736(20)31526-9.
6. Newton-Cheh C, Zlotoff DA, Hung J, Rupasov A, Crowley JC, Funamoto M. Case 24-2020: a 44-year-old woman with chest pain, dyspnea, and shock. *N Engl J Med*. 2020;383(5):475–484. https://doi.org/10.1056/NEJMcpc2004975.
7. Jones I, Bell LC, Manson JJ, Last A. An adult presentation consistent with PIMS-TS. *Lancet Rheumatol*. 2020;2(9):e520–e521. https://doi.org/10.1016/S2665-9913(20)30234-4.
8. Fox SE, Lameira FS, Rinker EB, Vander Heide RS. Cardiac endotheliitis and multisystem inflammatory syndrome after COVID-19. *Ann Intern Med*. 2020;173(12):1025–1027. https://doi.org/10.7326/L20-0882.
9. Kofman AD, Sizemore EK, Detelich JF, Albrecht B, Piantadosi AL. A young adult with COVID-19 and multisystem inflammatory syndrome in children (MIS-C)-like

illness: a case report. *BMC Infect Dis*. 2020;20(1):1−4. https://doi.org/10.1186/s12879-020-05439-z.
10. Multisystem inflammatory syndrome in adults (MIS-A). Center for disease Control and prevention website. Updated November 13, 2020 https://www.cdc.gov/mis/mis-a.html. Accessed February 1, 2022.
11. Mycoplasma pneumoniae infections. Center for disease Control and prevention website. Updated on August 24, 2022 https://www.cdc.gov/pneumonia/atypical/Mycoplasma/hcp/antibiotic-treatment-resistance.html. Accessed September 22, 2022.
12. Huang AC, Huang CG, Yang CT, Hu HC. Concomitant infection with COVID-19 and Mycoplasma pneumoniae. *Biomed J*. 2020; 43(5):458−461. https://doi.org/10.1016/j.bj.2020.07.002.
13. Plebani A, Meini A, Cattalini M, et al. Mycoplasma infection may complicate the clinical course of SARS-Co-V-2 associated Kawasaki-like disease in children. *Clin Immunol*. 2020 Dec;221:108613. https://doi.org/10.1016/j.clim.2020.108613.