Received: 9 August 2022 | Revised: 29 September 2022 | Accepted: 4 November 2022

DOI: 10.1002/ccr3.6676

CASE REPORT

Clinical Case Reports Open Access WILEY

Pneumonia in the presentation of Kawasaki disease: The syndrome or a sequence of two diseases?

Lana Khoury[1] | **Galit Livnat**[2,3] | **Mohamad Hamad Saied**[4] | **Karin Yaacoby-Bianu**[2]

[1]Department of Pediatrics, Carmel Medical Center, Haifa, Israel

[2]Pediatric Pulmonology Unit and CF Center, Carmel Medical Center, Haifa, Israel

[3]B. Rappaport Faculty of Medicine, Technion–Israel Institute of Technology, Haifa, Israel

[4]Pediatric Rheumatology Unit, Carmel Medical Center, Haifa, Israel

Correspondence
Karin Yaacoby-Bianu, Pediatric Pulmonology Unit and CF Center, Carmel Medical Center, 7 Michal St, Haifa 3436212, Israel.
Email: karinya@clalit.org.il

Abstract

Two cases of Kawasaki disease (KD) presented as persistent lung consolidation associated with *Group A Streptococcus* and *Influenza A* co-infection, which resolved following intravenous immunoglobulin. Thus, pediatricians should consider the diagnosis of KD in the presence of pneumonia that is nonresponsive to antibiotic therapy with prolonged fever and inflammatory reactions.

KEYWORDS

case report, *Influenza*, Kawasaki disease, pulmonary involvement, *Streptococcus*

1 | BACKGROUND

Kawasaki disease (KD) is a medium vessel multi-systemic vasculitis that predominantly affects children younger than 5 years old.[1] The diagnosis of classic KD is based on the presence of ≥5 days of fever and ≥4 of five principal clinical features: polymorphous exanthem, extremity changes, mucosal involvement of the lips and oral cavity, bilateral non-purulent conjunctivitis, and unilateral cervical lymphadenopathy.[2] These criteria unfortunately do not identify all children with the illness. Therefore, patients who do not have sufficient clinical findings can be diagnosed with incomplete or atypical KD by fulfilling some of the typical criteria with other clinical, laboratory, or echocardiographic findings.[3] The reported prevalence of incomplete presentation is 15 to 36.2% among patients with KD.[4,5] There is no definitive diagnostic test for KD, thus accurate and timely diagnosis of KD is challenging for clinicians.

The etiology of KD remains unclear, though various hypotheses regarding the role of an infectious trigger and bacterial super antigens have been postulated.[2] Although inflammation of the coronary arteries is the most dreaded clinical outcome, KD is characterized by systemic inflammation in all medium-sized arteries and in multiple organs and tissues during the acute febrile phase,[6] leading to increased vascular permeability and associated clinical findings.

The pulmonary manifestations of KD may be diverse but are distinctly uncommon and often unrecognized. Careful monitoring is essential because some features of KD may evolve over time. Early identification of patients who have pulmonary presentation may prevent delays in diagnosis and allow early initiation of appropriate therapy. Singh et al,[7] showed that only 1.83% of children diagnosed with KD (11/602) had a predominant pulmonary presentation, and the first sign of KD was noted at a mean duration

Lana Khoury, Galit Livnat, Equal contribution.

of 14.5 days from the onset of symptoms. Lee et al,[8] in the largest cohort of KD patients with pulmonary involvement, reported 15% with concurrent pneumonia (54/358).

Chest radiological findings in patients with KD are uncommon (~20%) in the acute phase and may include an interstitial pattern, alveolar infiltrates, subsegmental atelectasis, and pleural effusion.[9]

In this article, we report two cases of KD presenting as pneumonia associated with GAS and *Influenza A* co-infection that were admitted to the Pediatric Department in Carmel Medical Center, Haifa, Israel, in February 2019 and January 2020, respectively.

2 | CASE PRESENTATIONS

2.1 | Case 1

A previously healthy and immunized 15-month-old girl presented with a 4 days history of fever, coughing, and vomiting. On the second day of fever, a rash was described. On admission to the emergency room (ER) she was weak, pale, and sleepy. Vital signs were 147 beats per minute, temperature 37.9°C, respiratory rate 32 per minute, and oxygen saturation 98% in room air. On physical examination, she had a diffused rash, pealing on the face and lips, signs of left otitis media, clear lung sounds, normal heart auscultation other than tachycardia. There were no meningeal signs or papilledema. Initial laboratory blood results (Table 1) revealed minimally elevated white blood cell count, mildly low hemoglobin level, normal platelet count, and elevated inflammatory markers: C-reactive protein (CRP) 24 mg/dL and erythrocyte sedimentation rate (ESR) of 48 mm/h. Due to her ill appearance, she underwent a lumbar puncture (Table 1), with no signs of infection.

An anteroposterior chest X-ray (CXR) (Figure 1A) showed blurring of the left diaphragm that was interpreted as left lower lobe (LLL) pneumonia. In addition, she was found to be *Influenza A* positive by a multiplex respiratory viruses polymerase chain reaction (PCR). She was started on a third generation Cephalosporin plus Oseltamivir for 5 days and was admitted to the pediatric department.

Bacterial cultures from blood, urine, and CSF were negative. On the third day following admission, a second CXR (Figure 1B) was performed and revealed a LLL consolidation with mild pleural effusion. Chest US supported the findings and revealed left moderate pleural effusion with growing septations on follow ups. Therefore, Clindamycin was added in combination with Ceftriaxone. Further workout ruled out *RSV, Metapneumovirus, Rhino virus, Adeno virus, Parainfluenza 1/2/3, Measles*, *EBV*, *CMV,* and *Enterovirus* infections. The patient underwent thoracentesis to characterize the pleural effusion on the 5th day (Figure 1C). Forty ml of sero-bloody fluid were drained. The laboratory findings of the fluid (Table 1) were compatible with exudative pleural effusion according to Light's criteria.[10] PCR from Pleural fluid revealed *Streptococcus pyogenes*.

On the 10th day of fever, despite administration of wide-spectrum antibiotics, Oseltamivir and thoracentesis, the child continued to appear ill and irritable. On physical examination, a strawberry tongue was observed, in addition to a pealing rash on her face, chest and genitalia. Repeated blood samples (Table 1) showed an elevation of acute phase reactants (ESR 90) and an extremely elevated thrombocyte count, up to 1025 K/ul.

A diagnosis of Incomplete Kawasaki was made by fulfilling the criteria of polymorphous exanthem, mucosal involvement of the oral cavity, elevated inflammatory markers, and laboratory findings: anemia, elevated platelets count, elevated white blood cell (WBC) count, and low albumin. The patient received a dose of 2 g/kg of intravenous immunoglobulin (IVIG), with immediate relief of fever and improvement in her general condition. Periungual desquamation of hands developed later. Aspirin was not administered due to risk of Reye Syndrome during active infection with *Influenza A*. Instead, nonsteroidal anti-inflammatory drugs (NSAID's, Ibuprofen 10 mg/Kg X3/day) were administered as an anti-inflammatory agent. She completed 3 weeks of antibiotics (switched to oral Amoxicillin when she clinically improved).

Heart Echocardiogram was obtained on admission and was normal. On the 10th day of fever, it showed mild to moderate pericardial effusion which resolved on Day 14, with intact coronary arteries at pediatric cardiology follow-up during the following 2 months. No pulmonary or cardiology sequelae were evident after one-year of follow-up.

2.2 | Case 2

A previously healthy and immunized 28-month-old girl presented with a 3 days history of fever, coughing, rhinorrhea, and vomiting. The parents described a sleepy child with no appetite. A chest X-ray performed on the second day of fever was normal.

On admission to the ER, she appeared ill, tachycardic with 156 beats per minute, had normal blood pressure, temperature of 38.6°C, oxygen saturation 98% in room air. On physical examination, she had no meningeal signs, a diffuse maculopapular rash was noticed on her face, chest, and back. Erythematous, swollen

TABLE 1 Laboratory results

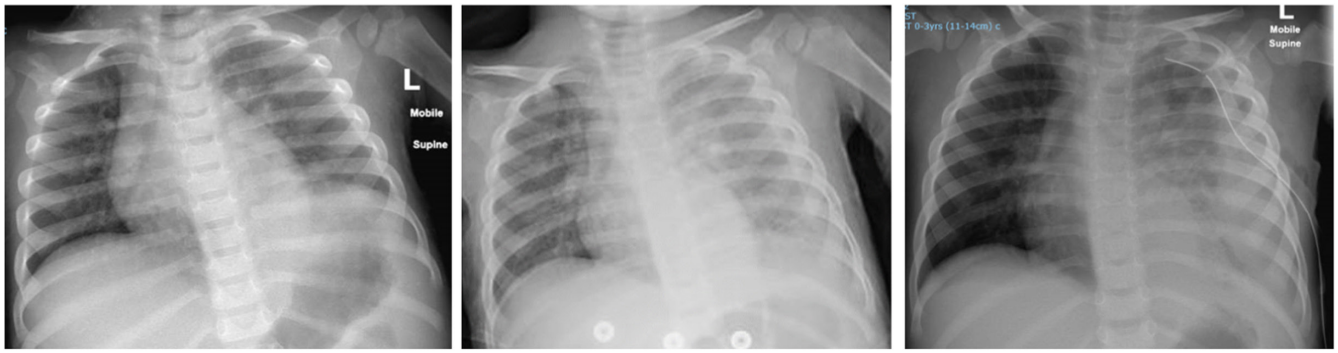
	Case 1		Case 2	
	At the day of admission	**At the 10th day of fever**	**At the day of admission**	**Normal range**
Blood				
WBC (k/μL)	**11.09**	**21**	**11.6**	1–32 months (6–14) 2–9 years (4–12)
ANC (k/μL)	**10.31**	**9.24**	**9.35**	3–5.8
Hb (g/dl)	**10.8**	**8.8**	**10.5**	1–32 months (10.5–14) 2–9 years (11.5–14.5)
Platelets (k/μL)	359	**1025**	**423**	150–400
Na (mEq/l)	**130**	138	**132**	134–144
K (mEq/l)	3.8	**5.4** (hemolytic)	4.2	3.3–4.6
Creatinine (mg/dL)	0.48	0.13	0.42	0.03–0.5
Urea (mg/dL)	**39**	17	16	5–18
AST (U/L)	**80**	16	23	20–60
ALT (U/L)	**52**	9	6	5–45
ALP (U/L)	**1600**	**810**	83	145–420
GGT (U/L)	15	17	9	5–32
LDH (U/L)	**1018**	**532**	**590**	150–500
Albumin (g/dL)		**2.7**		3.4–4.2
Ferritin (ng/mL)		**412**		10–60
CRP (mg/dL)	**24**	**16**	**9.6**	0.08–0.79
ESR (mm/h)	**48**	**90**		0–10
CSF			Two days following admission	
WBC (cells/μL)	2		1	0–5
RBC (cells/μL)	4		**45**	0
Glucose (mg/dL)	107		122	>50
Protein (mg/dL)	9		10	<45
Pleural fluid				
WBC (cells/μL)	720			
Segments percentage (out of total WBC)	60%			
pH	7.51			
Glucose (mg/dL)	41			
LDH (blood at same time) (mg/dL)	2829 (715)			
Protein (blood at same time) (mg/dL)	4.2 (6.7)			

Abbreviations: ALP, alkaline phosphatase; ALT, alanine transaminase; ANC, absolute neutrophilic count; AST, aspartate transaminase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; GGT, gamma-glutamyl transferase; Hb, hemoglobin; K, potassium; LDH, lactic dehydrogenase; Na, sodium; pH, power of hydrogen; RBC, red blood cells; WBC, white blood cells.

Bold values are values above or below the normal range for that value.

tonsils were noticed with no white exudate or cervical lymphadenopathy. On lung auscultation, decreased air entry over the left lung was heard, without crackles or wheezing. Initial laboratory blood results (Table 1) revealed an elevated white blood cell count, minimally elevated platelet count, and elevated CRP 9.6 mg/dL. CXR showed bilateral peri-hilar peri-bronchial thickening. *Influenza A* was positive by a multiplex respiratory viruses PCR. She was admitted to the pediatric department with Oseltamivir treatment, for further evaluation and supportive care.

Two days following admission, she was lethargic; LP was performed and was normal (Table 1). Bacterial cultures from blood and CSF and serology for EBV and CMV



(A) On admission (4th day of fever) **(B)** On 3rd day (7th day of fever) **(C)** On 5rd day (9th day of fever)

FIGURE 1 Chest X-rays of case 1

were negative. Physical examination revealed pus eruption from her left ear, and crackles were heard over the lower part of the left lung. Thus, a second CXR was performed, which revealed LLL consolidation. In addition, a throat culture and an exudate ear culture were both positive for *group A*. *Streptococci* (GAS). In order to treat the lobar pneumonia, purulent otitis media, and GAS tonsillitis, she was started on a third generation Cephalosporin in addition to Oseltamivir.

On the 4th day of hospitalization (7th day of fever), new signs on physical examination were found, including cervical lymphadenopathy, red swollen lips with strawberry tongue, extremity edema, pealing perineal skin, and a rash that was described at admission. Laboratory examination revealed CRP 19 mg/dL, ESR 78 mm/h, albumin 2.6 g/dL, and platelet count of 562 K/ul. Heart echocardiogram was performed and was normal.

A diagnosis of complete Kawasaki was made by fulfilling the criteria of polymorphous exanthem, extremity changes, mucosal involvement of the lips and oral cavity, and unilateral cervical lymphadenopathy. The patient received 2 g/kg of IVIG, with decrement of fever. Thirty-six hours following the end of the IVIG infusion the fever relapsed. Our assumption was that she had KD with first dose IVIG resistance. On the contrary, the patient was persistently lethargic therefore to rule out a brain abscess, a CT scan of the brain was done and was normal. A second dose of IVIG was administered, and immediate relief of fever was observed. In addition, she was treated with NSAID's (Ibuprofen 10 mg/Kg X3/day) as an anti-inflammatory agent, and Aspirin as an anti-thrombotic, especially since her platelet count was elevated up to 1111 K/ul. She completed a week of Ceftriaxone and then 10 days of Amoxicillin. The pediatric cardiology follow-up visits over the next 2 months were all normal. Aspirin administration was stopped 6 weeks after the diagnosis. No pulmonary or cardiology sequelae were evident after one-year of follow-up.

3 | DISCUSSION

We reported on two cases of KD that presented with pulmonary involvement which is an uncommon presentation of KD. These children with predominant pulmonary manifestations often have a significant delay in diagnosis, since the patient has a "source" of infection in the CXR, and the physician attributes the fever and the ill appearance to a lung infection.

In order to diagnose these deceptive cases, the physician should have a high index of suspicion especially in cases where the patient does not respond to conventional treatment with antimicrobials, and the patient has persistent fever and ill appearance together with elevated inflammatory markers. In these types of cases, a diagnosis of inflammatory disease such as KD is a possibility in the differential diagnosis whether it is a typical or an atypical disease.

Arslanoglu Aydin et al.,[11] in a recent review of 16 articles identified only 20 patients with pleural effusion due to KD, and only 11 of them presented with respiratory symptoms. Was KD triggered by the infection of the pleural space, or were the pulmonary findings features of the inflammation of KD? These are questions that should be investigated further by examining more cases of KD with pleural effusion.

The two cases described, are associated with *GAS* and *influenza A* co-infection. The etiology of KD remains unclear, and no consistent association between any infectious agent and KD has been demonstrated to date, more than half a century since the discovery of KD. While approximately 35% of children with KD have a concurrent infection,[12] the presence of infection does not rule out the diagnosis of KD and ascribing the fever to such an infection or to KD requires clinical judgment.

In an English language literature search, only two case reports were found[13,14] describing the comorbidity of atypical KD and *GAS* pleural effusion in healthy children.

TABLE 2 Comparison between the two reported cases of KD (*GAS* and *influenza A* co-infection) and cases with influenza or Streptococcus only infections

	Case 1	Case 2	Influenza and KD Joshi et al. (15)	Influenza and KD Wang et al (16)	Strep and KD Hendaus et al (13)	Strep and KD Leahy et al. (14)
Pathogen	**GAS and influenza A**	**GAS and influenza A**	**influenza A (H1N1/09)**	**influenza A (H1N1)**	**GAS**	**GAS**
Age (years)	1	2	5	1	3	3
Gender	F	F	M	M	F	M
Cough	+	+	+	+	+	+
Respiratory Rate (per minute)	32		24	38	50	
Heart Rate (bpm)	147	156	122	138	130	190
Oxygen Saturation	98%	98%	99%	94%	90% (on 2 L oxygen)	
Duration of fever till diagnosis (days)	10	7	9	7	13	14
Examination findings suggestive of KD:						
(a) Polymorphous exanthem	+	+	+	+	−	−
(b) Mucosal involvement	+	+	+	−	−	−
(c) Bilateral non-purulent conjunctivitis	−	−	+	−	−	−
(d) Cervical lymphadenopathy	−	+	+	−	−	−
(e) Extremity changes	Late desquamation	+	+	+	Late desquamation	Late desquamation
WBC (10^9/L)	11	11.6	16.1	11.1	24	30.1
Platelets count (10^9/L)	359	423	191	195	206	804
CRP (mg/L)	240	96	148	>50	100	214
ESR (mm/min)	48		134	69	65	103
Treatment	IVIG	2 doses of IVIG	IVIG	IVIG	IVIG	IVIG
Echo findings during the acute phase	Mild to moderate pericardial effusion	Normal	Normal	The inner diameter of the left main coronary artery was dilated, and the double coronary artery was not smooth.	Prominent left anterior descending artery and left main coronary artery	Prominent left anterior descending artery and left main coronary artery
Echo findings following 2 months	Normal	Normal	Normal	Normal	Normal	Persistent dilation of the left anterior descending artery and left main coronary artery

(Continues)

TABLE 2 (Continued)

	Case 1	Case 2	Influenza and KD Joshi et al. (15)	Influenza and KD Wang et al (16)	Strep and KD Hendaus et al (13)	Strep and KD Leahy et al. (14)
Pathogen	GAS and influenza A	GAS and influenza A	influenza A (H1N1/09)	influenza A (H1N1)	GAS	GAS
Chest X-Ray findings	LLL consolidation with mild pleural effusion	LLL consolidation	Minor peribronchial wall thickening bilaterally but no focal collapse or consolidation	Normal	Right middle and lower lobar consolidation with moderate pleural effusion	Large right-sided, lower lobe pneumonia with associated loculated pleural effusion

Regarding *Influenza* A with concomitant KD there are two case reports.[15,16] Table 2 shows a comparison between our two cases and these four cases.

In a retrospective study[17] that reported a 4.8% rate of KD with concomitant *Influenza*, patients in the KD and Flu group exhibited longer time to KD diagnosis, longer duration of fever, and higher CRP and ESR values compared with patients in the only Flu or only KD groups.[17] To the best of our knowledge, this is the first description of cases of KD with GAS and *influenza A* co-infection. Influenza infection is potently immunomodulatory at the bronchial epithelial surface, leading to sustained desensitization to subsequent pathogen challenge and is associated with changes in bacterial colonization patterns.[18,19] This supports the hypothesis that the development of KD is dependent on a permissive genetic and environmental host phenotype that may be facilitated by previous or concurrent infection. Hence, the potential contribution of GAS and *influenza* co-infection to KD etiology requires further study.

Atypical clinical course and infectious-like manifestations can cause delays in the accurate diagnosis and treatment of KD. The lack of response to prolonged wide spectrum parenteral antibiotic treatment and the complete response to IVIG also support the diagnosis of KD. In general, IVIG should be initiated as early as possible, within the first 10 days of fever onset as soon as the diagnosis can be established. Timely treatment is necessary in order to decrease the risk of coronary artery lesions which may develop in ~15–25% of untreated children and may lead to ischemic heart disease or sudden death.[1] Indeed, in these two cases no cardiac aneurisms were reported in the cardiologic follow-up, perhaps due to the relatively early IVIG treatment.

Two dilemmas arose while treating these two patients. The first is whether to continue the administration of antibiotics after the diagnosis of KD. Both patients were treated with wide spectrum antibiotics and there was no reduction of fever. Therefore, if the pulmonary manifestation is a total KD presentation, there is no need for treating the patient with antibiotics. On the contrary, bacterial super-antigens have been associated with the development of KD,[20] therefore, KD may be a post infectious manifestation. Thus, treating the patient with antibiotics may be essential. In these two cases we decided to treat the patients with a full antibiotic regime for bacterial pneumonia.

The second dilemma was the use of high dose aspirin as an anti-inflammatory agent as part of KD treatment. Reye syndrome is a non-inflammatory encephalopathy associated with fatty degeneration of the liver,[21] and it may occur in children receiving aspirin while experiencing active infection with *varicella* or *influenza*.[22] Therefore, in a patient who presents with *influenza A* and KD, administration of high-dose IVIG with NSAIDs, without high

dose (anti-inflammatory) aspirin should be considered. Amarilyo et al. found no significant clinical benefit in using IVIG with high dose Aspirin in KD compared with IVIG with low dose Aspirin.[23] Therefore, it was reasonable for us to treat the inflammatory phase of KD with NSAIDs rather than Aspirin in combination with IVIG in order to eliminate the risk for Reye syndrome.

4 | CONCLUSIONS

KD requires a high index of suspicion and awareness of its unusual presentations. In our cases, KD mimicked pneumonia that was nonresponsive to antibiotic therapy with prolonged inflammatory reaction. Thus, KD should be included in the differential diagnosis by pediatricians in patients with febrile pneumonia who do not respond to antibiotics. This can prevent delay in diagnosis, shorten the hospital stay and prevent detrimental sequelae. The potential mechanism underlying pathogen infection-mediated KD requires further investigation, which may provide scientific evidence for the pathogenesis of KD.

AUTHOR CONTRIBUTIONS

Galit Livnat: Writing – review and editing.

ACKNOWLEDGMENTS

None.

CONFLICT OF INTEREST

The authors declare that they have no competing interests.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analyzed during the current article.

ETHICAL APPROVAL

Not applicable.

CONSENT

Written informed consent for publication was obtained from the parents of both cases (including images which are entirely unidentifiable).

ORCID

Lana Khoury https://orcid.org/0000-0003-2757-6766
Mohamad Hamad Saied https://orcid.org/0000-0001-7450-4146

REFERENCES

1. McCrindle BW, Rowley AH, Newburger JW, et al. Diagnosis, treatment, and long-term management of Kawasaki disease: a scientific statement for health professionals from the American Heart Association. *Circulation*. 2017;135:e927-e999. doi:10.1161/CIR.0000000000000484
2. Son MBSR. Kawasaki Disease. In: Petty RE, Laxer RM, Lindsley CBWL, eds. *Textb. Pediatr. Rheumatol*. Elsevier Saunders; 2015:467-483.
3. De Graeff N, Groot N, Ozen S, et al. European consensus-based recommendations for the diagnosis and treatment of Kawasaki disease-the SHARE initiative. *Rheumatol (United Kingdom)*. 2019;58:672-682. doi:10.1093/rheumatology/key344
4. Witt MT, Minich LL, Bohnsack JF, Young PC. Kawasaki disease: more patients are being diagnosed who do not meet American Heart Association criteria. *Pediatrics*. 1999;104(1):e10. doi:10.1542/PEDS.104.1.E10
5. Barone SR, Pontrelli LR, Krilov LR. The differentiation of classic Kawasaki disease, atypical Kawasaki disease, and acute adenoviral infection: use of clinical features and a rapid direct fluorescent antigen test. *Arch Pediatr Adolesc Med*. 2000;154:453-456. doi:10.1001/ARCHPEDI.154.5.453
6. Amano S, Hazama F, Kubagawa H, Tasaka K, Haebara H, Hamashima Y. General pathology of Kawasaki disease: on the morphological alterations corresponding to the clinical manifestations. *Pathol Int*. 1980;30:681-694. doi:10.1111/j.1440-1827.1980.tb00966.x
7. Singh S, Gupta A, Jindal AK, et al. Pulmonary presentation of Kawasaki disease—a diagnostic challenge. *Pediatr Pulmonol*. 2018;53:103-107. doi:10.1002/ppul.23885
8. Lee MN, Cha JH, Ahn HM, et al. Mycoplasma pneumoniae infection in patients with Kawasaki disease. *Korean J Pediatr*. 2011;54:123. doi:10.3345/kjp.2011.54.3.123
9. Monedero Picazo MD, Fernández-Montes JG, Molina Fábrega R, Vallcanera Calatayud A, Alabau Vázquez G, Revert Ros MA. Radiologic findings in the lungs of patients with Kawasaki disease. *Radiologia*. 2006;48:14-18. doi:10.1016/S0033-8338(06)73124-1
10. McGraw MD, Robison K, Kupfer O, Brinton JT, Stillwell PC. The use of light's criteria in hospitalized children with a pleural effusion of unknown etiology. *Pediatr Pulmonol*. 2018;53:1101-1106. doi:10.1002/ppul.24065
11. Arslanoglu Aydin E, Demir S, Aydin O, Bilginer Y, Ozen S. Pleural effusion as an atypical presentation of Kawasaki disease: a case report and review of the literature. *J Med Case Reports*. 2019;13(1):344. doi:10.1186/s13256-019-2284-4
12. Fernández-Cooke E, Tascón AB, Antón-López J, Grasa Lozano CD, Sánchez-Manubens J, Calvo C. Previous or coincident infections with suspected Kawasaki disease. Should we change our approach? *An Pediatría (Engl Ed)*. 2019;90:213-218. doi:10.1016/J.ANPEDE.2018.06.011
13. Hendaus M, Alhammadi A. Comorbidity of Kawasaki disease and group a streptococcal pleural effusion in a healthy child: a case report. *Int J Gen Med*. 2013;6:613-616. doi:10.2147/ijgm.s49510
14. Leahy TR, Cohen E, Allen UD. Incomplete Kawasaki disease associated with complicated streptococcus pyogenes pneumonia: a case report. *Can J Infect Dis Med Microbiol = J Can des mal Infect La Microbiol Medicale*. 2012;23:137-139. doi:10.1155/2012/638357
15. Joshi AV, Jones KDJ, Buckley AM, Coren ME, Kampmann B. Kawasaki disease coincident with influenza a H1N1/09 infection. *Pediatr Int*. 2011;53:e1-e2. doi:10.1111/j.1442-200X.2010.03280.x

16. Wang J, Sun F, Deng HL, Liu RQ. Influenza a (H1N1) pdm09 virus infection in a patient with incomplete Kawasaki disease: a case report. *Medicine (Baltimore)*. 2019;98:e15009. doi:10.1097/MD.0000000000015009
17. Huang X, Huang P, Zhang L, et al. Influenza infection and Kawasaki disease. *Rev Soc Bras Med Trop*. 2015;48:243-248. doi:10.1590/0037-8682-0091-2015
18. Wadowsky RM, Mietzner SM, Skoner DP, Doyle WJ, Fireman P. Effect of experimental influenza a virus infection on isolation of Streptococcus pneumoniae and other aerobic bacteria from the oropharynges of allergic and nonallergic adult subjects. *Infect Immun*. 1995;63:1153-1157. doi:10.1128/iai.63.4.1153-1157.1995
19. Short KR, Habets MN, Hermans PWM, Diavatopoulos DA. Interactions between Streptococcus pneumoniae and influenza virus: a mutually beneficial relationship? *Future Microbiol*. 2012;7:609-624. doi:10.2217/fmb.12.29
20. Leung DYM. Superantigens related to Kawasaki syndrome. *Springer Semin Immunopathol*. 1996;17:385-396. doi:10.1007/BF01795136
21. Reye RDK, Morgan G, Baral J. Encephalopathy and fatty degeneration of the viscera. a disease entity in childhood. *Lancet*. 1963;282:749-752. doi:10.1016/S0140-6736(63)90554-3
22. Takahashi M, Mason W. Kawasaki syndrome, Reye syndrome, and aspirin. *Pediatrics*. 1986;77:616-617.
23. Amarilyo G, Koren Y, Brik Simon D, et al. High-dose aspirin for Kawasaki disease: outdated myth or effective aid? *Clin Exp Rheumatol*. 2017;35(Suppl. 103):209-212.

How to cite this article: Khoury L, Livnat G, Hamad Saied M, Yaacoby-Bianu K. Pneumonia in the presentation of Kawasaki disease: The syndrome or a sequence of two diseases? *Clin Case Rep*. 2022;10:e06676. doi:10.1002/ccr3.6676