Case Report

SAGE Open Medical Case Reports

SAGE Open Medical Case Reports
Volume 9: 1–5

DOI: 10.1177/2050313X21989465
journals.sagepub.com/home/sco
SAGE

Pediatric COVID-19 presenting as supraglottitis with vocal cord hypomobility

Meghana Nadiger[1,2], Manette Ness-Cochinwala[1,2], Carolina Sanchez-Vegas[2,3], Prithvi Sendi[1,2], Brian Ho[4], Balagangadhar R Totapally[1,2] and Ramesh Sachdeva[1,2]

Abstract

In the United States, an estimated 7.3% of confirmed cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (COVID-19) are among persons aged less than 18 years. Data regarding clinical manifestations in this age group are still evolving. An upper airway predilection has been reported in children. We describe the case of a 15-year-old female with supraglottitis and unilateral hypomobility of vocal cord with concern for critical airway, associated with COVID-19. She was managed by a multidisciplinary team including critical care, infectious diseases, and otolaryngology. This report adds to the sparse but evolving body of literature on the clinical presentation of COVID-19 disease in children.

Keywords

COVID-19, pediatric, supraglottitis, vocal cord hypomobility

Date received: 4 July 2020; accepted: 29 December 2020

Introduction

In the United States, an estimated 7.3% of confirmed cases of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection (COVID-19) are among persons aged less than 18 years.[1] Clinically significant infection is far less common in children than in adults. We describe the case of a 15-year-old female with COVID-19 presenting with dysphonia and odynophagia secondary to supraglottitis and vocal cord hypomobility. Supraglottitis is a potentially life-threatening condition characterized by inflammation of the supraglottic larynx, including the arytenoids, false vocal cords, aryepiglottic folds, laryngeal ventricles and the epiglottis.[2] In the post-*Hemophilus influenzae* (type B) vaccine era, it tends to occur in older children.[3] This is one of the first few reports of COVID-19 cases with this presentation and adds to the growing body of literature on upper airway manifestations of COVID-19.[4,5]

The case

A 15-year-old female presented to the emergency room in mid-April 2020, with a 2-day history of worsening cephalalgia, hoarseness and muffling of voice, difficulty swallowing and globus pharyngeus. She had nausea and generalized malaise with the above symptoms. She was otherwise in good health with normal swallowing and normal phonation prior to the onset of these symptoms. Past medical history was significant for an emergency room visit 2 years prior, with lip swelling and itchiness, with no rashes and no known exposures. The symptoms had resolved with an oral antihistamine and a 3-day course of oral steroids.

On examination, she was afebrile with normal vital signs and oxygen saturation. There was tenderness over the right submandibular area, but no neck swelling, posterior pharyngeal erythema, palpable lymphadenopathy, stridor, facial swelling or rashes noted. Complete blood count, liver function test, renal function test, lipase level and urinalysis were all within normal limits. A throat swab for rapid streptococcal antigen was negative. A soft tissue computed tomography (CT)

[1]Division of Critical Care Medicine, Nicklaus Children's Hospital, Miami, FL, USA
[2]Herbert Wertheim College of Medicine, Florida International University, Miami, FL, USA
[3]Division of Infectious Disease, Nicklaus Children's Hospital, Miami, FL, USA
[4]Division of Otolaryngology, Nicklaus Children's Hospital, Miami, FL, USA

Corresponding Author:
Ramesh Sachdeva, Division of Critical Care Medicine, Nicklaus Children's Hospital, 3100 SW 62nd Avenue, Miami, FL 33155, USA.
Email: ramesh.sachdeva@nicklaushealth.org

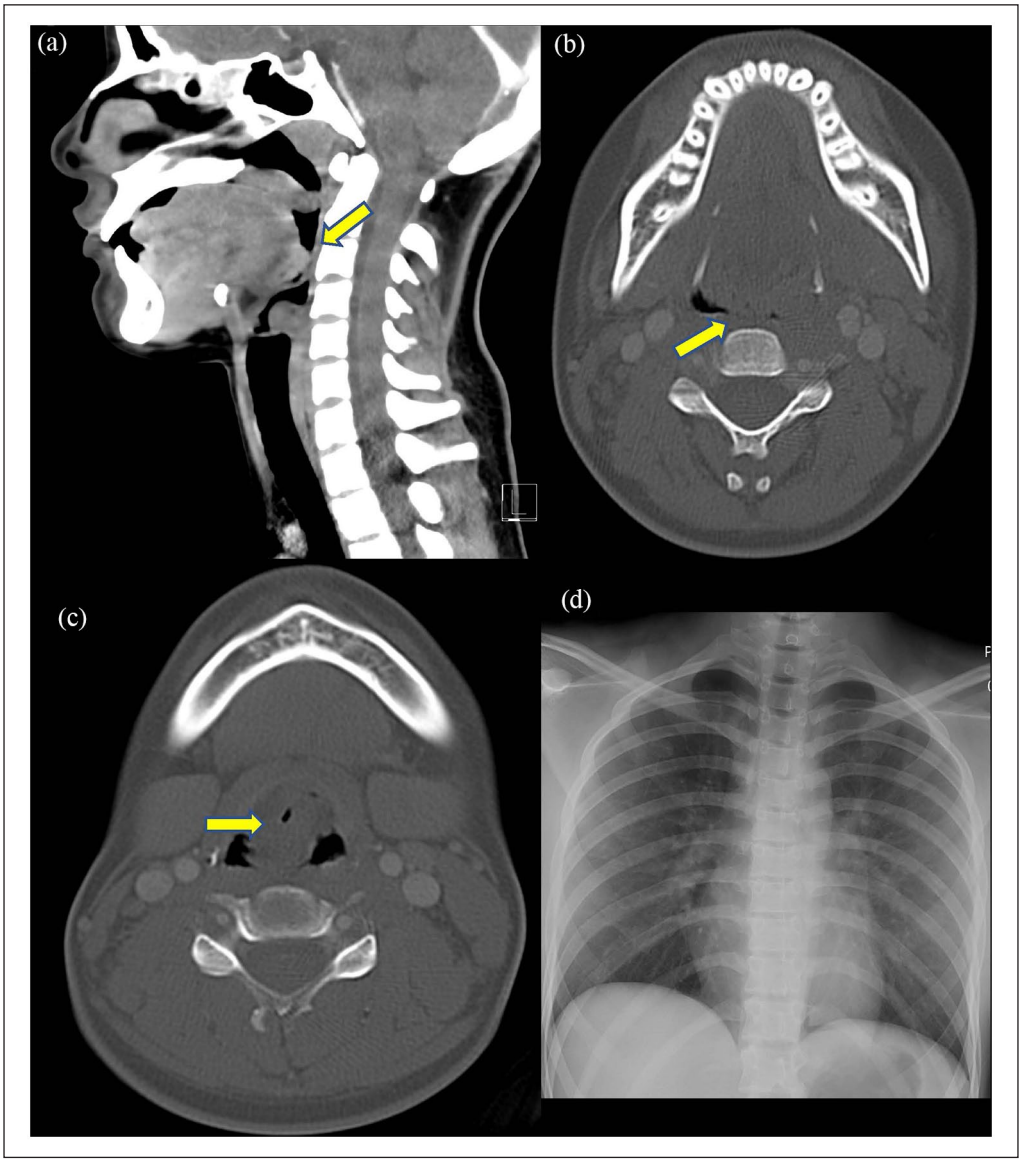

Figure 1. Panels (a)-(c): Contrast enhanced CT scan of soft tissues of the neck. Panel (a): Sagittal image showing tongue base resting against the posterior oropharynx, Panel (b): Axial image showing tongue base obstructing a large portion of the oropharyngeal airway, Panel (c): Arytenoid edema, with small glottis opening and Panel (d): Anteroposterior chest radiograph with subtle atelectasis in the right lung field.

scan of the neck showed a prominence of lingual tonsils, supraglottic laryngeal edema with focal airway narrowing, concerning for supraglottitis; no cervical lymphadenopathy was noted (Figure 1(a)-(c)). She was evaluated by the otolaryngologist with flexible laryngoscopy in the emergency room, which showed prominent lingual tonsils and arytenoid edema and pooling of secretions limiting visualization of the posterior cricoid region. There was no glottic (vocal cord) edema but the right vocal cord was hypomobile. The epiglottis was normal in shape with no significant edema. Due to the history of subjective fevers, malaise, chills, emesis, and ongoing transmission of SARS-CoV2 in the community, a decision was made to obtain COVID-19 testing, which came back positive.

She was treated with dexamethasone for airway edema. Ceftriaxone and clindamycin were started in order to cover for possible bacterial supraglottitis, after blood and throat cultures were sent. She was admitted to the COVID-19 isolation unit in the pediatric intensive care unit for high-risk airway monitoring. In view of her COVID-19 status, azithromycin and hydroxychloroquine were added. A chest radiograph showed subtle atelectasis in the right lung (Figure 1(d)). Initial labs showed a normal leukocyte count with bandemia, monocytosis, and a normal lymphocyte count. D-dimer level was elevated with normal coagulation profile. Ferritin, C-reactive protein, and interleukin-6 levels were normal. Liver function, renal function and urinalysis were normal.

Table 1. Serial laboratory values.

Measure	Reference range	HD 1	HD 2	HD 3	HD 4	HD 5
SARS-CoV-2 PCR	–	Positive	–	–	–	–
Respiratory pathogen panel PCR*	–	–	Negative	–	–	–
White blood cell count (10,000/μL)	4.8–10.8	8.3	4.8	5.0	11.3	9.8
Hemoglobin (g/dL)/hematocrit (%)	12–16/36–46	13.2/40.4	13.6/40.8	13.6/39.6	14.2/40.2	13.4/39.9
Platelet count (10,000/μL)	150–500	283	284	278	315	305
Neutrophils (%)	34–64	48	76	83	88	80
Bands (%)	0–6	12	0	0	1	0
Absolute lymphocyte count	1200–4900	2656	1152	700	904	1470
D-dimer (μg/mL)	0.27–0.41	–	1.03	1.17	1.06	1.04
Prothrombin time (s)	11.6–15.4	–	14.6	14.2	14.5	14.2
International normalized ratio	–	–	1.17	1.13	1.16	1.13
Activated partial thromboplastin time (s)	22.8–38.2	–	33.5	28.6	30.5	29.7
C-reactive protein (mg/dL)	0–1	–	1	–	–	–
Procalcitonin (ng/mL)	0–0.1	–	<0.05	<0.05	<0.05	<0.05
Interleukin-6 (pg/mL)	<1.8	–	1.5	–	–	–
Ferritin (ng/mL)	9–125	–	15	19	–	–
Lactate dehydrogenase (IU/L)	340–670	–	489	525	418	392
Alanine aminotransferase (IU/ L)	5–35	16	15	14	–	–
Aspartate aminotransferase (IU/ L)	5–30	40	28	35	–	–
Total protein (g/dL)	6.1–8	8.3	7.9	8.7	–	–
Albumin (g/dL)	3.1–4.8	4.8	4.6	4.9	–	–
Brain-type natriuretic peptide (pg/mL)	15–75	–	<15	–	–	–

On hospital day 2, she had odynophagia with drooling of saliva, worsening hoarseness of voice, with difficulty saying more than a few words, but without stridor, desaturations or tachypnea. A trial of racemic epinephrine was not effective, and her steroid frequency was increased. A repeat chest radiograph was normal, with resolution of previously seen atelectasis. A lateral soft-tissue neck radiograph was similar to the initial CT scan with signs of inflammation of supraglottic structures. Serial labs showed leukopenia and worsening lymphopenia (Table 1). A respiratory pathogen panel sent on admission was negative for common pathogens. Mycoplasma pneumoniae polymerase chain reaction (PCR) was negative. EBV panel was sent and the serologic results indicated a recent but not acute Epstein-Barr virus (EBV) infection, anytime in the preceding 6 weeks to 6 months.[6] EBV PCR test was sent and it returned negative, confirming that the EBV infection was not active. Throat culture and blood cultures were negative for bacterial growth. In view of her prior history of facial swelling with no known allergies or exposures a C1 esterase inhibitor functional assay was sent which was normal, thus ruling out hereditary angioedema.[7]

During her 7 days of hospital stay, she remained afebrile, with normal saturations of above 97% in room air. She completed a 5-day course of azithromycin and hydroxychloroquine. After day 3, her symptoms gradually improved. By day 7, she had minimal residual hoarseness of voice with no breathing difficulty and mild dysphagia, but able to tolerate a full liquid diet. Antibiotics were stopped. She was discharged home on a high-calorie, soft-liquid diet and tapering doses of oral prednisone. She followed-up with Pediatric Infectious Diseases and Otolaryngology specialists as an out-patient via Tele-health. As on 2 months post-discharge, she still has residual but improving hoarseness of voice and dysphagia more for solids than liquids. She was seen by the Neurologist, who recommended an magnetic resonance imaging (MRI) of the cervical spine. Her neurologic examination and MRI cervical spine were normal. She is receiving out-patient speech therapy for persistent but improving problems with swallowing solid food.

Discussion

COVID-19 presents with myriad clinical findings, with children being mostly asymptomatic or having mild lower respiratory symptoms.[8–10] Usual clinical manifestations noted are fever, dry cough, and fatigue along with other upper respiratory findings, such as runny nose, nasal congestion and sore throat.[8–10] To our knowledge, this is the first reported case of isolated supraglottic inflammation and vocal cord hypomobility, without any fever or lower respiratory involvement. It is notable that although fever is described as frequent manifestation in pediatric COVID-19, our patient was afebrile throughout her course, despite having moderate to severe supraglottitis.

In our patient, COVID-19 related laryngeal and pharyngeal inflammation led to edema that was significant enough to cause airway concerns. In a recent report from Manchester, United Kingdom, McGrath et al.[11] described

the potential of SARS-CoV2 in causing subtle to overt airway and laryngeal inflammation, in some cases causing post-extubation failure. They suggest treating the airway of a COVID-19 patient as technically challenging at the outset, due to this reason.

Another finding that heightened clinical concern for a potential airway emergency in our patient was vocal cord hypomobility. Vocal cord/ vocal fold hypomobility has been reported in association with viral upper respiratory tract infections, especially with nasopharyngeal and laryngeal inflammation. In their review of patient data spanning 10 years, Bhatt et al.[12] report that pharyngitis and laryngitis are the most common symptoms of concurrent upper respiratory infection and idiopathic vocal fold paralysis. Vocal cord paralysis without a history of intubation has been reported in association with herpes simplex virus, varicella zoster virus, EBV and cytomegalovirus.[13] Mechanical and neurogenic mechanisms have been proposed, however, causality or an exact pathogenic mechanism have not been established. Extension of adjacent inflammation can cause unilateral or bilateral reduction in mobility of the small muscles and joints of the larynx. This is seen as reduced mobility of the vocal folds on laryngoscopy. Another rare mechanical cause which is unlikely but worth mentioning is inflammation of the cricoarytneoid (CA) joint itself. It is associated with many of the features seen in our patient including odynophagia, hoarseness, a globus sensation, tenderness to local palpation and arytenoid edema on flexible laryngoscopic examination.[14] CA joint inflammation has been described as manifesting with croup-like symptoms in various systemic inflammatory states. However, it is exceedingly rare, not reported in previously healthy children and is difficult to diagnose on CT scan.

Another possible explanation of vocal cord hypomobility is a viral neuropathy. There have been several anecdotal and published reports of self-reported anosmia and dysgeusia with COVID-19, especially in the early stages of disease.[15] Possible mechanisms suggested include binding of the virus to the taste receptors[16] and trans-neural penetration of the virus, as found in mouse models for SARS-CoV.[17] This raises the question whether the reduced vocal cord mobility noted on flexible laryngoscopy in our patient could have been due to neuronal involvement, although such a hypothesis needs to be rigorously tested in future clinical studies.

Conclusion

As the COVID-19 pandemic continues to spread, it is important for clinicians caring for acutely ill hospitalized children, to be aware of unusual patterns of presentation. Our patient had isolated supraglottic inflammation and vocal cord hypomobility, without fever or significant lower respiratory involvement. This is one of the first few reports of upper airway involvement in pediatric patients with COVID-19. This report highlights the unique presentation of a novel disease and adds to the evolving body of literature on the clinical picture of pediatric COVID-19.

Declaration of conflicting interests

The author(s) declared the following potential conflicts of interest with respect to the research, authorship, and/or publication of this article: Dr. Ramesh Sachdeva is uncompensated for his role as Chief Scientific Officer for Virtual Pediatric Systems (VPS), LLC; however, his travel expenses for VPS related meetings were paid by VPS, LLC. Drs. Nadiger, Sachdeva and Totapally are participating in the "Overcoming COVID-19: Influenza and other emerging respiratory pathogens surveillance registry" study funded by the CDC at Nicklaus Children's Hospital, however none of them receive any compensation for this activity.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series

Informed consent to participate

We thank the patient and her parents for giving us their written informed consent to publish this report with the relevant patient information and images.

Informed consent for publication

Written informed consent was obtained from the patient and her mother for anonymized patient information to be published in this article.

ORCID iD

Meghana Nadiger https://orcid.org/0000-0002-1027-6962

References

1. CDC COVID-19 Response Team. Coronavirus disease 2019 in children—United States, February 12-April 2, 2020. *MMWR Morb Mortal Wkly Rep* 2020; 69(14): 422426.
2. Eskander A, de Almeida JR and Irish JC. Acute upper airway obstruction. *N Engl J Med* 2019; 381(20): 19401949.
3. Navaratnam AV, Smith ME, Majeed A, et al. Adult supraglottitis: a potential airway emergency that can present in primary care. *Br J Gen Pract* 2015; 65(631): 99–100
4. Venn AMR, Schmidt JM and Mullan PC. A case series of pediatric croup with COVID-19 (S0735-6757(20)). *Am J Emerg Med*. Epub ahead of print 15 September 2020. DOI: 10.1016/j.ajem.2020.09.034.
5. Pitstick CE, Rodriguez KM, Smith AC, et al. A curious case of group: laryngotracheitis caused by COVID-19. *Pediatrics*. Epub ahead of print 10 September 2020. Doi: 10.1542/Peds.2020-012179.
6. American Academy of Pediatrics. Epstein-barr virus infections. In: Kimberlin DW, Brady MT, Jackson MA, et al. (eds) *Red book: 2018 report of the committee on infectious diseases*. Itasca, IL: American Academy of Pediatrics, 2018, pp. 334-338.

7. Longhurst HJ and Bork K. Hereditary angioedema: an update on causes, manifestations and treatment. *Br J Hosp Med (Lond)* 2019; 80(7): 391398.
8. Castagnoli R, Votto M, Licari A, et al. Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in children and adolescents: a systematic Review [published online ahead of print, 2020 Apr 22]. *JAMA Pediatr* 2020; 174: 882-889.
9. Ludvigsson JF. Systematic review of COVID-19 in children shows milder cases and a better prognosis than adults [published online ahead of print, 2020 Mar 23]. *Acta Paediatr* 2020; 109: 1088-1095.
10. Shekerdemian LS, Mahmood NR, Wolfe KK, et al. Characteristics and outcomes of children with coronavirus disease 2019 (COVID-19) infection admitted to US and Canadian pediatric intensive care units [published online ahead of print, 2020 May 11]. *JAMA Pediatr* 2020; 174: 868-873.
11. McGrath BA, Wallace S and Goswamy J. Laryngeal oedema associated with COVID-19 complicating airway management [published online ahead of print, 2020 Apr 17]. *Anaesthesia* 2020; 75: 972.
12. Bhatt NK, Pipkorn P and Paniello RC. Association between upper respiratory infection and idiopathic unilateral vocal fold paralysis. *Ann Otol Rhinol Laryngol* 2018; 127(10): 667–671.
13. Marie JP, Keghian J, Mendel I, et al. Post-intubation vocal cord paralysis: the viral hypothesis. *Eur Arch Otorhinolaryngol* 2001; 258(6): 285–286.
14. Chalmers A and Traynor JA. Cricoarytenoid arthritis as a cause of acute upper airway obstruction. *J Rheumatol* 1979; 6(5): 541542.
15. Giacomelli A, Pezzati L, Conti F, et al. Self-reported olfactory and taste disorders in SARS-CoV-2 patients: a cross-sectional study [published online ahead of print, 2020 Mar 26]. *Clin Infect Dis* 2020; 71: 889-890.
16. Xu H, Zhong L, Deng J, et al. High expression of ACE2 receptor of 2019-nCoV on the epithelial cells of oral mucosa. *Int J Oral Sci* 2020; 12(1): 8.
17. Netland J, Meyerholz DK, Moore S, et al. Severe acute respiratory syndrome coronavirus infection causes neuronal death in the absence of encephalitis in mice transgenic for human ACE2. *J Virol* 2008; 82(15): 7264–7275.