Serial Computed Tomography Findings in a Child with Coronavirus Disease (COVID-19) Pneumonia

Published online: April 09, 2020;
PII: S097475591600158

Novel coronavirus disease (COVID-19) is a highly infectious disease with its outbreak in China in late 2019 [1]. The novel coronavirus is reportedly affecting more adults than children [2,3]. Here, we provide computed tomography **(**CT) findings in a typical pediatric case with confirmed COVID-19 infection.

An 11-year-old boy, the close contact of confirmed COVID-19 infected father, presented to hospital with high fever for 10 days. He was confirmed COVID-19 infection by throat swab specimen test using Realtime RT-polymerase chain reaction (RT-PCR) method.

His symptoms relieved somewhat after interferon α-2b combined with aerosol therapy in a local hospital. On admission, arterial blood gas analysis showed a low $PaO_2$of 69.6 mmHg. Chest CT was performed, which showed patchy ground-glass opacities in left lower lobe with air bronchogram (***Fig.*** **1a**). He was diagnosed as COVID-19 pneumonia. During hospitalization, the child received recombinant human interferon alpha-2b (rhIFNα2b) twice-a-day through nebulization combined with Complementary and alternative medicines. Supportive care including nasal cannula (maximum oxygen requirement 2L/min) was administered. CT done one week later (day 7) showed scattered ground-glass opacities in left lower lobe (***Fig.*** **1b**). After two weeks of therapy, only slight sporadic ground-glass opacities in left lower lobe were found in repeat chest CT (***Fig.*** **1c**). Realtime RT-PCR on two throat swab specimens was negative for the COVID-19 at 14 weeks, 48 hour apart. The boy made a complete recovery.

This communication underscores the course of CT findings in COVID-19 pneumonia in a child without any co-morbidity, who improved after treatment.

Authors' contributions: GH,JC: conceptualized the study, collected data, WS: conceptualized the study, drafted the initial manuscript, and reviewed and JW: carried out the analyses; all authors reviewed and revised the manuscript, and approved the final manuscript as submitted.

Competing interests: none stated. *Funding*: Wenzhou Municipal Science and Technology Bureau (ZY202004).
Published online: April 09, 2020; *PII*: S097475591600158

GUIQING HE[1], WENJIE SUN[2], JING WU[3] AND JING CAI[4]*
[1]Department of Infectious Diseases and Infectious Diseases Laboratory, and [4]Department of Comprehensive Medicine, Wenzhou Sixth People's Hospital, Wenzhou Central Hospital Medical Group, Wenzhou, China;
[2]The Second Affiliated Hospital of Fujian Traditional Chinese Medical University, Fuzhou, China; and
[3]Department of Infectious Diseases, Huashan Hospital, Fudan University, Shanghai, China.
michellecai666@hotmail.com

REFERENCES

1. Zhu N, Zhang D, Wang W, Li X, Yang B, Song J, *et al*. A novel coronavirus from patients with pneumonia in China,

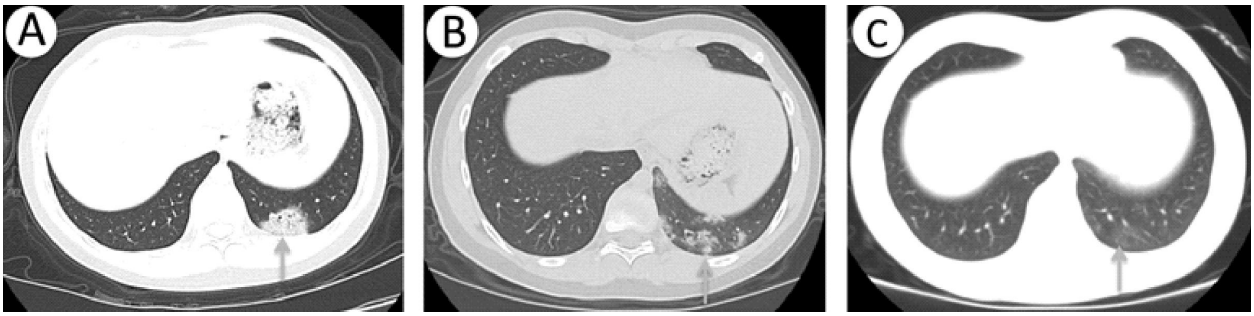

Fig. 1 *Chest computed tomography (CT) scans in an 11-year-old boy with coronavirus disease-19. a) Chest CT performed on the day of admission shows patchy ground-glass opacities in left lower lobe with air bronchogram; b)Follow-up CT obtained on day 7 shows scattered ground-glass opacities in left lower lobe which were partly resolved; c)Follow-up CT obtained on day 14 shows slight sporadic ground-glass opacities in left lower lobe which have significantly resolved.*

2019. N Engl J Med. 2020;382:727-33.
2. Cai J, Xu J, Lin D, Yang Z, Xu L, Qu Z, *et al.* A case series of children with 2019 novel coronavirus infection: Clinical and epidemiological features. Clin Infect Dis. 2020 Feb 28 [Online ahead of print]. Available from:*https://academic.oup.com/cid/advance-article/doi/10.1093/cid/ciaa198/5766430*. Accessed April 2, 2020.
3. Guan WJ, Ni ZY, Hu Y, Liang WH, Ou CQ, He JX, *et al*. Clinical characteristics of coronavirus disease 2019 in China. N Engl J Med. 2020 Feb 28. Available from: *https://www.nejm.org/doi/full/10.1056/NEJMoa2002032*. Accessed April 2, 2020.

Urolithiasis due to Hereditary Xanthinuria Type II: A Long-term Follow-up report

Hereditary xanthinuria (HX) is a rare autosomal recessive disorder of purine metabolism. It results from deficiency of the enzyme 'xanthine dehydrogenase/oxidase (XDH/XO)' which catalyzes the final two steps in the purine degradation pathway (conversion of hypoxanthine and xanthine to uric acid). The resultant plasma accumulation and excess urinary excretion of xanthine is responsible for the arthropathy, myopathy, crystal nephropathy, urolithiasis, and renal failure seen in this disorder. Most patients belong to Middle East or Mediterranean region, and the disorder is rare in other parts of the world [1,2]. Two types of HX have been described; type I and type II, based on distinct mutation loci. It is difficult to distinguish the two subtypes on clinical and biochemical grounds, and molecular testing is needed for accurate phenotyping [1].

A 13-month-old male child, presented with recurrent episodes of orange colored graveluria and hematuria since the age of nine months. The child was born of a second-degree consanguineous marriage and family history was negative. Examination revealed a healthy appearing child with length 78 cm (75th centile) and weight 8.8 kg (10th-25th centile). The general and systemic examination was unremarkable.

Investigations revealed serum calcium 10.2 mg/dL, phosphorus 5.6mg/dL, alkaline phosphatase 678 IU/L, creatinine 0.4 mg/dL, blood urea 10 mg/dL, hypouricemia (serum uric acid <0.01 mg/dL) and hypouricosuria (24 hour urinary uric acid 0.4 mg/day, normal 250-750 mg/day; 24 hour urinary creatinine 88 mg/day, normal 88-106 mg/day). Radiograph of kidney ureter and bladder was normal, while ultrasonography revealed two calculi in the urinary bladder and concretions in the lower pole of left kidney. In view of low serum and urinary uric acid levels and radiolucent nature of renal stones, xanthinuria was suspected. His hospital course was complicated by urethral obstruction which was relieved by catheterization followed by cystolithotomy at a later date. The bladder stones retrieved were subjected to *X*-ray diffraction study, revealing them to be of xanthine origin. A targeted gene sequencing revealed compound heterozygous mutation in the enzyme molybdenum cofactor sulfurase (*MOCOS*) gene [heterozygous two base pair deletion in exon 6 (chr18:33785104_33785105delCT) and heterozygous nonsense mutation in exon 11 (chr18:33831134T>G)]. Patient was diagnosed to be having HX type II and advised dietary purine restriction (avoidance of purine-rich foods including red and organ meat, shell fish, oily fish, seafood, sweetened beverages such as fruit juices and colas, yeast and mushroom, spinach, peas and whole pulses), and adequate oral hydration.

On follow-up, he had no further episodes of renal colic, graveluria or hematuria. The child maintained good compliance to dietary restrictions advised. His height and weight at nine years were 138.7cm (75th-97th percentile), and 32.1 kg (75th-97th percentile), respectively. Serial annual ultrasonography imaging and renal functions have remained normal with serum uric acid <0.01 mg/dL.

HX is a rare disorder of the purine metabolism that leads to urolithiasis. Renal stones can occur at any age, even in infancy [2]. The stones are radiolucent, and are seen in about 40-50% patients with this disorder. The diagnosis may be established with stone analysis, demonstration of an elevated urinary xanthine or hypoxanthine excretion, and measurement of XDH/XO activity in liver or intestinal biopsy sample. The finding of an orange-brown urinary sediment, orange-stained diapers, and profound hypouricemia are other important indicators. However, it is difficult to characterize the exact phenotype of the disorder (type I or II) based on these clinical and biochemical indicators alone, necessitating the use of molecular tests.

The mainstay of treatment is institution of a low-purine diet, and intake of plenty of oral fluids [3]. Urinary