OPEN ACCESS Freely available online

ISSN: 2329-6925
Journal of Vascular Medicine & Surgery

Case Report

Kawasaki Disease with Intestinal Ischemia/Mucous Membrane Necrosis in a 16 Months Old Boy: A Case Report and literature Review

Xiao-Zhe Cui, Hong Wang*

Department of Pediatric, Shengjing Hospital of China Medical University, Shenyang, China

ABSTRACT

Background: Kawasaki Disease (KD) is a common vasculitis in children under 5 years old, and it may cause multiple system complications. In the case of the digestive system complication, KD can cause abdominal pain, diarrhea, hypoalbuminemia, liver damage, intussusception, cholecystitis, limit of peritonitis, and intestinal stenosis. So far there have been no reports on KD with bowel necrosis. Here, we report the first case of a KD patient with significant intestinal mucous membrane necrosis.

Case presentation: Patient is a 16 months old boy, presented with fever for five days, along with rashes, congestive conjunctiva, red and cracked lips, left cervical lymphadenopathy, edema on hands and feet for one day. He had vomiting, progression of abdominal distension, along with poor mental state and irritability for 2 days.

Physical examination: His abdomen was distended and the sounds of bowel movement were weakened. The boundary of pulmonary vs. liver was clears. Neck rigidity was observed. Abdominal DR showed the intestine at left side filled with fluid and gas. Ultrasound showed pneumatosis in liver around portal vein, dilated intestine, enlarged gallbladder, and stasis of bile in the gall bladder on Day 5 of illness. Abdominal enhanced CT showed hydronephrosis at the left kidney, dilated intestinal tube. Pneumatosis at the lower middle intestinal wall was detected on Day 7 of illness, but recovered almost normal on Day 10. He was diagnosed with KD complicated with extensive intestinal mucous membrane necrosis. He was treated with IVIG, oral aspirin, fasting, rehydration, short term of methylprednisolone, and anti-inflammatory drugs. There was no surgery intervention needed. He recovered without coronary artery damages.

Conclusion: In KD patients, severe intestinal necrosis at the acute stage can jeopardize patients' lives. Here we report a case that a KD patient had digestive system involvement. Physical exam, lab tests, ultrasonic and CT tests suggested intestinal necrosis in mucosal layer. We provided comprehensive treatments immediately after the assessment. The patient recovered without the need of surgical intervention.

Keywords: Intestinal necrosis; Kawasaki disease; Children; Treatment; Prognosis

Abbreviations: KD: Kawasaki Disease; IVIG: Intravenous Immunoglobulin; ASA: Acetyl Salicylic Acid; CRP: C-Reactive Protein; ESR: Erythrocyte Sedimentation Rate; PCT: Procaicitonin; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; TBIL: Total Bilirubin; D-BIL: Direct Bilirubin; CSF: Cerebrospinal Fluid; DR: Digital Radiography; CT: Computed Tomography; HIV: Human Immunodeficiency Virus; EBV: Epstein-Barr Virus; HSV: Herpes Simplex Virus; MP: Mycoplasma Pneumonia; CP: Chlamydia Pneumonia; HAV, HBV, HCV, and HEV: Hepatitis A, B, C, and E Virus.

INTRODUCTION

Kawasaki Disease (KD) is a common vasculitis in children, characterized by inflammation in medium-sized arteries, with involvement of almost all systems. It is the most common cause of acquired heart disease in the developed countries [1]. The prevalence of KD in China is gradually increased. KD mainly affects children younger than 5 years old, most in boys, without any definite causes [2].

The prevalence of digestive system involvement in KD is 2.3% [3]. Clinical aspects are reported to include nausea, vomiting, diarrhea, hypoproteinemia, liver dysfunction, cholestasis, cholecystitis, pancreatitis, limited peritonitis, and intussusception and so on [4-6]. However, KD with intestinal necrosis has been never reported. Here we report a case of 16 months old boy who was diagnosed

Correspondence to: Hong Wang, Department of Pediatric, Shengjing Hospital of China Medical University, Shenyang, China, E-mail: wanghong_64@sina.com

Received: 01-Sep-2022, Manuscript No. JVMS-22-17914; **Editor assigned:** 05-Sep-2022, PreQC No. JVMS-22-17914 (PQ); **Reviewed:** 19-Sep-2022, QC No. JVMS-22-17914; **Revised:** 26-Sep-2022, Manuscript No. JVMS-22-17914 (R); **Published:** 06-Oct-2022, DOI: 10.35248/2329-6925.22.S10.476.

Citation: Cui XZ, Wang H (2022) Kawasaki Disease with Intestinal Ischemia/Mucous Membrane Necrosis in a 16 Months Old Boy: A Case Report and Literature Review. J Vasc Surg. S10:476.

Copyright: © 2022 Cui XZ, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

with KD and developed intestinal mucous membrane necrosis. The patient recovered completely, neither with surgical intervention to remove affected segment of intestine nor with coronary artery damages.

CASE PRESENTATION

A 16-months-old previously healthy boy presented with fever for five days, along with rashes, congestive conjunctiva, red and cracked lips, left cervical lymphadenopathy, edema on hands and feet for one day. He had vomiting, progressed abdominal distension, along with poor mental state and irritability for 2 days. He had no family medical history and no history of allergies.

Physical examinations

His body temperature was 38°C and his vital signs were stable. There were scarlet rashes on his trunk and legs. He had congestive conjunctiva without purulent discharges. His lips were red and cracked, and he had strawberry tongue. Ultrasound revealed a left lymphadenopathy about 1.8 cm × 0.7 cm. His abdomen was distended and his bowel movement sounds were weakened. He had edema on hands and feet. Neck rigidity was observed. Other systems were normal.

Laboratory results

A normal blood routine results (PLT 283 × 10^9/L), elevated C-Reactive Protein (CRP) 85.2 (<8 mg/dl), Erythrocyte Sedimentation Rate (ESR) 94 (<15 mm/h), Procalcitonin (PCT) 3.26 (<0.05 ng/ml), Interleukin-6 (IL-6) 477.3 (<1.72 pg/ml). Hepatic function tests revealed elevation in Alanine Aminotransferase (ALT) 80 (0-40 U/L), Aspartate Aminotransferase (AST) 40 (5-34 U/L), Total Bilirubin (TBIL) 86.1 (3.4-20.5 umol/L), Direct Bilirubin (D-BIL) 74.3 umol/h (0-8.6 umol/L). Albumin was decreased to 29.8 g/L. Potassium was 2.7 mmol/l. D-dimer was 4810 (252 ug/L), and NT pro-BNP was 3524 (<300 pg/ml). Renal function test was normal. In the examination of Cerebrospinal Fluid (CSF), pressure was 90 drops/min, protein, glucose, chloride and the amount of cells were normal. Pathogen tests were negative. Abdominal Digital Radiography (DR) showed there was gas at left abdominal intestine (Figure 1). Abdominal Ultrasound revealed pneumatosis in the liver (Figure 2a), red arrow located around portal vein (Figure 2a), green arrow, partially dilated intestinal tube (Figure 2b), red arrow, enlarged gallbladder (Figure 2c), red arrow, and stasis bile in gallbladder (Figures 2a-2d). Pathogen tests were negative for Human Immunodeficiency Virus (HIV), Syphilis, Epstein-Barr Virus (EB virus), Herpes Simplex Virus (HSV), Mycoplasma Pneumonia (MP), Chlamydia Pneumonia (CP), Hepatitis A, B, C, and E Virus (HAV, HBV, HCV, and HEV).

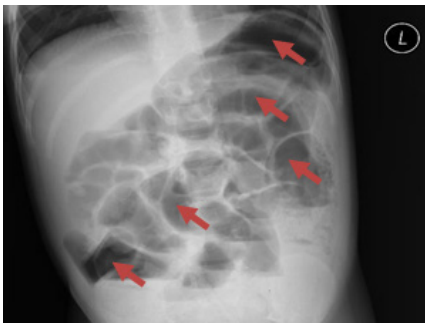

Figure 1: Abdominal DR showed the intestine is flush with fluid and gas most at left abdomen on Day 5 of illness.

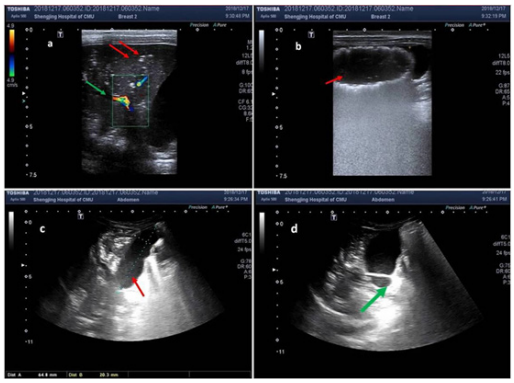

Figure 2: Abdomen ultrasound showed pneumatosis in liver. 2A: (red arrow), around portal vein (green arrow), dilated intestine (B), enlarged gallbladder (C), and stasis bile in gallbladder (D) on Day 5 of illness.

Treatments

He was treated with intravenous infusion of immunoglobulin (1 g/Kg) for 2 days, and second generation of cephalosporin. He was given oral aspirin at 30 mg/kg/day, divided into 3 times, and metronidazole and gentamicin suppositories as soon as KD was diagnosed after he was admitted. Meanwhile, potassium and albumin were infused. Meanwhile he was maintained on a liquid diet.

Disease progression

He continued to have fever on Day 7 of illness. His abdominal distension was worse, and bowel movement sounds diminished. Enhanced CT of the abdomen revealed hydronephrosis in the left kidney (Figure 3a), red arrow, dilated intestinal tube, pneumatosis, and hydrocele (Figure 3b), green arrow (Figures 3a and 3b). There was air accumulated in the small intestine wall (intestinal wall necrosis) at the middle and lower abdomen (Figure 3b), yellow arrow (Figure 3b). The rectal wall was slightly thickened and there was mild interstitial edema around portal vein. Echocardiography showed normal coronary arteries. Electrocardiogram result was normal.

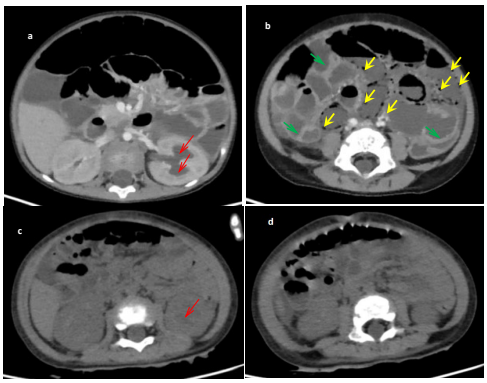

Figure 3: Abdominal enhanced CT showed hydronephrosis of left kidney (A), dilated intestinal tube. 3B: (green arrow) and pneumatosis (yellow arrow) at middle-lower abdomen intestinal wall on Day 7 of illness. Abdominal CT showed hydronephrosis of left kidney (C) and almost normal intestine (D) on Day 10.

Additional treatments and examinations

He was on water intake restriction, gastrointestinal decompression, and parenteral nutrition. He was given intravenous infusion of methylprednisolone (2 mg/Kg for 2 days and 1 mg/Kg for one

day). About 67 hours later after fasting, abdominal distension diminished and bowel movement sounds recovered. On Day 10 of illness, repeated abdominal CT showed the same hydronephrosis in the left kidney (Figure 3c), red arrow, but almost normal intestines (Figures 3c and 3d). His fever subsided on Day 12 of the illness. On Day 13 of illness, there was skin peeled around fingernails. On Day 15 of illness, he was discharged after CRP subsided.

Follow up

We have followed up with this patient routinely for 2 years. At about 2 months of illness, a deep transverse groove was developed across his finger nails (Beau's lines). Echocardiography repeatedly showed normal coronary arteries. He stopped taking oral aspirin at 2 months.

RESULTS AND DISCUSSION

The boy met the criteria of classic KD. In addition, he presented with vomiting, progressive abdominal distension. His symptoms and radiography results indicate intestinal ischemia or mucous membrane necrosis. It has been shown that portal or mesenteric venous gas strongly suggests the presence of bowel infarction and necrosis [7,8]. Superior mesenteric artery infarction or mesenteric vein thrombosis can result in the intestinal ischemia, presented with detectable gas. KD is essentially inflammation-mediated small vasculitis. Despite the mechanism difference behind intestinal complication, the bowel gas accumulation in KD patients is also considered a manifestation of intestinal mucosal necrosis. A persistent and significant elevation of D-Dimer level could also suggest cardiac infarction or intestinal necrosis [9]. Recovery is possible as long as there is no occurrence of transmural necrosis (intestinal gangrene) and perforation of the intestine. For this patient, the surgeon recommended conservative treatment rather than surgical removal of the ischemic or necrotic bowel segments due to KD related intestinal arteritis.

In our center, it is common to see KD patients with digestive system involvement. Clinical aspects include abdominal pain and/or distension, hepatic dysfunction, gallbladder wall edema, and other forms of gastrointestinal involvement such as intussusception, ischemic colitis, hepatic necrosis, splenic infarct, intestinal obstruction, colitis, and colon edema but much less frequently. Hypoproteinemia, a high risk factor for coronary aneurysms, can occur during fever due to KD-related vasculitis that causes plasma albumin leaking. Thus, if possible, we monitor food intake in these patients, and keep them at least on liquid food. However, in the case of intestinal paralysis and incomplete intestinal obstruction, food intake may result in intussusception or food retention in the expanded intestinal cavity, thus facilitates bacteria growth in intestine, aggravates intestinal paralysis, and, as a result, leads to intestinal necrosis.

Therefore, it requires clinicians to monitor the scale of food intake in patients. When KD children develop abdominal distension, hypokalemia caused by poor feeding should be concerned at first. Once confirmed, intravenous potassium supplementation must be applied as soon as possible. Based on the concentration limit of potassium supplementation, oral potassium is also a good supplement choice. However, with added potassium, if abdominal distension is not relieved or continues to progress, the toxicity of intestinal paralysis should be considered, and the relevant examinations should be performed, including tests for blood amylase, lipase, urinary amylase and abdominal DR and ultrasonic imaging. Once the diagnosis of intestinal paralysis is established, antibiotics for gram-negative bacteria and anaerobic bacteria need to be used in treatment. If above treatments for anti-inflammatory, fluid, potassium supplementation and albumin supplementation fail to improve the abdominal distension, along with lack of the intestinal movement sound, clinicians should consider possibilities of the intestinal obstruction and/or intestinal necrosis.

Abdominal ultrasound is the first choice for non-invasive examination of cholecystitis, pancreatitis and intussusception, and abdominal enhanced CT is the first choice for evaluating intestinal necrosis diagnosis. Whether diagnosis of intussusception or intestinal necrosis is established, patients must be on fasting and given nutrition intravenously. The most important part is to give albumin to KD children, because they have higher risk for IVIG-resistance and coronary aneurysm development [10]. As for hydronephrosis, it occurred before the patient had KD. As the result of comprehensive treatment, this patient did not develop damages in coronary arteries, which is the key outcome measurement for a long-term quality of life in pediatric KD patients.

We input key words "Kawasaki Disease and intestinal or gastrointestinal" when searching in PubMed. In recent 20 years, there are 34 cases of KD with intestinal involvements, including 9 girls, 23 boys, and 2 with unknown gender [11-37]. About 44.1% (15 cases) of these patients had coronary artery involvements, 20.6% (7 cases) with coronary artery aneurysm, 8.8% (3 cases) with both left and right coronary arteries lesion. One 7 months old child with coronary artery aneurysm developed complete occlusion 2 years later [26]. Six of them recovered in the follow-up. Total 7 cases (20.6%) had pericardial effusion, and all recovered in one month of the illness. The incidence of coronary artery lesion in KD with intestinal involvement is 43.8% (21/48), which is much higher than the average incidence of 0.3%-5.3% in KD [38,39].

This is mainly due to the leakage of proteins caused by intestinal arteritis and obstruction to protein absorption and utilization due to inflammatory edema in intestinal walls. In consequence, hypoproteinemia leads to coronary artery edema and exacerbates coronary artery lesion. According to these case reports, some of patients presented intestinal symptoms at first as primary symptoms, which may result in delaying diagnosis of KD in these patients. Among the 34 reported cases with intestinal involvement, there were 13 with intestinal obstruction, and the most common complication in patients was pseudo-obstruction. 7 with abdominal effusion, 2 with intestinal wall edema, 2 with ischemic intestinal stenosis, 2 with limited peritonitis, 2 with cholestasis, 1 with intestinal dilatation, 1 with intussusception, 1 with descending duodenal perforation, 1 with pancreatitis, 1 with appendicitis, and 1 with sigmoid colitis. Though their inflammation and lesion were relatively limited, without prompt IVIG treatment, these patients tend to develop coronary artery complications. This patient presented with vomiting, progression of abdominal distension.

Physical examination found his abdomen was distended and his bowel movement sounds were weakened and eventually diminished after treatment. Laboratory test showed significantly increased levels of inflammatory markers. Abdominal ultrasound showed dilated intestinal tubes. Enhanced abdominal CT revealed dilated abdominal intestinal tubes, pneumatosis, and gallbladder wall edema. There was extensive air accumulated in the small intestine wall (intestinal ischemia or mucosal necrosis) at the middle and lower abdomen, along with slightly thickened rectal wall and mild interstitial edema around portal vein. Test results showed

that D-Dimer level was 4810 ug/L (<252 ug/L) at admission and continued to increase to 7384 ug/L five days later. After treatment, D-Dimer level decreased to 1423 ug/L (Figure 4). Thus, he met the criteria for intestinal ischemia/necrosis [8,40].

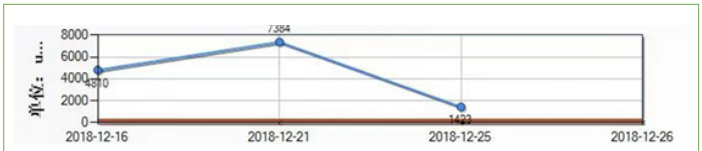

Figure 4: The dynamic changes of D-Dimer.

In this case, enhanced CT revealed necrotic gas accumulation in the wall of the large area in small intestine. If the patient did not receive timely treatment, the intestine involvement could progress to necrotic enterocolitis. In that case, the necrotic intestine would need to be surgically removed, which may lead to short bowel syndrome. Therefore, timely diagnosis combined with appropriate comprehensive treatment is the keys to noninvasively treat the intestinal complication in KD patients.

CONCLUSION

In our center, we have seen significant number of KD patients who have complication in digestive system. In cases with mild symptoms, once KD patients were treated timely, the mild symptoms diminish. However, in cases with severe complications, patients need comprehensive treatment. When a child presents with remittent fever, rashes, hypoproteinemia, intestinal symptoms, especially with pseudo-ileus observed in radiography examination, a diagnosis of KD with intestinal complication should be considered. If intestinal necrosis is limited to the mucosal layer, prompt diagnosis and proper comprehensive treatment can prevent complication progressing.

DECLARATIONS

Ethics approval and consent to participate

We have submitted a statement on ethics approval and consent.

CONSENT FOR PUBLICATION

Written informed consent for the publication of this case report and figures was obtained from the parents.

AVAILABILITY OF DATA AND MATERIALS

All data generated or analyzed during this study are included in this published article.

COMPETING INTERESTS

The authors declare that they have no competing interests.

FUNDING

No

AUTHORS' CONTRIBUTIONS

XZ C, the case observation, data collection, manuscript editing. H W*, the case diagnosis, treatment, and the main points discussed. All authors have read and approved the manuscript.

ACKNOWLEDGEMENTS

Not applicable.

REFERENCES

1. Menikou S, Langford PR, Levin M. Kawasaki disease: The role of immune complexes revisited. Front Immunol. 2019;10:1156.
2. McCrindle BW, Rowley AH, Newburger JW, Burns JC, Bolger AF, Gewitz M, 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(17):e927-e999.
3. Yaniv L, Jaffe M, Shaoul R. The surgical manifestations of the intestinal tract in Kawasaki disease. J Pediatr Surg. 2005;40(9):e1-e4.
4. Wang H. Paediatric kawasaki disease: Clinical analysis and cases. Springer Nature. 2021;pp:1-6.
5. Koca T, Aslan N, Akaslan Kara A, Pektas A, Ozen M, Akcam M. Kawasaki disease in a 9-year old girl presenting with febrile cholestasis: Case report and review of literature. Int J Rheum Dis. 2018;21(11):2046-2049.
6. Lydia K, Evangelos K, George P, Kyriaki K. A girl with extremely refractory Kawasaki disease: An instructive case with unusual course and outcome. Cardiol Young. 2012;22(4):461-465.
7. Morasch MD, Ebaugh JL, Chiou AC, Matsumura JS, Pearce WH, Yao JS. Mesenteric venous thrombosis: A changing clinical entity. J Vasc Surg. 2001;34(4):680-684.
8. Chen H, Wu Q, Fang H, Liang B, Fang L. Intestinal necrosis cannot be neglected in a patient with hepatic portal vein gas combined with appendicitis: A rare case report and literature review. BMC surg. 2019;19(1):1-6.
9. Hu JK. D-dimer test may contribute to detect acute mesenteric ischemia and intestinal necrosis. World J Surg. 2015;39(6):1584-1585.
10. Fabi M, Corinaldesi E, Pierantoni L, Mazzoni E, Landini C, Bigucci B, et al. Gastrointestinal presentation of Kawasaki disease: a red flag for severe disease? PloS One. 2018;13(9):e0202658.
11. Beiler HA, Schmidt KG, Von Herbay A, Loffler W, Daum R. Ischemic small bowel strictures in a case of incomplete Kawasaki disease. J Pediatr Surg. 2001;36(4):648-650.
12. Krohn C, Till H, Haraida S, Kurnik K, Boehm R, Grantzow R, et al. Multiple intestinal stenoses and peripheral gangrene: A combination of two rare surgical complications in a child with Kawasaki disease. J Pediatr Surg. 2001;36(4):651-653.
13. Fang SB, Lee HC, Huang FY, Chen MR. Intestinal pseudo-obstruction followed by major clinical features of Kawasaki disease: Report of one case. Acta Paediatr Taiwan. 2001;42(2):111-114.
14. Zulian F, Falcini F, Zancan L, Martini G, Secchieri S, Luzzatto C. Acute surgical abdomen as presenting manifestation of Kawasaki disease. J Pediatr. 2003;142(6):731-735.
15. Akikusa JD, Laxer RM, Friedman JN. Intestinal pseudo obstruction in Kawasaki disease. Pediatrics. 2004;113(5):e504-e506.
16. Thabet F, Bellara I, Tabarki B, Kchaou H, Selmi H, Yacoub M, et al. Ischemic colitis and hemophagocytosis complicating Kawasaki disease. Arch Pediatr. 2004;11(3):226-228.
17. Tiao MM, Huang LT, Liang CD, Ko SF. A typical Kawasaki disease presenting as intestinal pseudo-obstruction. J Formos Med Assoc. 2006;105(3):252-255.
18. Pavone P, Nicolini A, Armenia R, D'Orazio A, Nunnari G, Nicolini E. Kawasaki's disease with paralytic ileus. A case report. Minerva Pediatr. 2006;58(2):193-197.

19. Maurer K, Unsinn KM, Waltner-Romen M, Geiger R, Gassner I. Segmental bowel-wall thickening on abdominal ultrasonography: An additional diagnostic sign in Kawasaki disease. Pediatr Radiol. 2008;38(9):1013-1016.

20. Yu XX, Li XM, Wang H, Zhang Y, Huo YL. KD with digestive system involvement. Paediatr Kawasaki Disease. 2021;pp:113-136.

21. Hussain RN, Ruiz G. Kawasaki Disease presenting with intussusception: A case report. Ital J Pediatr. 2010;36(1):1-3.

22. Lin YL, Chang TJ, Lu KC, Hu WL, Ke TY. Surgical treatment of Kawasaki disease with intestinal pseudo-obstruction. Indian J Pediatr. 2011;78(2):237-239.

23. Miyamoto K, Yamazaki Y, Okamoto K, Tsuboi T, Hirao JI, Arisaka O. Kawasaki disease: Relationship between acute surgical abdomen and cytokine profiles. Pediatr Infect Dis J. 2013;32(11):1299.

24. Miyahara M, Hirayama M. Kawasaki disease resembling acute colitis. BMJ Case Rep. 2013;2013:bcr2012007384.

25. Garnett GM, Kimball S, Melish ME, Thompson KS, Puapong DP, Johnson SM, et al. Appendicitis as the presenting manifestation of Kawasaki disease. Pediatr Surg Int. 2014;30(5):549-552.

26. Godart F, Bakhti OM, Bonnevalle M. Intestinal ischemia as a severe presentation of Kawasaki disease leading to short-bowel syndrome. Cardiol Young. 2014;24(3):567-570.

27. Losada V, Montali C. Acute abdomen at onset of incomplete and atypical Kawasaki disease: Case report. Arch Argent Pediatr. 2015;113(2):e88-e93.

28. Masoumi K, Forouzan A, Saidi H, Javaherizadeh H, Khavanin A, Bahadoram M. Spontaneous duodenal perforation as a complication of Kawasaki disease. Case Rep Pediatr. 2015;2015:689864.

29. Trapani S, Montemaggi A, Simonini G, Calabri GB, Messineo A, Resti M. Surgical abdomen with intestinal pseudo-obstruction as presenting feature of atypical Kawasaki disease. J Paediatr Child Health. 2016;52(11):1032-1034.

30. Lone YA, Menon J, Menon P, Vaiphei K, Rao KL, Thapa B, et al. Kawasaki disease presenting as acute intestinal obstruction. J Indian Assoc Pediatr Surg. 2017;22(3):170-172.

31. Zhuang Y, Zhou Y, Gu Y. Intestinal pseudo-obstruction as the presenting manifestation of Kawasaki disease. Indian J Pediatr. 2017;84(8):647-648.

32. Ohnishi Y, Mori K, Inoue M, Satake N, Yano M. A case of Kawasaki disease presenting as sigmoid colitis. Journal of Medical Ultrasonics. 2018;45(2):381-384.

33. Kaman A, Aydın-Teke T, Gayretli-Aydın ZG, Oz FN, Metin-Akcan O, Eris D, et al. Two cases of Kawasaki disease presented with acute febrile jaundice. Turk J Pediatr. 2017;59(1):84-86.

34. Botti M, Costagliola G, Consolini R. Typical Kawasaki disease presenting with pancreatitis and bilateral parotid gland involvement: A case report and literature review. Front Pediatr. 2018;6:90.

35. Koca T, Aslan N, Akaslan Kara A, Pektas A, Ozen M, Akcam M. Kawasaki disease in a 9-year old girl presenting with febrile cholestasis: Case report and review of literature. Int J Rheum Dis. 2018;21(11):2046-2049.

36. Karunakar P, Gunasekaran D, Ramamoorthy JG, Krishnamurthy S, Anantharaj A, Sasidharan A, et al. Kawasaki disease presenting with intestinal pseudo-obstruction. Indian J Pediatr. 2020;87(7):569-570.

37. Osman RS, Salum HM, Noorani M. Kawasaki disease presenting after intussusception: A case report. J Med Case Rep. 2021;15(1):1-5.

38. Colomba C, La Placa S, Saporito L, Corsello G, Ciccia F, Medaglia A, et al. Intestinal involvement in Kawasaki disease. J Pediatr. 2018;202:186-193.

39. Abou Sherif S, Ozden Tok O, Taşkoylu O, Goktekin O, Kilic ID. Coronary artery aneurysms: A review of the epidemiology, pathophysiology, diagnosis, and treatment. Cardiovasc Med. 2017;4:24.

40. Bala M, Kashuk J, Moore EE, Kluger Y, Biffl W, Gomes CA, et al. Acute mesenteric ischemia: Guidelines of the World Society of Emergency Surgery. World J Emerg Surg. 2017;12(1):1-1.