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SCIENTIFIC LETTER

Upper gastrointestinal bleeding in a patient with Kawasaki disease: A case report and literature review

Sangrado del tubo digestivo alto en un paciente con enfermedad de Kawasaki: reporte de caso y revisión de la literatura

Kawasaki disease is a systemic inflammatory disease that manifests as vasculitis, predominantly affecting medium-caliber arteries, in particular, the coronary arteries.[1]

Diagnostic criteria are based on the presence of fever $\geq$ 5 days and $\geq$ 4 of the clinical criteria: erythema or cheilitis of the lips, edema or desquamation of the hands and feet, cervical lymphadenopathy, and polymorphous exanthema and/or conjunctival injection.[2] Of the digestive tract manifestations, gastrointestinal bleeding has been reported, but it is a very rare presentation worldwide.

A previously healthy one-year-old boy had disease onset 3 days prior to his arrival at the emergency service, presenting with fever (38.2 °C), conjunctival injection, and diarrheic stools with no mucus or blood, as well as edema in his lower limbs; 24 hours before going to the emergency room, he had melenic stools on 3 occasions. He had no history of self-medication or nonsteroidal anti-inflammatory drug use prior to his symptoms. Laboratory tests were ordered, strikingly revealing severe anemia (hemoglobin 6.1 mg/dl) that was microcytic (73.3 fl) and hypochromic (25.1 pg), as well as platelets at 186 $\times$ 10^3/mcl, a prothrombin time of 10.8%, with an INR of 0.95, and a partially activated thromboplastin time of 30.5 s. Management was started with a blood product transfusion and proton pump inhibitor.

During hospitalization, the patient presented with perioral cheilitis and polymorphous exanthema, meeting the criteria for Kawasaki disease. Complementary test results reported C-reactive protein above 24 mg/l, and ESR 0 mm/h; urinalysis: leukocytes 24 cells/mcl, negative nitrites (sterile pyuria); transthoracic echocardiogram: valvulitis (slight mitral, aortic, and pulmonary insufficiency), and mild pericardial effusion. Immunoglobulin G was administered (2 g/kg/dose), without an antiplatelet agent.

Panendoscopy was performed, as part of the approach to the gastrointestinal bleeding, revealing 6 minor 5 mm ulcers at the level of the gastric body and fundus, 2 with scant bleeding and hyperemic and erythematous mucosa. Biopsies were taken (Fig. 1).

Pathology report: In the gastric body and fundus, the gastric mucosa had a slight increase in lymphocytes and plasma cells; there were areas of superficial erosion and recent bleeding. In the gastric antrum, the mucosa showed a moderate increase in lymphocytes, plasma cells, and the formation of lymphoid follicles. Spiral-shaped bacilli (*Helicobacter pylori* [*H. pylori*]) were identified (Fig. 2). In the duodenum, there was a slight increase in lymphocytes and plasma cells.

The patient presented with adequate clinical evolution and was released. At present, he has no symptomatology of *H. pylori* infection, but the pathology report indicated chronic gastritis data, and so he is receiving eradication therapy as an outpatient. The patient is currently in follow-up at the cardiology and rheumatology services, with no reported events, as well as at the gastroenterology service, where the verification of *H. pylori* eradication is pending. He has not presented with a new gastrointestinal bleeding episode.

A systematic review of the topic was carried out on the Medscape®, PubMed®, Scopus®, and ScienceDirect® search engines, finding a total of 6 articles that reported gastrointestinal bleeding in Kawasaki disease. Table 1 describes patient clinical characteristics reported in previous years.

The latest case of Kawasaki disease with gastrointestinal bleeding prior to anticoagulant therapy was described at the Hangzhou Hospital of the University of Zhejiang, in China, in 2020.[8]

Gastrointestinal manifestations in this entity are rare, but are important to consider, so that a timely diagnosis can be made and treatment started, thus preventing future complications.

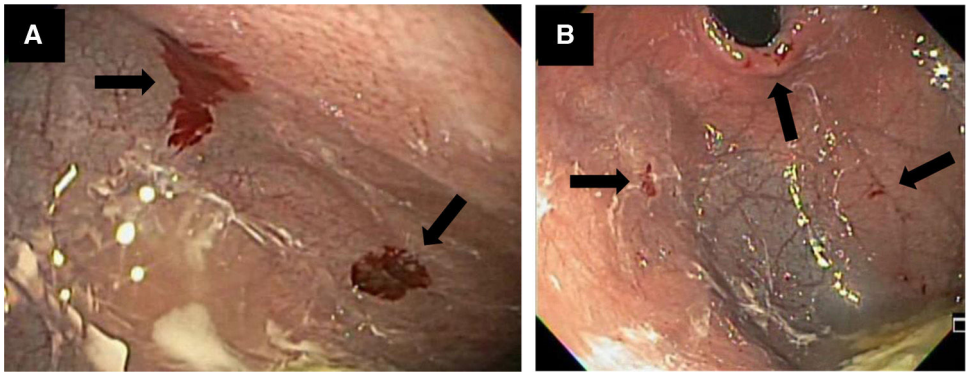

Figure 1 A) Bleeding ulcer in the gastric body. B) Ulcers in the gastric fundus.

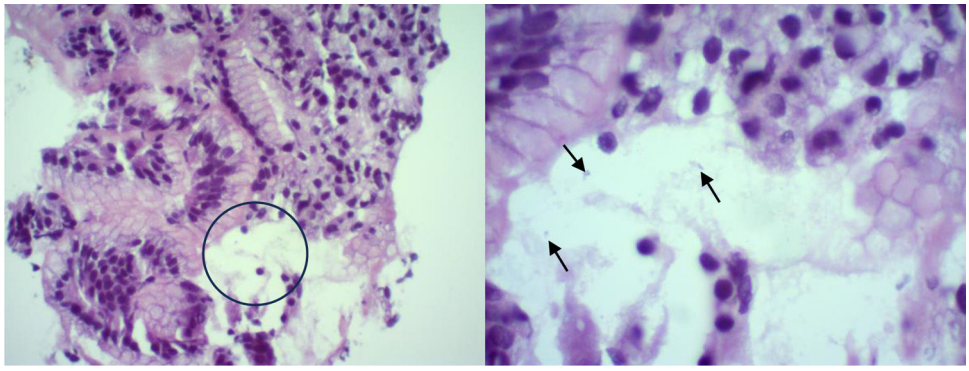

Figure 2 *Helicobacter pylori* in glycocalyx.

Table 1 Clinical characteristics reported in patients with gastrointestinal bleeding in Kawasaki disease.

Year	Lead author	Age/sex	Gastrointestinal manifestation
1996	Matsubara et al.[3]	2 years old (M)	Hematemesis after starting treatment with aspirin
1996	Matsubara et al.[3]	4 years old (F)	Melena after starting treatment with aspirin
2003	Zulian et al.[4]	20 months old (M)	Hematemesis one week after disease onset, without receiving aspirin
2004	Chang et al.[5]	5 years old (M)	Melena on day 6 from disease onset, without receiving aspirin
2007	Singh et al.[6]	4 years old (M)	Hematemesis with bleeding shock, without receiving aspirin
2019	Asada et al.[7]	7 years old (M)	Hematemesis and melena on day 5 of disease onset, without receiving aspirin
2020	Hu and Yu[8]	4 years old (M)	Hematemesis and melena on day 5 of disease onset, without receiving aspirin

M: male; F: female.

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Ethical considerations

This work meets the current bioethical research norms and was not authorized by an ethics committee, given that it contains no information that could identify the patient. Nevertheless, informed consent was obtained from the patient's guardians.

Conflict of interest

The authors declare that there is no conflict of interest.

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Hemostatic clip use for the control of ulcer bleeding after esophageal variceal ligation

Uso de clip hemostático para el control del sangrado por úlcera posligadura de várices esofágicas

Variceal upper gastrointestinal bleeding (UGIB) is one of the complications of portal hypertension. It is associated with high morbidity and mortality at 6 weeks, in around 15-20% of patients.[1] Endoscopic variceal ligation (EVL) plays an important role in treating said bleeds.[2,3] Despite its efficacy and safety profile, the procedure can cause potentially life-threatening bleeding secondary to ulceration, following band ligation.[4,5]

A 57-year-old woman had a past medical history of diabetes mellitus, high blood pressure, and Child B cirrhosis of the liver, with a MELD score of 10. She was diagnosed in 2023, when she presented with her first episode of variceal UGIB.

She was admitted to our hospital in June 2024 and programmed for EVL as an outpatient. In the gastroduodenal endoscopy (GDE), 3 large esophageal varices and one medium-sized varix were found. EVL was carried out using 3 elastic bands, with no immediate complications (Fig. 1). Ten days later, the patient arrived at the emergency service, presenting with hematemesis at a volume of 1000 cc, melena, and syncope.

Physical examination revealed that she was hemodynamically unstable, with a Glasgow coma scale score of 14. Laboratory tests showed a decrease in hemoglobin, compared with the previous outpatient control value (from 10.5 to 7.6 g/dl).

The patient was admitted to the intensive care unit and medical management of the variceal UGIB was started. During the first hours of hospitalization, she presented with a new episode of hematemesis, greater hemodynamic instability, and a decrease in hemoglobin to 5.9 g/dl. She was intubated and vasopressor support was started.

GDE was performed, revealing an abundant amount of blood in the esophagus (Fig. 2A) arising from an actively bleeding post-ligation ulcer. The previously placed elastic band was becoming detached (Fig. 2B) and came completely detached during the procedure (Fig. 2C). In addition, 3 large varicose cords that were distally ligated were identified. A new ligation at the level of the post-ligation ulcer was unsuccessfully attempted. With the aid of an endoscopic cup, a hemostatic clip was placed, achieving complete hemostasis (Fig. 2D).

The patient's clinical evolution was good, and she was released after 5 days, with no complications.

The incidence of post-ligation bleeding (PLB) is from 2. 3 to 7 .3%,[3] with a mortality rate of 22.3-24.5%.[2,5]

After EVL, the band remains attached to the esophageal wall for 3 to 7 days and thrombi develop in the strangulated vessels.[5,6] The band then detaches, resulting in an ulcer that generally heals in 2 to 3 weeks.[3] Premature detachment of the band exposes the vessel inside the ulcer, leading to PLB.[7] The time from EVL to PLB is an average of 11 days.[2]

There is no consensus on bleed predictors after EVL.[2] Some studies [3,5,6] have found that factors associated with PLB were hepatocellular carcinoma, higher Child-Pugh score, MELD score above 10, high prothrombin time, and suboptimum dose of propranolol. In the study by Reji et al., other associated factors were peptic esophagitis and a low hemoglobin level.[2] Our patient had a Child B score of 7, a MELD score of 10, and mild anemia.

Other factors related to the endoscopic procedure are emergency EVL, the use of a higher number of bands, high-risk varices, larger varices, a higher number of esophageal varices,[8,9] and associated gastric varices.[3] Of those factors, emergency EVL is the strongest predictor for PLB.