

“KISSING-ULCER” – ONSET WITH COMPLICATIONS IN SMALL AGE

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ABSTRACT

Ulcer disease is a pathology with low incidence in pediatric population, with an unspecific symptomatology or even absent, thus leading to the development of complications. We present the case of 2 years and 10 month-old male patient, admitted in the regional hospital with the diagnosis of febrile acute gastroenteritis, who in evolution (after 5 days) associated digestive hemorrhage, thus being transferred in the Pediatrics Clinic I, Targu-Mures. The superior digestive endoscopy performed after the hemodynamic and metabolic re-equilibration, pointed out 2 ulcers in the duodenal mucosa, one with active bleeding. The treatment with proton pump inhibitors and the adequate diet initiated determined the patient's favorable evolution. The particularity of the case consists in the appearance of 2 duodenal ulcers of the duodenal mucosa in a small child, without significant personal history, with adequate life conditions, without associated risk factors and without infection with *Helicobacter pylori*.

Keywords: “kissing-ulcer”, child, superior digestive hemorrhage

INTRODUCTION

Ulcer represents a loss of substance at the level of gastric or duodenal mucosa, being the final result of the inflammation caused by the disequilibrium between the protective and aggressive factors at the level of the gastric or duodenal mucosa. “Kissing-ulcer” is a form of ulcer that consists in the presence of 2 ulcers, “in mirror” of the duodenal mucosa. This form of ulcer disease presents a small incidence in adults, being unusual in children, probably also due to the difficulty of diagnosis establishment, reason for which there aren't any recent studies that asses this clinical form in children. The etiology includes sepsis, shock of different etiologies, Zollinger-Ellison syndrome, stress, the administration of non-steroid anti-inflammatories, severe burns (Curling ulcer) or head trauma (Cushing ulcer) (1). Even though the most frequent etiology involved in the development of ulcer disease, especially the duodenal ones, is represented by the infection with *Helicobacter pylori*, in 15-20% of the cases, these ulcers do not have an obvious etiology, being named idiopathic ulcers, which

have a more increased recurrence rate than the ulcers with identifiable etiology (1). The gold-standard method used in order to establish the diagnosis remains the superior digestive endoscopy that can be performed at any age. The identification of multiple or “kissing-ulcers” in children is uncommon, thus the duodenal wall must be carefully explored during the digestive endoscopy (2). The complications such as perforation or superior digestive hemorrhage are the most frequent in case of this form of ulcer disease, with a fatal risk of 1.7-5% for upper digestive hemorrhage among children (3,4). The treatment can include drugs: proton pump inhibitors associated or not with an eradication regimen of the infection with *Helicobacter pylori*, or surgery, reserved only for the complicated cases.

CASE PRESENTATION

We present the case of small male child, 2 years and 10 month-old admitted in the regional hospital with vomiting, diarrhea and fever, associating af-

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terwards black stools initially, then with fresh blood, thus being transferred in the Pediatrics Clinic I, Targu-Mures. From the familial history we mention the following: maternal grandmother with arterial hypertension, breast cancer; maternal grandfather with the diagnosis of multiple sclerosis; paternal grandmother with gastric ulcer. The physiological history without special importance, excepting the fact that 6 months ago, the child presented several episodes of vomiting, which remitted without treatment. The actual disease begins 5 days before the admission in our clinic, with vomiting, diarrheic stools and fever, therefore being admitted in the regional hospital, where the fever disappears, but one day before the transfer in our clinic, the patient presented melena, afterwards stools with fresh blood, with the sudden decrease of hemoglobin level from 12.9 g/dl to 6.6 g/dl, an inferior digestive hemorrhage being suspicioned, an abdominal radiography is performed without obvious pathological elements, reason for which he is referred to the Pediatric Clinic I, Targu-Mures, Pediatrics Gastroenterology Compartment in order to widen the spectrum of investigation and to administer the adequate treatment. We mention that both, the stool culture and the Rotavirus infection test from the stools, performed at the regional hospital were negative. The clinical exam performed at the moment of admission points out altered general status, sorrowful facies, pale skin, diminished cutaneous turgor, persistent skinfold, hyperemia of the pharynx, equilibrated from the cardio-respiratory point of view, slightly distended abdomen, apparently without pain at palpation, accelerated bowel movements, decreased consistency stools, with fresh blood, normal liver and spleen, present diuresis, without meningeal irritation signs, W: 12 kg.

The laboratory tests performed at the moment of admission point out: severe anemia (Hb 6.6 g/dl, VEM 76.3 fL, Hem 2,490/ μ L), hyponatremia and hypopotassemia (Na 134.8 mmol/L, K 2.72 mmol/L), reasons for which we decide to administer erythrocyte mass and rehydration perfusions with saline substances supplemented with potassium chloride. Also, we associated in the treatment proton pump inhibitor, anti-emetic and prokinetic drugs, and also, taking under consideration the onset with fever and the associated hemorrhagic complication, introduced antibiotic in order to prevent a potential sepsis. In the first 24 hours after admission, he presents 3 stools with decreased consistency, the first 2 with fresh blood, and the last one colored in black. We performed gastric lavage with Etamsilat, but without pointing out fresh blood in

the lavage liquid. The abdominal ultrasound does not show any pathological elements. During the *superior digestive endoscopy*, we noticed in the initial part of the duodenum a circular lesion with active hemorrhage and at the same level, in opposition with this one, we observed another lesion, “in mirror”, circular, with prominent margins, well defined, covered by white deposit, without active hemorrhage, without other pathological elements of the stomach or esophagus. We took multiple biopsies from the duodenum, gastric antrum and corpus. Thus, the established *diagnosis* was “*Kissing-ulcer*” with active bleeding.



FIGURES 1, 2. Duodenal “kissing-ulcer”, endoscopic aspect

The evolution during admission was favorable under the administered treatment, with the normalization of the stools aspect after approximately 72 hours, the hydro-electrolytic reequilibration, with the normalization of potassium after 48 hours, he started to consume by mouth, without presenting vomiting. The hemoglobin performed after the administration of erythrocyte mass was 9 g/dl, in-

creasing progressively to 10.6 g/dl after approximately 6 days. The rest of the blood tests performed during admission, such as ESR, CRP, coagulogram, stool culture and Rotavirus test from the stools were negative. The patient was discharged in good general status, with the following recommendations: hygienic and dietary regimen according to his pathology (avoiding the aliments with increased content of fats, concentrated sweets, fresh fruits and vegetables, sweet drinks, with acid, consuming the aliments only if they are boiled, divided in 5 meals per day, 3 main meals and 2 meals), treatment with proton pump inhibitor for one month, endoscopic reevaluation in one month or in emergency in case of acute hemorrhage. After one month from discharge, the evolution was favorable, the patient did not present any vomiting or modified aspect stools, with good appetite, good general status, without other significant complaints. The endoscopic reevaluation shows the same aspect of the duodenum, but without spontaneous bleeding, and the ulcers presented epithelization areas. We recommended the continuation of the hygienic and dietary regimen and the treatment with proton pump inhibitor for another 4 weeks, with further reevaluation. *The particularity of the case* consists in the appearance of “kissing-ulcer” in a small child (2 years and 10 months) without significant pathological history, without associated risk factors, with adequate life conditions, whose onset was complicated by an upper digestive hemorrhage.

DISCUSSIONS

Duodenal ulcer disease is a pathology with low incidence in pediatric patients. A study performed in multiple centers on 694 children showed an incidence of 8.1% of ulcers and/or erosions, with higher frequency in the second decade of life (5). Even though the etiology of most cases of ulcers is represented *Helicobacter pylori* infection or by the intake of non-steroid antiinflammatory drugs, thus predominating the secondary ulcers among the children (6), in the case of the above presented patient we could not identify any obvious cause in the history, and the infection with *Helicobacter pylori* was absent in the histopathological exam. The only potential involved factor in the onset of the digestive hemorrhage, but not in the development of the two ulcers, being the gastroenteritis installed approximately 7 before the onset of hemorrhage. Similarly to our case, Wilson et al described a case of a 7 year-old patient, admitted with the diagnosis of gastroenteritis, who during admission developed

a perforation of the duodenal ulcer (7). Even though steroid or non-steroid anti-inflammatory drugs are often incriminated in the etiology of this pathology, it seems that they are not the only drugs that can lead to the development of ulcer disease, thus being described the case of patient with the age of 6 years and 6 months, diagnosed with thalassemia major, for which he received treatment with deferasirox, oral iron chelation drug (8). The clinical signs of the ulcer disease vary with the age, thus approximately one half of the small children with ulcer will present hematemesis or melena, while the school-aged ones will present symptoms similar to adults, like: nausea, loss of appetite, vomiting (1). Thus, the symptomatology is as unspecific as the patient's age is smaller, being possible to present as in our case with digestive hemorrhage or even perforation, like in the case described by Lee, of a male child, with the age of 2 years and 6 months, who presented only severe hematochezia and intense anemia (9). Progressively with child's ageing, starting from the school age, the symptomatology of the disease tends to be similar with those encountered in the adults, but this fact is not mandatory because though there are cases in which the symptoms can lack, like in the 12 year-old patient described by Mbarushimana, who presented the onset of the disease only 4 hours before admission with bilious vomiting and severe abdominal pain (10). Due to these diverse and unspecific clinical pictures, the diagnosis of this pathology is often established only at the moment when the children develop complications, and the low incidence at the pediatric age is another factor that leads to the omission the gastric or duodenal ulcer from the differential diagnosis lists among this group of patients (11-14).

CONCLUSIONS

Even though the ulcer disease has a low incidence at small ages, being associated in over 80% of the patients with the presence of *Helicobacter pylori*, in case of the patient presented above, this pathology appeared at the age of 2 years and 10 months, without infection with *Helicobacter pylori* in the histopathological exam. Also, the onset with digestive hemorrhage underlines the fact that the symptomatology of this disease in case of pediatric aged patient is unspecific or sometimes could even lack. The genetic factors, like the innate immunity receptors, can represent a potential etiology of the idiopathic cases of ulcer, further studies being needed in order to identify their contribution.

REFERENCES

1. **Blanchard S.S., Czinn S.J.** Peptic Ulcer Disease in Children. In: Kliegman RM. Nelson Textbook of Pediatrics, Elsevier, 2016: 1816-1819.
2. **Gershman G., Ravelli A.** Diagnostic upper gastrointestinal endoscopy. In Gershman G., Thomson M., Ament M. Practical Pediatric Gastrointestinal Endoscopy, Wiley-Blackwell, 2012: 41-82.
3. **Dehghani S.M., Haghghat M., Imanieh M.H., Tabebordbar M.R.** Upper gastrointestinal bleeding in children in Southern Iran. *Indian J Pediatr* 2009; 76:635–638.
4. **Colle I., Wilmer A., Le Moine O. et al.** Upper gastrointestinal tract bleeding management: Belgian guidelines for adults and children. *Acta Gastroenterol Belg* 2011; 74:45–66.
5. **Kalach N., Bontems P., Koletzko S. et al.** Frequency and risk factors of gastric and duodenal ulcers or erosions in children: a prospective 1-month European multicenter study. *Eur J Gastroenterol Hepatol* 2010; 22(10):1174-1181.
6. **Huang S.C., Sheu B.S., Lee S.C., Yang H.B., Yang Y.J.** Etiology and treatment of childhood peptic ulcer disease in Taiwan: a single center 9-year experience. *J Formos Med Assoc* 2010; 109(1):75-81.
7. **Wilson J.M., Darby C.R.** Perforated duodenal ulcer: an unusual complication of gastroenteritis. *Arch Dis Child* 1990; 65:990-991.
8. **Yadav S.K., Gupta V., Kohly A.E., Fadhli W.A.** Perforated ulcer: A rare complication of deferasirox in children. *Indian J Pharmacol* 2013; 45(3):293-294.
9. **Lee N.M., Yun S.W., Chae S.A. et al.** Perforated duodenal ulcer presenting with massive hematochezia in a 30-month-old child. *World J Gastroenterol* 2009; 15(38):4853-4855.
10. **Mbarushimana S., Morris-Stiff G., Thomas G.** Atypical presentation of perforated peptic ulcer disease in a 12-year-old boy. *BMJ Case Rep* 2014, doi: 10.1136/bcr-2014-204716.
11. **Ameh E.A.** Duodenal ulcer in childhood in developing countries. *Indian Pediatr* 2003; 40:272.
12. **Sisil Kumara P.D., Weerawardena W.A., Esufali S.T.** A perforated duodenal ulcer in a child. *Ceylon Med J* 2000; 45:133-134.
13. **Barandica R., Patel M.** Pediatric duodenal perforation missed on computed tomography. *Ann Emerg Med* 1997; 30:545–547.
14. **Grosfeld J.L., Molinari F., Chaet M. et al.** Gastrointestinal perforation and peritonitis in infants and children: experience with 179 cases over ten years. *Surgery* 1996; 120: 650-655.