**Diagnosis and Management of Ménétrier Disease in Children: A Case Series Review**

**Jasmina Krikilion, Elvira Ingrid Levy, and Yvan Vandenplas**
KidZ Health Castle, Universitair Ziekenhuis Brussel, Vrije Universiteit Brussel, Brussels, Belgium

**ABSTRACT**

**Purpose:** Ménétrier disease (MD) was first described in 1888, and 50 cases have been reported until now. We aimed to discuss the etiology, diagnostics, and management of MD in children.

**Methods:** We searched for case reports published from 2014 till 2019 in English using PubMed. Articles were selected using subject headings and key words of interest to the topic. Interesting references of the included articles were also included.

**Results:** The pathophysiology of MD is still uncertain. However, overexpression of transforming growth factor alpha with transformation of the gastric mucosa has been observed, which may be mediated by genetics and provoked by an infectious trigger. Clinically, MD is diagnosed by abdominal pain, vomiting, anorexia, and edema secondary to hypoalbuminemia. A gastroscopy with biopsy is the gold standard for the diagnosis of MD.

In children, the disease is self-limiting and only requires supportive treatment. In general, children have a good prognosis and recover spontaneously within a few weeks.

**Conclusion:** Few pediatric cases of MD have been described in recent years, and with all different etiology. Endoscopy with biopsy remains the golden standard for the diagnosis of MD, and in children, the disease is self-limiting.

**Keywords:** Ménétrier disease; Hypo-proteinemia; Hypertrophy mucosal folds; Transforming growth factor alpha

**INTRODUCTION**

Ménétrier disease (MD) was described by the French pathologist Pierre Ménétrier in 1888 [1,2]. MD is an uncommon acquired self-limiting disorder in children, of which the pathogenesis and etiology are not yet fully understood [3,4].

Until now, there have only been approximately 50 pediatric cases of MD (Table 1) [1-9] reported in the literature, the majority of which are case series. In this review, we discuss the etiology and propose guidance for the diagnosis and management of MD.
CLINICAL MANIFESTATIONS

MD requires a clinical-pathological diagnosis since there are no known pathognomonic features to diagnose MD. The symptoms described in adults (males are more commonly affected than females) include vomiting, nausea, abdominal pain, diarrhea, weight loss, malnutrition, and peripheral edema secondary to hypoalbuminemia [1,5]. Children with MD often demonstrate a prodromal phase caused by a transient viral infection, followed by edema and gastro-intestinal symptoms, including emesis, epigastric pain, anorexia, diarrhea, vomiting, and abdominal pain (Table 2) [3,4,6,7,10-35]. Edema is caused by hypoalbuminemia as a result of protein loss as a consequence of edema of the gastric mucosa [4]. The average age of affected children is 2–5 years [6]; however, a case series from Gökçe and Kurugöl [4] describes two cases of neonatal MD, both with edema as a major symptom [4]. Since spontaneous remission is common in children, it is possible that the disease is associated with *Helicobacter pylori* infection or transient infections such as cytomegalovirus (CMV) [4,5]. These associations will be discussed later in this review.

There is a wide variation in the clinical manifestations of MD depending on the age of the patient; thus, it is important to consider MD in the differential diagnoses of edema that occurs in combination with gastro-intestinal symptoms.

PATHOPHYSIOLOGY AND ETIOLOGY

The pathogenesis of MD is not yet fully understood [3,4]. However, observational studies in transgenic mice showed a potential relationship between the overexpression of transforming growth factor alpha (TGF-α) and the development of gastric changes that are characteristic of MD [5]. TGF-α inhibits gastric acid production and stimulates the growth of gastric epithelial cells [1]. Furthermore, TGF-α mediates signal transduction by binding to the epidermal growth factor receptor (EGFR), which leads to increased cellular proliferation [5]. More specifically, in MD, overexpression of TGF-α redirects the gastric progenitor cells to surface mucous cell differentiation at a disadvantage to parietal and chief cell differentiation [5]. Remarkably, the gastrin levels in serum are normal, despite lower gastric acidity, which is a stimulus for increased production of gastrin [1].

In children, MD is transient and is generally believed to be associated with infections such as herpes simplex virus, Giardia lamblia, Mycoplasma pneumonia, CMV, and *H. pylori* [3-6]. One possible pathogenic mechanism is the abnormal accumulation of local TGF-α as a result of damage to the gastric mucosa caused by infection [5]. CMV infection in the stomach causes elevation of intracellular messengers and activation of proto-oncogenes, both of which cause an increase in the production of TGF-α in mucosal cells [6]. Some case reports have shown an association between MD and some medications and allergy [4]. Indeed, several patients have MD with CMV and *H. pylori* co-infection, although it has been proposed that *H. pylori*...
### Table 2. Pediatric cases of Ménétrier disease

| Author, year | Cases | Age | Sex | Medical history | Diagnosis | Treatment | Comments |
|--------------|-------|-----|-----|-----------------|-----------|-----------|----------|
| Gökçe and Kurugöl, 2017 [4] | 3 | 11 yr | M | Edema, vomiting, appetite loss | Clinical examination, hypoalbuminemia, gastric endoscopy and biopsy, CMV positive | Albumin infusion and furosemide | All patients tested positive for CMV infection |
| | 3 mo | M | Edema, ichthyosis, hydronephrosis | Clinical examination, hypoalbuminemia, X-ray barium swallow, CMV positive, gastric endoscopy and biopsy | Albumin infusion and furosemide, Ganciclovir, and H2 receptor blockers |
| | 2 mo | M | Pallor, edema | Clinical examination, hypoalbuminemia, CMV positive, gastric endoscopy and biopsy | Albumin infusion, furosemide, Valgancyclovir |
| Yoo et al., 2013 [3] | 1 | 3 yr | M | Anorexia, vomiting, facial and peripheral edema | Clinical examination, hypoalbuminemia, hypoproteinemia, gastric endoscopy and biopsy, C13 urea breath test positive, CMV positive | H. pylori eradication with lansoprazole, Co-infection with H. pylori amoxicillin, and clarithromycin; albumin infusion, fluid restriction, diuretics, and high-protein diet |
| Baker et al., 1986 [10] | 4 | 2.5 yr | M | Edema, vomiting, diarrhea | Clinical examination, hypoalbuminemia, hypoproteinemia, upper gastrointestinal X-rays, endoscopy biopsy | No treatment |
| | 6.5 yr | M | Congestion, nausea, anorexia, abdominal swelling | Clinical examination, hypoalbuminemia, hypoproteinemia, chest X-ray, upper gastrointestinal X-rays, endoscopy and biopsy | High-protein diet |
| | 2.5 yr | M | Vomiting, edema, weight gain | Clinical examination, hypoalbuminemia, upper gastrointestinal X-rays, endoscopy biopsy | No treatment |
| | 5 yr | F | Abdominal pain, anorexia, vomiting | Hypoalbuminemia, hypoproteinemia, upper gastrointestinal X-rays, endoscopy biopsy, Gastrotomy | High-protein diet |
| Canan et al., 2008 [11] | 1 | - | - | - | - | - | Turkish article |
| Hong et al., 2018 [12] | 1 | 22 mo | M | Vomiting and poor oral intake; cough, rhinorrhea, and fever developed 1 week prior to presentation. Lethargic and decreased urination at presentation | Clinical examination (edema, icteric sclera, and hepatosplenomegaly), hypoalbuminemia, hypoproteinemia, abdominal ultrasonography, gastric endoscopy and biopsy, CMV positive | Albumin infusion and lansoprazole | Acute CMV infection |
| Megged and Schlesinger, 2008 [13] | 8 | 1 yr | F | Fever, vomiting | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| | 3.5 yr | F | Edema, jaundice | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion, Ganciclovir |
| | 2 yr | M | Vomiting, diarrhea | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion, and Ganciclovir |
| | 3 yr | F | Abdominal pain, diarrhea, edema | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| | 3.5 yr | M | Anemia, vomiting | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| | 4 yr | M | Edema | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| | 4.5 yr | F | Diarrhea, edema | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| | 2.5 yr | M | Fever, edema | Hypoalbuminemia, CMV positive | Supportive care and albumin infusion |
| Floret et al., 1978 [14] | 1 | 2 yr | F | - | Hypoproteinemia associated with gastritis | - | Only abstract available |
| Gilles et al., 1994 [15] | - | - | - | - | - | - | No abstract available |
| Oderda et al., 1990 [16] | 2 | - | - | - | - | - | Only abstract available |
| Roussel et al., 1990 [17] | 1 | 7 yr | M | Complete digestive intolerance and protein loss, resulting in major hypoalbuminemia and edema | Upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive | Albumin infusions |
| Cardona Barberán et al., 2006 [18] | 1 | 15 mo | - | Intractable vomiting, weight gain, and generalized progressive edema | Clinical examination, hypoproteinemia, hypoalbuminemia, hyponatremia, abdominal ultrasound, gastric endoscopy and biopsy, CMV positive | Albumin infusion, high-protein diet antacids | (continued to the next page) |
## Table 2. Pediatric cases of Ménétrier disease

| Author et al., year | Cases | Age | Sex | Medical history | Diagnosis | Treatment | Comments |
|---------------------|--------|-----|-----|----------------|-----------|-----------|----------|
| Occena et al., 1993 | 2 | 3.7 yr | M | Vomiting, edema, sore throat, low-grade fever, diarrhea, abdominal distention | Clinical examination, stool culture positive for Salmonella type B, hypoproteinemia, hypoalbuminemia, chest X-ray, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive | Diuretics, fluid and salt restriction, albumin infusion, high-protein diet, rifampin, trimethoprim/sulfamethoxazole | Developed sepsis and died from brain stem infarction |
| Coad and Shah, 1986 | 1 | 4 yr | M | Congestion, vomiting and anorexia, edema, abdominal distention | Clinical examination, hypoalbuminemia, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive | High-protein diet | |
| Fishbein et al., 1992 | 1 | 3 mo | M | Diarrhea, vomiting, failure to thrive | Characteristic radiological, pathological, and functional abnormalities of the stomach, hypoproteinemia, CMV positive | Prednisone, cyclosporine A, diuretics, high-protein diet | No treatment association between Ménétrier’s disease and a bezoar |
| Hillman et al., 2013 | 1 | - | - | Edema and ascites | Possible CMV infection | Self-limiting supportive care | Only abstract available |
| Chang et al., 2000 | 1 | 4 yr | M | Two-week history of vomiting and periorbital edema | Clinical examination, hypoalbuminemia, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive | Fluid restriction, albumin infusion, furosemide, PPI, valganciclovir | No treatment |

(continued to the next page)
| Author, year | Cases | Age | Sex | Medical history | Diagnosis | Treatment | Comments |
|-------------|-------|-----|-----|----------------|-----------|-----------|----------|
| Tard et al., 2019 [6] | 2 | 7 yr | F | Epigastric pain and vomiting, facial edema | Clinical examination, hypoalbuminemia, gastrointestinal endoscopy and biopsy, CMV positive | Esomeprazole | |
| | 5.5 yr | F | Facial edema and abdominal pain with vomiting and anorexia | Clinical examination, hypoalbuminemia, abdominal ultrasonography, upper gastrointestinal endoscopy and biopsy | IV hydration, anti-vomiting and pain medication | |
| Imataki et al., 2018 [29] | 1 | 13 yr | M | Anemia, no other symptoms | Anemia, *H. pylori* positive, hypoproteinemia | *H. pylori* eradication therapy | |
| Iwama et al., 2010 [7] | 1 | 18 mo | M | Abdominal distention, edema, fever, diarrhea, cough | Clinical examination, hypoproteinemia, hyperglobulinemia, elevation of alfa-1-antitrypsin in stool, abdominal ultrasonography, endoscopy and biopsy, CMV positive, *H. pylori* positive | Albumin infusion, gamma globulin infusion | |
| Kraut et al., 1981 [30] | 2 | 11 yr | M | Abdominal pain, decreased exercise tolerance, pallor | Clinical examination, anemia, gastric endoscopy and biopsy, hypoalbuminemia | Transfusion, oral ferrous sulphate, gastric resection | No specific treatment |
| | 7 yr | M | Vomiting, malaise, edema | Clinical examination, hypoalbuminemia, hypogammaglobulinemia, chest X-ray, abdominal X-ray, upper gastrointestinal X-ray, gastric endoscopy and biopsy, CMV positive | Fluid restriction, high-protein diet, albumin infusion, furosemide | |
| Blackstone and Mittal, 2008 [31] | 1 | 3 yr | F | Edema, decreased activity, decreased appetite, abdominal distention, emesis | Clinical examination, hypoproteinemia, hypoaalbuminemia, hypotremia, chest X-ray, elevated fecal alfa-1-antitrypsin, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive | Supportive treatment: plasma and albumin infusions | Only abstract available |
| Ricci et al., 1996 [32] | 3 | 3 mo-3 yr | | Protracted vomiting, generalized edema, colitis (one case) and elevated serum aminotransferases (one case) | Typical endoscopic and histological picture of the gastric mucosa (two cases). Typical radiological findings (one case). High fecal alpha-1-antitrypsin excretion in all patients. Evidence of primary CMV infection (two cases). | Supportive treatment: plasma and albumin infusions | Only abstract available |
| Zhang et al., 2020 [33] | 1 | 4.5 yr | M | Vomiting, abdominal pain, hypoproteinemia, edema | Hypoproteinemia, hypoaalbuminemia, hypogammaglobulinemia, clostridium difficile positive, abdominal ultrasound, abdominal CT, gastrointestinal endoscopy with biopsy | Albumin infusion, Vancomycin | Infection with clostridium difficile |
| Pederiva et al., 2006 [34] | 1 | - | - | Vomiting, weight loss, abdominal pain | Hypoaalbuminemia, gastric endoscopy with biopsy, CMV positive | - | Only abstract available |
| Wilches-Luna et al., 2018 [35] | 2 | 23 mo | M | Flu, fever, vomiting, bloating, diarrhea, asthenia, adynamia, loss of appetite, edema, weight gain | Clinical examination, hypoproteinemia and hypoaalbuminemia, positive for CMV, abdominal ultrasound, gastric endoscopy with biopsy | Diuretics, albumin infusion, esomeprazole | |
| | 5 yr | M | Abdominal pain, vomiting, diarrhea, edema, oliguria | Clinical examination, abdominal CT, hypoproteinemia, hypoaalbuminemia, chest X-ray, abdominal ultrasound, gastric endoscopy and biopsy | Diuretics, albumin infusion, antisecretory management and nutritional management | |

M: male, F: female, CMV: cytomegalovirus, *H. pylori*: *Helicobacter pylori*, IV: intravenous, PPI: proton pump inhibitor.
has the most causative role in the disease [4,7]. However, given the high incidence of *H. pylori* infection, these associations may be coincidental. A case series of two siblings with CMV-associated MD proposed the hypothesis that genetic factors may stimulate increased production of TNF-α in response to CMV infection [6].

A genetic predisposition to develop MD has been proposed following the discovery of a unique, four-generation pedigree with an autosomal dominant gastropathy exhibiting the typical clinical, endoscopic, and pathological MD-like findings, although in the absence of protein loss and with no increase in the levels of gastric TGF-α [8].

Although the pathogenesis of MD still has to be explored further, there is evidence of overexpression of TGF-α and transformation of the gastric mucosa, possibly mediated by genetics and provoked by an infectious trigger.

**DIAGNOSTICS AND HISTOLOGICAL FINDINGS**

The diagnosis of MD starts with a thorough history of the patient, in which contact with family members with possible *H. pylori* infection is investigated. Gastroscopy, biopsies, and cultures must be performed to confirm the diagnosis of MD. MD is also characterized by endoscopic findings of thickened gastric mucosal folds that are predominantly present in the body and the fundus of the stomach, relatively sparing the antrum (Table 1) [1-9]. The most striking feature of MD, a histological sine qua non, is foveolar hyperplasia (expansion of the mucosal cell surface), which leads to thickening of the gastric mucosa. There is also a loss of parietal cells due to atrophic oxyntic glands, which subsequently leads to an increase in the gastric pH; the normal pH of gastric fluid is 1–3, while that in MD is 4–7 [1,5]. Additionally, deep glands are often dilated, forming cysts. Histologically, there is chronic inflammatory cell infiltration at the lamina propria with the presence of eosinophils and plasma cells, hyperplasia of smooth muscle, and edema [1,5].

Other diseases with similar endoscopic findings include hypertrophic lymphocytic gastritis, eosinophilic gastritis, Zollinger-Ellison syndrome, polyposis syndrome, gastric malignancies, and lymphoma [5,6]. Interestingly, a new mechanism that involves TGF-β-SMAD 4 pathway inactivation and TGF-α overexpression related to *H. pylori* infection has been proposed to explain the association of juvenile polyposis syndrome with MD [8].

In conclusion, the golden standard for the diagnosis of MD is to perform gastroscopy with biopsy and the typical histological findings.

**TREATMENT**

The management of MD in children is often supportive as most of the reported cases are associated with transient infections. As infection resolves spontaneously, MD usually resolves within several weeks to months [4,5]. If there is evidence of *H. pylori* infection, eradication can be considered, although a previous report described a case in which MD resolved without the use of antibiotics [3,6]. As *H. pylori* is the only known causative organism that is not a transient infection, we believe that the association between MD and *H. pylori* is a coincidence.
Supportive treatment of MD includes albumin infusion to correct hypo-albuminemia, as well as diuretics, fluid restriction, and a high-protein diet [2,3]. Furthermore, acid inhibitors, such as proton pump inhibitors and H2 receptor blockers, and anticholinergic agents are used to protect the stomach; no preference of acid inhibitors has been reported. Ganciclovir treatment can be considered if there is evidence for active CMV infection, and if the patient is immunocompromised, very young, or if spontaneous improvement does not occur [4,6]. In adults and adolescents with chronic and severe MD chirurgical therapy, such as partial or total gastrectomy, can be considered [2,5]. Further clinical trials with cetuximab, an immunoglobulin that binds to EGFR and prevents the binding of TGF-α, have shown promising results with rapid improvement of symptoms after the first administration in adults [1].

In conclusion, the treatment of MD in children is mainly supportive, although in some cases, correction of hypoalbuminemia with albumin infusions and administration of diuretics is needed (Table 1) [1-9].

CONCLUSION

MD is a rare condition in children, and our knowledge of its pathophysiology and etiology remains incomplete. New possible mechanisms and the involvement of genetics in the pathophysiology of MD have been suggested but require further investigation. Moreover, some viral, bacterial, and parasitic infections are associated with the condition. The disease can only be diagnosed by gastroscopy and histology of gastric biopsies. MD in children is self-limiting, and supportive therapy is advised.

REFERENCES

1. Coffey RJ Jr, Tanksley J. Pierre Ménétrier and his disease. Trans Am Clin Climatol Assoc 2012;123:126-33; discussion 133-4.
2. Azer M, Sultan A, Zalata K, Abd El-Haleem I, Hassan A, El-Ebeidy G. A case of Menetrier’s disease without Helicobacter pylori or hypoalbuminemia. Int J Surg Case Rep 2015;17:58-60.
3. Yoo Y, Lee Y, Lee YM, Choe YH. Co-infection with cytomegalovirus and Helicobacter pylori in a child with Ménétrier’s disease. Pediatr Gastroenterol Hepatol Nutr 2013;16:123-6.
4. Gökçe Ş, Kurugöl Z. Cytomegalovirus-associated Menetrier disease in childhood. Clin Pediatr (Phila) 2017;56:382-4.
5. Huh WJ, Coffey RJ, Washington MK. Ménétrier’s disease: its mimickers and pathogenesis. J Pathol Transl Med 2016;50:10-6.
6. Tard C, Madhi F, Verlhac S, Hagège H, Epaud R, Jung C. Protein-losing gastropathy associated with cytomegalovirus in two sisters: case reports and review of the literature. Arch Pediatr 2019;26:232-5.
7. Iwama I, Kagimoto S, Takano T, Sekijima T, Kishimoto H, Oba A. Case of pediatric Ménétrier disease with cytomegalovirus and Helicobacter pylori co-infection. Pediatr Int 2010;52:e200-3.
8. Piepoli A, Mazzoccoli G, Panza A, Tirino V, Biscaglia G, Gentile A, et al. A unifying working hypothesis for juvenile polyposis syndrome and Ménétrier’s disease: specific localization or concomitant occurrence of a separate entity? Dig Liver Dis 2012;44:952-6.
9. Strisciuglio C, Corleto VD, Brunetti-Pierri N, Piccolo P, Sanguerano R, Rindi G, et al. Autosomal dominant Ménétrier-like disease. J Pediatr Gastroenterol Nutr 2012;55:717-20.

10. Baker A, Volberg F, Sumner T, Moran R. Childhood Menetrier’s disease: four new cases and discussion of the literature. Gastrointest Radiol 1986;11:131-4.

11. Canan O, Özçay F, Bilezikçi B. Ménétrier’s disease and severe gastric ulcers associated with cytomegalovirus infection in an immunocompetent child: a case report. Turk J Pediatr 2008;50:291-5.

12. Hong J, Lee S, Shon Y. Ménétrier’s disease as a gastrointestinal manifestation of active cytomegalovirus infection in a 22-month-old boy: a case report with a review of the literature of Korean pediatric cases. Clin Endosc 2018;51:89-94.

13. Megged O, Schlesinger Y. Cytomegalovirus-associated protein-losing gastropathy in childhood. Eur J Pediatr 2000;167:1217-20.

14. Floret D, Renaud H, Hage G, Aymard M, Monnet P. [Gastritis with hypoproteinemia in children. Relation to Menetrier’s disease and cytomegalic inclusion disease]. Arch Fr Pediatr 1978;35:82-9. French.

15. Gilles I, Chevallier B, Gelez J, Gompel H, Lagardère B. [Benign hypertrophic gastropathy associated with cytomegalovirus infection in children]. Presse Med 1994;23:182. French.

16. Oderda G, Cinti S, Cangiotti AM, Forni M, Ansaldi N. Increased tight junction width in two children with Ménétrier’s disease. J Pediatr Gastroenterol Nutr 1990;11:123-7.

17. Roussel M, Dupont C, Sidibe T, Andre C, Barbet P, Badoual J. [Benign hypertrophic gastritis associated with cytomegalovirus infection]. Arch Fr Pediatr 1990;47:271-3 French.

18. Cardona Barberán A, Sorní Hubrecht A, Hostalot Abás A, Rosal Roig J, Mercé Gratacós J, Izuel Navarro JA. [Ménétrier’s disease of childhood and acute cytomegalus virus infection]. An Pediatr (Barc) 2006;64:478-80 Spanish.

19. Ouennou RO, Taylor SF, Robinson CC, Sokol RJ. Association of cytomegalovirus with Ménétrier’s disease in childhood: report of two new cases with a review of literature. J Pediatr Gastroenterol Nutr 1993;17:217-24.

20. Coad NA, Shah KJ. Menetrier’s disease in childhood associated with cytomegalovirus infection: a case report and review of the literature. Br J Radiol 1986;59:615-20.

21. Fishbein M, Kirschner BS, Gonzales-Vallina R, Ben-Ami T, Lee PC, Weisenberg E, et al. Menetrier’s disease associated with formula protein allergy and small intestinal injury in an infant. Gastroenterology 1992;103:1664-8.

22. Hochman JA, Witte DP, Cohen MB. Diagnosis of cytomegalovirus infection in pediatric Menetrier’s disease by in situ hybridization. J Clin Microbiol 1996;34:2588-9.

23. Kirberg BA, Rodríguez VB, Donoso VE, Kirhman TM, Noriel VM. [Hypertrophic protein-losing gastropathy: Ménétrier disease. A clinical case]. Rev Chil Pediatr 2014;85:80-5. Spanish.

24. Cieslak TJ, Mullet CT, Puntel RA, Latimer JS. Menetrier’s disease associated with cytomegalovirus infection in children: report of two cases and review of the literature. Pediatr Infect Dis J 1993;12:340-3.

25. Hillman MM, Meinarde LL, Furnes RA, Daruich ML, Riva V, Cuestas E. [Protein losing gastroenteropathy and possible relationship to cytomegalovirus infection: Ménétrier disease in a child]. Arch Argent Pediatr 2013;111:446-9. Spanish.

26. Chang KW, Lin SJ, Hsueh C, Kong MS. Menetrier’s disease associated with cytomegalovirus infection in a child. Acta Paediatr Taiwan 2000;41:339-40.
27. Tagliaferro G, Llera J, Orsi M. [Ménétrier’s disease in pediatric patients secondary to cytomegalovirus infection: presentation of two clinical cases in a high complexity center]. Arch Argent Pediatr 2019;117:e158-62. Spanish. 

28. Anandpara KM, Aswani Y, Hira P. An unusual association of Ménétrier’s disease with a gastric bezoar. BMJ Case Rep 2015;2015:bcr2014207087. 

29. Imataki O, Uchida S, Yokokura S, Uemura M, Kadowaki N. Anemia and hypogammaglobulinemia caused by Ménétrier’s disease. Int J Hematol 2018;107:3-4. 

30. Kraut JR, Powell R, Hruby MA, Lloyd-Still JD. Menetrier’s disease in childhood: report of two cases and a review of the literature. J Pediatr Surg 1981;16:707-11. 

31. Blackstone MM, Mittal MK. The edematous toddler: a case of pediatric Ménétrier disease. Pediatr Emerg Care 2008;24:682-4. 

32. Ricci S, Bonucci A, Fabiani E, Catassi C, Carlucci A, Bearzi I, et al. [Protein-losing gastroenteropathy (Ménétrier’s disease) in childhood: a report of 3 cases]. Pediatr Med Chir 1996;18:269-73. Italian. 

33. Zhang J, Wang Y, Liu H, Xiao Y, Zhang T. Ménétrier’s disease in childhood: a case report from China. BMC Pediatr 2020;20:110. 

34. Pederiva C, Ruscitto A, Brunetti I, Salvini S, Sala M. [Cytomegalovirus-induced protein-losing gastropathy: a case report]. Pediatr Med Chir 2006;28:42-7. Italian. 

35. Wilches-Luna A, Osorio G, Oviedo N, Higuíta J, Cardona AC. Two cases of Menétrier’s disease in children. Rev Colomb Gastroenterol 2018;33:312-7.