Pediatr Gastroenterol Hepatol Nutr. 2021 Jan;24(1):109-117
https://doi.org/10.5223/pghn.2021.24.1.109
pISSN 2234-8646·eISSN 2234-8840

Pediatric Gastroenterology, Hepatology & Nutrition
pghn

Letter to the Editor

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

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Received: May 24, 2020
Revised: Jul 27, 2020
Accepted: Aug 5, 2020

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Conflict of Interest
The authors have no financial conflicts of interest.

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.

Table 1. Summary of Ménétrier disease in children [1-9]

Symptoms	Edema, emesis, epigastric pain, anorexia, diarrhea, vomiting, and abdominal pain
Diagnostics	Endoscopy in combination of biopsy and cultures
Triggers	Herpes simplex virus, Giardia lamblia, Mycoplasma pneumonia, Cytomegalovirus, and *Helicobacter pylori*
Treatment	Self-limiting
	Supportive therapies: Albumin, diuretics, fluid restriction, high-protein diet, acid inhibitors, Ganciclovir

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 [3]. 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*

https://doi.org/10.5223/pghn.2021.24.1.109

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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, amoxicillin, and clarithromycin; albumin infusion, fluid restriction, diuretics, and high-protein diet	Co-infection with *H. pylori* and CMV
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	
		2 yr	M	Vomiting, diarrhea	Hypoalbuminemia, CMV positive	Supportive care, 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	-	-	-	Gastric endoscopy and biopsy, CMV positive	-	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	

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Table 2. (Continued) Pediatric cases of Ménétrier disease

Author, year	Cases	Age	Sex	Medical history	Diagnosis	Treatment	Comments
Occena et al., 1993 [19]	2	3.7 yr	M	Vomiting, edema, sore throat, lowgrade fever, diarrhea, abdominal distention	Clinical examination, stool culture positive for Salmonella type B, hypoalbuminemia, hypoproteinemia, hypogammaglobulinemia, 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, ranitidine, trimethoprimsulfamethoxazole	
		3.7 yr	M	Edema, abdominal distention, sore throat, colicky abdominal pain, vomiting, malaise, anorexia	Clinical examination, hypoalbuminemia, hypoproteinemia, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive	Albumin infusion, high-protein and low-fat diet, cimetidine	
Coad and Shah, 1986 [20]	1	4 yr	M	Congestion, vomiting and anorexia, edema, abdominal distention	Clinical examination, hypoalbuminemia, hypoproteinemia, chest X-ray, abdominal ultrasound, diagnostic tap of ascites fluid, chrome-labeled albumin test, upper gastrointestinal X-rays.	High-protein diet	
Fishbein et al., 1992 [21]	1	3 mo	M	Diarrhea, vomiting, failure to thrive	Characteristic radiological, pathological, and functional abnormalities of the stomach. Upper gastrointestinal X-rays, gastric endoscopy and biopsy, colonoscopy, abdominal ultrasound, alpha1-antitrypsin positive, hypoalbuminemia	Prednisone, cyclosporine A, gastrostomy (drainage gastric fluid), feeding with transpyloric jejunal tube	Developed sepsis and died from brain stem infarction
Hochman et al., 1996 [22]	1	7 yr	M	Abdominal pain, abdominal distention, facial edema	Clinical examination, hypoalbuminemia, hypoproteinemia, abdominal ultrasound examination, gastric endoscopy and biopsy, CMV positive	High-protein diet	
Kirberg et al., 2014 [23]	1	6 yr	F	Edema, abdominal pain, abdominal distention, malaise, sore throat, fever	Hypoalbuminemia, abdominal ultrasound, gastric endoscopy and biopsy, *H. pylori* and CMV positive	Albumin infusion, antacid, diuretics, prednisone, high protein diet	
Cieslak et al., 1993 [24]	2	22 mo	F	Diarrhea, emesis, edema, abdominal distension	Hypoproteinemia, hypoalbuminemia, abdominal ultrasound, Upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive	No treatment Cimetidine	
		25 mo	M	Abdominal distention, edema, congestion, diarrhea, emesis, fever	Clinical examination, hypoproteinemia, hypoalbuminemia, abdominal ultrasound, chest X-ray, upper gastrointestinal x-rays, gastric endoscopy and biopsy, CMV positive		
Hillman et al., 2013 [25]	1	-	-	Edema and ascites	Possible CMV infection	Self-limiting supportive care	Only abstract available
Chang et al., 2000 [26]	1	4 yr	M	Two-week history of vomiting and periorbital edema	Upper gastrointestinal endoscopy and biopsy, CMV positive	Omeprazole	
Tagliaferro et al., 2019 [27]	2	11 mo	-	Diarrhea, vomiting and edema	Clinical examination, hypoalbuminemia, hyponatremia, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive	Fluid restriction, albumin infusion, furosemide, PPI, valganciclovir	
		4 yr	-	Fever, vomiting, abdominal distention, and edema	Clinical examination, hypoalbuminemia, hypoproteinemia, hyponatremia, abdominal ultrasound, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive	Albumin infusion, diuretics, PPI	
Anandpara et al., 2015 [28]	1	14 yr	M	Epigastric discomfort, vomiting, inability to gain weight, pedal edema	Clinical examination, hypoproteinemia	No treatment	Association between Ménétrier's disease and a bezoar

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Table 2. (Continued) Pediatric cases of Ménétrier disease

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, hypoalbuminemia, 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	
		7 yr	M	Vomiting, malaise, edema	Clinical examination, hypoalbuminemia, hypogammaglobulinemia, chest X-ray, abdominal X-ray, upper gastrointestinal X-ray, gastric endoscopy and biopsy, explorative laparoscopy	No specific treatment	
Blackstone and Mittal, 2008 [31]	1	3 yr	F	Edema, decreased activity, decreased appetite, abdominal distention, emesis	Clinical examination, hypoproteinemia, hypoalbuminemia, hyponatremia, chest X-ray, elevated fecal alfa-1-antitrypsin, upper gastrointestinal X-rays, gastric endoscopy and biopsy, CMV positive	Fluid restriction, high-protein diet, albumin infusion, furosemide	
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, hypoalbuminemia, 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	Hypoalbuminemia, 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 hypoalbuminemia, 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, hypoalbuminemia, 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.

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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.

https://doi.org/10.5223/pghn.2021.24.1.109

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.

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