Treatment of Congenital Microgastria

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Abstract

Introduction Congenital microgastria is an extremely rare birth defect. The aim of this study was to present an overview of existing literature on the treatment of microgastria.

Materials and Methods The term "microgastria" was used in a PubMed and Medline search. Since merely case reports were found, only a narrative synthesis with limited statistical analysis can be given. Data of different treatment modalities were collected and divided into two groups: conservative or less invasive treatment (C/LT, i.e., modified diet or a gastrostomy/jejunostomy) and extensive gastric surgery (EGS, i.e., Hunt–Lawrence pouch or total esophageal gastric dissociation). Clinical outcome parameters (nutrition, growth pattern, and mortality) were compared.

Results Out of 73 articles published from 1973 to 2019, 38 articles describing 51 cases were included. In four patients, microgastria was an isolated anomaly (8%). Type of treatment was described in only 46 patients, 19 were treated by C/LT. Mortality was 9/19 (47%) in the C/LT group versus 4/27 (15%) in the EGS group (chi-square = 5.829, $p = 0.016$, Fisher = 0.022). There was a negative correlation between the invasiveness of the treatment and both mortality ($r = -0.356$, $p = 0.015$) and comorbidity ($r = -0.506$, $p < 0.001$). Patients in the C/LT group had significantly more comorbidity than in the EGS group (mean = 4.32 vs. 2.26, $p = 0.001$). There was a positive correlation between comorbidity and mortality ($r = 0.400$, $p = 0.006$). Median follow-up was 42 months (range: 1–240). Type and way of nutrition were poorly described. In at least 9 of the 33 surviving patients, oral feeding was reported as normal, of whom 8 belonged to the EGS group. In all patients, growth could be acknowledged, but in comparison to peers, final body length was less. There was no difference in final body length between the two treatment groups.

Conclusion In patients with congenital microgastria, only minimal differences in clinical outcome in terms of type of nutrition and body growth were found when C/LT was compared with treatment by EGS. Mortality was significantly higher in the first group as well as the amount of comorbidities.

Keywords

- microgastria
- congenital microgastria
- Hunt–Lawrence pouch
- total esophageal gastric dissociation
- intestinal malrotation

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Introduction

Throughout the literature, less than 100 cases of congenital microgastria have been described.[1] Microgastria is a rare condition and often associated with other anomalies, such as asplenia, intestinal malrotation, diaphragmatic hernia, cardiopulmonary anomalies, renal anomalies, limb defects, and laryngotracheobronchial clefts.[2–36] Patients can show symptoms of food intolerance, gastroesophageal reflux, vomiting, recurrent aspiration pneumonia, and failure to thrive. Several treatment options have been described, but since 1980, various more extensive surgical procedures are being performed.[4] However, long-term results of these surgical interventions remain unclear. In this study, we present an overview of existing literature on the outcome of both more extensive surgical and minimal invasive or nonsurgical treatment options.

Materials and Methods

A literature search was conducted. Congenital microgastria is not a known MeSH term; therefore, a PubMed and Medline search was performed using the full-text term "microgastria." Study relevance was evaluated by screening title and abstract. Articles not written in English or without online availability were excluded, as well as articles on microgastria not describing its treatment. A graphic resume is presented as a flowchart in ►Fig. 1. After the final selection of articles was determined, all data were pooled according to the type of treatment. Due to the fact that only case reports were being found and due to the small number of patients, PRISMA guidelines were found not suitable to analyze the results.[37] Therefore, a narrative synthesis is given.

To compare the different treatment modalities, we divided them into two groups: conservative or less invasive treatment (C/LT, i.e., modified diet, gastrostomy or jejunostomy) and treatment by extensive gastric surgery (EGS, i.e., Hunt–Lawrence pouch or total esophageal gastric dissociation [TEGD]). For statistical analysis, we compared the different groups using correlation tests, *t*-tests, chi-square tests, and Fisher's exact tests in SPSS version 25.

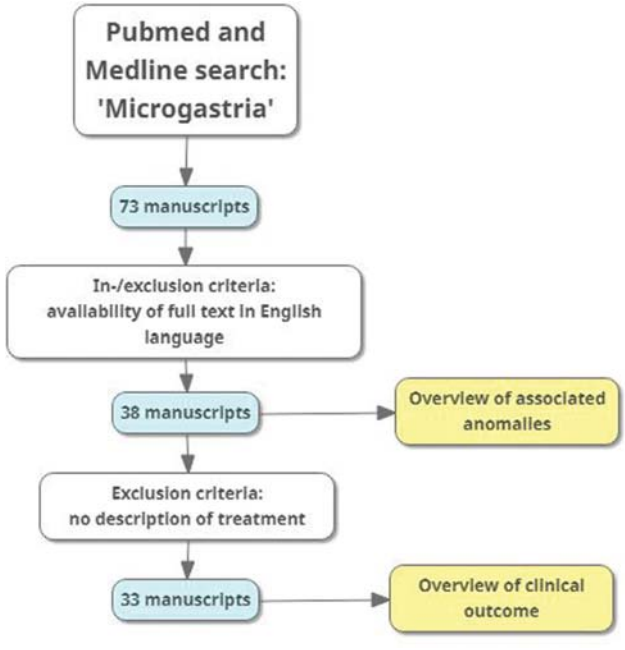

Fig. 1 Flowchart.

Results

Online literature search identified 71 articles published between 1973 and 2019. All articles not written in English or without online availability were excluded, after which an overview of associated anomalies, as presented in ►Table 1, was composed. Noteworthy is the fact that in only four patients (4/51; 8%), microgastria was present as an isolated anomaly.[1,16,38,39] The most common associated anomalies are defects of the spleen and the limbs.

As 5 of the remaining 38 articles did not describe any form of treatment of congenital microgastria,[6,12,15,18,31] 33 relevant articles remained for a narrative synthesis of clinical outcome and limited statistical synthesis. In total, 46 patients with congenital microgastria were found, of whom 19 were treated conservatively (►Table 2). ►Table 3 summarizes the clinical outcome in the two treatment groups. Mortality was 9/19 (47%) in the C/LT group versus 4/27 (15%) in the EGS group which was confirmed as a significant difference (chi-square = 5.829, $p = 0.016$, Fisher = 0.022). There was a negative correlation between the invasiveness of the treatment and both mortality ($r = -0.356, p = 0.015$) and comorbidity ($r = -0.506, p < 0.001$). Patients in the C/LT group had significantly more comorbidity than in the EGS group (mean = 4.32 vs. 2.26, $p = 0.001$). Comorbidities that were significantly associated with the invasiveness of the treatment were the categories "other intra-abdominal anomalies" (chi-square = 5.906, $p = 0.015$, Fisher = 0.025) and "central nervous system defects" (chi-square = 5.888, $p = 0.015$, Fisher = 0.032). There was a positive correlation between comorbidity and mortality ($r = 0.400$, $p = 0.006$). The only significant association between a certain category of comorbidity and mortality was found for anorectal malformations (chi-square = 8.147, $p = 0.004$, Fisher = 0.019).

Looking at the deceased patients, we found that they have significantly more comorbidity in the categories mentioned in ►Table 1 than the group of surviving patients (mean = 4.38 vs. 2.61, $p = 0.032$). In the C/LT group, mortality was mostly related to comorbidity and not to the microgastria itself, whereas in the EGS group, the highest mortality rate was found in patients who underwent several other operations.[26,27] The most frequently found EGS treatment was the Hunt–Lawrence pouch. In reports describing the reconstruction of a Hunt–Lawrence pouch, a relatively low mortality rate of 6% was found.

Of all surviving patients, outcomes of nutrition and growth are shown in ►Table 3. In general, the exact amount and type of feeding were poorly described. In the C/LT group, only 6/10 could be enterally fed, either in frequent small amounts ($n = 3$), through enteral feedings ($n = 2$) or normally ($n = 1$).[22] In four patients, no details on enteral feeding were available. In the EGS group, data on enteral feeding were available in 18/23 patients and consisted of a normal

Table 1 Associated anomalies ranked by category

Comorbidity	Total (%; $N = 51$)	C/LT (%; $N = 19$)	EGS (%; $N = 27$)	NUT (%; $N = 5$)
Spleen anomalies	20 (39)	7 (37)	9 (33)	4 (80)
Limb anomalies	19 (37)	10 (53)	6 (22)	3 (60)
Urogenital malformations	18 (35)	11 (58)	5 (19)	2 (40)
Facial dysmorphia	16 (31)	6 (32)	7 (26)	3 (60)
Other intra-abdominal anomalies	16 (31)	9 (47)	5 (19)	2 (40)
Cardiovascular anomalies	15 (29)	6 (32)	8 (30)	1 (20)
Intestinal malrotation	14 (27)	6 (32)	6 (22)	2 (40)
CNS anomalies	10 (20)	7 (37)	2 (7)	1 (20)
Lung anomalies	9 (18)	3 (16)	3 (11)	3 (60)
Esophageal atresia	7 (14)	4 (21)	2 (7)	1 (20)
Congenital hearing loss	6 (12)	3 (16)	3 (11)	0 (0)
Diaphragmatic hernia	6 (12)	2 (11)	3 (11)	1 (20)
Vertebral anomalies	6 (12)	4 (21)	2 (7)	0 (0)
Anorectal malformation	4 (8)	3 (16)	0 (0)	1 (20)
No other anomalies	4 (8)	0 (0)	4 (15)	0 (0)
Metabolic disorder	1 (2)	1 (5)	0 (0)	0 (0)

Abbreviations: C/LT, conservative or less invasive treatment; CNS, central nervous system; EGS, extensive gastric surgery; NUT, no or unknown treatment.
Note: Of 51 reported cases, most patients have multiple comorbidity.

diet in 8 patients (35%) and the need for additional nocturnal feedings in 4 patients (13%). The patients in both groups seemed to grow adequately, although they remain relatively small compared with their peers when looking at the World Health Organization or national growth charts. Median follow-up was 42 months (range: 1–240).

Discussion

Congenital microgastria is an early defect in the embryological development of the foregut and often associated with other anomalies, the most frequent being anomalies of the spleen and limbs. Both facial dysmorphisms and limb anomalies are often seen in syndromal disorders[5–7,9,15–18,21,23,26,29,30,33–36]; however, only a few syndromal cases have been reported in the literature, for example, the Pierre Robin sequence.[23,30,33,35] The foregut starts to grow from the level of the pharynx, forming esophageal and gastric precursors as well as the lungs. In the fifth week, the stomach originates from the dorsal mesogastrium along with the spleen. This explains the association with lung anomalies, esophageal atresia, diaphragmatic hernia, and asplenia. Thus, the clinical manifestation can be determined by the moment in the embryological timeline where the defect finds its origin. In most cases of microgastria, the stomach is nothing more than a small saccular, or tubular, midsagittal structure with minimal reservoir capacity. If not diagnosed at an early age, a dilated esophagus can be found as a result of compensation for the small reservoir.

Histological analysis usually shows a normal cell differentiation with a lagging cell number.[8] Depending on whether the cell differentiation was entirely completed or not, the stomach shows some functionality and produces a certain amount of acid and intrinsic factor. Therefore, the stomach appears to be both anatomically and functionally rudimentary at birth and it is hard to predict possible growth and functional outcome after birth.

In 1980, the first surgical treatment for microgastria was published, and before 1980, many patients with microgastria died at an early age.[4] Currently, several treatment options exist, varying from conservative (such as a modified nutrition with or without a nasoduodenal tube) or less invasive surgery (i.e., gastrostomy, jejunostomy) to EGS (i.e., a Hunt–Lawrence pouch, TEGD, or a combination thereof). A specific treatment is best chosen depending on the comorbidity and the failure of gastric and/or enteral feeding. It has been recommended to consult a clinical geneticist in cases of microgastria with several other anomalies to rule out specific syndromes. Anomalies that are part of these syndromes could cause a higher chance of mortality, and in some patients, they were the direct cause of death and not the presence of the microgastria.[8–11,21,26,29] The mortality rate of the C/LT group was significantly higher than in the EGS group. We suspect that this could be due to the fact that patients with this treatment had an a priori worse outcome assignable to their comorbidity and that no major surgery was considered. The fact that there was significantly more comorbidity in the C/LT group than in the EGS group, the positive correlation between comorbidity and mortality and the fact that the group of deceased patients had significantly more comorbidity support this hypothesis.

Neifeld et al were the first to report for a successful use of the Hunt–Lawrence pouch in 1980. It consists of a double-lumen jejunal pouch which is attached to the greater curvature of the stomach with a distal Roux-en-Y jejunojejunostomy.[4] Since

Table 2 Treatment overview and mortality rate

Year	Authors	*N*	Conservative or less invasive treatment		Extensive gastric surgery		
			Nonoperative	Gastrostomy/ Jejunostomy	Hunt–Lawrence	TEGD	Other
1973	Blank and Chisolm	1		1			
1974	Hochberger and Swoboda	1	1				
1980	Neifeld et al	1			1		
1983	Anderson and Guzzetta	2			2		
1987	Dorney et al	1			1 (1)		
1990	Velasco et al	4	1 (1)		3		
1992	Meinecke et al	1	1 (1)				
1993	Cunniff et al	3	1 (1)		2		
1993	Hasegawa et al	1		1 (1)			
1994	Hoehner et al	1			1		
1994	Moulton et al	1			1		
1996	Ramos et al	1			1		
1997	Hernáiz Driever et al	1	1				
1997	Sarin et al	1	1				
1998	Kroes and Festen	1			1		
1999	al-Gazali et al	2	1 (1)	1 (1)			
1999	Murray et al	1		1			
2000	Giurgea et al	1					1[a]
2002	Stewart et al	1	1				
2003	Menon et al	1			1		
2004	Herman and Siegel	1		1			
2005	Kawaguchi et al	6		2 (2)		2 (1)	2 (1)[b]
2005	Sharma and Menon	1					1 (1)[c]
2006	Lall et al	1				1	
2007	Jones and Cohen	1			1		
2008	Filippi et al	1	1 (1)				
2008	Laurie and Wakeling	1		1			
2010	Dicken et al	2			2		
2011	Vasas et al	1		1			
2011	Kunisaki et al	1				1	
2014	Roberts et al	1		1			
2016	Hattori et al	1				1	
2017	Filisetti et al	1			1		
Mortality			5/9 (56%)	4/10 (40%)	1/18 (6%)	1/5 (20%)	2/4 (50%)
			9/19 (47%)		4/27 (15%)		

Abbreviation: TEGD, total esophageal gastric dissociation.
Note: Number presented within () denotes mortality rate.
[a]Duodenal diversion, fundojejunal anastomosis in a jejunal pouch + gastrostomy.
[b]Combination of stomach division, a fundoplication and a Roux-en-Y jejunostomy ($N = 2$).
[c]Diamond-shaped side-to-side anastomosis between distal esophagus and stomach (to bypass the stenosis of the gastroesophageal transition).

then, this procedure has been applied in roughly 18 cases with variable success.[1,4,5,7,8,10,13,14,16,20,32,38,39] Kawaguchi et al introduced the use of the TEGD.[26] Up until now, this procedure has been performed five times, with good outcome being reported on gastroesophageal reflux symptoms.[26,28,33,36] Growth and nutrition have not been described properly in all cases but known data mentioned adequate growth and normal nutrition.

Table 3 Treatment overview, mortality rate, and outcome

	C/LT		EGS			NUT
	Nonoperative	Gastrostomy/ jejunostomy	Hunt–Lawrence	TEGD	Other	
No. of patients	9	10	18	5	4	5
No. of mortality	5	4	1	1	2	2
Percentage	56	40	6	20	50	40
Total mortality[a]	9/19 (47%)		4/27 (15%)			2/5 (40%)
Nutrition[b]	Normal diet (1/10) Frequent small amounts (3/10) Enteral feedings (2/10) No information (4/10)		Normal diet (9/23) Frequent small amounts (1/23) Additional nocturnal feedings (6/23) Nutrition via tube (2/23) No information (5/23)			Not applicable or not described
Median growth[b]	Length: p35 (range: p25–50) Weight: p25 (range: p15–50)		Length: p10 (range: p3–50) Weight: p11 (range: p3–90)			Not applicable or not described

Abbreviations: C/LT, conservative or less invasive treatment; EGS, extensive gastric surgery; NUT, no or unknown treatment; TEGD, total esophageal gastric dissociation.[a]Difference in mortality was significant between C/LT and EGS groups (chi-square 5.829, $p = 0.016$, Fisher = 0.022).
[b]Of surviving patients.

Although the C/LT group had a higher mortality rate, the patients who survived had a good outcome on nutrition and growth (►Table 3). The EGS group had a lower mortality rate with similar growth and slightly better outcome of nutrition; however, case series are all small and the true benefit of major surgery in relation to possible enteral nutrition and growth remains to be determined. Extensive surgery comes with the burden of a large procedure in a vulnerable infant, generally with comorbidity of other organ structures as well. Overall, there is a relatively growth retardation observed in almost all patients with microgastria compared with their peers in the normal population. This seems to be independent of any surgical procedure.

Limitations of the study were the overall small number of patients and the fact that only case reports and case series have been published. Furthermore, these reports often showed incomplete data on nutrition and outcome of growth and development. Another important pitfall of this overview is the chance of publication bias. There are only a few cases seen in all surgical centers what comes with the possibility that the patients who are treated unsuccessfully are not published in the literature. Furthermore, there is also the probability that some cases of microgastria have been left unfound due to an early death of the patient. Then, the comparison of mortality between the different treatment groups should be interpreted carefully since the described follow-up time was varied. Finally, a fair comparison was hard to make because of the heterogeneity between both the treatment groups, considering the more complex comorbidity present in the C/LT group.

Conclusion

In patients with congenital microgastria, only minimal differences in clinical outcome in terms of type of nutrition and body growth were found when C/LT was compared with treatment by more EGS. Mortality was higher in the former group, but this may be related to severe comorbidities. To rule out possible associated syndromes, we recommend consultation of a clinical geneticist. Some form of registration in a database for rare anomalies seems desirable to improve the care of these patients. The recent start of European Reference Networks, including one for rare inherited and congenital anatomical anomalies (ERNICA: European Reference Network on rare Inherited and Congenital Anomalies), may be the first step to accomplish this.[40] ERNICA: European Reference Network on Rare inherited and congenital anomalies

Based on these findings, it seems appropriate to adhere to a tailored treatment strategy and to only consider EGS when conservative or less invasive options have been deemed unsuccessful.

Conflict of Interest

None declared.

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