Objectively diagnosing rumination syndrome in children using esophageal pH-impedance and manometry

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Key points:

1. Based on ambulatory combined pH-impedance and manometry measurement, we showed that rumination patterns in children are comparable to those seen in adults, albeit with lower gastric pressure increase.

2. We propose that the diagnosis of rumination syndrome in children without pathological gastroesophageal reflux disease should be based on demonstration of retrograde bolus flow extending to the proximal esophagus closely related to a gastric pressure increase of > 25mmHg.

Abstract

Background: Rumination syndrome is characterized by recurrent regurgitation of recently ingested food into the mouth. Differentiation with other diagnoses and reflux disease in particular is difficult, recently new objective criteria were proposed for adults. The aim of this study was to determine diagnostic criteria using ambulatory gastroesophageal pH-impedance(pH-MII) and manometry in children.

Methods: Clinical data and 24-hour pH-MII and manometry recordings of children with a clinical suspicion of rumination syndrome were reviewed. Recordings were analyzed for retrograde bolus flow extending into the proximal esophagus. Peak gastric and intraesophageal pressures closely related to these events were recorded and checked for a pattern compatible with rumination. Events were classified into primary, secondary and supragastric belch-associated rumination.

Results: Twenty-five consecutive patients (11 male, median age 13.3 years (IQR 5.9-15.75)) were included and recordings of 18 patients were suitable for analysis. Rumination events were identified in 16/18 patients, with 50% of events occurring <30 minutes postprandially. Fifteen of 16 patients showed ≥1 gastric pressure peak >30mmHg, whilst only 50% of all events was characterized by peaks >30mmHg and an additional 20% by peaks >25mmHg. Four patients, had evidence of acid gastroesophageal reflux disease (GERD), all showing secondary rumination.

Conclusion: Combined 24-hour pH-MII and manometry can be used to diagnose rumination syndrome in children and to distinguish it from GERD. Rumination patterns in children are similar compared to adults, albeit with lower gastric pressure increase. We propose a diagnostic cutoff for gastric pressure increase >25mmHg associated with retrograde bolus flow into the proximal esophagus.

List of abbreviations

FGID   Functional gastrointestinal disorder

GER   Gastroesophageal reflux

GERD   Gastroesophageal reflux disease

HRM   High-resolution manometry

IQR   Interquartile range

LES   Lower esophageal sphincter

pH-MII   pH-impedance

RI   Reflux index

Introduction

Rumination syndrome is a functional gastrointestinal disorder (FGID) of unknown etiology, characterized by recurrent regurgitation of recently ingested food into the mouth, not preceded by retching or nausea and in absence of structural disease.[1, 2] Rumination syndrome was first described in mentally disabled children and although it is now recognized in individuals of all ages and cognitive abilities it tends to remain one of the less commonly recognized FGIDs in children.[3-6] Epidemiological data on rumination syndrome in children are scarce. One large school-based survey was conducted in Sri Lanka, reporting a prevalence of rumination symptoms in 5.1% of children 10-16 years.[7] One large questionnaire-based study performed in the US on the prevalence of FGIDS according to the Rome III criteria in 1447 children (age range 4 – 18 years), showed that 23.1% of children qualified for at least one FGID, however none qualified for rumination syndrome.[8]

According to the recent Rome IV criteria, a clinical diagnosis of rumination syndrome can be made after exclusion of other diagnoses that could explain the symptoms.[1, 9] However, to exclude gastroesophageal reflux disease (GERD), functional vomiting disorders and upper gastrointestinal motility disorders such as gastroparesis can be challenging. This leads to patients visiting multiple physicians, undergoing extensive diagnostic testing and being treated with several pharmacological therapies before the final diagnosis is reached.[10] The cornerstone of treatment of rumination syndrome is thorough explanation of the condition and its underlying mechanism. Although this resolves symptoms in a substantial part of patients, behavioral therapy aimed at suppressing the increase of gastric pressure is often indicated.[10-12]

With the use of new diagnostic tools, including combined pH-impedance (pH-MII) and (high-resolution) manometry measurement, studies in adults have shown that patients with rumination syndrome exhibit characteristic patterns during their regurgitation episodes.[13-17] These episodes are typically induced by a rise in intragastric pressure, generated by a voluntary, but often non-intentional, contraction of the abdominal wall musculature. Recently, Kessing et al. proposed diagnostic criteria for rumination syndrome in adults based on comparison of results of combined pH-MII and manometry measurement in patients with rumination. They proposed that the diagnosis of rumination syndrome could be based on the demonstration of retrograde bolus flow extending to the proximal esophagus, closely associated with an abdominal pressure increase > 30mmHg. Applying this criterion, they additionally identified three distinct rumination patterns: i) primary rumination, in which the abdominal pressure

increase precedes retrograde flow, ii) secondary rumination, consisting of an increase in abdominal pressure following the onset of a reflux event and iii) supragastric belch-associated rumination, consisting of a supragastric belch immediately followed by a rumination event.[13]

To date, there are no studies evaluating combined pH-MII and manometry measurements in children that present with clinical symptoms of rumination. Objective diagnostic criteria to establish the diagnosis of rumination syndrome in children may contribute to earlier diagnosis. If subtypes of rumination syndrome are present in children, this would create opportunities for better targeted treatment. In this study, our aim was therefore to first explore gastroesophageal pressure and flow characteristics of rumination episodes in children by quantitative analysis of pH-MII recordings combined with dual esophageal and gastric pressure measurements. Our second aim was to determine objective diagnostic pH-MII and manometry criteria for rumination syndrome in children based on this analysis

Methods

Combined 24-hour pH-MII and manometry recordings of 25 pediatric patients (< 18 years) with a clinical suspicion of rumination syndrome were extracted from a database of studies conducted at the Motility Center of the AMC, Amsterdam, The Netherlands, between December 2010 and February 2016. Patients with a known major esophageal motor disorder based on the adult Chicago Classification V3 criteria were excluded.[18] Clinical data, including duration of symptoms, predominant symptoms, prior diagnostic interventions and medication use, current medication use and potential life-events were extracted by chart review and are reported for all patients that were offered a 24-hour pH-MII and manometry test. All recordings were manually re-analyzed by two of the authors (MS and JO). Reviewers were blinded to the patient's clinical characteristics and results of initial analysis. In cases of disagreement, the recordings were adjudicated by a third reviewer (AB) in order to reach consensus.

Ambulatory pH-MII and manometry

All subjects fasted for at least 3 hours prior to the study and were studied off acid-suppressive and esophageal motility influencing drugs. A solid-state manometry catheter was placed, which consisted of two pressure sensors that were located at 5 cm above and 5 cm below the upper border of the

lower esophageal sphincter (LES). Secondly, a pH-impedance catheter fitted with six impedance recording segments and one ion-sensitive field-effect transistor pH-electrode (Unisensor AG, Attikon, Switzerland) was placed. Impedance recording segments were located at 2-4, 4-6, 6-8, 8-10, 14-16 and 16 – 18cm above the upper border of the LES. The position of the LES from the nares was determined by either stationary esophageal HRM measurement or chest radiography. Impedance, pH and manometry signals were recorded for 24 hour at sample rates of 50Hz, 1Hz and 8Hz respectively using a digital datalogger (Ohmega, Medical Measurement Systems, Enschede, The Netherlands).

Data analysis

Impedance tracings were analyzed for the occurrence of episodes of retrograde bolus flow according to previously published criteria.[19] Furthermore, a supragastric belch was defined as a rapid antegrade movement of gas (rise in impedance level to at least 1000 Ohms followed by a quick expulsion of gas in retrograde direction resulting in the baseline impedance level returning to baseline (Figure 1C).[20] Supragastric belches less than 5 seconds apart were considered as a single episode. The reflux index (RI), or acid exposure time, was calculated as the percentage of time during which the pH was < 4 in both upright and supine position, excluding meal periods. The pH-study was defined as abnormal if the RI was ≥ 5% in patients aged > 1 year and ≥ 10% in those aged < 1 year.[21]

All retrograde bolus movements that reached the proximal esophagus were identified and the peak gastric and esophageal pressures recorded during these events were subsequently analyzed. Individual events with a gastric pressure increase of at least 20mmHg from baseline were classified as rumination events. Furthermore, the temporal alignment of retrograde bolus movement onset (>50% impedance drop from baseline) with the onset and peak of gastric and esophageal pressure increases was determined. Rumination events were grouped into i) primary rumination, ii) secondary rumination and iii) supragastric belch-associated rumination (as previously described). The dominant sub-type of rumination seen in each patient defined their typical rumination profile.

Statistical analysis

Distribution of data was evaluated using the Kolmogorov-Smirnov test. Parametric data are expressed as mean ± standard deviation (SD), and nonparametric data as median, interquartile range (IQR). Comparison between patients exhibiting one of the rumination profiles and patients with no signs of

rumination were analyzed using Mann-Whitney U test. Statistical tests were performed using IBM

SPSS Statistics 23 (SPSS Inc, Chicago, Illinois). Differences were considered statistically significant

when $P < 0.05$.

Results

Twenty-five patients (11 male (44%), median age at time of pH-MII measurement 13.3 years (IQR 5.9

– 15.75)) with a clinical diagnosis of rumination syndrome were enrolled. Symptoms at time of

presentation are shown in Table 1. A median time of 12 months (IQR 9 – 24, data available for 16

patients) had passed between the onset of symptoms and the combined pH-MII and manometry

investigation. For twelve patients, possible predisposing life-events/illnesses that could have

contributed to the onset of their symptoms were noted. Of these, three reported personal or family

matters, two had experienced an upper airway infection, two had a history of an eating disorder

(anorexia nervosa) and one had a history of cured cancer. Patients had undergone between 0 – 7

other diagnostic tests prior to the current investigation. Past investigations included barium swallow (n

= 11), upper GI endoscopy (n = 9) and H. Pylori diagnostics (n = 9). Twenty patients had been

previously prescribed proton-pump inhibitor therapy. None of the patients had attended a speech

pathologist or behavioral therapist prior to the current investigation.

Nine of the 25 patients were excluded due to poor tolerance of the procedure (n = 4), technical failure

of one or both of combined recordings (n = 3), or the absence of rumination symptoms during the test

(n = 2) we therefore present pH-MII and manometry data from the 16 remaining patients.

Combined ambulatory pH-impedance and manometry monitoring

Median duration of measurement was 21 hours (IQR 20 – 23 hours). All three rumination patterns that

were previously described in adults were present (Table 2; Figure 1). Fifteen out of 16 patients had at

least one gastric pressure peak of amplitude > 30mmHg and all patients had at least one peak >

25mmHg. Primary rumination occurred in 13 (81%) patients and was the predominant mechanism in

five (38%). Secondary rumination occurred in 15 (91%) patients and was the predominant mechanism

in six patients (40%), of which three patients had an abnormal RI. Secondary rumination was the only

mechanism in three patients (20%), of which one patient had an abnormal RI. In one of these patients, all rumination events were preceded by acid GER and in the other patient, rumination events were preceded by both acid (48.4%) and non-acid GER. Supragastric belching associated with rumination occurred in four (25%) patients, and was the only mechanism in one patient. The median number of supragastric belches recorded in these patients was 13.0 (1.5 – 34.0).

Characteristics of individual rumination events

Of a total of 266 rumination events were registered (97 primary, 160 secondary and 9 supragastric belching induced), 141 (53%) occurred within 30 minutes after a meal, 183 (69%) within 60 minutes and 217 (82%) within 90 minutes. In two patients, none of the rumination events occurred within 30 minutes after a meal.

During primary rumination events, the onset of the abdominal pressure increase was recorded 0.00s (0.00 – 0.01) prior to registration of a drop in impedance signal in the most distal channel (> 50% with regard to baseline). In secondary rumination events, the onset of the abdominal pressure increase was recorded 0.03s (0.02 – 0.05) after registration of a drop in impedance signal in the most distal channel.

One hundred and thirty-one (49.2%) of the individual gastric pressure peaks during proximal episodes of retrograde bolus flow had an amplitude of > 30mmHg and 184/266 (69.2%) of > 25mmHg. The median amplitude of all gastric pressure peaks prior to rumination was 30 (25 – 42) mmHg, 29 (24 – 38) mmHg in the primary rumination events, compared with 32 (25 – 46) mmHg and 29 (24 – 35) mmHg in the secondary and supragastric belching associated events respectively ($P = 0.089$). Overall, esophageal pressure peaks had a median amplitude of 25.50 (20 – 35) mmHg. The median amplitude was 24 (21 – 31) mmHg in the primary rumination events, compared with 26 (20 – 37) mmHg and 32 (23.5 – 36.5) mmHg in the secondary and supragastric belching associated events respectively ($P = 0.186$). Median gastric and esophageal pressure peaks did not differ significantly between patients with and without pathologic reflux based on RI (35.6 (29.4 – 64.2) and 27.3 (25.5 – 50.3) mmHg vs 35.3 (26.7 – 44.7) and 28.8 (24 – 37.6) mmHg respectively). Esophageal pH during primary rumination events was 3.8 (2.8 – 5.5), compared to 3.4 (2.6 – 4.6) during secondary and 6.1 (4.1 – 6.7) during supragastric belching associated events ($P < 0.01$).

Discussion

This is the first paper to describe objective criteria for the diagnosis of rumination syndrome in children using 24-hour pH-impedance and dual channel manometry (Table 4). In contrast to adult rumination, we showed that a substantial number of rumination episodes are initiated by a gastric pressure rise to < 30 mmHg. Our findings underline the clinical utility of using pH-MII with dual channel manometry to diagnose rumination syndrome in children. By providing an unequivocal diagnosis, pH-MII manometry can shorten the time to diagnosis and can direct adequate treatment. As shown in our cohort, the diagnosis of rumination syndrome is challenging and can be falsely made when based on clinical suspicion alone. On the other hand, excluding other diagnoses may lead to many children being exposed to multiple diagnostic tests and empiric treatments for prolonged periods of time. This delay can lead to functional impairment, emotional distress, but also weight loss, malnutrition and electrolyte imbalance.[22-24]

Our criteria have been adapted from the adult criteria for rumination syndrome. In adults, patients with GERD also exhibit gastric pressure peaks up to 30mmHg without further evidence of rumination syndrome. A cut-off value < 30mmHg would thus also identify adult GERD patients as having rumination.[13] In the present study we did not evaluate gastric pressure peaks in a separate group of pediatric GERD patients. However, as we use 24-hour pH-impedance testing, we can also confirm or rule out GERD. The current study suggests that a lower, 25mmHg, cut-off may more optimally identify rumination episodes in children.

Using our criteria, we were able to confirm the diagnosis of rumination syndrome in 10 out of 18 children. Two patients referred to our clinic had no rumination episodes and showed no evidence of GERD. Of these, one was later diagnosed with severe functional constipation and symptoms improved after laxative treatment. In the other patient, no explanation for complaints was found and this patient was lost to further clinical follow-up. The remaining six patients were diagnosed with GERD, either because of prolonged RI or because all rumination episodes were initiated by GER episodes.

In the 10 patients with rumination syndrome, three distinct rumination patterns were identified as is consistent with reports in adult patients (Table 2).[10] These subtypes suggest different pathophysiological mechanisms, which in turn may require different treatment strategies. Rumination is thought to be a learned behavior, therefore the cornerstone of current treatment is an explanation of the condition, with initiation of a behavioral therapy regime.[10, 13] A close temporal association of

abdominal symptoms before the appearance of rumination or belching has been reported earlier in adults also confirms the possible behavioral etiology of these conditions.[25] Clinically, this may aid in providing patients a clear explanation of the cause for their symptoms by showing them the measurement and may help patients to come to terms with the diagnosis, understand the underlying mechanism of their symptoms and engage with behavioral treatment. A recent study in adults diagnosed with rumination syndrome based on clinical symptoms and HRM showed that diaphragmatic breathing, aided with biofeedback by HRM, can be effective by averting the gastroesophageal pressure disturbance that precipitates rumination.[26]

Secondary ruminators demonstrate an abnormal strain response to episodes of GER, this sub-group may therefore respond to anti-reflux therapies. In five patients, rumination was present, but this was predominantly secondary rumination initiated by acid GER episodes. This suggests that acid suppressive therapy may be a first targeted treatment step to improve symptoms in these patients, although this will not prevent the occurrence of weakly acid and alkaline reflux episodes. We would therefore advise to repeat measurement on PPI therapy if symptoms persist despite therapy. If acid suppressive treatment fails or if GER episodes are non-acidic as in one patient in our cohort, behavioral interventions might lead to alleviation of symptoms. Biofeedback therapy could, in theory, decrease a patient's level of response to the sensation experienced when a reflux episode occurs. Additionally, he GABA-b receptor agonist baclofen has been shown to significantly enhance postprandial pressure at the LES and to suppress transient LES relaxations in both adults and children and may have a role in treating secondary rumination.[27, 28] Anti-reflux surgery has also being shown effective in a pilot study in adult patients who did not respond to behavioural therapy.[29] However, currently, there is no evidence showing symptomatic improvement with the use of any anti-reflux therapy in children with rumination syndrome.

This case series has some limitations which are important to acknowledge. First, clinical data was not always complete and symptoms of regurgitation were inconsistently documented by many patients during the ambulatory measurement. Therefore, we were unable to analyze measurements for concurrent associations of symptoms and clinical events with rumination patterns. Another limitation of this study is the limited size of our study population. However, the results from our study describe a unique cohort of consecutive patients with a clinical suspicion of rumination syndrome, referred for combined pH-MII and dual channel manometry measurement. Whilst we did not perform HRM to

diagnose rumination, we note the findings by Kessing et al., comparing both stationary and ambulatory measurement approaches, and suggesting that both techniques are equally effective in diagnosing rumination syndrome.[13] In our study we found that almost half of all events occurred > 30 minutes postprandial and a third of all events even > 60 minutes postprandial, suggesting that a short stationary measurement might often not be sufficient to detect rumination. An additional advantage of using combined pH-MII and manometry is the ability to identify (acid) GER as the underlying mechanism attributing to secondary rumination events.

In conclusion, we performed a systematic analysis of combined pH-MII and dual channel manometry measurements of a cohort of pediatric patients with a clinical suspicion of rumination syndrome. The typical pattern of brief, gastric pressure increases associated with the occurrence proximal retrograde bolus flow as seen in adults was also typically found in children. In addition, three distinct rumination patterns as described in adults could also be identified, which include primary rumination, secondary rumination and supgragastric belch-associated rumination. Application of combined pressure-impedance monitoring may thus improve, the sometimes equivocal, clinical diagnosis of rumination syndrome and may moreover help distinguish rumination syndrome from secondary rumination as a result of pathologic GERD in children. On the other hand, it may prevent children from extensive diagnostic testing to rule out alternative diagnoses and may additionally provide better targeted treatment options. We propose that the diagnosis of rumination syndrome in children should be based on demonstration of retrograde bolus flow extending to the proximal esophagus closely related to a gastric pressure increase of > 25mmHg, in the absence of any other underlying medical condition that could explain the symptoms, including GERD.

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Figure 1. The rumination variants as measured by combined pH-impedance and manometry monitoring. Arrows indicate: 1 = impedance drop, starting in most distal channel, marks start of retrograde bolus flow; 2 = return of impedance signal back to baseline, starting in most proximal channel, marks end of retrograde bolus flow; 3 = rise in intragastric and intraesophageal pressure channels, marks rumination event; 4 = supragastric belch, characterized by sudden rise in impedance signal starting in most proximal channel, followed by an expulsion of air in the oral direction.
A) Primary rumination: an increase in gastric pressure is followed by the flow of gastric content. Peak gastric pressure is observed during the flow of gastric content. Pressure in the esophageal lumen increases during the retrograde flow of gastric content. **B)** Secondary rumination: similar to primary rumination but preceded by a spontaneous gastroesophageal reflux event. In this particular patient occurring during a long period of acidic stasis in the esophagus. **C)** Supragastric belch-induced rumination. Two subsequent supragastric belches, the second one inducing a rumination event. Initially, a movement of the diaphragm in aboral direction and a sub-atmospheric pressure in the esophageal lumen is observed. The subsequent inflow of air is indicated by an antegrade rise in impedance and causes a rise in the esophageal pressure. Thereafter, the esophageal air is immediately expulsed during which an increase in gastric pressure is observed. Subsequent flow of gastric content into the esophagus is observed during the increase in gastric pressure. This can be seen as a drop in impedance compared to the initial impedance baseline preceding the supragastric belch.

Table 1. Patient characteristics

Characteristics	
Median age (years)	13.3 (IQR 5.9-15.8)
Male	11 (44.0%)
Mean BMI[1]	18.7 (± 2.6)
Median time between onset of symptoms and pH-MII manometry (months)[2]	12.0 (IQR 9.25- 24.00)
Life event reported[3]	7 (58.3%)
Presenting symptoms*	
Regurgitation	24 (96.0%)
Belching	5 (20.0%)
Heartburn	5 (20.0%)
Bloating	3 (12.0%)
Nausea	1 (4.0%)
Previous Diagnostics*	22 (88.0%)
(Timed) barium swallow	11 (44.0%)
H. Pylori diagnostics	9 (36.0%)
Upper GI Endoscopy	9 (36.0%)
Gastric emptying test	8 (32.0%)
Abdominal ultrasound	7 (28.0%)
CT or MRI of the brain	5 (20.0%)
pH-metry	3 (12.0%)
Abdominal X-ray	3 (12.0%)
Routine laboratory examination	2 (8.0%)
Chest X-ray	2 (8.0%)
Triple feces test	1 (4.0%)
Previous Treatment*	20 (80.0%)
Omeprazole	13 (52.0%)
Domperidone	10(40.0%)
Ranitidine	7 (28.0%)
Esomeprazole	5 (20.0%)
Erythromycin	4 (16.0%)
Hypnotherapy	3 (12.0%)
Alginate	2 (8.0%)
Baclofen	2 (8.0%)
Laxative	2 (8.0%)

Percentages are valid percentages. *Multiple symptoms/investigations/treatments possible per patient. Data available for [1]n = 15 patients, [2]n = 16 patients, [3]n = 12 patients.

Table 2. Rumination profile of each patient with rumination episodes[1]

	Total number of episodes	Primary Rumination	Secondary Rumination	Supragastric-belch associated rumination
Subject 1	19	21%	**79%**	0%
Subject 2	31	0%	**100%**	0%
Subject 3	79	42%	**57%**	1%
Subject 4	18	**39%**	28%	33%
Subject 5	51	43%	**57%**	0%
Subject 6	6	**67%**	33%	0%
Subject 7	6	33%	**66%**	0%
Subject 8	11	0%	**100%**	0%
Subject 9	1	0%	0%	**100%**
Subject 10	5	40%	**60%**	0%
Subject 11	2	0%	**100%**	0%
Subject 12	4	**75%**	25%	0%
Subject 13	7	43%	**57%**	0%
Subject 14	9	**78%**	22%	0%
Subject 15	14	**64%**	43%	0%
Subject 16	3	33%	33%	33%

[1]Bold indicates predominant rumination pattern; in subject 16 rumination patterns were equally distributed

Table 3. Median outcomes of ambulatory combined pH-MII/manometry monitoring during the 24-h assessment in all patients with rumination episodes

	Median (IQR)
Upright RI	3.1 (0.7 – 8.9)
Supine RI	0.2 (0 – 2.4)
Total RI	1.9 (0.4 – 11.0; range 13.6 – 19.3%)
Number of patients with abnormal RI - All had predominant secondary rumination	N=4 (22%)
Retrograde bolus movements	62.5 (52.5 -110.3)
Proximal events	32.0 (13.5 – 60.3)
Acid events	20.5 (6.3 – 43.5)
Weakly acid events	36.0 (22.3 – 59.8)
% proximal events with a rise in gastric pressure of > 30mmHg	30.6 (17.5 – 87.7)
% proximal events with a rise in gastric pressure of > 25mmHg	72.6 (57.7 – 100.0)

RI = reflux index; IQR = interquartile range; pH-MII = pH-impedance

Table 4. Suggested criteria for diagnosing rumination syndrome in children

1. **Fulfills Rome IV criteria** (all below)

    I. Repeated regurgitation and rechewing or expulsion of food that:

    a) Begins soon after ingestion of a meal

    b) Does not occur during sleep

    II. Not preceded by retching

    III. After appropriate evaluation, symptoms cannot be fully explained by another medical condition (including GERD)

OR

2. **Fulfills combined clinical and pH-MII/manometry criteria (all below)**

    I. Clinical suspicion of rumination syndrome, but not fulfilling strict Rome IV criteria (e.g. due to atypical history or the fact that GERD has not formally been excluded)

    II. Presence of retrograde bolus flow extending into the proximal esophagus on MII

    III. Close relationship between retrograde bolus flow and abdominal pressure increase > 25mmHg in combination with an esophageal pressure increase

    IV. Above pattern not solely initiated by gastroesophageal reflux episodes (secondary rumination)[1]

    V. Normal RI on 24-hour pH-MII

Rumination syndrome can be diagnosed in two ways. Either according to Rome IV criteria or when there is a clinical suspicion, but Rome IV criteria are not fully met, by using our suggested pH-MII/manometry criteria.
[1]If secondary rumination is present, patients generally fulfill criteria for GERD (i.e. GER episodes causing troublesome symptoms and/or complications.[30] These patients should first be treated for GERD, especially when acid GER episodes cause the rumination episodes. However, if this treatment fails or GER episodes are non-acidic, behavioral interventions might lead to alleviation of symptoms.

GER = Gastroesophageal reflux; GERD = gastro-esophageal reflux disease; MII: multichannel intraluminal impedance; pH-MII = pH-impedance; RI = reflux index.