CASE REPORT

Gut and Liver, Vol. 7, No. 3, May 2013, pp. 377-381

Reinterpretation of Follow-Up, High-Resolution Manometry for Esophageal Motility Disorders Based on the Updated Chicago Classification

Jun Young Song, Moo In Park, Do Hyun Kim, Chan Hui Yoo, Seun Ja Park, Won Moon, and Hyung Hun Kim

Department of Internal Medicine, Kosin University College of Medicine, Busan, Korea

The aim of this study was to assess changes between primary classification of esophageal motility disease and follow-up classification by high resolution manometry (HRM) and to determine whether previously classified diseases could be recategorized according to the updated Chicago Classification published in 2011. We reviewed individual medical records and HRM findings twice for each of 13 subjects. We analyzed primary and follow-up HRM findings based on the original Chicago Classification. We then reclassified the same HRM findings according to the updated Chicago Classification. This case series revealed the variable course of esophageal motility disorders; some patients experienced improvement, whereas others experienced worsening symptoms. Four cases were reclassified from variant achalasia to peristaltic abnormality, one case from diffuse esophageal spasm to type II achalasia and one case from peristaltic abnormality to variant achalasia. Four unclassified findings were recategorized as variant achalasia. In conclusion, esophageal motility disorders are variable and may not be best conceptualized as an independent group. Original classifications can be recategorized according to the updated Chicago Classification system. More research is needed on this topic. **(Gut Liver 2013;7:377-381)**

Key Words: Esophageal motility disorders; Manometry; Esophagus; Classification

INTRODUCTION

Esophageal motility disorders consist of a complicated array of disturbances associated with dysphagia, gastroesophageal reflux, and noncardiac chest pain.[1] The named esophageal motility disorders—achalasia, diffuse esophageal spasm, nutcracker esophagus, and the hypertensive lower esophageal sphincter—are characterized by esophageal dysmotility, which is responsible for these symptoms.[1,2] The clinical presentation may be perplexing, especially when heartburn or chest pain are the presenting symptoms instead of dysphagia.[2] Although the diagnosis and treatment of achalasia are well-defined, the pathophysiology of the other esophageal motility disorders has not been identified. Until now, it is unclear as to whether this collection of diagnoses is an independent group of diseases or incidental phenomena caused by other diseases.

Esophageal manometry is considered the gold standard for assessing esophageal motor function.[3] The aim of esophageal motility testing is to reveal abnormalities by assessing the function of the esophagus and its sphincters.[4] Although conventional manometry has been widely used to evaluate esophageal motor function, this test is not capable of explaining esophageal symptoms. A recent study showed poor intraobserver and interobserver reproducibility of manometric tracing interpretation.[5] The detailed display of esophageal motor function and dysfunction by high resolution manometry (HRM), on the other hand, allows for better classification of esophageal motility disorders. This test uses a practical manometric device with 36 solid-state, circumferentially sensitive sensors spaced at 1 cm intervals coupled with a designated computer (ManoScan; Sierra Scientific Instruments, Los Angeles, CA, USA) and custom software for topographic pressure plotting and analysis (ManoView; Sierra Scientific Instruments). The most recent meeting of the HRM Working Group in Ascona, Switzerland in April 2011 announced an updated Chicago Classification system.[6]

Here, we report case series of 13 subjects who received follow-up, HRM between July 2009 and April 2012. The goal of this study was to assess the changes between previous classifications of esophageal motility diseases and follow-up classifications by HRM and to determine whether the previously classified disease could be recategorized in the framework of the

Correspondence to: Moo In Park
Department of Internal Medicine, Kosin University College of Medicine, 262 Gamcheon-ro, Seo-gu, Busan 602-702, Korea
Tel: +82-51-990-5205, Fax: +82-51-990-5055, E-mail: mipark@ns.kosinmed.or.kr
Received on October 8, 2012. Revised on November 13, 2012. Accepted on November 13, 2012.
pISSN 1976-2283 eISSN 2005-1212 http://dx.doi.org/10.5009/gnl.2013.7.3.377

updated Chicago Classification system.

CASE REPORT

This case series consisted of six males and seven females (Table 1). Mean age was 35 years with an age range of 19 to 71 years. They were referred to or consulted at Gospel Hospital manometry laboratory for the evaluation of specific symptoms including dysphagia and acid regurgitation. All subjects had symptoms of esophageal motility disorders for a long period (up to 10 years) that caused serious consequences on physical health and significant distress with impairments in social, family, and academic lives. Eight patients experienced dysphagia, and five presented predominantly with gastroesophageal reflux disease symptoms such as heartburn or acid regurgitation (Table 1).

Table 1. Patients Characteristics at Baseline

Patient no.	Age	Gender	Main symptom
1	71	M	Dysphagia
2	62	M	Acid regurgitation
3	51	F	Dysphagia
4	59	F	Dysphagia
5	55	F	Dysphagia
6	58	M	Acid regurgitation
7	34	M	Acid regurgitation
8	19	F	Dysphagia
9	49	M	Dysphagia
10	43	F	Dysphagia
11	48	F	Acid regurgitation
12	33	M	Heartburn
13	69	F	Dysphagia

M, male; F, female.

Primary HRM results of 13 patients were classified according to original Chicago Classification and updated Chicago Classification (Table 2). Numbers of patients with each diagnosis of the original Chicago Classification were as follows: absent peristalsis, one; intermittent hypotensive peristalsis, one; frequent hypotensive peristalsis, two; distal esophageal spasm, one; spastic achalasia, three; and functional esophago gastric junction (EGJ) obstruction, nine. Four patients were determined to have unclassified findings. In addition, the number of patients with each diagnosis of the updated Chicago Classification were as follows: absent peristalsis, one; weak peristalsis with small peristaltic defects, two; weak peristalsis with large peristaltic defects, one; distal esophageal spasm, one; and type III achalasia, three. Five patients were classified as achalasia variant.

Follow-up HRM results from the 13 patients were also classified (Table 3). Numbers of patients with each diagnosis of the original Chicago Classification were as follows: absent peristalsis, one; frequent hypotensive peristalsis, five; distal esophageal spasm, one; spastic achalasia, three; functional EGJ obstruction, one; and achalasia with esophageal compression, one. One patient was determined to have unclassified findings. In addition, the number of patients with each diagnosis of the updated Chicago Classification was as follows: absent peristalsis, one; weak peristalsis with small peristaltic defects, four; weak peristalsis with large peristaltic defects, one; rapid contraction, one; type II achalasia, one; and type III achalasia, three. Two patients were classified as achalasia variant.

The updated classification applied to unclassified findings of previous HRM is shown in Table 4. Four patients were determined to have unclassified findings according to original Chicago Classification. However, all of these unclassified find-

Table 2. Findings of Primary, High-Resolution Manometry Matching with the Updated Classification

Patient no.	Exam date	Original classification	Updated classification
1	24/11/09	Unclassified	Achalasia variant
2	19/5/10	Hypotensive peristalsis (frequent)	Weak peristalsis with small peristaltic defects
3	29/12/10	Unclassified	Achalasia variant
4	15/2/11	Spastic achalasia	Type III achalasia
5	23/9/11	Spastic achalasia	Type III achalasia
6	7/9/09	Absent peristalsis	Absent peristalsis
7	22/11/11	Functional EGJ obstruction	Achalasia variant
8	1/7/11	Unclassified	Achalasia variant
9	30/8/11	Spastic achalasia	Type III achalasia
10	16/11/09	DES (segmental)	DES
11	23/7/09	Hypotensive peristalsis (intermittent)	Weak peristalsis with large peristaltic defects
12	14/1/11	Hypotensive peristalsis (frequent)	Weak peristalsis with small peristaltic defects
13	29/7/11	Unclassified	Achalasia variant

EGJ, esophagogastric junction; DES, distal esophageal spasm.

Table 3. Findings of Follow-Up, High-Resolution Manometry Matching with the Updated Classification

Patient no.	Exam date	Original classification	Updated classification
1	23/3/10	Hypotensive peristalsis (frequent)	Weak peristalsis with large peristaltic defects
2	24/5/10	Hypotensive peristalsis (frequent)	Weak peristalsis with small peristaltic defects
3	22/8/11	Hypotensive peristalsis (frequent)	Weak peristalsis with small peristaltic defects
4	22/9/11	Spastic achalasia	Type III achalasia
5	2/1/12	Spastic achalasia	Type III achalasia
6	18/4/11	Absent peristalsis	Absent peristalsis
7	18/4/12	DES (segmental)	Weak peristalsis with small peristaltic defects
8	20/9/11	Unclassified	Achalasia variant
9	31/1/12	Spastic achalasia	Type III achalasia
10	20/12/10	Achalasia with esophageal compression	Type II achalasia
11	1/6/12	Functional EGJ obstruction	Achalasia variant
12	18/5/11	Hypotensive peristalsis (frequent)	Weak peristalsis with small peristaltic defects
13	14/12/11	Hypotensive peristalsis (frequent)	Rapid contraction

DES, distal esophageal spasm; EghaGJ, esophagogastric junction.

Table 4. New Classification Applied to Previously Unclassified Findings of Previous High-Resolution Manometry

Patient no.	Exam date	Original classification	Updated classification
1	24/11/09	Unclassified	Achalasia variant
3	29/12/10	Unclassified	Achalasia variant
8	1/7/11	Unclassified	Achalasia variant
	20/9/11	Unclassified	Achalasia variant
13	29/7/11	Unclassified	Achalasia variant

ings were categorized as achalasia variant under the updated Chicago Classification.

Among 13 patients with esophageal motility disorders, patient 7 had typical symptom improvement based on follow-up, HRM findings (Fig. 1). He had weak peristaltic contractions with large peristaltic defects in the body of the esophagus following wet swallows, with variable contraction amplitude ranging from 30 to 170 mm Hg and variably incomplete lower esophageal sphincter (LES) relaxation with a high basal LES pressure. Additionally, high-resolution manometry revealed an elevated mean integrated relaxation pressure (IRP) of 16 mm Hg. The revised Chicago Classification published in 2011 classifies this presentation as variant achalasia after ruling out the mechanical obstruction and hypercontractility. At follow-up after 5 months of calcium channel blocker therapy, the patient was asymptomatic for dysphasia. The repeat HRM at that time showed weak peristaltic contractions, with a mean IRP of 14.8 mm Hg and a near completely relaxed LES with a normal basal LES pressure.

On the other hand, patient 11 had typical symptom worsening at the follow-up HRM findings (Fig. 2). Primary study showed weak peristaltic contractions in the body of the esophagus with a mean IRP of 10.8 mm Hg and 87% swallow with small breaks in the 20 mm Hg isobaric contour. After supportive care, a follow-up HRM revealed an elevated mean IRP of 22.6 mm Hg and weak peristalsis. We have been able to diagnose this presentation as variant achalasia more specifically after ruling out the mechanical obstruction and hypercontractility.

DISCUSSION

Esophageal manometry is considered the gold standard for assessing esophageal motor function.[3] The aim of esophageal motility testing is to assess the function of the esophagus and its sphincters to reveal abnormalities. The development of micromanometric, water-perfused assemblies[7] and miniaturized, solid-state pressure sensors[8] has made HRM possible. HRM has several advantages in interpreting esophageal function compared to conventional manometry: 1) HRM has many pressure sensors on the manometric assembly, and this can lead to a spatial continuum of intraluminal pressure after interpolating between adjacent sensors; 2) pressure sensors on HRM have a very rapid response time, allowing HRM to follow the dynamic movement and function of the pharyngeal swallow; 3) each sensor is circumferentially sensitive to overcome directionality limitations inherent in conventional water-perfused systems; and 4) sophisticated plotting algorithms of HRM enable us to see the accurate and dynamic imaging of intraesophageal pressure as a continuum along the length of the esophagus with pressure magnitude depicted by a spectral color scale and isobaric conditions among regions indicated by isocoloric areas.[9]

Esophageal motility disorders appear in various forms, such as achalasia, diffuse esophageal spasm, and nutcracker esophagus. Unfortunately, only achalasia has a known cause and

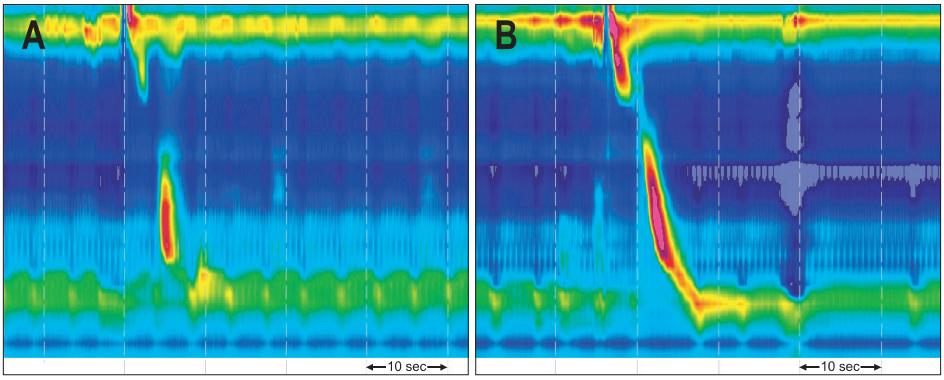

Fig. 1. Typical improved case. (A) A primary, high resolution manometry tracing of patient 7 showed an achalasia variant based on the updated Chicago Classification. (B) A follow-up high resolution manometry of the same patient showed weak peristalsis with small peristaltic defects pattern after 5 months of calcium channel blocker therapy.

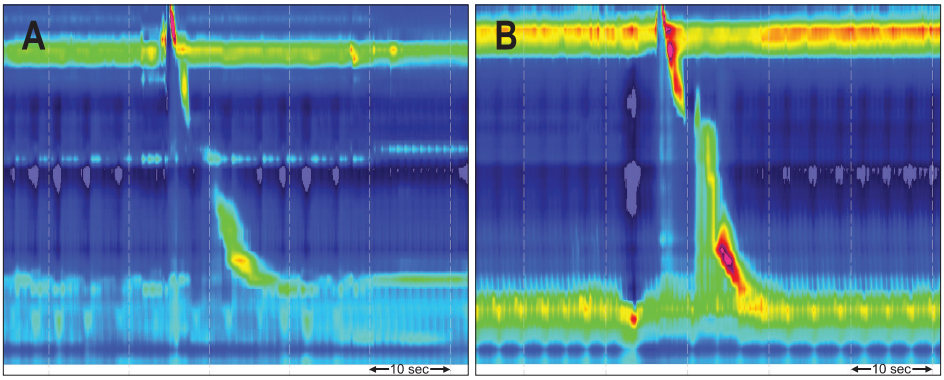

Fig. 2. Typical worsened case. (A) A primary, high resolution manometry tracing of patient 11 showed weak peristalsis with small peristaltic defects based on the updated Chicago Classification. (B) Follow-up high resolution manometry after supportive care showed variant achalasia.

pathophysiology.[2] Sifrim *et al.*[10] reported that the spectrum of primary esophageal motility disorders is an expression of a progressively failing deglutitive inhibition based on an inverse relationship between the degree of inhibition and the propagation velocity of the deglutitive contraction among achalasia, diffuse esophageal spasm, and intermediate forms. These results demonstrate the possibility of transition among primary esophageal motility disorders according to failing deglutive inhibition.[10] Currently, the varied severities of esophageal motility disorders as seen in the visible spectrum are thought to be variants of the disease rather than distinct diseases. This is based on the frequent occurrence of intermediate types of motility disorders and the transition from nutcracker esophagus to diffuse spasm or from diffuse spasm to achalasia.[11-13]

This case series revealed a variable course of follow-up with either improved or worsened symptoms. Four cases of the transition from achalasia variant to peristaltic abnormality, one case of the transition from diffuse esophageal spasm to type II achalasia, and one case of the transition from peristaltic abnormality to achalasia variant were reinterpretations according to the updated Chicago Classification system. Four cases that were previously unclassified are now categorized as achalasia variant according to the new system. Four unclassified cases showed elevated IRP and some instances of intact peristalsis or weak peristalsis.

In conclusion, six out of 13 esophageal motility disorder cases had a different follow-up classification according to the updated Chicago Classification system. Four previously unclassified cases were reclassified as achalasia variant. Although sample size is small, this study demonstrates that esophageal motility disorders may not be best conceptualized as an independent group. In the future, studies involving more patients are needed, along with efforts to investigate the pathophysiology of esophageal motility disorders.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

REFERENCES

1. Richter JE. Oesophageal motility disorders. Lancet 2001;358:823-828.
2. Patti MG, Gorodner MV, Galvani C, et al. Spectrum of esophageal motility disorders: implications for diagnosis and treatment. Arch Surg 2005;140:442-448.
3. Pandolfino JE, Kahrilas PJ; American Gastroenterological Association. AGA technical review on the clinical use of esophageal manometry. Gastroenterology 2005;128:209-224.
4. Spechler SJ, Castell DO. Classification of oesophageal motility abnormalities. Gut 2001;49:145-151.
5. Nayar DS, Khandwala F, Achkar E, et al. Esophageal manometry: assessment of interpreter consistency. Clin Gastroenterol Hepatol

2005;3:218-224.
6. Bredenoord AJ, Fox M, Kahrilas PJ, et al. Chicago classification criteria of esophageal motility disorders defined in high resolution esophageal pressure topography. Neurogastroenterol Motil 2012;24 Suppl 1:57-65.
7. Chen WH, Omari TI, Holloway RH, Checklin H, Dent J. A comparison of micromanometric and standard manometric techniques for recording of oesophageal motility. Neurogastroenterol Motil 1998;10:253-262.
8. Clouse RE, Parks T, Haroian L, Zakko SF. Development and clinical validation of a solid-state high-resolution pressure measurement system for simplified and consistent esophageal manometry. Am J Gastroenterol 2003;98:S32-S33.
9. Park MI. Clinical usefulness of high-resolution manometry. Korean J Neurogastroenterol Motil 2009;15:107-115.
10. Sifrim D, Janssens J, Vantrappen G. Failing deglutitive inhibition in primary esophageal motility disorders. Gastroenterology 1994;106:875-882.
11. Cha SW, Lee JS, Im HH, et al. Factors involved in the transition from achalasia to nutcracker esophagus or diffuse esophageal spasm after intrasphincteric injection of botulinum toxin. Korean J Gastrointest Motil 2001;7:188-196.
12. Millan MS, Bourdages R, Beck IT, DaCosta LR. Transition from diffuse esophageal spasm to achalasia. J Clin Gastroenterol 1979;1:107-117.
13. Vantrappen G, Janssens J, Hellemans J, Coremans G. Achalasia, diffuse esophageal spasm, and related motility disorders. Gastroenterology 1979;76:450-457.