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High resolution manometric findings in patients with Chagas' disease esophagopathy

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ABSTRACT

Objective: To describe high resolution manometry features of a population of symptomatic patients with Chagas' disease esophagopathy (CDE). **Methods:** Sixteen symptomatic dysphagic patients with CDE [mean age (54.81±13.43) years, 10 women] were included in this study. All patients underwent a high resolution manometry. **Results:** Mean lower esophageal sphincter (LES) extension was (3.02±1.17) cm with a mean basal pressure of (15.25±7.00) mmHg. Residual pressure was (14.31±9.19) mmHg. Aperistalsis was found in all 16 patients. Achalasia with minimal esophageal pressurization (type 1) was present in 25% of patients and achalasia with esophageal compression (type 2) in 75%, according to the Chicago Classification. Upper esophageal sphincter (UES) mean basal pressure was (97.96±54.22) mmHg with a residual pressure of (12.95 ±6.42) mmHg. **Conclusions:** Our results show that LES was hypotensive or normotensive in the majority of the patients. Impaired relaxation was found in a minority of our patients. Aperistalsis was seen in 100% of patients. UES had impaired relaxation in a significant number of patients. Further clinical study is needed to investigate whether manometric features can predict outcomes following the studies of idiopathic achalasia..

1. Introduction

Chagas disease (CD) is a tropical infectious malady caused by the protozoan *Trypanossoma cruzi*. It has been first described by the Brazilian physician Carlos Chagas in 1909[1]. CD is highly prevalent in South America. It is estimated that 8 million people are infected by *Trypanossoma cruzi* in Latin America[1].

Insects of the subfamily Triatominea act as vectors of the protozoan, sheltering trypomastigotes in their digestive system. Humans contract trypanossomiasis when bitten by vector species. The protozoan present in the insect's stools infiltrates man's scratched skin or permissive mucosa, where a lymphoreticular response occurs. This local inflammatory response may be clinically apparent as an inoculation chagoma or as the Romaña's sign. Circulating forms (trypomastigotes) are taken to peripheral tissues, such as liver, spleen, lymphatic ganglia, skeletal and heart muscle, where they form pseudocysts of amastigotes. Pseudocysts' rupture triggers inflammatory reaction with muscle and neuron cell damage. The inflammatory reaction and cell destruction are maintained by the presence of *Trypanossoma cruzi* or its fragments and by the DNA of the parasite, with a late hypersensitivity reaction that results in esophageal dysperistalsis and dilation[2,3]. In Chagas disease esofagopathy (CDE) there is always some degree of destruction of the autonomic nervous system, which is presumed to precede the changes in esophageal motility[4].

Non-vectorial mechanisms such as blood transfusion, solid organ and bone marrow donation, ingestion of infected food and vertical transmission also play important role in CD's spread nowadays[1,4]. CD spread in non-endemic areas is also ascribable to populational movements[1]. It is estimated that a few hundred chagasic patients live today in Europe, USA and Asia[4].

CD is characterized by an acute phase, which is asymptomatic in most cases. The majority (60%-70%) of infected individuals will never manifest the disease (indeterminate form of CD). More than one third of these will manifest chronic involvement of cardiac or gastrointestinal system, or both, throughout their lives[1]. Every year, an

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estimated 0.33% of patients with indeterminate form of CD progress to CDE[4].

Gastrointestinal dysfunction (megacolon or megaesophagus) occurs in 10%–15% of patients infected by *Trypanossoma cruzi*[4]. CDE is characterized by slowly-progressive dysphagia, malnutrition and weight loss. Some patients may also experience regurgitation, heartburn, chest pain and pulmonary complications due to alimentary aspiration.

The aim of this paper is to describe high resolution manometry (HRM) characteristics of a population of symptomatic patients with CDE.

2. Materials and methods

2.1. Population

Sixteen symptomatic dysphagic patients with CDE [mean age (54.81±13.43) years, 10 women] were included in this study. CD's diagnosis was confirmed either by positive serologic test for CD or by typical manifestations of CD in other target organs, such as the heart and the colon. CDE diagnosis was made on the basis of typical clinical manifestations and findings at the esophagogram, such as contrast retention, esophageal dilation, sigmoid esophagus and bird's beak appearance of esophagogastric junction[5]. Patients who had undergone previous surgical treatment for achalasia were excluded.

2.2. Tests

All patients underwent upper digestive endoscopy to rule out malignant or premalignant lesions.

All individuals underwent barium esophageal study (esophagogram) for better evaluation of esophageal dilatation grade. One patient had a grade Ⅰ megaesophagus (maximum esophageal diameter < 4 cm), 2 had a grade Ⅱ (maximum esophageal diameter 4–7 cm), 4 had grade Ⅲ (maximum esophageal diameter 7–10 cm) and five patients had grade Ⅳ megaesophagus (maximum esophageal diameter > 10 cm or sigmoid esophagus).

2.3. HRM

HRM data of all patients have been acquired using a solid-state HRM assembly with 36 solid-state sensors spaced at 1 cm intervals (Sierra Scientific Instruments Inc., Los Angeles, (CA). The HRM assembly was positioned transnasally. All studies have been performed with patients in left lateral position, after a minimum fasting period of eight hours. The manometric protocol included a 20-second period to assess basal sphincters pressures and ten 5 mL water swallows. Manometric data were analyzed using ManoView analysis software (Sierra Scientific Instruments Inc., Los Angeles, CA). The following manometric parameters were studied: lower esophageal sphincter (LES) length, basal and residual pressures, esophageal body peristalsis, and upper esophageal sphincter (UES) basal and residual pressures.

3. Results

LES was not transposed in 3 patients. Therefore, LES has been studied in 13 patients. Mean LES extension was (3.02±1.17) cm (range 1.6–4.7 cm); short LES was detected in 46% of patients. Mean basal pressure was (15.25±7.00) mmHg (range: 2.1–26.2 mmHg); LES was hypotensive in 39% and normotensive in 61% of patients. Residual pressure was (14.31±9.19) cm (range: 2.0–30.9 mmHg), LES impaired relaxation was found in 23% of patients.

Aperistalsis was found in all 16 patients. Achalasia with minimal esophageal pressurization (type 1) was present in 25% of patients and achalasia with esophageal compression (type 2) in 75%, according to the Chicago Classification[6, 7].

UES was analysed in all patients. UES mean basal pressure was (97.96±54.22) mmHg (range: 29.7–207.6 mmHg), LES was hypotensive in 37%, normotensive in 19% and hypertensive in 37% of patients. Residual pressure was (12.95±6.42) mmHg (range: 2.9–23.2 mmHg) and UES impaired relaxation was present in 44% of patients(Figure 1).

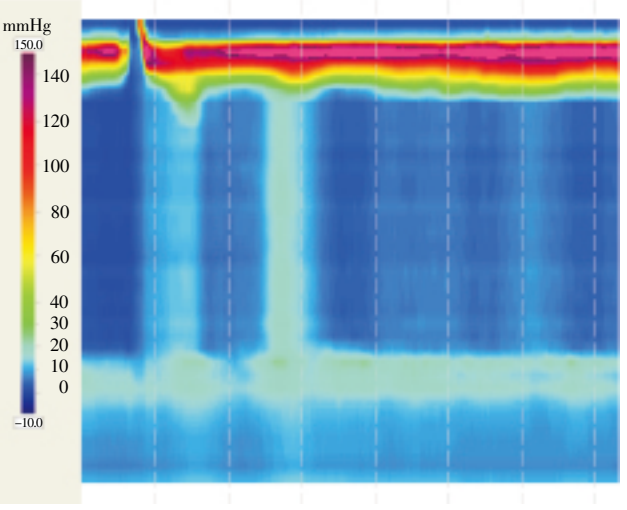

Figure 1. High resolution manometry tracing of Chagas' disease esophagopathy.

4. Discussion

Our results show that LES was hypotensive or normotensive in the majority of the patients. Impaired relaxation was found in a minority of our patients. Aperistalsis was seen in 100% of the patients. UES had impaired relaxation in a significant number of patients.

CDE and idiopatic (primary) achalasia share several similarities[8]. In both diseases the main feature is aperistalsis of the esophageal body[8–10]. The LES shows a defective relaxation[9]; however, nowadays most patients receive surgical therapy after some sort of endoscopic therapy, either forceful dilatation or botulinum toxin injection, rendering the LES findings unusable. LES basal pressure can be either hypotonic, normotonic or hypertonic[10]. UES has not been enough studied in patients

with CDE or idiopathic achalasia and no clinical conclusions can be drawn.

Few papers analyzed the preoperative esophageal manometry as a prognostic factor for outcomes. Arain *et al*[11] found better results after myotomy and fundoplication in patients with a hypertonic LES, although others did not report the same results[12].

We previously reported our experience with conventional manometry in CDE patients and found that the resting pressure of the upper and lower esophageal sphincter, and amplitude and propagation of peristalsis of the esophageal body did not correlate with surgical outcome[13-15].

HRM is a new technology recently available for functional study of esophageal motility[16]. One of its most advantageous features is simultaneous data acquisition from hypopharynx to stomach. Furthermore, manometric data display as pressure topography plots enhances ability to recognize motility disorders, notably focal ones, which could otherwise be missed by analysis of conventional manometry tracing. The current study is the first report of HRM in patients with CDE.

HRM is probably the best method for LES relaxation assessment since motion artifacts are minimized and diaphragm contractions can be discriminated from LES pressure[17]. We found that the LES was hypotensive or normotensive in the majority of the patients. Impaired relaxation was found in a minority of our patients, although all of them presented with dysphagia at the moment of the test. This feature strongly contrasts with literature data regarding LES manometric characteristics in achalasia, as the expected finding is impaired LES deglutitive relaxation with elevated residual pressures.

The esophageal body at the light of HRM shows aperistasis in all patients. The Northwestern University group classified the aperistalsis based on esophageal pressurization[6,7]. They found that patients with idiopatic achalasia with esophageal compression (type Ⅱ) are more likely to have favourable response to therapy than patients with classic achalasia (type Ⅰ) and patients with achalasia with spasm (type Ⅲ) have the worst response to therapy. We did not find type Ⅲ in our series. We are still unsure if the Chicago classification can be applied to CDE.

Interestingly, we found a significant number of patients with hypertonic and non-relaxing UES. It is known that the UES is composed of striated muscle that is not affected by CDE; however, our findings may be a physiologic response to prevent aspiration.

Our results show our initial experience with HRM in the evaluation of CDE. Most patients were not treated yet and those operated have a very short-term follow-up. Thus, it is still impossible to ascertain prognostic inference from our studies. Further clinical experience will state whether manometric features can predict outcomes following the studies of idiopathic achalasia.

Conflict of interest statement

We declare that we have no conflict of interest.

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