Abdominal bloating and distension: is it a motility or visceral hypersensitivity problem?

Fernando Azpiroz
Digestive System Research Unit, University Hospital Vall d’Hebron; Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (Ciberehd); Department of Medicine, Universitat Autònoma de Barcelona, Barcelona, Spain

RESUMEN

La hinchazón y la distensión abdominales con frecuencia se atribuyen al gas intestinal, pero el mecanismo por el cual el gas puede producir los síntomas no está claro. En la mayoría de los casos, la tasa de la producción de gas, así como del volumen de gas intestinal, se encuentra dentro del rango normal. Además, estudios detallados que miden el volumen de contenido intestinal no gaseoso no han detectado diferencias consistentes relacionadas con distensión y distensión abdominal. En este contexto, la hinchazón puede estar relacionada con la hipersensibilidad intestinal. Por otro lado, la visible distensión abdominal parece una respuesta de comportamiento, con una contracción diafragmática y descenso con protrusión de la pared anterior. De hecho, la distensión abdominal se puede corregir mediante biorretroalimentación guiada por electromiografía abdominofrénica. Sin embargo, el motivo por el cual estos pacientes adquieren este comportamiento anormal sigue siendo un misterio. Del mismo modo, los factores que desencadenan los episodios de distensión abdominal no han sido identificados, pero presumiblemente la percepción de la sensación de hinchazón puede jugar un papel determinante. El diagnóstico fisiopatológico adecuado parece importante para el manejo de estos pacientes.

Palabras clave: Distensión abdominal. hinchazón abdominal. gas intestinal. respuestas viscerosomáticas.
ABSTRACT

Abdominal bloating and distension are frequently attributed to intestinal gas, but the mechanism by which gas might produce the symptoms is not clear. In most cases, the rate of gas production, as well as the volume of intestinal gas, is within the normal range. Furthermore, detailed studies measuring the volume of non-gaseous intestinal content have not detected consistent differences related to abdominal bloating and distension. In this context, bloating may be related to gut hypersensitivity. On the other hand, visible abdominal distension seems a behavioral response, featuring a diaphragmatic contraction and descent with anterior wall protrusion. Indeed, abdominal distension can be corrected by abdomino-phrenic electromyography-guided biofeedback. However, why these patients acquire this abnormal behavior remains unknown. Likewise, the factors that trigger the episodes of abdominal distension have not been identified, but conceivably, the perception of bloating sensation may play a role. Proper pathophysiological diagnosis seems important for the management of these patients.

Corresponding author: Fernando Azpiroz, azpiroz.fernando@gmail.com

Key words: Abdominal distension. Abdominal bloating. Intestinal gas. Viscerosomatic responses.

INTRODUCTION

Conventionally, the terms abdominal bloating and distension were imprecise and bloating was used as the subjective sensation of abdominal distension. More recently the term bloating has been defined as “sensation of increased abdominal pressure,” and abdominal distension as an objective, visible increase in girth. Using this terminology the relation of bloating to distension has become clearer, an area that resulted confusing in the previous literature. A substantial proportion of patients, particularly those with constipation-predominant irritable bowel syndrome (IBS), exhibit both subjective bloating sensation and visible abdominal distension, but other patients do not, and this lack of correlation has been evidenced in patients with diarrhea-predominant IBS or those with visceral hypersensitivity.

Recent data indicate that the sensation of bloating and objective abdominal distension may originate by different mechanisms (Fig. 1). It is important to emphasize that abdominal distension may be related to various organic disorders, and depending on the geographic region, parasitic diseases, and food allergies should be considered.

This review will specifically focus on abdominal bloating and distension in patients with functional digestive disorders. These symptoms may be present in several functional digestive syndromes, such as functional dyspepsia and IBS. Functional disorders frequently overlap, so that abdominal bloating/distension may be associated with a variety of symptoms. In the absence of associated digestive symptoms, the diagnosis of functional abdominal bloating is used.
GENERAL CLINICAL FEATURES

Many patients with functional gastrointestinal symptoms complain of abdominal bloating and distension, and these symptoms are more frequent in women than in men. The severity of bloating may vary from very mild to severe and uncomfortable. An important aspect of the anamnesis is to determine whether bloating is associated with other abdominal symptoms, such as abdominal discomfort or pain. As stated above, in a substantial proportion of patients, the bloating sensation is associated with visible abdominal distension, and it is important to record the patients’ own impression about the presence and severity of objective (visible) abdominal distension. Some patients complain of bloating but acknowledge no physical evidence of abdominal distension. Others, point toward a “distended” abdomen that the examining physician appreciates as normal. Bloating/distension may be localized in the upper abdomen, sometimes associated with dyspeptic symptoms, or in the lower abdomen, as part of IBS related syndromes; many patients describe bloating of the whole abdomen.

Bloating/distension may be related to food intake. A substantial number of patients with bloating, up to 82%, describe that it develops or worsens in the early postprandial period. Some patients complain of being even unable to complete a full meal because of bloating. This effect is more pronounced when eating large and especially fatty meals. Postprandial bloating is a characteristic feature in specific subgroups of patients, such as dyspepsia and binge eaters. Some patients claim specific food intolerances in connection with bloating/distension, but the putative mechanism of the intolerance, or even whether this is real or imaginary, remains unclear. High fiber foods or fiber supplements are frequently reported to worsen bloating. Dairy products are frequently reported as deleterious, but only an undetermined fraction of these cases is truly due to genuine lactose intolerance; lactase deficit is unlikely when the symptoms occur in response to low quantities of dietary lactose. Fatty foods and carbonated drinks are also frequently reported as offending.

Circadian variations are a common feature of abdominal distension. In most patients, distension progressively develops during daily activity and tends to diminish or disappear after night rest. This has probably a physiological base because continuous measurement of girth in healthy subjects has shown that girth progressively increases during the day and returns to basal at night. Meals are other factors that have shown to increase girth in healthy subjects. Girth is also larger in the upright position than in supine, and likewise,
bloating tends to be alleviated by lying down. Stress is reported by some patients (about one third in one study) to worsen abdominal distension, and patients tend to feel better when relaxed. In some patients bloating/distension are associated with tiredness and sleeping difficulties, and these symptoms altogether impair quality of life. Some patients characteristically describe the rapid onset of distension after a precipitating event, <60 s in 35%, and < 10 min in another 26%. As discussed below, such abrupt development would fit with a muscular mechanism of bloating. The abdominal rumbling is a frequent associated feature, but passing flatus or stool does not necessarily alleviate bloating.

In up to 40% of women, bloating gets worse before and during the menstrual period. Furthermore, bloating is one of the most frequent menstrual symptoms. Neurohormonal factors have been implicated, but the prevalence of bloating is the same in pre- and post-menopausal women. Furthermore, some data indicate that in healthy women, during the menstrual period, bloating may progressively increase during the daytime, but is not paralleled by objective girth increments.

**VISCERAL SENSITIVITY AND REFLEXES**

Bloating sensation may originate from the viscera and may be related to impaired gut motility. Some studies have shown that patients complaining of bloating have impaired propulsion and delayed clearance of intraluminal gas, due to the abnormal reflex control of gas transit. This motor dysfunction cannot be identified by conventional techniques, such as intestinal manometry or scintigraphic measurement of solid/liquid chime transit. Probably the dysfunction is more subtle and may be related to increased resistance to flow and/or impaired accommodation in discrete gut segments. Alternatively, other components of chime may be the offending element that triggers bloating. An interesting study illustrated how different intraluminal elements, gas or not gas, may induce bloating. In a group of healthy subjects lactulose, which is not absorbed in the small intestine and is fermented into the colon releasing hydrogen, induced flatulence and bloating. Interestingly, non-fermentable methylcellulose produced no hydrogen release detectable by breath test and did not induce flatulence, but still induced the sensation of abdominal bloating. Hence, probably little amounts of either gas or fiber, not likely to induce changes in girth, are sufficient to trigger the bloating sensation.

In recent years, the relation of abdominal bloating/distension to gut contents has been carefully evaluated by imaging studies using computed tomography (CT) and magnetic resonance imaging scanning. In patients with functional gut disorders symptoms are not related to major changes in gut content, but some minor alterations have been detected in some of them. Gut hypersensitivity, a characteristic feature of IBS patients, could explain these symptoms. Experimental studies have shown that probe stimuli, such as gut distension, tend to reproduce the customary symptoms in patients complaining of abdominal bloating, and this can be triggered by small volume balloon distension. In patients with functional gut disorders, altered sensitivity combined with impaired
control of gut motility, and both dysfunctions may interact to produce their bloating sensation. Basically, in case of a hypersensitive gut, even normal intraluminal contents would induce sensation of abdominal bloating (Fig. 1).

**DISTORTED INTERPRETATION**

Bloating sensation without distension may be also related to a distorted interpretation. A double-blind study was performed on people who regarded themselves as severely lactose intolerant and complained of bloating after consumption of even small amounts of dairy products; they reported negligible symptoms when consuming 250 ml milk, regardless of whether it was lactose-hydrolyzed or not. These data indicate that the milk-related symptoms in these patients had an imaginary origin. Some patients, usually with persistent bloating, just exhibit a prominent, fatty abdomen. A prospective study reported that patients with bloating were more likely to have experienced recent weight gain than healthy controls, despite similar age, sex, and body mass index between both groups. Thus, fat accumulation in the abdomen may favor the development of the awareness of bloating as a symptom. Some of these patients experience IBS-type symptoms and believe they are due to intestinal gas retention.

**NORMAL ABDOMINAL ACCOMMODATION**

The volume of intra-abdominal contents exhibits physiological variations in relation to ingestion and bladder/rectal evacuation. For that purpose the walls of the abdomen actively adapt to its content by tight control of their muscular tone, a phenomenon termed abdominal accommodation. In healthy subjects, an increase in intra-abdominal content induces relaxation and ascent of the diaphragm, which permits cephalic expansion of the abdominal cavity with minor protrusion of the anterior wall. The diaphragmatic ascent produces a decrease in lung height, which is compensated by intercostal muscle contraction, elevation of the coastal wall and an increase in pulmonary cross-section, to prevent a reduction in lung volume; hence, the accommodation of intra-abdominal content involves a well-coordinated response of the abdominothoracic walls as a whole. The results obtained in acute animal experiments support the existence of viscerosomatic reflexes that modulate the abdominal wall contraction and, conceivably, this type of reflexes govern abdominal accommodation.

Abdominal accommodation has been studied measuring the responses of the abdominal wall muscles (by means of electromyography [EMG]) to experimental increments in intestinal content (modeled by colonic gas infusion). A considerable intra-abdominal overload (1.4 L colonic gas infusion) produces in healthy subjects a relatively small increment in girth, because of the muscles in the anterior abdominal wall contract, while the diaphragm relaxes, and this abdomino-phrenic coordination controls the intra-abdominal distribution of the volume load. Obviously, a very large intra-abdominal volume increment will result in visible abdominal distension with significant girth increment, and this can be seen in patients with massive intestinal retention due to intestinal obstruction or pseudoobstruction or in patients with ascites.
ABNORMAL VISCEROSOMATIC RESPONSES AND DISTENSION

Some studies showed that patients with functional or IBS-related bloating exhibit abnormal responses to colonic gas infusion \(^{18,23}\). On the one hand, patients with IBS and bloating had reduced tolerance to colonic gas infusion and reported significantly more severe symptoms than healthy subjects; this is conceivably related to intestinal hypersensitivity with increased perception of mechanical stimuli characteristic of these patients\(^{14,24-27}\). At the same time, colonic gas infusion induced exaggerated abdominal distension in patients with IBS-related bloating. In them, intra-abdominal volume increments in the physiological range, induce a paradoxical relaxation of the anterior abdominal wall and a contraction of the diaphragm, and this abdomino-phrenic incoordination is associated to exaggerated abdominal distension\(^{28}\). Abnormal wall responses to intra-abdominal volume increments may explain the characteristic meal-related abdominal distension in some patients.

Meal ingestion produces a minimal girth increment in healthy subjects, due to the efficacy of the normal abdominal accommodation mechanism that activates diaphragmatic relaxation, providing extra space in the upper abdominal cavity, and a compensatory contraction of the anterior wall muscles preventing distension in the upright position. By contrast, dyspeptic patients experience a significantly larger increment in girth due to aberrant abdominal accommodation, featuring paradoxical contraction of the diaphragm and relaxation of the anterior wall\(^{29}\). The contraction of the diaphragm induced by meal loads in dyspepsia is comparable to that observed in IBS during colonic gas infusion\(^{30}\). However, the response of the anterior wall in dyspepsia and IBS is site-specific. Dyspeptic patients react to meal ingestion with the relaxation of the upper anterior wall, i.e., upper rectus and external oblique, as opposed to contraction in healthy subjects; no changes in the lower muscles, i.e., lower rectus and internal oblique, were observed either in dyspeptic patients or healthy subjects. By contrast, IBS patients relax the internal oblique in response to colonic gas.

MORPHOLOGICAL AND FUNCTIONAL CORRELATIONS

Abdominal distension has been investigated using an original program for volumetric measurements by CT imaging analysis. Careful studies comparing CT images from the same patients during basal conditions and during episodes of severe bloating have demonstrated first that most patients complaining of abdominal distension really have an objective increase in girth and anterior abdominal wall protrusion.

Discrete episodes of real-life abdominal distension in patients with functional bloating were associated with a relatively modest increase in net intra-abdominal volume, but an aberrant somatic response leads to abdominal distension\(^{23,28}\), specifically, a paradoxical contraction of the diaphragm that pushed abdominal contents downward and relaxation of the anterior abdominal wall, resulting in redistribution of the contents with ventral expansion of the abdominal cavity (Fig. 1). These findings were similar to the response to provocative stimuli described above.
Recent studies have shown a paradoxical activity of the chest during abdominal distension. Indeed, in contrast to healthy subjects, in whom the coastal wall follows the diaphragm to compensate lung function, the diaphragmatic descent during episodes of abdominal distension in patients is associated with elevation of the coastal wall and chest hyperinflation. The fact that other individuals with increased lung volumes, such as patients with chronic obstructive pulmonary disease or even healthy subjects during exercise, do not report significant abdominal distension suggests that the mechanism of abdominal distension may involve both thoracic and abdominal contributions. The breathlessness reported by some patients during episodes of abdominal distension might be related to hyperinflation of the chest rather than thoracic compression by a distended abdomen.

**UNDERLYING MECHANISMS OF ABDOMINAL DISTENSION**

The mechanism of the abdomino-phrenic dysfunction in patients with abdominal distension has not been demonstrated yet. Since, these patients’ exhibit abnormal viscerovisceral reflexes, impairment of viscerosomatic reflexes would be conceivable. However, some data suggest that abdomino-phrenic incoordination and abdominal wall dystonia may be related to learned responses. In a classic article, Alvarez described in great detail a series of patients in which pronounced abdominal distension was, in his view, related to the muscular activity of the abdominal wall\(^31\), and used the term “hysterical type of nongaseous abdominal bloating.” This hypothesis is substantiated by the fact that in some patients visible abdominal distension has a very rapid onset\(^4,31\) and resolves instantaneously by gentle abdominal palpation while asking the patients to relax or by anesthesia\(^31\). Severe abdominal distension also resolves during hypnotic induction.

Using an EMG-guided biofeedback technique, it has been shown that patients effectively learn to control abdomino-thoracic muscular activity, and correction of postural tone is associated with a reduction in girth back to baseline levels\(^32,33\). In these studies, patients learned to correct their postural tone during the treatment sessions and reported a subsequent improvement in distension. Furthermore, the change was significantly greater than with placebo treatment. Moreover, after placebo failure in a control group, subsequent biofeedback produced the same effect as in patients originally allocated to the biofeedback treatment.

The effect of biofeedback provides persuasive evidence that the aberrant somatic activity associated with abdominal distension is under voluntary control. Indeed, patients with abdominal distension were trained to control abdomino-thoracic activity and thereby correct distension\(^32,33\). These data further suggest that abnormal abdomino-thoracic activity associated with distension may represent a conditioned response. Other frequent somatic

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<th>TABLE 1. ABDOMINAL DISTENSION: FACILITATING AND TRIGGERING FACTORS</th>
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<td><strong>Facilitators</strong></td>
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<td>Visceral hypersensitivity (abdominal sensations)</td>
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<td>Food intake (intolerance)</td>
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<td>Circadian influences</td>
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manifestations of functional gut disorders, such as rumination and aerophagia, may have a similar conditioning-related origin. However, what determines the acquisition of the conditioned response and what the conditioning stimuli are, i.e., why patients learned to produce it in the first place and in response to what, remain unknown (Table 1). It could be speculated that in patients with increased intestinal sensitivity, even minor increases in intestinal content may induce a bloating sensation and, in conditioned patients, this sensation triggers the abnormal abdomino-thoracic response that produces abdominal distension, which may further reinforce the bloating sensation.

**MANAGEMENT STRATEGIES**

Based on the data discussed above, tests, such as CT scanning and EMG evaluation, are only indicated in the indication of specific testing in these patients (by CT or EMG) may be restricted to rare cases were intestinal dysmotility and luminal pooling are suspected. The initial line of treatment in patients with abdominal distension would target potential triggers, such as increased intestinal load, either in relation to diet or constipation. Since perception of symptoms, particularly bloating, seems a key factor frequently associated with distension and treatment of abdominal symptoms by conventional IBS therapy would seem reasonable. If identifiable, other triggers, such as emotional factors or anxiety, could also be targeted. As described above, a complex biofeedback technique has been proven useful to correct the abdominal postural tone and resolve the distension. The indication of this treatment is restricted by its complexity and cost. Conceivably, a simpler and cheaper conductual technique could be developed and, if proven really effective, might become the standard second-line treatment of abdominal distension for patients unresponsive to standard dietary or pharmacologic modalities.

**CONCLUSION**

Abnormal abdomino-phrenic adaptation to intra-abdominal content in patients with bloating may be a behavioral response, analogous to aerophagia or rumination. The faulty response may be triggered by uncomfortable abdominal sensations. Indeed, it has been consistently shown that patients with bloating have impaired transit of intestinal gas due to abnormal gut reflexes. Given the characteristic intestinal hypersensitivity in these patients, even modest retention may induce bloating sensation and objective abdominal distension. Indeed, the amount of intestinal gas measured during spontaneous bloating is relatively small. Conceivably, other factors such as meals, fatigue or stress, either independently or in conjunction, may also contribute to trigger the abnormal response in real-life distension. Correction of the inappropriate abdomino-phrenic activity by biofeedback treatment may resolve abdominal distension.

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DISCLOSURES

None.

REFERENCES


