

## REVIEW ARTICLE

# ACUTE GIARDIASIS AND CHAPMAN REFLEXES: MUSCULOSKELETAL SYMPTOMS PRECEDING, DURING AND AFTER INFECTION

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Giardiasis is an acute infection caused by *Giardia lamblia*, which produces profuse secretory diarrhea that can lead to dehydration and electrolyte derangement. Musculoskeletal manifestations resulting because of giardiasis occur due to prolonged inflammation and viscerosomatic reflexes of the pathophysiology for this disease process. By treating the parasitic infection with an antiparasitic agent, as well as treating the somatic dysfunctions with osteopathic manipulative treatment, analgesics and a home exercise program, the patient in the following article experienced an uneventful course of treatment and a complete recovery including resolution of the pain.

## INTRODUCTION

The organism *Giardia lamblia* is most often transmitted through contaminated water or food, or by the fecal-oral route.<sup>1</sup> Clinical presentation of giardiasis can vary; approximately 50% of patients exposed remain asymptomatic, and the other 50% will develop gastrointestinal symptoms.<sup>2</sup> Gastrointestinal symptoms include loose diarrhea with foul-smelling, non-bloody stools, in addition to flatulence, abdominal cramps, bloating, loss of appetite, nausea and weight loss within 1–2 weeks of exposure.<sup>2</sup> Malabsorption, dehydration and substantial weight loss are hallmarks of the infection, in addition to the infectious diarrhea.<sup>2</sup> Approximately half of infections resolve without treatment within 4 weeks of onset, and the other half require antibiotic and/or antiparasitic therapy. The diagnosis of giardiasis is made by stool analysis revealing cysts. *Giardia* antigens can be detected in stool specimens using monoclonal antibodies or direct fluorescent assays; serologic studies are not useful because they cannot distinguish between active and recovered infection.

The prevention of infection with *G. lamblia* should focus primarily on the avoidance of contaminated water. Outbreaks of giardiasis have usually been associated with contaminated surface water or shallow wells. Vigorous hand-washing and proper disposal of soiled diapers should be practiced in day care settings. Boiling water that may be contaminated with *Giardia* cysts is useful for

eradication, but chlorination of the water is not effective. The cysts associated with *Giardia* transform into the trophozoite form in the gastrointestinal tract. The *Giardia* genus will lead to decreased expression of brush border enzymes, structure changes to the microvilli, increased intestinal permeability to water and death of small intestinal epithelial cells. Cysts and trophozoites are unable to survive outside of the gastrointestinal tract.<sup>2,3</sup>

## EPIDEMIOLOGY

Giardiasis is present all over the world, affecting nearly 8% of children and 2% of adults in developed countries, and 33% of persons living in developing countries have had giardiasis. In the United States, *Giardia* infection is the most common intestinal parasitic disease affecting humans. Those at greatest risk include those who travel to countries where giardiasis is common, those in child care settings with exposure to soiled diapers, those who work in the vicinity of or ingest contaminated water, and those engaged with anal intercourse (including men who have anal sex with men).<sup>2</sup>

## CASE REPORT

A 43-year-old male developed musculoskeletal symptoms of the bilateral hips, back pain, abdominal pain and thigh pain. He experienced 2 days of sharp bilateral pain along the greater trochanteric region, as well as radiation to the lateral thighs bilaterally. He did not have any specific or identifiable triggers to cause the pain, nor had he sustained an acute injury, although he did report swimming in 2 different lakes and had visited 2 different water parks 2 weeks prior to the onset of symptoms. He

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reported that the pain was insidious at the onset and gradually became more severe, to the point where he experienced difficulty sleeping at night due to significant pain when lying on either side. He initially attributed his symptoms to a musculoskeletal cause and used over-the-counter analgesics without relief.

Two days after the onset of symptoms, he noted the onset of severe watery diarrhea. He reported experiencing at least 12–15 episodes of this secretory diarrhea within the first 24 hours after onset as well as a fever of 102.2°F (39°C) at home. Associated symptoms included generalized weakness and malaise. He tolerated oral fluids and mild bland food but felt that post-meal transit time was accelerated. He attempted fluid resuscitation at home and was unsuccessful, becoming near-syncopal and requiring evaluation at the emergency department. He was treated with intravenous hydration and had labs performed, including a complete blood count and a basic metabolic panel. Other than slight hypokalemia with a potassium level of 3.4, the remainder of the labs were unremarkable. Stool studies were not ordered at this initial visit. His past medical history was notable for gastroesophageal reflux disease, chronic gastritis and *Helicobacter pylori* infection. His past surgical history was remarkable for an appendectomy. His family history is non-contributory. Social history was unremarkable for alcohol, tobacco or drug use. Home medications included one 30 mg lansoprazole tab daily and one 10 mg loratadine tab daily. He denied any medication allergies. His vital signs and physical exam were unremarkable, though his mucous membranes were dry at the time of presentation. He was discharged home and diagnosed with a viral gastrointestinal illness.

On the first post-discharge day he continued to experience several episodes of watery diarrhea. He reported another 10–15 episodes within these 24 hours. As a result of his ongoing symptoms, he was directly admitted to the hospital by his gastroenterologist. Upon admission, laboratory values were repeated and were not significantly different from the prior emergency department visit. Imaging of the bilateral hips with x-ray was unremarkable. A computed tomography scan of the abdomen and pelvis was also unremarkable. He demonstrated no acute peritoneal signs. However, at this time, stool samples were obtained for studies. Stool culture was negative for enteric pathogens. Toxin studies were negative for *C. difficile*. Ova and parasite studies were positive for *G. lamblia*. There were live parasites noted within the stool. *Giardia* enzyme-linked immunosorbent assay in the stool was positive, and the *Cryptosporidium* screen was positive as well, but his HIV antibody test was negative. He was treated with a course of nitazoxanide. Though he clinically improved, the musculoskeletal symptoms persisted.

## DISCUSSION

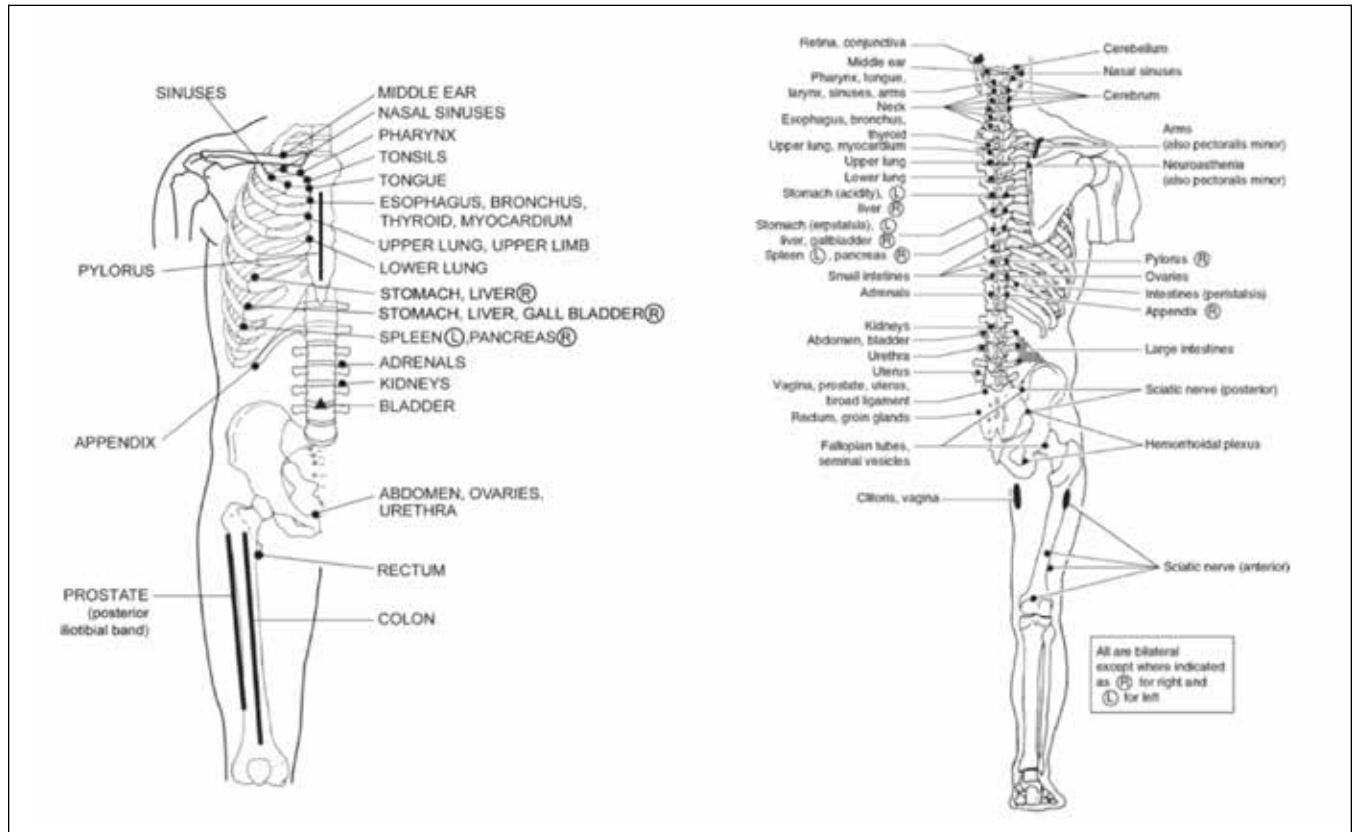
The musculoskeletal findings that preceded and accompanied the symptoms of this disease process provide the osteopathic physician with an additional clue to the underlying disorder. A consideration of the physiologic and pathophysiologic mechanisms involved is appropriate. The autonomic nervous

system mediates the interactions between the paravertebral ganglia and the prevertebral ganglia, as well as somatic structures leading to viscerosomatic reflexes. The paravertebral ganglia, also known as the sympathetic trunk, are found paraspinally to the sympathetic trunk of the spinal levels of T1–L2. The prevertebral ganglia are associated with the large vessels of the abdominal cavity: the celiac ganglia, the superior mesenteric ganglia and the inferior mesenteric ganglia. A viscerosomatic reflex is caused by irritation of the viscera, causing a signal to be sent to somatic structures in the local area as the result of a referred pain. Signal pathways by which these reflexes occur originate from afferent signals entering through the dorsal horn of the spinal cord (posteriorly) and efferent signals leaving through the ventral horn (anteriorly).<sup>4</sup> When signals enter through the visceral afferent pathway, nerves are very close to each other, resulting in signals being carried to higher brain centers, which then 1) travel back down to the efferent output, 2) form an immediate signal arc from the dorsal horn to the ventral horn in the brain matter, or 3) both. This can result in referred pain. It is often difficult to determine the origin of the input (ie, muscle versus viscera); therefore, the result is referred to muscular tissue. Specifically, an imbalance of sympathetic versus parasympathetic dominance can exacerbate expected symptoms associated with the respective autonomic division. Chapman reflexes are a type of viscerosomatic reflex mediated by sympathetic nerves. These reflexes represent lymphatic stasis secondary to diseased, stressed or irritated organs. Chapman points are located on both the anterior surface and the posterior surface. Anterior Chapman points are typically located in intercostal spaces, with the rib segments corresponding to the sympathetic innervations of the involved viscera, and are utilized for diagnosis. Posterior Chapman points are found in the soft tissue between the spinous process of a vertebrae above and a transverse process of a vertebra below and are utilized for treatment.<sup>4,5</sup>

*Giardia* affects primarily the lower gastrointestinal tract because of epithelial dysfunction in the small intestine. The posterior Chapman points associated with the gastrointestinal tract are located at T1–L2 levels at the spinous and transverse processes of the corresponding levels of the spine. The anterior Chapman point for the lower gastrointestinal tract occurs along the anterior iliotibial band bilaterally. The patient's hip and thigh pain occurred due to spinal facilitation, which is the maintenance of a pool of neurons in a state of partial or sub-threshold excitation. The prolonged inflammation of the small intestine due to resolving infection will certainly manifest in the musculoskeletal symptoms this patient was experiencing.

Innervation of the GI tract should be considered in addition to viscerosomatic reflexes and Chapman reflexes. The prevertebral ganglia are associated with the large vessels of the abdominal cavity: the celiac ganglia, the superior mesenteric ganglia and the inferior mesenteric ganglia. The GI tract and nearby structures can be grouped into the following regions based on the corresponding ganglia and nerves:

FIGURE 1:

Anterior and posterior Chapman points<sup>4,5</sup>

- 1) Upper GI tract: T5–T9 levels (celiac ganglion and greater splanchnic nerve): stomach, liver, gallbladder, spleen, and portions of the pancreas and duodenum.
- 2) Middle GI tract: T10–T11 levels (superior mesenteric ganglion and lesser splanchnic nerve): portions of the pancreas and duodenum, jejunum, ileum, ascending colon and proximal 2/3 of the transverse colon (the right colon). Also classified here are the kidneys and the upper ureters.
- 3) Lower GI tract: T12–L2 levels (inferior mesenteric ganglion and least splanchnic nerve): distal 1/3 of the transverse colon, descending colon and sigmoid colon (the left colon), as well as the rectum. Also classified here are the lower ureters. The transverse colon does not fit neatly into the above classification; the sympathetic innervation of the transverse colon for the proximal 2/3 is by the T10–T11 spinal levels and the distal 1/3 by the T12–L2 spinal levels. The parasympathetic innervation of the proximal two-thirds of the transverse colon is by the vagus nerve and the distal 1/3 by the pelvic splanchnic nerves.<sup>4,6</sup> The viscerosomatic reflexes will show tissue texture changes at the T5–T10 levels for the small intestine and T12–L2 for the length of the colon with respect to sympathetic autonomic innervation. The vagus and pelvic splanchnic nerves supply parasympathetic autonomic innervation to the GI tract.<sup>4,6</sup>

Of interest are the musculoskeletal manifestations of viscerosomatic reflexes occurring because of giardiasis. A literature search did not reveal any prior examples of either diagnosis of giardiasis using Chapman points or treatment of symptoms or sequelae using osteopathic manipulation. The patient continued to experience intermittent abdominal cramping for up to 4 weeks after completing the initial treatment. He continued to experience persistent bilateral hip and lateral thigh pain. The pain was more severe nocturnally and frequent sleep interruption was experienced. Acetaminophen was utilized for pain.

A comprehensive examination of the 10 body regions of somatic dysfunction—cranial, cervical, thoracic, ribs/sternum/clavicle, lumbar, sacrum, hips/pelvis/innominates, upper extremity, lower extremity and abdomen—was performed. Affected regions included thoracic, lumbar, sacrum, abdomen and hips/pelvis/innominate. The following somatic dysfunctions and structural abnormalities were identified:

- 1) T5–T7 neutral, side bent left, rotated right
- 2) Anterior Chapman points on the right in the fifth and sixth intercostal spaces and along the bilateral iliotibial bands
- 3) L2–L4 neutral, side bent right, rotated left
- 4) L5 neutral, side bent left, rotated right
- 5) A left-on-left sacral torsion

6) A hypertonic left piriformis muscle

7) A hypertonic right psoas muscle

8) A right anterior innominate

9) Hypertonic celiac collateral ganglion musculature

On a 10-point scale, the patient noted pain of 7.

Osteopathic manipulative medicine was engaged as an adjunctive treatment modality, and these treatments provided relief and were targeted to the affected biomechanical, autonomic and lymphatic dysfunctions. Treatments were performed twice at 2 consecutive visits. The autonomic dysfunction was treated with release and OA release (to normalize parasympathetic tone, in particular, the vagus nerve), rib raising (to normalize sympathetic tone) and sacral rocking (to normalize parasympathetic tone, in particular, the pelvic splanchnic nerves S2–S4). Collateral ganglia myofascial release (MFR) techniques were used to target the respective ganglion, particularly the superior (T10–T11) and inferior (T12–L2) mesenteric ganglia for this patient. MFR and muscle energy techniques were applied to the thoracic spine, lumbar spine, sacrum, piriformis and psoas muscles, with treatment of posterior Chapman points invoked. In addition, the patient was prescribed a home exercise program with lateral hip stretches. After the initial treatment, only the bilateral hip and thigh pain remained and was diminished, compared to the initial presentation. All the gastrointestinal and musculoskeletal symptoms took a total of 6 weeks to entirely resolve. The pain gradually improved, and the patient has had no residual effects. At the end of the 6 weeks, his pain reduced to 1/10. The resolution of posterior Chapman points additionally demonstrated a successful treatment. Lymphatic considerations and treatments were insignificant in this scenario; thus, those techniques were not performed here.<sup>6</sup>

## CONCLUSION

Infection with *Giardia lamblia* leading to acute giardiasis and secretory diarrhea shows associated musculoskeletal manifestations, including bilateral hip and thigh pain. Considerations of the underlying biomechanical and autonomic dysfunctions can suggest osteopathic manipulative treatment as an adjunctive to treating the underlying parasitic infection during convalescence and potentially help to shorten the course of disease if employed at onset of musculoskeletal symptoms. Due to the persistent inflammation, a multifaceted treatment modality would be beneficial in such situations. The patient was treated with an antiparasitic, analgesics and osteopathic manipulative treatment. He recovered without further sequelae.

## AUTHOR DISCLOSURE(S)

No relevant financial affiliations or conflicts of interest. If the authors used any personal details or images of patients or research subjects, written permission or consent from the patient has been obtained. This work was not supported by any outside funding.

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