

# Osteopathic Manipulative Treatment in the Management of Biliary Dyskinesia

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Financial Disclosures:  
None reported.

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Submitted  
July 2, 2013;  
revision received  
August 8, 2013;  
accepted  
August 20, 2013.

**Biliary dyskinesia is a functional gastrointestinal disorder of the gallbladder and sphincter of Oddi. Diagnosis is made on the basis of symptoms of biliary colic in the absence of cholelithiasis and gallbladder inflammation. Palpatory findings of tissue texture changes at midthoracic levels (T6-T9) may correspond to visceral dysfunction related to the biliary system. Osteopathic manipulative treatment (OMT) of the T6-T9 segments can remove the feedback related to the somatic component, thereby affecting nociceptive facilitation at the spinal level and allowing the body to restore autonomic balance. Few reports in the current literature provide examples of treatment for patients with biliary dyskinesia using OMT. The author describes the case of a 51-year-old woman who presented with symptoms consistent with biliary dyskinesia. Her biliary colic completely resolved after OMT. Osteopathic evaluation and OMT should be considered a safe and effective option for conservative management of biliary dyskinesia.**

*J Am Osteopath Assoc.* 2014;114(2):129-133  
doi:10.7556/jaoa.2014.027

Gastroenterologists define *biliary dyskinesia* as a motility disorder of the biliary system.<sup>1</sup> Diagnosis is made on the basis of the presence of typical biliary colic symptoms, including postprandial right upper quadrant pain, nausea, fatty food intolerance, vomiting, and bloating without cholelithiasis (ie, gallstones).<sup>1,2</sup> Incidence and prevalence are difficult to determine, as biliary dyskinesia does not have a distinct diagnostic code. In a 2013 study, Bielefeldt<sup>3</sup> found that the *International Classification of Disease, Ninth Revision (ICD-9)*<sup>4</sup> code 575.8 (“gall-bladder disease not elsewhere specified”) was used for biliary dyskinesia in 81% of cases using this code. Between 1997 and 2010, admissions with a primary diagnosis code ICD-9 575.8 tripled.<sup>3</sup>

Biliary dyskinesia must be distinguished from more serious suspicions of acute cholecystitis or other causes, and therefore patients with persistent biliary colic are often subject to extensive and expensive medical imaging and procedures, which often include ultrasonography, upper gastrointestinal series, esophagogastroduodenoscopy, computed tomography, and intravenous pyelography. Furthermore, results of a 2012 study<sup>1</sup> showed that 30% of patients with biliary dyskinesia underwent cholecystectomy. Of the patients who underwent cholecystectomy, almost half sought medical attention for recurring gastrointestinal complaints after surgery. The results of the study suggest that there is renewed need for careful patient selection and counseling for potential limitations of cholecystectomy, especially regarding treatment for patients with biliary dyskinesia.

In the present report, I describe a case of biliary dyskinesia that resolved after osteopathic manipulative treatment (OMT). I also review relevant physiologic and pathologic processes of the biliary system, diagnosis and standard management of biliary dyskinesia, and the concept of nociception in osteopathic medicine as it relates to biliary dyskinesia.

## Report of Case

A 51-year-old woman presented to the Osteopathic Manipulative Medicine Clinic with a 1-year history of intermittent postprandial right upper quadrant pain. After meals, the pain radiated into her mid-to-low back and epigastrium. The patient's symptoms worsened after eating greasy foods, though she attempted to keep a low-fat, vegetarian diet. She also noted intermittent diarrhea and constipation.

The patient had been evaluated by a general surgeon at the time of symptom onset. The general surgeon ordered a comprehensive metabolic panel, the results of which were within normal limits. An ultrasonographic image of the right upper quadrant revealed no abnormalities: the gallbladder contained no stones, no gallbladder wall thickening or pericholecystic fluid was found, the biliary tract was not dilated, and the common hepatic duct measured within normal limits (5 mm) at the porta hepatis. Evaluation of the pancreas by means of ultrasonography was also unremarkable. One month after presentation to the general surgeon, the patient underwent a nuclear medicine hepatic iminodiacetic acid (HIDA) scan using technetium (Tc99m) mebrofenin with cholecystokinin stimulation. Results were normal, with gallbladder ejection fraction at 30 minutes calculated at approximately 97%. Two months after presentation, biliary fluid was obtained via endoscopic retrograde cholangiopancreatography. Results contained no cholesterol crystals or biliary sludge. Also at 2 months after presentation, a gastroenterologist conducted an esophagogastroduodenoscopy and a colonoscopy. Results of a small bowel biopsy were negative for celiac disease, and results of a gastric biopsy showed slight chronic superficial gastritis

but no evidence of *Helicobacter pylori*. In addition, *H pylori* antibody test was also performed a few days before the gastric biopsy and was found to be normal, suggesting no infection or peptic ulceration.

On presentation to the Osteopathic Manipulative Medicine Clinic, past medical history also included hypothyroidism, seasonal allergies, and history of occasional tension-type headaches. Medications were levothyroxine (100 µg once daily), topiramate (50 mg twice daily), cetirizine (5 mg daily as needed for allergic rhinitis), selenium (200 µg once daily), pseudoephedrine (60 mg, 4-6 hours as needed for nasal congestion), multivitamin (once daily), and B-complex vitamin (once daily). On review of systems, the patient denied weight loss, vomiting, hematemesis, dysuria, urinary hesitancy, and bowel or bladder incontinence. The patient also denied tobacco, alcohol, or illicit drug use. On presentation, the patient's blood pressure was 110/70 mm Hg; pulse rate, 60 beats per minute; and respirations, 12 breaths per minute.

Physical examination revealed a healthy-appearing woman with a body mass index of 28. Her abdomen was soft and nontender to palpation without rebound or guarding. Osteopathic structural examination revealed boggy tissue texture changes at the level of the T6-T9 vertebrae on the right with right rotation, left side-bending. Inferior fascial drag over this segmental region was increased. Motion over the region of the sphincter of Oddi was palpated to have counterclockwise rotation. Tissue congestion was also found in the region of the gallbladder anteriorly. The superior third of the linea alba was restricted. The sacrum was in a left-on-right backward torsion pattern, and L5 was flexed, rotated, and sidebent right. Findings from the remainder of her physical examination were otherwise normal.

After providing verbal informed consent, the patient was treated with OMT on the day of presentation to the clinic by a third-year neuromusculoskeletal medicine/osteopathic manipulative medicine resident (K.H.). Osteopathic manipulative treatment included muscle energy to the thoracic region and sacrum and balanced ligamentous tension and myofascial release to the abdominal and lumbar regions. The patient tolerated the treatment well

without complication. Pain in the epigastric region and back was improved after treatment. Segmental somatic dysfunction was notably improved, though inferior fascial drag at T6-T9 was somewhat increased. At the completion of the visit, the patient was instructed to begin magnesium supplementation (325 mg daily) to decrease muscle and nerve irritation and digestive enzymes (1 with each meal; pancreatin, 70 mg; pepsin, 35 mg) to help break down proteins and disaccharides. In addition, the patient was instructed to begin gentle piston breathing (rhythmic abdominal/diaphragmatic breathing) for home exercise to help mobilize the rib cage region. She was asked to return in 2 weeks for reevaluation.

On return to the clinic 2 weeks after presentation, the patient reported that her right upper quadrant pain had completely resolved. She stated it initially resolved in the front and then resolved in the mid and low back region. In addition, she noted that her intermittent diarrhea and constipation ceased and she began having daily soft, formed bowel movements. She reported having been compliant with the digestive enzymes with each meal, as well as magnesium. At this follow-up visit, the musculoskeletal examination findings revealed residual fascial drag at T6-T9 on the right without rotational or side-bending dysfunction. Visceral pull to the gallbladder was present but much improved, and the sphincter of Oddi had a clockwise rotation. There was a left-on-left forward sacral torsion. The patient was again treated with gentle OMT using balanced ligamentous tension and myofascial release to the thoracic region and abdomen and muscle energy to the sacrum. She was instructed to continue the digestive enzymes with meals for at least 3 months. It was also recommended that the patient continue the magnesium supplementation and the home piston breathing exercise. She was told to return to the clinic on an as-needed basis; however, the patient did not return.

## Comment

### Physiologic and Pathologic Processes

Biliary colic is thought to be caused by increased intra-organ pressure and pain as the gallbladder contracts

against the gallbladder outlet or cystic duct opening in response to hormonal or neuronal stimulation. Biliary colic is manometrically measured as an increase in the sphincter of Oddi basal pressure. As pressure in the sphincter rises, resistance to bile flow from the common bile duct into the duodenum also increases.<sup>5</sup> In 1 study<sup>6</sup> involving 10 patients with suspected sphincter of Oddi dysfunction, all had abnormally high sphincter of Oddi basal pressure.

Biliary colic is most commonly located in the right upper quadrant but can be found in the epigastrium or in the chest,<sup>7,8</sup> and it is often associated with abdominal bloating, nausea, dyspepsia, vomiting, and fat intolerance.<sup>8</sup> The differential diagnosis includes, but is not limited to, peptic ulcer disease, gastroesophageal reflux disease, irritable bowel syndrome, nonulcer dyspepsia, acute mesenteric ischemia, cholecystitis, cholangitis, pancreatitis, hepatitis, subdiaphragmatic abscess, or pleurisy from pneumonia or empyema.<sup>9,10</sup>

On physical examination in patients with uncomplicated biliary colic, the pain is typically found to be visceral in origin and thus is less well localized. Patients will not present with a positive Murphy sign, as is usually found with acute cholecystitis.<sup>11</sup>

### Diagnosis

During both asymptomatic and symptomatic periods, laboratory results are typically normal in patients with uncomplicated biliary colic. Nonetheless, there are reasonable screening tests that can be helpful for excluding other differential diagnoses. A complete blood cell count is useful to evaluate for leukocytosis or anemia, amylase and lipase tests help to rule out pancreatitis, liver biochemical tests (aspartate transaminase, alanine transaminase, total bilirubin, alkaline phosphatase) are useful to evaluate for underlying hepatic disease, and urinalysis can be used to help identify underlying renal disease.<sup>7,12</sup>

For further evaluation of a patient with a medical history suggestive of biliary dyskinesia, imaging studies are used to look for the presence of gallstones, sludge, or gravel in the gallbladder. A systematic review<sup>13</sup> estimated that sensitivity of ultrasonography for determining the

presence of gallstones was 84% and specificity was 99%. If the diagnosis remains unclear after ultrasonography, a HIDA scan is recommended.<sup>13</sup> The HIDA scan has a sensitivity of 97% and a specificity of 90%.<sup>13</sup> Compared with computed tomography, magnetic resonance imaging shows better sensitivity and accuracy for gallstones.<sup>14</sup>

In patients with biliary colic and no gallstones at ultrasonography, bile microscopy can be performed to determine the presence or absence of microlithiasis or symptomatic sludge. Bile microscopy is the reference standard for confirming the presence of microlithiasis, with a sensitivity of 65% to 90%.<sup>15</sup> For patients with biliary-type pain, biliary dyskinesia is typically diagnosed after other conditions have been excluded.<sup>12</sup>

### Standard Management

Management of biliary dyskinesia involves pharmacologic pain control. Intramuscular ketorolac (30 to 60 mg, as adjusted for age or renal function) in an emergent setting can usually ameliorate symptoms within 20 to 30 minutes.<sup>16</sup> The use of nonsteroidal anti-inflammatory drugs or ibuprofen (400 mg orally) can also provide analgesic benefit for typical biliary pain.<sup>16</sup> In the present case, magnesium was suggested for pain control, as magnesium blocks central neuronal excitation mediated by the *N*-methyl-D-aspartate receptor and can potentiate muscle relaxation.<sup>17,18</sup>

There is some thought that cholecystectomy can help relieve symptoms of biliary dyskinesia in patients with a low gallbladder ejection fraction, despite the lack of gallstones or sludge on imaging studies. However, a systematic review<sup>19</sup> revealed that the ejection fraction is not a reliable indicator of positive clinical outcomes in such patients.

### Nociception in Osteopathic Medicine

A crucial piece of an osteopathic evaluation is the structural examination of the patient to determine the presence of somatic or visceral dysfunction. Chemical mediators from underlying inflammation stimulate nociceptive nerve endings of local tissue, which send signals to the spinal cord and result in altered neuronal activity. Effects of en-

hanced or altered spinal outflow are manifested at the somatic and autonomic levels as somatic dysfunction. With the use of palpation, somatic dysfunction can be identified as tissue texture changes, decreased range of motion, asymmetry, or increased sensitivity of the affected area.<sup>20,21</sup> Although there are case reports of OMT used to manage disease processes such as functional dyspepsia, peptic ulcer disease, and gastroesophageal reflux disease, a review of the literature did not provide any examples of OMT for patients with biliary dyskinesia.<sup>22-24</sup>

Biliary dyskinesia represents a disease state involving abnormal contraction of the gallbladder in response to hormonal or neural stimulation. Primary afferent nociceptors (PANs) are the small-caliber fibers found, among other places, in the walls of viscera that are activated by tissue-damaging stimuli. Facilitation of the spinal cord by PANs can lead to altered output on the ventral roots, which contain both somatic efferent axons innervating skeletal muscle and visceral efferent fibers innervating smooth muscle of organ walls, as well as surrounding fascia.<sup>20,25</sup>

Excessive synaptic drive from the PANs can serve to facilitate the spinal cord coming from dysfunction related to either the soma, the viscera, or both. The sympathetic nerve supply of the gallbladder originates from the T6-T9 segments. In the present case, the somatic dysfunction findings of boggy tissue texture changes at T6-T9 and visceral dysfunction of altered sphincter motion resulted from facilitation of the spinal cord by PANs. The aim of OMT is to remove excessive synaptic drive from the PANs to restore the homeostatic condition of the system. Allostasis is a pathologic condition that results from activation of the neural, endocrine, and immune systems, which function together to help fend off disease states. Chronic exposure to the allostatic response can result in the gradual destruction of effective feedback pathways meant to reestablish homeostasis.<sup>26</sup> Visceral stressors, such as those suspected in biliary dyskinesia, are a strong drive on the allostatic mechanism. As in the case described herein, the patient received OMT to the areas of somatic dysfunction with an aim toward reestablishing a more natural homeostatic condition by defacilitating allostatic drive. This method repre-

sents a unique piece of the osteopathic approach to disease processes encountered in medicine. As noted, when the patient returned for reevaluation, the areas of facilitation were notably improved and the patient's symptoms had completely resolved.

## Conclusion

Diagnostic and therapeutic procedures for biliary dyskinesia, including medical, endoscopic, and surgical options, can be invasive and expensive. In addition to assessing the symptomatic complaints of the patient and following review of diagnostic laboratory studies and procedures, the osteopathic physician is taught to consider the structure and function of the system. The osteopathic approach is aimed at normalizing these processes by reducing allostatic load, thereby moving toward a more natural homeostatic condition. As exhibited in the present case report, this approach is both effective and efficient. Osteopathic manipulative treatment can and should be considered in the management of biliary dyskinesia.

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