Osteopathic Family Physician (2010) 2, 144-147

acofp Osteopathic Family Physician

Osteopathic manipulative treatment in a patient with idiopathic dysautonomia: a case presentation

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KEYWORDS:
Dysautonomia;
Facilitation;
Somato-visceral reflex;
Viscero-somatic reflex

Functional gastrointestinal disorders are a common ailment that often results in extensive and costly diagnostic workups as well as significant chronic suffering to the afflicted. The following is a case review and discussion of the use of osteopathic manipulative treatment in a patient with unresolved functional gastrointestinal ailments.

Disorders of the gastrointestinal (GI) tract as a whole affect one in three people. Of those who seek further investigation from a gastroenterologist, one in five will not reach an etiologic explanation of their symptoms.[1] Many of these patients fall into the category of "functional gastrointestinal disorders," which includes such diagnoses as irritable bowel syndrome and functional dyspepsia. These diagnoses are reached through extensive history given by the patient and require that "organic" etiologies be ruled out.[2] Because the functional GI disorders have no identified etiology, patient management consists primarily of supportive therapies directed at the patient's unpredictable symptoms. These patients often have conditions similar to many chronic disease patients, such as anxiety and depression. Many times they are frustrated and searching for support, so sufficient care requires tact, time, and patience.[1,3] A select subsets of patients can have additional unexplained symptoms spanning multiple organ systems, which has led some to believe that an autonomic etiology may be causing the viscera to function inappropriately.[2,4,5] Because of chronic autonomic stimulation of the viscera, there may be somatic segmental facilitation, resulting in a viscerosomatic reflex. This is exemplified by musculoskeletal complaints or somatic dysfunction at the same spinal levels as the visceral innervations. Osteopathic manipulative treatment can be used to interrupt the viscerosomatic reflex, resulting in improvement of both the visceral and musculoskeletal symptoms.

The following is a case of a patient who presents with a long history of unexplained GI, cardiovascular, and neurologic symptoms. Having had extensive investigations in neurology, cardiology, and gastroenterology, the patient was frustrated and looking for relief from his chronic symptoms.

Case report

A 48-year-old Caucasian male administrator presented with a nine-year history of vague GI disturbance, transient generalized fasciculations and paresthesias, and diffuse back pain. His GI symptoms included early satiety, sensation of bloating, abdominal cramping, nausea and vomiting, and increased flatus that occurred approximately 30 minutes after every meal and usually lasted for hours. The functional debilitation caused by the symptoms resulted in eating very little throughout the day. Over the previous decade, the patient had seen gastroenterologists at multiple institutions in which a battery of radiographic, endoscopic, and func-

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1877-573X/$ -see front matter © 2010 Published by Elsevier Inc.
doi:10.1016/j.osfp.2010.03.002

tional tests were performed, with no definitive diagnosis. Regarding the patient's neurologic symptoms, neurology had been consulted to rule out known organic etiology of his fasciculations and paresthesias. Studies included numerous magnetic resonance imaging scans, computed tomography scans, and electromyograms, all of which resulted equivocally. At the time of presentation, the patient's most severe back pain was sharp and located over his right sacroiliac joint, which he equated to a football injury 10 years prior; however, he stated that over the previous five years, his back pain had migrated between his cervical, thoracic, and lumbar regions in a waxing and waning manner for weeks at a time. His pain was aggravated by lifting, standing, or bending forward. Alleviators included heat, massage, mobility, physical therapy, and stretching. He did not relate any bladder or bowel incontinence, or saddle anesthesia.

The patient was an active, married individual who participated in both aerobic and strength training programs at least three to four times/week. He did not smoke tobacco or drink alcohol and there was no history of illicit drug use. He did, however, consume two cups of coffee every day, which he admitted was decreased from seven cups two years prior. His family was healthy, although he recalled that both his maternal and paternal grandmothers complained of GI disturbances similar to his. Past medical history included premature birth, mild sleep apnea, and postural orthostatic tachycardia syndrome (POTS). According to the patient, the cardiologist who originally diagnosed him with POTS referred to his condition as "dysautonomia," of which POTS was only a component. The patient had two right inguinal surgeries for an undescended testis, which resulted in orchiectomy.

The patient was taking three medications at the time of presentation. Ramelteon 8 mg at bedtime and temazepam 15 mg at bedtime were taken to aid sleep. Polyethylene glycol was used daily for constipation. Although he could not remember the names of the previous drugs he had taken for his condition, some information was gained on previous treatment. He had been on numerous antacids, both proton pump inhibitors (PPIs) and H2-antagonists, and pro-motility agents, which offered minimal relief. At one point, he remembered being prescribed an anticholinesterase agent—although he could not recall the name—that also gave minimal relief. In an attempt to identify a common food-based etiology to his GI disturbance, the patient had tried both gluten-free and lactose-free diets. Previous testing for celiac sprue had been negative; however, elimination of gluten protein from his diet had resulted in the most relief, although his symptoms were still functionally debilitating on a daily basis.

The review of systems showed few findings beyond the history of his present illness. At times the patient felt weak and had myalgias. These generally go hand-in-hand with the fatigue and lethargy associated with his sleep apnea. A five-system physical examination was within normal limits. Pertinent findings on neurological examination included no sensory loss, weakness, or abnormal reflexes. All cranial nerves were intact and deep tendon reflexes were equal bilaterally.

Osteopathic treatment was based on a find-and-treat approach, with the primary focus directed on key lesions given the patient's current complaints. Treatment techniques were chosen based on the response of the patient to the technique. Different modalities were used until key lesions were corrected. The only limitations were that high-velocity, low-amplitude techniques not be implemented based on patient preference. At the initial visit, the screening musculoskeletal examination showed tissue texture, asymmetry, restriction, and tenderness (TART) changes interspersed over the entirety of the spine and pelvis. Key lesions included: occipitolantal (OA) SrRl; L2-5 SlRr; right sacroiliac (SI) restriction; right piriformis spasm; left sacrotuberous ligament restriction; right iliolumbar (IL) tenderpoint. Treatment of these somatic dysfunctions was achieved through soft tissue, myofascial release, muscle energy, and ligamentous articular strain techniques. Soft tissue techniques included stretching, kneading, and inhibition directed at muscular and fascial structures. Myofascial release and integrated neuromuscular release are more specific soft-tissue techniques in which both direct and indirect approaches are used to engage static and dynamic barriers in three dimensions to reflexively release restriction patterns. Muscle energy techniques are patient interactive techniques, where the patient activates specific muscle groups against the physician's counterforce to mobilize restricted joints.[6] Ligamentous articular strain techniques disengage injured tissues through either compression or decompression. The injured tissues are carried into the original position of injury and maintained at a balance point until the tissues release and return to their original functional position.[6,7]

The second office visit showed the patient pleased with treatment for his low back pain, given vast improvement. However, he relayed that the pain was now focused in his midthoracic area as well as at the base of the occiput. He had not seen noticeable improvement of his GI symptoms at that time. Key lesions included: C3 SrRr; T3-4 SrRl; T10-12 SrRl; bilateral sacrotuberous ligament restriction; bilateral SI joint restriction; left innominate anterior. Treatment of these somatic dysfunctions was achieved through soft tissue, myofascial release, muscle energy, and ligamentous articular strain techniques.

At the following office visit, the patient stated that he was doing well. His GI symptoms, although not absent, were the least problematic they had been in a number of years. However, he noted that his low thoracic back pain and neck pain, although better for a number of days, seemed to have returned. He located his thoracic pain at T10-L1 and described it as a tight band that wrapped around his flanks. Key lesions included: OA SrRl; C2-5 RlSl; C6 RrSr; T9 SlRr; T10 FBSrRr; T11-L3 SlRr; left SI restriction; superior mesenteric ganglion (SMG) and inferior mesenteric ganglion (IMG) tenderness and restriction. Treatment of these somatic dysfunctions was achieved through muscle energy, articulatory, ligamentous articular strain, and integrated

neuromuscular release techniques. Articulatory techniques directly engage the restrictive barrier by repetitively forcing the implicated tissues through their range of motion until the barrier is reduced.[6] It was notable that while performing myofascial release on the patient's exquisitely tender SMG and IMG, pain began to radiate back through the band of tightness previously described by the patient, resulting in significant bilateral paravertebral spasm at that level. Once the myofascial structures were restored to resting function, the pain and spasm in the low thoracic spine abated.

On the fourth office visit, the patient relayed that both his back pain and visceral symptoms were vastly improved initially. Unfortunately, as time progressed, his visceral symptoms and neck pain began to return. Key lesions included: OA SrRl; C3-6 RlSl; T9-12 SlRr. Treatment of these somatic dysfunctions was achieved through muscle energy, myofascial release, ligamentous articular strain, and facilitated positional release techniques. Facilitated positional release techniques are indirect. The region of somatic dysfunction is placed in a neutral position of flexion or extension and the tissues are positioned at the point of ease. An activating force is then applied to shorten the restricted tissues and facilitate further release.[6] It was notable that all dysfunctional findings were palpably decreased from initial dysfunction treated in the clinic. Cervical somatic dysfunction showed the most significant TART changes, which even still were decreased from initial findings.

Over the course of his visits at the clinic, this patient experienced vast improvement of both his visceral and musculoskeletal symptoms. Although he experienced improvement, because of the intermittent nature of his condition, he must follow up for symptomatic management.

Discussion

Previous studies have shown that disorders of the autonomic nervous system innervating the gut manifest as GI dysmotility, and patients who have functional GI disorders also show autonomic abnormalities extending beyond the GI tract.[2,4,5] Other areas showing autonomic dysregulation include cardiovascular, pulmonary, neurologic, and integumentary systems.[2,5] Autonomic instability has even been correlated with increased incidence of musculoskeletal abnormalities such as scoliosis and kyphoscoliosis.[8,9] Although correlations have been drawn, the role of autonomic dysfunction and GI dysmotility in the development of functional GI disorders remains uncertain. Autonomic dysfunction as a whole is most commonly secondary to other known disease processes including diabetes, autoimmune disorders, amyloidosis, renal failure, and liver failure.[10,11] Rarely, however, autonomic neuropathy occurs as a primary disorder, particularly in the GI tract. Unfortunately, because of the vague symptoms associated with autonomic dysfunction in the GI tract, as well as the difficulties of directly testing autonomic function, it is unlikely that the autonomics be used as a diagnostic target for identifying functional GI disorders.[10] Although this correlation may not be useful diagnostically, appropriate application of osteopathic principles could prove helpful in the symptomatic management of the patients' disease process.

The concept of facilitation of spinal segments is integral in the basis of many osteopathic modalities of practice. It has been shown that areas of the spinal cord receiving chronically elevated stimulus via primary afferent nociceptors (PANs), somatic or visceral, results in the facilitation (sensitization) of the surrounding nerves both afferent and efferent. Stimulation of the involved somatic pathways has demonstrated masking of the visceral perception of these facilitated segments, indicating established somatic and visceral pathways.[12] In addition, this has been evidenced by elevated sympathetic activity in dermatomes corresponding with associated myofascial irritation or musculoskeletal abnormalities.[13,14] Once the facilitation has occurred, hyperirritability of these segments remains despite removal of the inciting PAN stimulus and my be easily reactivated through normal activity.[15-19] Areas of segmental facilitation reveal themselves palpably as "somatic dysfunction," represented by TART changes. These palpable changes result in deformation of involved nerves through compression, torsion, stretching, and angulation. Because nerves are trophic to their innervated tissues, disruption of these mechanisms would alter normal structure and function.[20,21] If somatic dysfunction is restored to its resting state, normal feedback between visceral and somatic afferents and efferents may be restored.[17] The restoration of the deformed tissues, and therefore normal nerve function, can be achieved through the application of various osteopathic manipulative treatments. Of course, if the initial cause of increased PAN stimulation is not removed, it is likely that the facilitated segment will return. However, in the instance of patients with primary dysautonomia, the etiology is unknown. Therefore, osteopathic treatment may be applied as regular management to balance the autonomic dysfunction and minimize facilitation and therefore visceral symptoms.

Like many patients at presentation, the initial assessment of the patient presented here reflects a lifetime of injuries and compensation, making decisions in treatment order and progression uncertain. Because corrections in specific dysfunctional segments will result in functional compensation, the physician may find unpredictable changes exposed at subsequent visits, much as one peals away the layers of an onion. Such went the treatment of our patient. The initial assessment showed the primary dysfunction to be low back pain from previous trauma, but once resolved, subsequent visits revealed classic patterns relating his visceral and somatic manifestations to abnormal autonomic function.

Much like dermatomes of the skin, all viscera has predictable segmental innervation from the autonomic nervous system. With the chronic GI disturbance, one could predict to find facilitation anywhere from T5-L2, representing sympathetic innervation, or at the occipito-atlantal junction and upper cervical vertebrae, representing parasympathetic in-

nervation.[22] This patient showed somatic dysfunction ranging from T9-L3, which on the third visit was found to correlate significantly to facilitation located at the SMG and IMG. Chronic facilitation of these segments suggests visceral disease ranging from the lower portion of the stomach to the right colon and correlates with his constellation of GI symptoms. Over the course of four office visits, we were able to identify and reduce somatic dysfunction most associated with his symptoms while offering modest improvement of both his chronic musculoskeletal and visceral complaints. Currently, he is managed intermittently when his symptoms flare up. Although manipulative treatment did not cure his problems, it offered relief that could not be found with other treatment modalities.

Conclusion

Among the population of those diagnosed with a functional GI disorder, there lies a subpopulation of patients with idiopathic dysautonomia. In addition to their GI complaints, numerous findings consistent with autonomic dysregulation span multiple organ systems. Although the etiology of most functional GI disorders is unknown, it is possible that in a subpopulation of patients it is the result of primary dysautonomia. By treating somatic dysfunction that results from chronic segmental facilitation, autonomic balance may be restored, thereby offering relief from visceral complaints. Like many patients with functional GI disorders, their symptoms often require lifestyle changes that are frustrating. Often times they have bounced from physician to physician and specialty to specialty searching for effective treatment for their unexplainable condition. Because of this difficulty, any consistently successful means to improve their symptomatic disease will be received with great appreciation.

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