

Introduction

One well-known complication of non-steroidal anti-inflammatory drug (NSAID) overuse is gastric ulcer formation and perforation. Perforation is a surgical emergency with a mortality rate of 30%¹. The involved inflamed viscera produce viscerosomatic reflexes, which cause palpable changes in the musculoskeletal system that can be diagnosed on an osteopathic structural exam (OSE)²⁻¹³. This case report describes a patient with a history of back pain and peptic ulcer disease (PUD) who presented after an ulcer perforation due to NSAID use. She had multiple prior medical visits for worsening mid-back pain and in these visits, recorded musculoskeletal exams were absent or lacking. An OSE performed post-operatively revealed findings that correlated with the patient's visceral pathology. We hypothesize that a proper OSE performed in earlier visits may have altered this patient's clinical course.

Case Description

History of Present Illness (HPI): A 64 year-old female with medical history as below and multiple recent medical visits for worsening back pain, presented to the Emergency Department (ED) with chief complaint of abdominal pain. The abdominal pain was severe and radiated to her back. She described a recent history of increased NSAID use. Imaging (see table 1) suggested a perforated gastric ulcer. The patient was taken for emergent exploratory laparotomy with graham patch repair of a 7mm perforated gastric ulcer near the pylorus of the stomach. The inpatient Osteopathic Manipulative Medicine (OMM) service was consulted by the Surgical Intensive Care Unit to optimize recovery of this patient in the postoperative setting. At the time of evaluation by the OMM team, the patient was lightly sedated and remained intubated secondary to concerns for her underlying interstitial lung disease (ILD).

Visit	Presenting Complaint	Findings/Intervention
30 days prior	Primary care office: Unspecified back pain	No musculoskeletal PE findings; Prescribed baclofen, naproxen, gabapentin
17 days prior	ED: "my doctor has given me some medication for my back but it is getting worse the last three days"	PE: Lumbar spine: No erythema, swelling, warmth or tenderness; Lumbar XR WNL; Directed to continue Naproxen and Prednisone
12 days prior	Primary care office: Back pain "worse than before"	No musculoskeletal PE findings; Prednisone dose increased, and given "short course of narcotics due to risk for NSAIDS"
Hospital day (HD) #0	ED: Severe abdominal pain radiating to back	CTA: pneumoperitoneum; Left upper quadrant inflammatory changes concerning for gastric perforation; Emergent exploratory laparotomy with graham patch repair

Table 1. Pre-admission history with medical exams and treatment.

Past Medical History: Chronic back pain, PUD requiring hospitalization one year prior for coffee ground emesis, rheumatoid arthritis, ILD diagnosed five months prior and requiring home oxygen and chronic corticosteroid use, hypertension, osteopenia, anemia.

Home Medications: Baclofen, Naproxen, Gabapentin, Prednisone and Plaquenil. Not using home oxygen or Plaquenil due to lack of insurance coverage.

Surgical History: Cesarean section x1, bilateral knee replacements.

Social History: Former 5 pack-per-year smoker, owned parakeets for 10 years.

Physical Exam (POD #1): Vitals: HR:90 BP:110/58 RR:24 Temp:98.5 O2 sat:99%

General: Intubated, lightly sedated on mechanical ventilation, cooperative.

Nose/Throat: Nasogastric tube with scant succus entericus, endotracheal tube in oropharynx.

Cardiovascular: RRR, no murmurs.

Pulmonary: Breath synchronous with ventilator, coarse breath sounds with rales diffusely, no wheeze or crackles.

Gastrointestinal: Abdomen obese, soft, non-distended; Surgical dressing from epigastrium to umbilicus, clean, dry; Jackson-Pratt drain with <5cc of serosanguinous fluid; hypoactive bowel sounds, + peri-incisional tenderness.

Region	Finding	Treatment
Head	Prolonged cranial extension phase, sphenobasilar synchondrosis compression	Balanced (BMT) membranous tension
Cervical	Bilateral paravertebral muscle (PVM) hypertonicity, Cervicothoracic junction FRSR	Myofascial release (MFR)
Thoracic	Left > right thoracic inlet fullness; Left > right T5-9 dry, ropy, hypertonic PVM with superimposed boggy of overlying tissues greater on right side, +tenderness to palpation bilateral T5-9	Balanced Ligamentous Tension (BLT), MFR, Thoracic inlet release
Lumbar	Decreased lumbosacral compliance, L5FRSL	BLT, MFR
Sacrum	ROR sacral torsion, decreased inherent motion	BMT
Pelvis	Right anterior innominate, pelvic diaphragm inhaled and with decreased amplitude of Primary Respiratory Mechanism	BLT, MFR
Ribs	Globally inhaled thoracic cage, reduced excursion and compliance, severe sternal fascial strain	BLT, MFR
Abdomen	Diaphragm inhaled bilaterally and severely restricted with significantly increased tension at crura. non-physiologic breathing pattern, notable abdominal congestion	BLT

Table 2. Osteopathic Structural Exam findings and treatment approach, HD #1/POD #1

Assessment and Plan

64 year-old female with gastric perforation, s/p emergent exploratory laparotomy with graham patch repair of a 7mm perforated gastric ulcer, POD#1.

The patient's OSE was notable for tenderness to palpation in the mid-thorax with severe, acute-on-chronic, somatic dysfunction and thoracic segmental facilitation. Also present were severely reduced excursion and compliance of the thoracic cage and diaphragms, with notable abdominal and supraclavicular congestion in an overall respiratory/circulatory pattern.

Gentle Osteopathic Manipulative Treatment (OMT) was applied to reduce acute thoracic segmental facilitation, facilitate lymphatic drainage, improve respiratory mechanics and promote Primary Respiration. See Table 2 for specific treatment approaches.

Hospital Course

After the first treatment the patient's diaphragm and thoracic cage excursion were noticeably improved. She was successfully extubated later that day. OSE on POD#2 was notable for reduced thoracic inlet fullness, significant improvement to acute thoracic spine reflexes and an overall reduction of inflammation, revealing underlying chronic, ropy, dry PVMs bilaterally in T5-9. It also revealed chronic cranial base and cervical spine restrictions, an exhaled thoracic cage, a severe sternomanubrial inhalation strain, and severely decreased lumbosacral compliance.

After OMT #2, the patient's breathing became less labored and she reported that breathing felt easier.

On POD #4, an upper gastrointestinal study was performed and no extravasation of oral contrast was observed. The patient reported passing flatus and bowel movements. A Pulmonologist deemed the patient was at her baseline respiratory function. She declined further OMT and was discharged from the hospital shortly after.

Discussion

Innervation to the stomach arises from the spinal roots of the 5-9th thoracic vertebral segments, primarily on the left^{1-3,5-7,9,11,14,15}. Nociceptive input from visceral afferent fibers enter the dorsal horn of the spinal cord to synapse with spinal interneurons in the upper gray matter^{2,3,5,10,13,15}. There, reflexive activation of corresponding regional sympathetic efferent and alpha motor activity occur^{2,3,5,10,13,14}.

- Sympathetic activation of the gut decreases motility, blood supply and secretion of bicarbonate, making the stomach susceptible to injury from chemical insult and changes in pH^{3,14}. Vasomotor, sudomotor, and pilomotor effects on the skin occur as well, leading to sweat gland activation and changes in temperature and skin texture¹⁴.
- Alpha motor activation stimulates regional paraspinal muscles to contract, leading to muscle spasm and pain sensitization^{10,13}.

Persistent visceral disease causes continuous activation of these reflex pathways, lowering the threshold of excitation and maintaining an exaggerated neural response to otherwise normal stimuli^{5,13,14}.

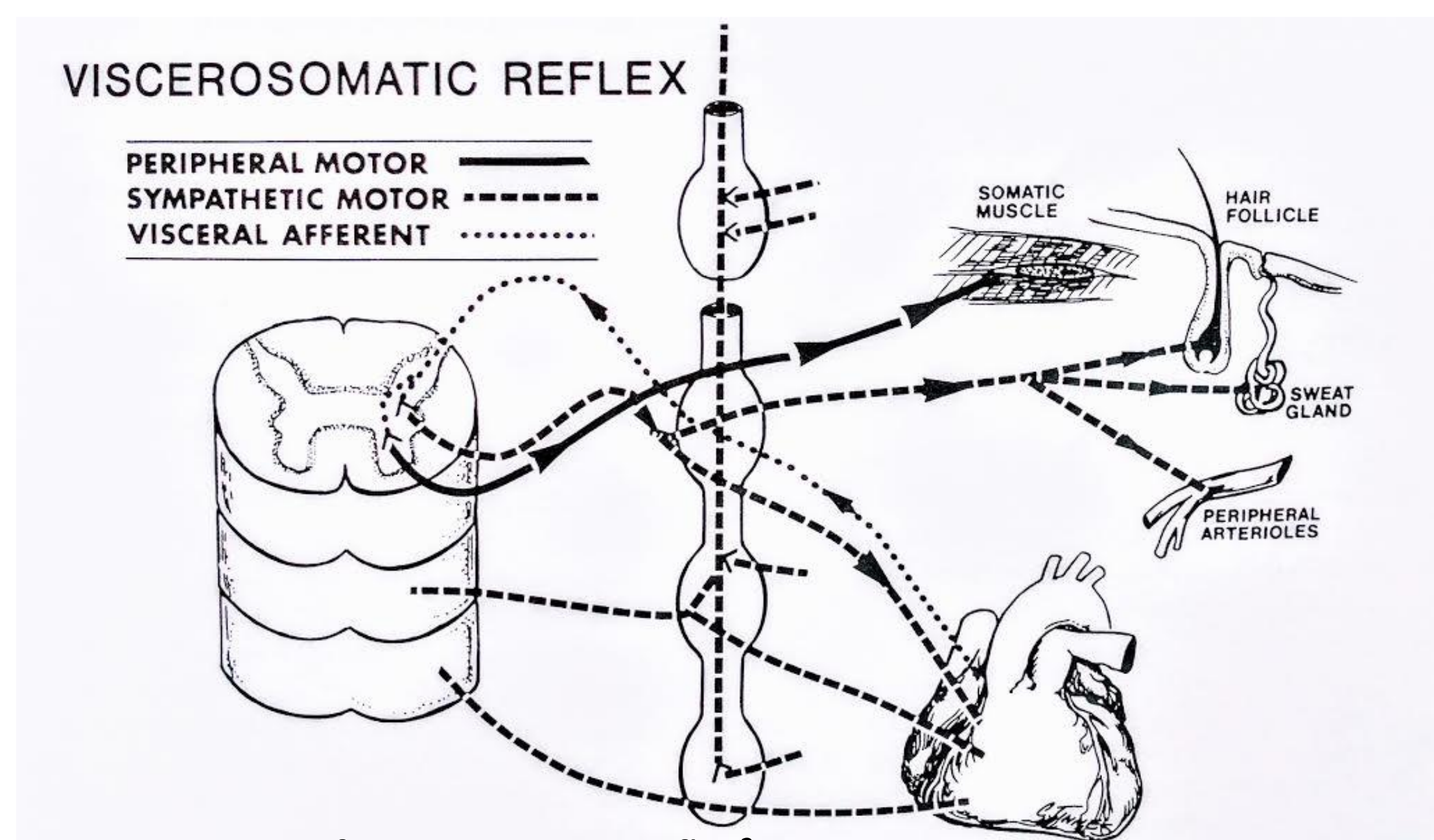


Figure 1. Representation of viscerosomatic reflex².

Viscerosomatic reflexes cause palpable phenomena in the musculoskeletal system that are well-documented in osteopathic literature²⁻¹⁵. Studies show that the number of segmental somatic findings increase in the presence of underlying visceral disease^{6,15}. Careful palpation allows for discernment of chronicity of visceral disease and can help clinicians distinguish this type of pathology from primary vertebral lesions^{4,6,14}. Warm and boggy tissue texture changes are found in acute reflexes, while cool and ropy tissue textures indicate a chronic underlying condition. In a viscerosomatic reflex, superficial tissues are typically hypertonic and often hypersensitive to touch¹⁶. Primary vertebral lesions are typically found in deeper spinal muscles and superficial muscles, if involved, are less seriously contracted and lack hypersensitivity¹⁶.

In this case, signs of acute-on-chronic facilitation were found on post-operative OSE in the T5-9 region, corresponding to sympathetic innervation of the stomach and consistent with the patient's history of PUD. In light of this relationship, it is likely that her initial back pain was due to a viscerosomatic reflex. Using the OSE to distinguish visceral from somatic etiology of pain during initial visits may have altered her outcome.

The OSE has long demonstrated its reliability in the osteopathic toolkit. Its adoption and implementation as a standard of care by all medical practitioners would benefit physicians and patients alike. Appreciating the relationship between structure and function enables better discernment of what maladies lay hidden below the patient's surface²⁻¹³ and may also lead to more appropriate selection of therapeutic modalities^{2,7}.

Limitations of this case report include multiple chronic co-morbidities in this patient that may have confounded the OSE findings. Future areas of research may include investigation into how targeted therapies, including OMT, effect change in OSE findings and whether these correlate with resolution of patient symptoms.

Contact

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