Osteopathic Manipulative Treatment for Patient With Severe Nausea and Vomiting in Pregnancy: A Case Study

Katherine Anne Markelz, OMS IV, and Janice Upton Blumer, DO

Abstract
A 27-year-old white female patient with unremarkable medical history who was experiencing her first pregnancy visited an osteopathic manipulative medicine clinic complaining of 8 weeks of progressively worsening severe nausea and vomiting in pregnancy (NVP). An osteopathic structural examination revealed multiple areas of somatic dysfunction, including cervical, thoracic, pelvic, and cranial diaphragms. Osteopathic manipulative treatment (OMT) was performed based on somatic dysfunction, after which the patient reported resolution of nausea and vomiting. Based on the Pregnancy-Unique Quantification of Emesis and Nausea (PUQE) index and the patient's Hyperemesis Impact of Symptoms Score (HISS), the patient reported 50% and 41% decreases of nausea and vomiting, respectively, after the first treatment and 58% and 68% overall decreases, respectively, after the second treatment. This case study suggests that OMT is a valuable treatment for patients with severe NVP.

Keywords
osteopathic manipulative treatment, hyperemesis, nausea, pregnancy, diaphragm, adjunct therapy

Introduction
In the United States, nausea occurs in up to 70% of women during pregnancy. While not well documented, severe nausea and vomiting occur in up to 1.3% of patients who are pregnant.¹

Severe nausea and vomiting in pregnancy (NVP) can negatively impact both the mother and the child. Mothers often admit to a lower perceived quality of life and a depressed or anxious mood. In a cohort of women with moderate to severe NVP, 47% reported possible anxiety, 11.4% were depressed in the last week of pregnancy, and 36% had a history of depression.² During pregnancy, patients may suffer from dehydration, electrolyte imbalances, malnutrition, and anorexia. If severe enough, hyperemesis may cause preterm delivery, low birth weight, low 5-minute Apgar scores, and fetal or congenital anomalies.³

The mainstay of treatment for patients with NVP is supportive and pharmacologic therapies. Initial therapy often involves dietary modifications, trigger avoidance, acupressure, and complementary alternative medications such as ginger and vitamin B₆. Acupressure has not been demonstrated to have significant efficacy compared to sham treatment for pregnant women with severe NVP.⁴ Some small studies have shown reduction of symptoms in NVP with ginger compared to placebo, but such evidence is limited.⁴

Primary pharmacologic therapy includes doxylamine succinate (10 mg) and pyridoxine hydrochloride (10 mg).³ Secondary therapy includes antihistamines, metoclopramide, ondansetron hydrochloride, phenothiazines, and corticosteroids, provided patients are past the first trimester. However, these treatments have limited efficacy, and they are further limited by adverse effects. Antihistamine use is often limited by sedation, especially in high doses. Metoclopramide increases the risk for tardive dyskinesia and serotonin syndrome. Ondansetron has been associated with increasing the interval between the Q and T waves on electrocardiograms, and current investigations are studying whether

(continued on page 14)
odansetron poses risks for birth defects such as cleft palate. To date, little attention has been given to the musculoskeletal manifestations of NVP. It is well known that pregnant women undergo many structural changes. These include ligamentous laxity, thoracic and pelvic diaphragm dysfunction, and passive tissue congestion. Often women are able to compensate for these changes by modifying their posture or their daily activity. However, when these patients’ structural integrity is stressed by repetitive, forceful vomiting, their compensatory patterns are easily altered, causing somatic dysfunction. The case presented here exemplifies how such somatic dysfunction may perpetuate clinical symptoms in NVP, especially as pertains to the neurological, respiratory and circulatory models.

Report of Case

History of Present Illness
A 27-year-old white female patient experiencing her first pregnancy visited an osteopathic manipulative medicine clinic complaining of severe nausea and vomiting. The patient was approximately 14 weeks pregnant, and she had experienced an overall weight loss of 5 lbs. In the initial interview, the patient described her nausea as a 9 on a 10-point scale, and she reported vomiting throughout the previous night. The patient also reported difficulty falling asleep and excessive sleepiness after waking (initial and terminal insomnia).

The patient stated that she had been struggling with nausea and vomiting since week 6 of her pregnancy. The episodes began once per day, but by 8 weeks, she was vomiting 7 to 10 times per day. The patient stated that she “could not keep anything down, not even water.” By 8 weeks, she had lost 5 lbs. On the recommendation of her obstetrician, she tried the natural remedy ginger, but it did not relieve her symptoms.

When she was 11 weeks pregnant, the patient sought pharmaceutical therapy. She received a prescription for 2 delayed-release tablets of the combination of doxylamine succinate (10 mg) and pyridoxine hydrochloride (10 mg) daily at bedtime for 3 weeks. At the same time, the patient began taking 4 mg of ondansetron orally in the morning and at bedtime. This protocol allowed her to tolerate crackers, cereal, soup, salad, and broth, and she began gaining weight.

At week 12, the patient began taking Unisom to relieve insomnia. With this pharmacologic intervention, she was able to sleep for 7 to 8 hours each night, averaging 1 waking episode per night.

The Pregnancy-Unique Quantification of Emesis and Nausea (PUQE) index and the Hyperemesis Impact of Symptoms score are two validated clinical tools for assessing the severity of NVP and

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motor deficits were present. The patient exhibited a steady gait with normal heel-to-toe strike and smooth, coordinated turns. A Romberg test was negative.

A musculoskeletal examination showed full range of motion with good tone and without tenderness in the upper and lower extremities bilaterally. Osteopathic somatic dysfunction was as follows:

- Cranial: Internal rotation of bilateral temporal bones with decreased cranial rhythmic impulse (CRI).
- Cervical: Occipito-atlantal joint flexed, sidebent right (sideslip right), rotated left.
- Suboccipital: Generalized hypertonicity, fourth cervical vertebrae flexed, rotated right, sidebent right (sideslip right).
- Thoracic: T1-4 and T8-9 hypertonicity in bilateral paravertebral muscles, thoracic diaphragm and thoracic inlet exhalation somatic dysfunctions (restricted in inhalation).
- Pelvic: Left anteriorly rotated innominate, exhaled pelvic diaphragm.
- Sacral: General reduced motion and bilaterally flexed sacrum.

Assessment
A 27-year-old white female with worsening severe nausea and vomiting during her first pregnancy had somatic dysfunctions of the cranial, cervical, suboccipital, thoracic, pelvic, and sacral regions.

Treatment
The patient was treated supine for the duration of the session (see Table). Her cranium was treated with balanced membranous tension (BMT) technique. Cervical treatments included atlantooccipital decompression, fourth cervical vertebrae high-velocity/hyperextension (HVT), and suboccipital myofascial release.

Patient History
The patient reported a predisposition to dehydration and heat stroke as a young child. The patient reported no other significant medical history. Past surgical intervention was limited to removal of third molars. Regular medication included prenatal vitamins. The patient has a family history of morning sickness (mother) and gestational hypertension (sister). There is no family history of diabetes mellitus. The patient lives with her husband, and she reported having a healthy, well-balanced diet and exercising regularly prior to the onset of NVP. The patient denied using tobacco products and alcohol.

Review of Systems
A review of the patient’s systems uncovered generalized fatigue, tension headaches, and constipation. The patient denied having muscle weakness, chest pain, shortness of breath, and depression. All other systems were negative.

Physical Examination
The 27-year-old female patient weighed 140 lbs, and she was 5 ft tall. She was well nourished and well dressed, and she was not in acute distress. She was alert, oriented, and cooperative. She appeared fatigued and was hunched over in discomfort. Her pupils were equally round and reactive to light. Extraocular movements were intact. A cardiovascular examination revealed normal pulses and good capillary refill. A respiratory examination showed good inspiratory effort. The patient’s gravid abdomen was appropriate to her due date. Cranial nerves II-XII were grossly intact bilaterally. No sensory or motor deficits were present. The patient exhibited a steady gait with normal heel-to-toe strike and smooth, coordinated turns. A Romberg test was negative.

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Table. The table below summarizes the structural diagnoses made and the osteopathic manipulative treatment techniques performed on the patient’s first visit.

<table>
<thead>
<tr>
<th>Region</th>
<th>Diagnosis</th>
<th>Technique</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cranium</td>
<td>Internal rotation of temporal bones and cranial extension somatic dysfunction</td>
<td>Balanced membranous tension</td>
</tr>
<tr>
<td>Cervical</td>
<td>C4 FR S4, OA FR S4, B/L suboccipital hypertonicity</td>
<td>High-velocity/low-amplitude to C4, balanced ligamentous tension, and suboccipital myofascial release</td>
</tr>
<tr>
<td>Thoracic</td>
<td>Thoracic inlet ESD, thoracoabdominal diaphragm ESD, and B/L hypertonicity (T1-4 and T8-9)</td>
<td>Balanced membranous tension</td>
</tr>
<tr>
<td>Pelvis</td>
<td>Pelvic diaphragm ESD</td>
<td>Lymphatic pelvic diaphragm release</td>
</tr>
</tbody>
</table>

C=cervical, F=flexed, R=rotated, S=sidebent, OA=occipito-atlantal joint, B/L=bilateral, ESD=exhalation somatic dysfunction, T=thoracic.
low-amplitude technique, and suboccipital release. The patient’s thoracic inlet and thoracoabdominal diaphragm were treated with BMT technique and myofascial release. The pelvic area was addressed with lymphatic pelvic diaphragm release bilaterally. The treatment concluded with an expansion of the fourth ventricle (EV4) technique.

Follow-up
At a follow-up visit 1 week after treatment, the patient reported that her initial nausea tapered off for 4 hours following treatment. The patient said that she “settled into her own body.” Although she was still taking ondansetron hydrochloride, she began to question whether she needed it. Between the initial treatment and the follow-up visit, she found she could go 3 to 4 hours in the morning without taking ondansetron hydrochloride.

The patient reported a significant increase in food intake 2 days following treatment. She dined out and ate items such as hummus, pita bread, hash browns, and eggs without vomiting. At the time of the follow-up visit, the patient had not vomited since the initial treatment. She continued to have bouts of nausea, but the severity decreased to 1 on a 10-point scale.

In addition, the patient’s activity level increased substantially. Two days after treatment, the patient traveled 90 minutes to Portland, Oregon, and walked around the city.

The patient also reported dramatically improved sleep. Following treatment, she was able to sleep 12 hours per night without waking.

At a follow-up visit 3 weeks after her first treatment, the patient reported she had tapered her use of ondansetron hydrochloride from 2 mg to 1 mg 2 days after the last treatment, and she reported infrequent nausea. The patient reported a total of 2 episodes of vomiting since her first treatment. Both episodes occurred in the evening on days in which the patient reported having overexerted herself at work. The patient reported less impairment after vomiting, without experiencing shaking or systemic fatigue as with prior episodes.

The patient continued using Unisom regularly.

At the 1-week follow-up visit, the patient scored 6 out of 16 on the PUQE index and a 10 out of 30 on HISS.

On second examination, the patient was smiling, talkative, well nourished, and well dressed, and she was not in acute distress. Although a physical examination revealed no significant change in findings, an osteopathic examination revealed improvement of somatic dysfunction as follows:

- Cranial: Good CRI throughout. Temporal restriction resolved.
- Thoracic: Hypertonicity of paravertebral musculature L>R, T1-T4. Hypertonicity bilateral (B/L) thoracolumbar junction, R>L.
- Pelvic: Inhalation somatic dysfunction of the pelvic diaphragm on the left. Exhalation somatic dysfunction of the pelvic diaphragm on the right.

Treatment consisted of BMT of the thoracic inlet and thoracoabdominal diaphragm, as well as lymphatic pelvic diaphragm release. Lastly, EV4 technique was performed.

At the 3-week follow-up visit, the patient scored 5 out of 16 on the PUQE index and 5.5 out of 30 on HISS (see Figure).

The patient reported that her constipation had completely resolved following her second treatment. In addition, although she had occasional tension headaches, they quickly resolved with water intake.

Following the initial treatment, the patient continued taking Unisom for sleep, and she regularly slept 10 to 11 hours overnight without nighttime awakenings.

Figure. The figure below shows the trend of the Pregnancy-Unique Quantification of Emesis and Nausea (PUQE) index and the Hyperemesis Impact of Symptoms score (HISS) throughout the patient’s osteopathic care.
Discussion
This case illustrates the benefits of osteopathic manipulative treatment (OMT) for women with NVP. Two 30-minute treatment sessions were associated with dramatically reduced symptoms and improved quality of life.

When caring for patients with NVP, the first model to consider is the neurologic model, which explains the connection between structure and neurologic function. In the upper gastrointestinal system, parasympathetic vagal afferents are responsible for carrying information about gastric electromechanical events to the area postrema and nucleus tractus solitarius. These areas interpret sensations as either normal or symptomatic, the latter of which may cause nausea. Sympathetic afferents mediate nociceptive stimuli and travel to the hypothalamus, where antidiuretic hormones and corticotropin-releasing hormones can be released. These hormones can then activate a vomiting reflex.\textsuperscript{11}

In this case, the authors postulate that somatic dysfunction and neurologic function were intimately related. The patient's somatovisceral reflex appears to have been that somatic dysfunction altered her neurological input, output, and reflexes, thus perpetuating her nausea and vomiting. As seen in this case, vomiting often leads to compression of the cranial base, resulting in impingement of the vagus nerve. This compression or impingement can lead to ineffective closure of the lower esophageal sphincter.

Additionally, increased tissue texture variations in the fascia and connective tissue surrounding neural bundles, such as the celiac ganglion, can alter neural signaling. This alteration may lead to improved function throughout organ systems and, ultimately, decreased pain.\textsuperscript{12}

In our patient's case, BMT applied to the suboccipital and thoracocaudal diaphragm contributed to decreased nausea and vomiting. We hypothesize that this was in part secondary to facial and connective tissue relaxation and unwinding that resulted in a more balanced neural input to the epigastric region. The patient's hypertonicity in the suboccipital region B/L was likely a manifestation of the parasympathetic component (occipito-atlantal joint, atlanto-axial joint) of a gastroesophageal viscerosomatic reflex, while her hypertonicity at T8-9 was likely a manifestation of the sympathetic component (T6-10) of the same gastroesophageal viscerosomatic reflex.\textsuperscript{7,13}

After considering the neurologic model, it is important to consider the respiratory and circulatory models. The body has 8 diaphragms, 7 of which serve as transition zones in a transverse plane parallel to the thoracocaudal diaphragm. Ideally, these diaphragms work in synchronization, propelling circulatory and interstitial fluid throughout the body. Sometimes the diaphragms can become tight or restricted, which can lead to diaphragms being out of phase with one another or becoming restricted in the exhalation or inhalation phase of respiration. With emesis, fluid is forced upward from the stomach, up the esophagus, and out of the body. This repetitive, forceful superior vector often causes exhalation somatic dysfunction in transition zones. The authors hypothesize that this is what the patient in this case experienced. By individually addressing each major diaphragm, the authors were able to offer significant relief.

The mechanism contributing to NVP is twofold. First, decreased arterial flow to the tissue limits the delivery of nutrients. Second, stagnancy in lymphatic and venous return results in the buildup of metabolic waste products, which can manifest as pain and discomfort. In this case, the buildup manifested as nausea and vomiting. Using OMT to remove these obstructions to flow restores hemodynamics.\textsuperscript{14,15}

By addressing the neurologic, respiratory, and circulatory models with OMT, the authors helped resolve the patient's symptoms and encouraged her body's inherent ability to heal itself.

Conclusion
Severe NVP negatively impacts women's physical, psychological, and social well-being. Currently, there are limited safe and effective treatment options for these patients. Osteopathic manipulative treatment may be an additional treatment that will benefit standard treatments such as pharmacological and supportive care for patients with severe NVP.

The patient in this case did not meet the Fairweather's criteria for hyperemesis gravidarum,\textsuperscript{16} which include ketonuria and severe electrolyte disturbances requiring hospitalization. But several of her symptoms, including severe vomiting, weight loss, and disability, are associated with hyperemesis gravidarum. Therefore, the authors believe this treatment protocol would benefit patients with hyperemesis gravidarum as well.

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Levodopa-induced dyskinesia (continued from page 23)

Conclusion
To the authors’ knowledge, this is the first published clinical observation of using OMT to manage a patient for LID. Although further study is warranted, the potential for using OMT as an adjuvant treatment for patients with motor control symptoms associated with PD is encouraging, especially in light of an increasing number of studies on the topic. The authors do not claim that OMT will cure either PD or LID, but they maintain concerns over the reliability and replicability of the effects of OMT in other cases. Further studies may do well to look at the effects of specific techniques on the various symptoms of PD and LID.

References

Severe nausea and vomiting in pregnancy (continued from page 18)


View From the Pyramids (continued from page 5)

References