Osteopathic Manipulative Treatment for Somatic Dysfunction After Acute Severe Traumatic Brain Injury

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Somatic dysfunction caused by traumatic brain injury (TBI) may be managed by osteopathic manipulative treatment (OMT). In this case report, the authors describe 2 patients with severe TBI who were each treated with OMT in a level-1 regional trauma center. Both patients received OMT beginning in the acute care phase of injury. Somatic dysfunction improved during the course of treatment, and no adverse effects of OMT were noted. More comprehensive research may clarify the efficacy and adverse effects of OMT as part of multimodal acute care of patients with severe TBI.

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The Centers for Disease Control and Prevention estimates that of the 1.4 million people who sustain traumatic brain injuries (TBIs) annually, 235,000 are hospitalized and 50,000 die.[1] The total number of TBIs in the United States has increased by 58% over the past decade, which leads to steadily increasing health care expenditures.[2] A multimodal approach is recommended for treatment and rehabilitation of patients after TBI, though the most effective combination of modalities has yet to be determined.[3]

Severe TBI may result in cranial and extracranial somatic dysfunction. Historically, acute severe TBI is considered a potential contraindication to OMT[4]; thus, research regarding the potential benefits and risks of OMT in this setting are lacking.[5,6] However, our regional trauma center supports an osteopathic manipulative medicine/neuromuscular medicine residency program that provides OMT for patients with injuries. Physicians in this program are regularly consulted by the trauma surgical service to provide OMT for the acute care of patients with severe TBI.

We describe 2 patients with severe TBI who were treated with OMT as a consistent part of acute inpatient multimodal care. To our knowledge, this is the first published description of OMT for managing acute severe TBI. We aim to stimulate interest in furthering the research into OMT for TBI-related somatic dysfunction.

Institutional review board approval was obtained from SBH Health System for these case reports (SBH IRB 2015.12 and 2015.83).

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Case 1

A 21-year-old otherwise healthy man fell from 4 stories and was admitted to the trauma center on the same day. Postresuscitation Glasgow coma score (GCS) was 7 on a scale of 3 (indicating worst eye, verbal, and motor response) to 15 (indicating best eye, verbal, and motor response). Results of computed tomography revealed multicompartmental epidural hematomas, subdural and intraparenchymal hematomas, diffuse cerebral edema (*Figure 1*), and multiple LeFort and cranial fractures. The patient's left eye was proptotic without reactivity. Extracranial injuries were limited to left cervical transverse processes, right clavicle fractures, and assorted soft tissue injuries.

During the osteopathic manipulative medicine/neuromuscular medicine consultation in the surgical intensive care unit on hospital day 4, his GCS was 6. Somatic dysfunctions were palpated, including substantially reduced motion at the cranial base, facial bone asymmetry, and asymmetry and restricted range of motion in the cervical spine, lumbar spine, ribs, and right upper extremity. Gentle OMT was used to manage areas of somatic dysfunction. Techniques used included balanced ligamentous tension and myofascial release.[7,8]

The patient received a total of 24 OMT sessions during his 42-day hospital stay. During his hospital course, the severity of asymmetry and restricted range of motion of the affected areas gradually diminished—palpatory evidence of improved somatic dysfunction. By hospital day 7, the patient's GCS had improved to 11. By hospital day 28, he became verbal, and by hospital day 31, he was fluent with speech. Vision in his left eye improved. No operative intervention was necessary during the patient's hospitalization, and he had no adverse outcomes associated with OMT. The patient was discharged home with a GCS of 15.

Case 2

A 54-year-old otherwise healthy man presented to the trauma center with complaints of dizziness and weakness that led to a fall that same day. Fifteen days earlier, he had been struck by a car and admitted to another local hospital with a 6-mm subdural hematoma in the right frontotemporoparietal area. After observation in the local hospital, he was discharged at his baseline mental status but with persistent complaint of dizziness.

Results of computed tomography after the subsequent fall revealed an increased right-sided subdural hematoma with a 10-mm midline shift and a new right parietal subarachnoid hemorrhage (*Figure 2*). The patient underwent an emergent right craniotomy with evacuation of the subdural hematoma and control of hemorrhage.

On initial consultation, the patient had a GCS of 14, and he complained of persistent headache and dizziness. Somatic dysfunction included substantially restricted range of motion of the cranial base, asymmetry of the temporal bones, asymmetric upper cervical vertebral rotation with preference of rotation to the right, and reduction of respiratory excursion of the thoracoabdominal diaphragm.

The patient was treated with OMT in the postsurgical intensive care unit on hospital day 1. Gentle OMT, including balanced ligamentous tension and myofascial release, were performed with focused attention to the cranium, diaphragm, and cervical vertebrae. The patient received 5 OMT sessions over the course of his 8-day hospitalization. By hospital day 3, he reported no headache or dizziness and no adverse events associated with OMT. During the course of treatment, his somatic dysfunction, particularly in the cervical spine, palpably improved. The patient was discharged home with a GCS of 15.

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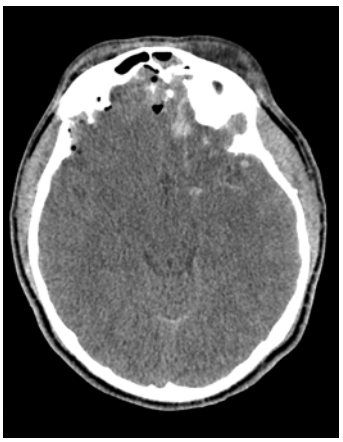

Figure 1.
Axial computed tomographic scan of brain in case 1, a 21-year-old otherwise healthy man who fell from 4 stories, demonstrating multicompartmental hemorrhages and diffuse cerebral edema after traumatic brain injury.

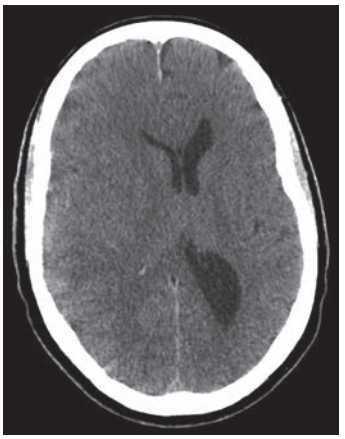

Figure 2.
Axial computed tomographic scan of brain in case 2, a 54-year-old otherwise healthy man with complaints of dizziness and weakness that led to a fall, demonstrating new onset subarachnoid hemorrhage and worsening subdural hematoma with 10-mm midline shift after traumatic brain injury. Craniotomy was required for decompression and control of hemorrhage.

Discussion

Osteopathic manipulative treatment was used in the acute management of the 2 current cases of severe TBI, including 1 patient treated after craniotomy whose cranial and extracranial somatic dysfunction improved. Although the addition of OMT to acute management of severe TBI is commonplace at our institution, this is the first published report of OMT for severe TBI in the acute care setting, to our knowledge.

Case reports cannot be used to draw conclusions of risk or benefit; however, there are multiple theoretical benefits of treating patients with somatic dysfunction in the setting of acute severe TBI that may inform future research.

Pain Modulation

Headache is common after craniotomy and is the most frequent type of pain after TBI.[9] The pathophysiologic mechanisms of headache after craniotomy or TBI are not well understood. Such pain may be in part caused by local inflammation, nociceptive firing from pericranial muscle and soft tissue, or damage to trigeminal periosteal or intracranial dural afferent nerves.[10,11]

Although the brain parenchyma lacks nociceptors, research has demonstrated that noxious stimuli such as surgical incisions or inflammation may cause central nervous system sensitization and promote the persistence of pain or hyperalgesia.[11,12]

Central nervous system sensitization at specific spinal cord segments can be palpated as tissue texture changes in corresponding myotomes of the paraspinal musculature. Decreased nociceptive firing thresholds measured by electromyography have been shown to correspond with these palpatory findings.[13] Muscle spindle length may be improperly set by enhanced motor neuron firing in a sensitized segment, and relaxation of paraspinal musculature using OMT may allow the muscle spindle length to be reset, which would decrease nociceptive and proprioceptive input into the spinal cord.[14]

In the 2 current cases, somatic dysfunction improved in parallel with improving pain, particularly headache. The exact mechanism of pain relief after management of somatic dysfunction with OMT requires further investigation and, to the authors' knowledge, has yet to be studied specifically in the setting of acute severe TBI. However, OMT has been shown to decrease pain in numerous settings, including postoperatively.[15-18]

Dizziness

A common complication of TBI, dizziness occurs in up to 80% of patients with TBI within the first few days of injury[19] and remains in approximately 18% of these patients for 2 years after injury.[20] Dizziness generally resolves after 2 months but may persist.[21] Osteopathic manipulative treatment has been shown to lessen symptoms of dizziness.[22]

For the patient in case 2, dizziness was a debilitating complication after his initial TBI, resulting in unsteady gait and subsequent severe TBI. This dizzi-

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ness persisted after undergoing craniotomy. A potential structural cause of this dizziness includes cervical somatic dysfunction: misfiring of proprioceptive signals in the upper cervical intervertebral joints, muscles, ligament insertions, and muscle spindles located in the deep cervical postural muscles.[23,24] The patient in case 2 was treated with OMT, and his dizziness resolved.

Fluid Drainage

In both of the current cases, we observed an improvement in the somatic dysfunction of the cranial bones, with more symmetry and greater range of motion of the cranial bones and dural system.

The dural venous sinuses are located in the bifurcated attachment of the dural membranes between the periosteal and meningeal layers; therefore, derangements in the tension of the dural membranes may result in derangements of venous sinus structure and suboptimal drainage.[25] Multiple attachments of the dural tissues to bony structures in the cranium create a complex housing of the superior and inferior sagittal sinuses.[26] Displacement of the temporal, occiput, or frontal bones can alter tensions through this dural system, causing the open oval shape of the lumen of the dural venous sinuses to narrow.[27]

In 2015, Louveau et al[28] identified functioning lymphatic vessels running parallel to the dural venous sinuses. These lymphatic vessels carry immune cells and fluid from the components of the cerebrospinal fluid and may act as the link between the intraparenchymal glymphatic and the extracranial lymphatic systems.[28]

By decreasing somatic dysfunction of the cranial bones, OMT may improve drainage of lymph, cerebrospinal fluid, and blood from the venous system for the maintenance of proper neurophysiologic function.[29] Some have suggested the role of OMT in altering such intracranial fluid and venous dynamics.[25,30] One study observed that compared with sham control, hemodynamic functioning in the cranial base improved in patients who received a venous sinus drainage OMT technique.[30]

Adverse Effects of OMT

The patients in the current 2 cases did not experience any notable adverse outcomes associated with OMT. Because data on OMT for TBI are rare, the risk of iatrogenesis cannot be fully elucidated or dismissed.

A case series[31] examining iatrogenesis of OMT for 55 patients with chronic TBI found 3 instances of clinically significant treatment reactions; 1 patient required hospitalization. Based on this case series, the incidence of iatrogenesis of OMT for TBI may be up to 5%.

A study implementing lymphatic drainage techniques on patients with acute TBI did not demonstrate any increase in intracranial pressure.[6] As amount of force and choice of technique may vary widely among physicians, studies are warranted to examine risk of iatrogenesis from OMT in the setting of severe TBI.

Injury after cervical OMT has been reported but is generally considered a rare complication, although vertebral artery dissection has occurred in patients treated with high-velocity, low-amplitude techniques.[32]

Retrospective Case-Matched Control Study Design

Study designs for severe TBI are predominantly retrospective and may draw from trauma registry data. At our institution, OMT in the setting of acute TBI is commonplace. We have developed a retrospective case-matched control study design that may be useful for other institutions with similar use of OMT.

The trauma registry may be interrogated to obtain a list of patients with TBI. They may be stratified by severity based on postresuscitation GCS. Generally, 1 or 2 years of TBI data are sufficient to generate cohorts for study. We recommend at least 30 patients per cohort; however, statistical power will be increased with higher numbers of relevant patients for study. Power analyses should be performed to optimize recruitment.

We recommend that focus be placed on patients who have had blunt-trauma TBIs because penetrating TBIs are much less frequent and have distinct characteristics that make them difficult to compare with blunt-trauma

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TBIs. Patients who died within 24 hours of injury should be excluded. Patients with polytrauma may also be excluded to create a more homogenous study population.

Patient characteristics, including demographics, injury patterns (eg, fall, assault, motor vehicle collision), injury scores (eg, injury severity score, revised trauma score, head abbreviated injury score), and postresuscitation GCS are essential to inform important distinctions between cohorts and to develop the most appropriate case-match. Computed tomography findings may be similarly useful, and we suggest quantifying results with devices such as the Marshall scoring system.

Case-match can be achieved by characterizing a cohort that received OMT. Each patient who received OMT should be matched with a patient who did not receive OMT using age, head abbreviated injury score or other injury scores, postresuscitation GCS, and, if available, Marshall score. The matched cohorts should be similar; data used for matching should be compared statistically and approximate each other by means and SDs. Data used for matching should demonstrate no statistical difference between the cohorts ($P>.05$). Once cohorts are matched, outcome differences, including subgroup analyses (eg, operative vs nonoperative), can be compared based on whether or not OMT was part of patients' acute care.

Areas of somatic dysfunction should be reported. A detailed description of the types of somatic dysfunction (eg, subluxations of individual spinal segments) are not required for adequate analysis. Number, type (eg, myofascial release, balanced ligamentous tension), and frequency of OMT should be described and quantified. If recorded, duration of OMT sessions may help inform cost-effectiveness in terms of physician relative value units.

Any complications associated with OMT should be reported and quantified. Known complications of OMT include pain, vascular lesions, and fracture.[33]

Outcomes observed should include major complications, hospital length of stay, mortality, discharge disposition (eg, subacute rehabilitation facility, skilled nursing facility, home), discharge GCS results, and, if available, Glasgow outcome score or Rancho Los Amigos scale score (both short term and long term). Other useful and more ubiquitous outcome data include results of Mini-Mental State Examination, ability to perform activities of daily living, and other information typically obtained from physical and occupational therapy assessments.

Comparisons of continuous variables should be performed with a signed rank test and of categorical values with symmetry tests.

Conclusion

Several theoretical benefits of managing somatic dysfunction as part of the multimodal acute care of patients with severe TBI exist. The field for research of OMT and TBI is largely unexplored. Comprehensive research is warranted to clarify optimal efficacy and potential adverse effects of OMT in the setting of acute TBI.

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