Decreasing Headache Pain Secondary to a Subarachnoid Hemorrhage with the use of Osteopathic Manipulative Medicine

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ORIGINAL RESEARCH

Abstract

Background: Controlling a headache (HA) secondary to a subarachnoid hemorrhage (SAH) can be challenging for most physicians. At Maine Medical Center in Portland, Maine, the neurointensivist and staff noticed a trend in decreasing HA pain caused by a SAH in patients treated with osteopathic cranial manipulative medicine and osteopathic manipulative medicine (OMM), more so than those treated solely with the traditional opioid approach. It was requested that a chart review of these patients be evaluated for an objective analysis of this observation.

Hypothesis: A decrease in HA caused by SAH will be observed in the group treated with OMM in comparison to those treated with opioids alone.

Methods and Materials: A retrospective, IRB approved, and exempted study reviewed 21 subjects with a SAH that were treated with OMM. This population was analyzed for a decrease in pain score following osteopathic treatment as well as for adverse events 6-month post treatment.

Results: Pain scores were consistently reduced when comparing pre-and-post OMM treatment. After the first treatment, pain scores decreased by an average of 4 points, after the second treatment scores decreased by an average of 3 points and after the third treatment pain scores decreased by an average of 2.5 points. The number of adverse events recorded were found to be less than the national averages.

Conclusion: The use of OMM as an adjunct with traditional treatments for a SAH can lead to a decrease in HA pain caused by a SAH. Minimal adverse events were observed.

Introduction

Controlling headache pain associated with subarachnoid hemorrhage (SAH) is a challenge for many physicians. Several forms of pain medication can be applied in various combinations to provide analgesia, but controlling a SAH headache still remains clinically difficult.[1] The use of osteopathic cranial techniques is a possible adjunct to opioids when treating SAH headaches, without increased morbidity and mortality, even though the study of such techniques is still nascent.

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Background

A SAH refers to the extravasation of blood into the subarachnoid space between the pia and arachnoid membranes.[2] Common causes of a SAH include head injury (most commonly caused by a fall in the elderly or a motor vehicle accident in the young),[3] bleeding disorders, blood thinners, arteriovenous malformations, and idiopathic. A SAH typically presents with a sudden onset of a severe headache (HA). It has often been coined "the worst headache of my life," as described by over 90% of patients.[4,5]

The sudden headache, caused by increased intracranial pressure, can be accompanied by nausea, vomiting, photophobia, meningismus, and loss of consciousness.[6,7,8] This sudden increase in intracranial pressure can lead to a stroke, most commonly from a ruptured saccular aneurysm (also known as a berry aneurysm).[2,9]

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In some studies, 30-day mortality rates for patients with SAH are close to 45% from stroke due to a SAH, with the majority of deaths occurring from initial hemorrhage.[10] In other studies, one in four patients with aneurysmal SAH die, with 50 % of survivors having a persistent neurological deficit.[11] Even after immediate treatment and stabilization, patients are often left with an excruciating headache not amenable to pain medication.[1] The headache typically lasts 1-2 weeks and can persist years beyond the initial bleed.[12] One longitudinal study, examining symptoms 4-7 years after a patient's initial SAH, found that of the 123 subjects, 16.5% still suffered from headaches, 41% reported memory issues, and 35% still experienced daytime sleepiness.[13]

Glisic et al[1] in 2016 published a retrospective review of headache prevalence, inpatient course, frequency of medication used to treat HA's, and clinical factors associated with HA severity after a non-traumatic SAH. A severe HA is defined as head pain for a duration of at least 2 days, a pain score of ≥8 out of 10, and treatment needing ≥3 different analgesic medications for at minimum 2 days. This 15-day study found that 73% of patients had a severe HA with minimal decrease in pain scores, despite the use of analgesic pain medications. Furthermore, they found that 75% of patients required a prescription for opioid medication upon discharge.[1]

Current interventional and non-analgesic treatment regimens for SAH-headaches include external ventricular drainage for symptomatic hydrocephalus, aneurysm stabilization with endovascular coiling or surgical clipping, nimodipine, atorvastatin, increased fluid intake (oral or I.V.), and maintenance of serum magnesium above 2.3 mg/dL. Analgesic regimens include stepwise progression with acetaminophen, codeine, tramadol, and piritramide, as well as acetaminophen , dexamethasone, Oxycodone, Fentanyl, Morphine, Butalbital/acetaminophen/caffeine, Hydromorphone, hydrocodone/APAP, and Ibuprofen, in decreasing order.[12] Glisic et al[1] found peak usage of opiates occurred at days 4-5, with about 18 mg of morphine equivalence being used; this despite minimal decreases in pain scores during the 15-day study period.

The efficacy of the use of OMM and OMM in the cranial field are long-standing osteopathic practices and are particularly suited to treating migraine and non-migraine headaches. Increases in cranial pressure can cause pain from the sensitive structures of the venous sinuses, meningeal/dural arteries, and portions of the cerebral arteries.[7,14,15,16] A SAH is an extravasation of blood between the arachnoid membrane and the pia mater into the subarachnoid space.[2,3] This extravasation of blood increases intracranial pressure. So, by addressing the venous and lymphatic system[17] to cause a slow drainage from the head with OMM and OMM cranial techniques seems to be a reasonable, noninvasive treatment approach to mitigate this pressure and decrease headache pain due to SAH.

There have been concerns in the past that using cranial OMM may not be a beneficial technique for SAH patients and have been deemed a relative contraindication. However, with newer studies showing changes in cerebral blood flow with transcranial doppler ultrasound (TCD) and OMM these viewpoints are being reexamined.[18,19]

Formes et. al.[20] showed that decreases in middle cerebral blood flow, as monitored by TCD, closely correlate with decreases in cerebral tissue oxygenation. Shi et al[19] demonstrated that performing suppression cranial OMM techniques caused decreased cerebral tissue oxygenation. Since the middle cerebral blood flow parallels cerebral tissue oxygenation, it would seem that suppression cranial OMM techniques could be applied to reduce increased intracranial pressure by slowly decreasing blood flow or volume.[19,20]

As the body has tensegrity, treating somatic dysfunction (impaired or altered function of related components of the *somatic* (body framework) system) and other congested areas of the body would allow for improved lymphatic drainage leading to an improved response to cranial OMM techniques.[14,21]

In an effort to consider how to decrease opioid usage at Maine Medical Center in cases of SAH-associated headaches, the neurointensivists and staff anecdotally noted their patients who receive treatment using OMM seemed to fare better than those who did not received OMM. The use of OMM was initially provided as part of the osteopathic consultative hospital service for inpatients. Thus, a retrospective study was discussed, to objectively examine this supposed phenomenon.

Hypothesis

The use of OMM will help decrease patient-reported headache pain due to SAH with no increase in adverse events.

Methods and Materials

The study population included a retrospective chart review of patients admitted to Maine Medical Center (Portland, Maine) with a diagnosis of headache secondary to nontraumatic SAH, confirmed by CT scan, from January 2015 to December 2017, and treated

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with OMM for SAH related headache. Patients with a diagnosis of SAH were excluded if they did not undergo treatment with OMM or had an incomplete medical record. The study was determined to be exempt from IRB review.

Subjects were asked to rate their pain on a scale of 1-10 both pre-and post-treatment as part of a standard osteopathic evaluation. Medical records were evaluated 6 months post-treatment to assess for any adverse events such as death, readmission to the hospital, new stroke, or new brain bleed/rebleeds. Pain scale ratings were compared pre- and post-treatment to determine improvements in pain immediately after OMM treatment. Structural Equation Modeling statistical analysis technique were used to summarize the data for the study population. All results and statistical analysis were performed by a licensed statistician.

All subjects were treated with OMM, in addition to traditional treatment methods including opioids for analgesia, cranial shunt placement, and neurosurgical drainage. Each subject received an OMM evaluation and treatment, of both cranial and non-cranial somatic dysfunctions. Treatment techniques included cranial techniques, myofascial release, and muscle energy. These techniques were applied to the head, thoracic area, pelvis, sacrum, and any other area of somatic dysfunction determined by the physician to contribute to the patient's headache. All subjects were treated by the attending physicians BLB or DDK with the assistance of the neuromusculoskeletal medicine/osteopathic manipulative medicine fellows.

Results

A total of 21 subjects ages 33-80 with a mean age of 53.4 (standard deviation (SD 14.70) met the inclusion and exclusion criteria. Time from admission to treatment with OMM ranged from 1-11 days with the mean start day of OMM treatment being hospital day 3.4 (SD 3.06). The total number of OMM treatments ranged from 1-6 treatments on separate days, with the mean number of OMM treatments per patient being 3 (SD 1.52) (Table 1).

When comparing pre-treatment to post-treatment values, a decrease in pain was noted immediately after each treatment. The average pre-treatment pain level was rated at 5.5 (SD 0.66), average post-treatment level was rated as a 1.8 (SD 0.93). Before the second treatment, the average pre-treatment pain level was rated a 4.7 (SD 0.69), and post-treatment pain level was rated as a 1.2 (SD 0.43). Before the third treatment, the average pre-treatment pain level was rated a 2.5 (SD 1.02), and post-treatment average pain level was 2.0 (SD 2) (Table 2). Because the number of treatments per patient varied more after three treatments, the sample size was not large enough to draw any significant conclusions as to the efficacy of more than 3 treatment sessions.

A summary of adverse outcomes 6 months after discharged was also analyzed for increases in morbidity and mortality. Out of the 21 subjects only 20 subjects had records after 6 months. Of the 20 subjects 0% deaths were noted. 20% (N=5) had readmission to the hospital (unrelated to the SAH), 5% had a stroke (N=1), and 10% experienced new onset brain bleed (N=2). Of note the mortality rate from this study had no in-patient deaths as well as no deaths at the 6-month mark for patients treated with OMM. This is clinically significant when comparing this to other Medicare national studies, of which all had either inpatient or 30-day mortalities reported (Table 3).[22,23,24,25]

Discussion

The purpose of this pilot study was to objectively analyze the data for possible correlations between the anecdotal reports of HA improve-

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Study Cohort n-21 Characteristics	Mean (SD)	Median	Range
Age	53.4 (14.70)	52	33-80
Time from Admission to OMM Treatment (Days)	3.4 (3.06)	2	1-11
Total SAH Hospitalized Treatment Duration (Days)	5.3 (3.36)	6	1-12
Total Number of OMM Treatments Sessions	3.0 (1.52)	3	1-6

Table 1. *Detailed list of demographics and treatment characteristics of the study cohort.*

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ment in SAH patients treated with OMM and actual reduction in HA pain. According to the preliminary data it seems that the use of OMM in conjunction with standard of care treatments may be beneficial for treating patients with headaches caused by a SAH. As such, a more formal regimented prospective study should be performed to verify the consistency of this information. Future studies should devise a control group of non OMM treated vs OMM treated groups along with a standardized OMM treatment protocol to ensure consistent treatment and documentation. Furthermore, a strict monitoring system of analgesic medication use, especially the morphine equivalent usage for controlling pain, should be observed as a dependent variable to examine if OMM can decrease the use of opiates as opiates have not been found to be very effective.[1,11] The reduction of opioid usage and the consistent treatment of headache pain would be beneficial to both patients and society.

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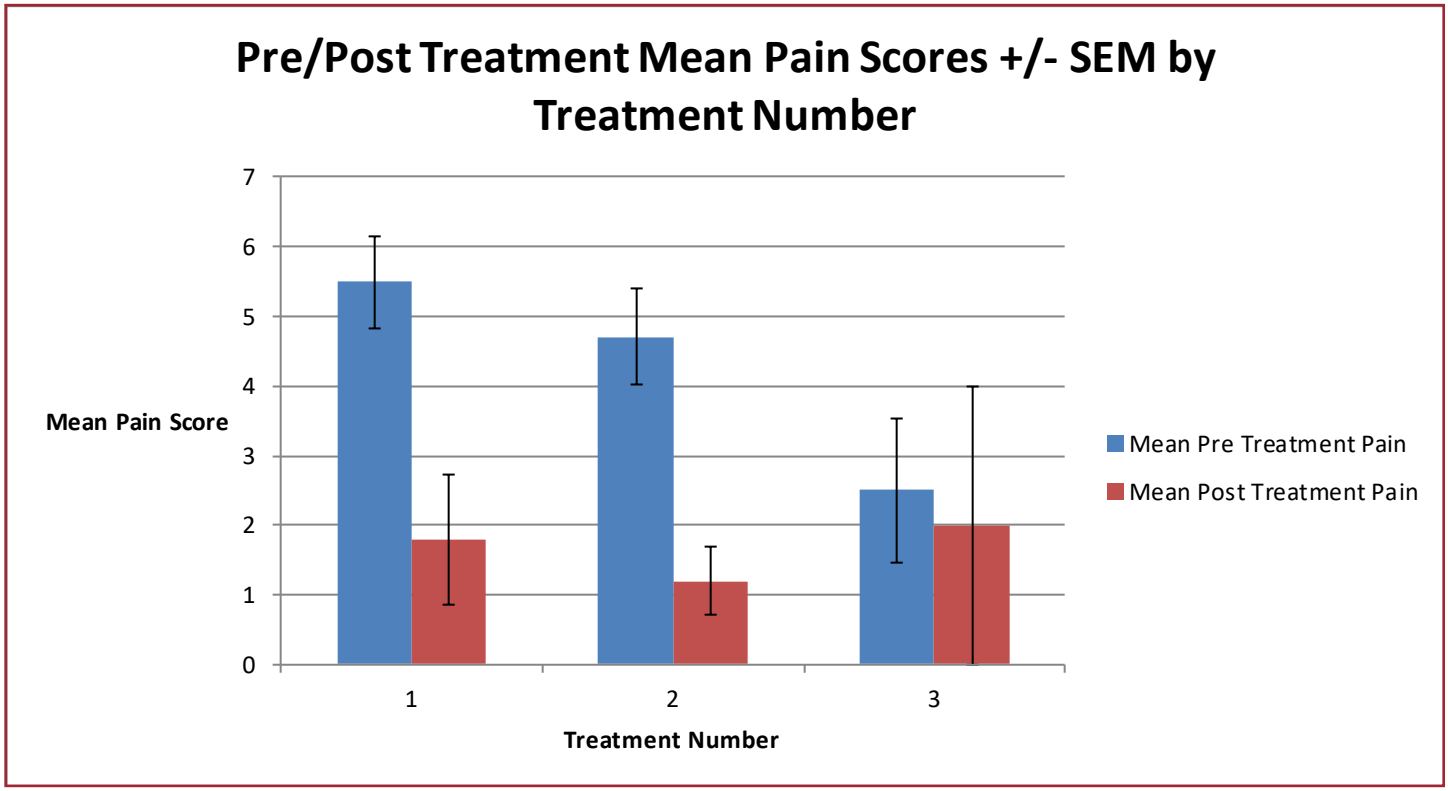

Table 2. *Comparison of the pre-and post-treatment pain scores using structural equation modeling (SEM) statistical analysis technique.*

Comparison of 6-month Adverse Events from the Study Cohort vs 30-day Adverse Events among Medicare Beneficiaries	6 months	30 days	30 days
	Study Cohort	**Study 1***	**Study 2****
Total number of subjects per study	n=20	n=2305	n=3387
Death within 6 months	0 (0%)	809 (35.1%)	419 (11%)
Readmission within 6 months	5 (20%)		346 (10.2%)
Stroke within 6 months	1 (5%)		
New Brain Bleed within 6 months	2 (10%)		48 (14.7%)

**Study 1= 30 day Mortality and Readmission after Hemorrhagic Stroke Among Medicare Beneficiaries in Joint Commission Primary Stroke Center-Certified and Noncertified Hospitals 2018.*[22]
***Study 2 = 30 day Mortality and Readmission after Hemorrhagic Stroke Among Medicare Beneficiaries in Joint Commission Primary Stroke Center-Certified and Noncertified Hospitals 2011.*[23]

Table 3. *Summary of Adverse Event Outcomes 6-month study cohort compared to 30 day outcomes for 2 other national studies.*

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Conclusion

A steady decrease in headache pain was noted with the adjunctive use of OMM, including OMM cranial techniques. The 6-month adverse events of death, stroke, and new onset brain bleed did not differ from expected SAH outcomes when compared to national data. Our analysis suggests OMM is a possible effective adjunctive treatment for decreasing HA pain associated with SAH and does not increase the number of adverse events already associated with SAH.

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