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Upper cervical chiropractic care for a 25-year-old woman with myoclonic seizures

Todd A. Hubbard DC^{a,*}, Casey A. Crisp DC^a, Brett Vowles DC^b

^a Assistant Professor, Academic Health Center, Palmer College of Chiropractic, Davenport, IA 52803 ^b Private Practice, Alpine, CA 91901

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Key indexing terms: Manipulation, Chiropractic; Myoclonic epilepsy, Juvenile; Cervical, Atlas; Menstrual cycle	 Abstract Objective: The purpose of this case report is to describe the chiropractic management using upper cervical techniques of a 25-year-old woman diagnosed with juvenile myoclonic epilepsy (JME). Clinical Features: A 25-year-old woman had a history of JME, which was diagnosed at the age of 14 years. Her seizure episodes began shortly after trauma to her cervical spine and the onset of menarche. Intervention and Outcome: After case history and physical examination, the patient received high-velocity, low-amplitude chiropractic spinal manipulation to her upper cervical spine using the Blair upper cervical chiropractic technique protocol. There was improvement in her seizure episodes and menstrual cycles following 12 weeks of chiropractic care.
	 conclusion: This case study demonstrated improvement in a young woman with a seizure disorder after she received upper cervical chiropractic manipulation. This case suggests the need for more rigorous research to examine how upper cervical chiropractic techniques may provide therapeutic benefit to patients with seizure disorders. © 2010 National University of Health Sciences.

Introduction

Current estimates are that 2 million Americans have epilepsy; 125 000 new cases develop each year, with 50% of these cases occurring in children and adolescents. The prevalence of epilepsy in those younger than 18 years is estimated to be 4.7 per $1000.^{1,2}$ Juvenile myoclonic epilepsy (JME) accounts for about 10% of all epileptic patients. It is the most common idiopathic generalized epileptic syndrome, having a prevalence of 10 to 20 per 100 000.^{2,3}

Very few case reports have been published on chiropractic management of JME. The purpose of this article is to describe the chiropractic management of a patient with an 11-year history of JME.

^{*} Corresponding author. Academic Health Center, Palmer College of Chiropractic, 1000 Brady St., Davenport, IA 52803. Tel.: +1 563 884 5184; fax: +1 563 884 5470.

E-mail address: todd.hubbard@palmer.edu (T. A. Hubbard).

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Case report

A 25-year-old female patient had been diagnosed with JME by the Michigan Institute for Neurological Disorders in 1998, 11 years before presentation for care, and had experienced seizures throughout this time. Her seizures began when she was 14 years of age, when her menstrual cycle began. This time also coincided with a trauma in which she fell out of bed and hit her head on a desk 2 to 3 months before the seizures began. At the time of her first chiropractic visit, she was taking 200 mg of lamotrigine per day to control the seizure activity. Over the previous 11 years, she experienced 1 to 2 seizures per month. She reported that her menstrual cycles lasted approximately 10 days, with 10 to 15 days between cycles. She began using an intrauterine device to control heavy flow and maintain hormone levels once she began her menstrual cycles.

She reported having a decrease in the frequency of seizures in the past with what she called a "more specific" chiropractic approach. However, she did not experience similar results from other types of chiropractic treatment.

Physical examination showed normal cervical spine range of motion with local neck pain upon the Soto-Hall maneuver. Cranial nerve, deep tendon reflexes, and



Fig 1. Base posterior radiograph. The right and left occipital condyles-lateral mass articulations are outlined to show the center of each joint. A, The right "condyle convergence angle." B, The left "condyle convergence angle."



Fig 2. The left Blair protractoview. The most lateral end of the superior articulating surface of the lateral mass is lateral to the most lateral tip of the inferior articulating surface of the occipital condyle. This is determined to be an ASL misalignment of the left occipitoatlantal articulation.

muscles strength tests were unremarkable. The Commission on the Outcome Measurement in Epilepsy states that there is no single standard outcome measure used to rate seizure activity and that the outcome used should focus on the health-related quality of life.⁴ Therefore, we used the Bournemouth Neck Questionnaire (BNQ) and the Headache Disability Index (HDI)^{5,6} and instructed her to replace the word *pain* with *seizures* when answering the questions. The BNQ scored 90%, and the HDI scored 76 overall (E = 44, F = 32).

The Blair upper cervical chiropractic technique protocols were used to guide treatment for this patient. This technique uses a base posterior radiograph (Fig 1) to find the occipital condyle convergence angle for the left and right condyle. From the occipital convergence angle, an oblique nasium radiograph, called the *Blair protractoview* (PV), is exposed down the center of the left (Fig 2) and right (Fig 3) condyles. This measurement attempts to determine occipitoatlantal alignment.

The patient's PV indicated a misalignment of the C1 vertebra to be anterior-superior-left (ASL), compared with the occiput. The PVs also showed the inferior articulating surface of her right condyle to be relatively steep (44°). With an inferior condyle articulation greater than 40°, Blair protocol is to manipulate (ie, adjust) the ASL misalignment from the right. With this particular



Fig 3. The right Blair protractoview. The most lateral occipital condyle and lateral mass articulation surfaces are under each other. There is no misalignment of the right occipitoatlantal articulation.

misalignment, the right posterior arch is used as a lever to manipulate the joint, pulling it toward the correct position. The spinal manipulation therapy (SMT) was given using a side-posture, toggle table, incorporating a modified high-velocity and low-amplitude Palmer Hole In One (HIO) toggle manipulation.

The patient was seen 11 times over a period of 12 weeks. She received 3 Blair spinal manipulations to her C1 during this time. She reported having 1 seizure a few days after the first manipulation. Since that time, she had reported no seizure episodes. She reported 6 episodes of minor twitching that did not progress into seizure. She also reported that her last 2 menstrual cycles lasted less than 7 days and were 28 days apart. Her follow-up BNQ and HDI questionnaire scores were 0 and 4 (E=4, F=0), respectively. Written consent from the patient was obtained allowing us to publish her personal health information without divulging personal identifiers.

Discussion

The onset of JME may occur from 6 to 22 years of age. Symptoms include myoclonic jerks, often associated with generalized tonic-clonic seizures and absence seizures.² There are typically no abnormalities seen on clinical examination or any intellectual deficits associated with JME. It is nonprogressive.² The etiology of JME is thought to stem from genetics.^{2,3,7} Patients report that certain factors such as sleep deprivation, menses, alcohol, and overall stress can increase their incidence of seizure.^{2,3,8} The most characteristic electroencephalographic pattern is generalized multiple high-frequency spikes followed by slower waves.^{2,8} Neuroimaging studies generally show no abnormalities. Atypical presentations are often misdiagnosed.²

The diagnosis of JME can be difficult because it can clinically mimic other disorders. Such disorders include forms of generalized epilepsy, lipid storage disorders, nonepileptic seizures, postanoxic myoclonus, and other seizure disorders. Thus, it is critical to perform a thorough patient history, focusing on precipitating seizure factors; it is also important to conduct an electroencephalogram.^{2,3,7} Medical approaches to management of JME involve antiepileptic drugs (AEDs), the most common of which is valproic acid. Other AEDs include lamotrigine, topiramate, zonisamide, levetiracetam, and benzodiazepines.^{2,3,8} Juvenile myoclonic epilepsy is a lifelong disorder. Treatment is often continued indefinitely to prevent recurrences, which are frequent. Seizure recurrence, which can return months to years after discontinuation of AEDs, may lead to status epilepticus.²

Alternative and complementary options to the medical treatment of JME are few. However, there is some evidence that spinal manipulation may be helpful in the treatment of JME. A case report by Goodman and Mosby⁹ discussed a 5-year-old girl who experienced seizures after head trauma. There was substantial occipital-atlantal-axial misalignment. After 1 month of care involving spinal manipulation to the upper cervical area, she experienced cessation of her seizures. Another case reported a patient with a history of low back pain and clonic-tonic seizures approximately every 3 hours.¹⁰ The patient received spinal manipulative care. At 1.5-year follow-up, she had intermittent short-duration seizures spaced up to 2 months apart.¹⁰ A literature review by Pistolese¹ states that a potential exists for a chiropractic management approach to epileptic seizures.

Christensen et al¹¹ conducted a population-based cohort study of more than 1.5 million people and found that the relative risk of epilepsy raised about 2-fold after mild head injury, which our patient had, and 7-fold after a severe head injury. Incidence was slightly greater in women than in men. Our patient fell out of bed, hitting her head on a nightstand, which may have caused trauma to her head and cervical spine. Trauma to the cervical spine has been assumed to be a cause of spinal segmental dysfunction.¹²

There are several theories that may explain how SMT may have been of benefit to this patient. The first involves displacement of the C1 vertebrae, placing pressure or torsion on the carotid sheath containing the common carotid artery; internal carotid artery; internal jugular vein; and glossopharyngeal, vagus, and spinal accessory nerves.^{13,14} A second theory arises from torsion or pressure upon the superior cervical ganglion located at the vertebral body of the axis and posterior to the carotid sheath.¹⁵ Vagal nerve stimulation, which is reported as an acceptable treatment of epilepsy, has been shown to reduce seizure frequency, even in pharmacoresistant generalized epilepsies.¹⁶ This may be a reason why we saw an improvement in the patient's seizure frequency following SMT to the C1 vertebrae. There is also the Grostic dentate ligamentcord distortion hypothesis, which describes the possibility of mechanical irritation and ischemia of the cord caused by abnormal tension on the dura when the upper cervical segmental dysfunction is present.¹⁷

Pickar¹⁸ states that there is evidence demonstrating that SMT evokes paraspinal muscle reflexes and alters motoneuron excitability. He states that "sensory input from paraspinal tissues can evoke visceral reflexes affecting the sympathetic nervous system and may alter end-organ function."¹⁸ Noxious paraspinal sensory input, which may be caused by the spinal segmental dysfunction, appears to have an excitatory effect on sympathetic outflow. Correcting the segmental dysfunction through SMT may normalize the sensory input, thus having an inhibitory effect on somatovisceral reflexes.¹⁸ A study conducted by Matsumoto et al¹⁹ found that women who suffered from more symptomatic premenstrual phases had a higher level of sympathetic nerve activity.

Levine²⁰ states that cervical spondylotic myelopathy may be caused by tensile stress transmitted to the spinal cord by the dentate ligaments that may cause stress at the lateral columns of the spinal cord. This stress may be the cause of noxious sympathetic input causing abnormal viscerosomatic effects. Noxious sympathetic input causing a higher level of sympathetic nerve activity may have been causing our patient's abnormal menstrual cycle, which is a known trigger for seizure activity.^{2,8}

Our patient also reported that her menstrual cycle normalized after treatment. She stated in her history that her menses started around the same time as her epileptic seizures. Alfradique and Vasconcelos² and Sokic et al⁸ have reported menses as a trigger for epileptic seizures. However, we cannot be sure if there was a direct link between the cessation of our patient's seizure episodes, SMT, and the return of a normal menstrual cycle. More research is needed to investigate this question.

The most common medical treatments have inconsistent success, which was the case here.³ Drug treatment of JME is mainly based on clinical experience and prospective and retrospective studies, with little evidence from randomized clinical trials.²¹ There are few head-to-head comparisons between old and new AEDs. Valproate is the drug of first choice in men with JME.²¹ In women, lamotrigine (which our patient was taking) would be considered first because of the teratogenicity and adverse effects of valproate. Levetiracetam is also effective. Recent data suggest that it may soon be used as a first-line treatment. Some AEDs can aggravate JME.^{2,21} In addition to AEDs, nonpharmacologic treatments are important in JME. Juvenile myoclonic epilepsy usually requires lifelong treatment because seizures nearly always return after withdrawal of therapy.²² Valproate and lamotrigine have shown to produce a complete remission of seizures in a majority of patients. It may be possible that our patient is experiencing a remission phase due to the medication she has been taking.^{2,3,8} However, our patient informed us that she has reduced her lamotrigine dosage to 50 mg/ d without a return of the seizures.

Limitations

Because this is a case report, it is not possible to generalize this treatment approach to all myoclonic seizure patients. Because of the cyclic nature of JME, we do not know if the improvements seen in this patient are from the SMT or if we are entering a cyclic phase sometimes seen with seizure disorders.²² A JME patient will need lifelong follow-up care for the possibility of reoccurrence of seizure episodes. This follow-up care would enable the patient to be treated if she had entered an absence phase of her seizures or reoccurring episodes begin; current literature states that this is likely.^{2,22}

In our attempt to use outcome measures for this study, we chose to use the BNQ and HDI and replace *pain* with *seizure*. Anytime an outcome instrument is revised or used in an alternate setting, its validity and reliability need to be studied. Therefore, although our pre- and postintervention scores for these outcome measures decreased substantially, we cannot view their validity and reliability as we would have if they had not been altered. Furthermore, the Commission on Outcome Measurement in Epilepsy states there is no one outcome measure that is recommended.⁴ Some of the current epilepsy-specific health-related quality of life instruments are the Liverpool Health-Related Quality of Life Battery, the Quality of Life in Epilepsy instruments, and the Impact of Childhood Illness Scale. Outcome measures related to global evaluation that are useful include the RAND 36-item Health Survey, the Nottingham Health Profile, the Sickness Impact Profile, and the Dartmouth COOP Function Charts. Future assessments of the epileptic patient should include one of these outcome measures.

Conclusion

This case is interesting and novel because the patient experienced a cessation of her JME seizures following upper cervical chiropractic treatment. The potential relationship between first cervical vertebrae misalignment and JME seizures is not yet known, and there are no studies that have been performed investigating this relationship. Based upon our clinical observations, there might be a correlation between the specific SMT and this patient's relief of symptoms. Future clinical trials need to be conducted to investigate the role of chiropractic care in the treatment of JME.

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