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CASE REPORT

Immediate stroke following chiropractic cervical spine manipulation: a case report with causal analysis

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ABSTRACT

Cervical spine manipulation (CSM) performed in the presence of vertebral artery dissection (VAD) has been implicated in immediate thromboembolic stroke. We report a 27-year-old male who developed immediate ischemic symptoms after CSM, with imaging confirming bilateral VAD and bilateral cerebellar infarctions. Craniectomy and decompression were required; despite rehabilitation, the patient remains permanently disabled.

Using an intuitive forensic approach, we determined that the VAD was most likely pre-existing from minor neck trauma sustained one week earlier in a mosh pit, compounded by fibromuscular dysplasia. A 3-step medicolegal causation analysis established that the immediate stroke was caused by CSM as more likely than not (biological plausibility, temporality, and absence of a more probable alternative explanation). Two breaches in the standard of care were identified: failure to diagnose and refer suspected VAD prior to manipulation, and failure to recognize and urgently refer acute stroke symptoms.

Clinical examination strategies to exclude VAD before CSM, coupled with improved training in vascular differential diagnosis for chiropractic physicians, could have prevented this outcome and can prevent future cases. This case underscores the importance of thorough history-taking, vital-sign assessment, and recognition of VAD "red flags" in chiropractic practice.

Introduction

Case reports of immediate stroke after cervical spine manipulation (CSM) have been published since 1947.[1] In 2001, a chiropractic researcher reviewed 255 cases of vertebrobasilar complications following CSM, 137 of which were immediate, occurring within moments or minutes.[2] Since 2001, additional case reports and case series documenting stroke immediately following CSM have continued to appear in the literature.[3–6]

Despite these reports, the causal relationship between CSM and stroke remains controversial. Population-based epidemiological studies, primarily authored by chiropractic researchers, have generally reported low overall risk.[7–10] However, these studies have important limitations when applied to the specific scenario of immediate post-manipulation stroke, including exclusion of the 0–1 day exposure window,[11,12] small numbers of stroke cases in younger patients (the population most at risk for vertebral and internal carotid artery dissection),[8,10] and warnings that they do not exclude CSM as a cause of stroke in individual cases.[7,8] Because randomized controlled trials to definitively establish causation are ethically impossible,[13] detailed forensic causal analysis of individual cases using probabilistic medicolegal frameworks provides essential evidence for clinical decision-making and risk communication.

We report a 27-year-old male who suffered an immediate stroke after chiropractic CSM. Imaging revealed bilateral vertebral artery dissection (VAD) and bilateral cerebellar infarctions affecting balance, coordination, and speech. Craniectomy and cerebellar decompression were required. Despite extensive occupational and speech therapy, the patient remains permanently disabled.

The objectives of this case report were to:

1. Perform a forensic analysis to determine the most likely causal mechanism of VAD.
2. Perform a forensic analysis to determine the most likely causal mechanism of the stroke.
3. Perform an analysis of the standard of care with the aim of determining how this case could have been prevented, and how future cases could be prevented.

Case Presentation

A 27-year-old male bartender presented as a new patient to a chiropractor at 4:30pm on October 27th.

Subjective examination

The patient reported a new complaint of recent onset sharp, constant, bilateral suboccipital neck pain (7/10) and dull, intermittent, bilateral occipital headache (3/10). Symptom onset was one week prior following "dancing at a concert." The chiropractor documented, "No trauma associated." Symptoms were worsening. The patient denied having these symptoms before, and denied nausea/vomiting, numbness, dizziness, difficulty walking, and difficulty speaking. Records include an assessment of Activities of Daily Living form which was not completed. The chiropractor did not obtain history of over-the-counter or prescription medication use, recreational or illegal drug use, alcohol use, or smoking.

Objective Examination

The patient was 5′ 10″ and weighed 185 pounds. Vital signs were not obtained. Palpation revealed mildly taut and tender fibers at C1 left and moderately taut and tender fibers at C5-C6 bilaterally. Subluxations were noted at the C5 and C6 levels. All cervical spine range of motion was noted as mildly restricted and moderately painful. Orthopedic examination consisted of Maximum Compression Testing positive bilaterally, Shoulder Compression Testing positive bilaterally, and Valsalva which was negative. Neurological and radiological examinations were not performed.

Diagnosis/Assessment

Documentation did not contain any diagnosis codes or diagnosis descriptions.

Procedure/Plan

Manual adjustments were performed at the C1, C5 and C6 levels. Procedure codes were not documented.

The Treatment Plan was 3 times a week for 1 week with a re-evaluation recommended at that time. The chiropractor noted that following the visit the patient felt "worse", however, there was no documentation of post-manipulation events.

POST-MANIPULATION EVENTS

Per the patient, immediately after CSM on one side, the patient told the chiropractor to stop because something was not right. The patient experienced increased neck pain and was feeling dizzy, lightheaded, and nauseous. However, the chiropractor proceeded to manipulate the other side of the neck. Immediately after the second manipulation, the patient could not use or feel his legs, was unable to walk, and began sweating profusely. The chiropractor advised the patient to drive home and sleep it off.

The patient knew that he could not drive home, so he called a friend to come and pick him up. Per the patient's friend, when he received the phone call, he had difficulty understanding the patient as he was slurring his speech. When the friend arrived, the chiropractor had him assist in carrying the patient through the lobby, out the front door and across the parking lot to his car. The patient could not use his legs, and they were dragging on the ground as he was being carried.

When the friend asked for an explanation, the chiropractor responded, "Everything is going to be OK. This happens all the time. Take him home, have him lie down, put an ice pack on his neck, he will be fine by tomorrow. He should be feeling better in a few hours, and he'll be completely normal by tomorrow." Trusting the advice of the chiropractor, the friend took the patient home to rest. Later that evening, the patient could barely sit up in bed and continued to be extremely dizzy. His friends carried him to the car and transported him to the emergency department (ED).

EMERGENCY DEPARTMENT EVENTS

At 6:38pm, over two hours after the 4:30pm chiropractic appointment, the patient was admitted to the ED. At 9:26pm, computed tomography angiography (CTA) neck examination was performed and interpreted by a double-board-certified neuroradiologist as unremarkable.

The 4.5-hour window for tPA (tissue plasminogen activator) treatment passed at 9:00pm. tPA treatment is a thrombolytic therapy, primarily used for acute ischemic stroke to restore blood flow by breaking up blood clots. It is most effective when given intravenously within 4.5 hours of stroke symptom onset.

The patient remained in the ED for six more hours until further imaging was done. At 3:10am, MRI Brain examination without contrast found restricted diffusion within the bilateral, left greater than right, cerebellum involving the posterior inferior cerebellar artery, anterior inferior cerebellar artery and superior cerebellar artery territories. (Figure 1) Impression was acute bilateral, left greater than right, cerebellar infarctions.

At 4:23am, review of the CTA neck imaging by a neurology resident showed, "There is a dramatic change in vascular caliber in the distal V3 segment of the left vertebral artery. (Figure 2) There appears to be full reconstitution in the proximal V4 segment, but there are well-visualized branches of the right PICA in the cerebellum with nearly no enhancing PICA branches in the left hemisphere. This is concerning for left vertebral artery dissection." The neuroradiologist noted, "Upon further review, there is irregularity and luminal narrowing of the bilateral distal V2 segments and V3 segments of the vertebral arteries. Finding consistent with dissection." Evidence of fibromuscular dysplasia was noted on the imaging.

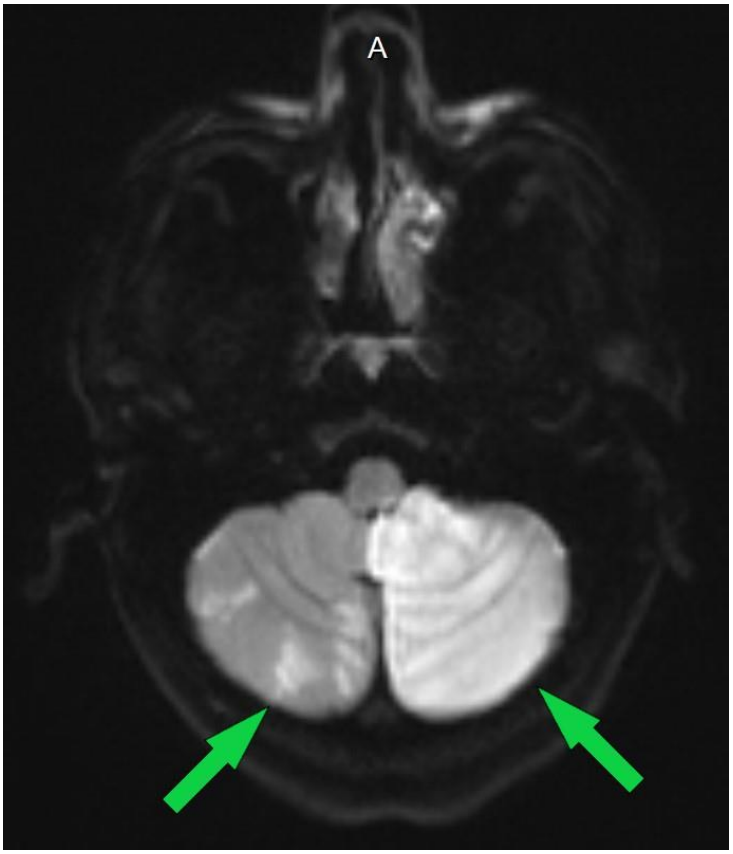

Figure 1: Brain MRI examination without contrast showing bilateral, left greater than right, cerebellar infarctions. (green arrows)

Figure 2: CTA neck examination showing change in vascular caliber of distal V3 segment of left vertebral artery. (right arrow)
Luminal narrowing of the V3 segment of the right vertebral artery can be seen in the transverse foramen of C1. (left arrow)

The patient was diagnosed with VAD and cerebellar infarct. At 2:30pm, he underwent a craniectomy and decompression to relieve intracranial pressure due to cerebellar swelling.

The patient underwent extensive occupational and speech therapy following the stroke. Follow-up showed the patient was permanently disabled and no longer able to work. He had lost balance, coordination, and strength, leading to falls and the use of a walker. He had difficulty using his arms and legs and difficulty speaking. His prognosis for further improvement was limited. See timeline of case events. (Table 1)

Table 1: Case timeline

Date	Time	Case event
October 20th	Unknown	Neck pain and headache following dancing at a concert
October 27th	4:30 pm	Immediate ischemic symptoms following neck manipulation
October 27th	6:38 pm	Patient admitted to emergency department
October 27th	9:00 pm	End of 4.5-hour window for IV tPA treatment
October 27th	9:26 pm	CTA neck read by neuroradiologist as unremarkable
October 28th	3:10 am	MRI Brain without contrast shows bilateral cerebellar stroke
October 28th	4:23 am	CTA neck read by neurology resident as bilateral V2-V3 VAD
October 28th	2:30 pm	Craniectomy & cerebellar decompression surgery

Methods

Consent for publication was obtained from the patient. Case information was taken from chiropractic and medical records. Information on post-manipulative events was provided by the patient and a friend who picked him up from the chiropractic office after the manipulation.

Causation of VAD was evaluated using an intuitive forensic approach.[14] This method relies on temporal association, known risk factors, plausible mechanisms, and the exclusion of alternative causes based on the best available evidence when only one biologically reasonable pathway exists.

Causation of the stroke was evaluated with a previously published 3-step medicolegal causation framework.[14–16] The three required criteria are (1) biological plausibility, (2) temporality, and (3) lack of a more probable alternative explanation. Each criterion was assessed against the clinical timeline, imaging, and peer-reviewed literature. Several alternative explanations (protopathic bias, toxin release, coincidence) were systematically evaluated and ruled out as less probable.

Causal Analysis

MOST PROBABLE CAUSE OF VERTEBRAL ARTERY DISSECTION

Dissections may be traumatic or spontaneous. Traumatic dissections result from severe trauma such as a motor vehicle collision or sports injury. Spontaneous dissections can occur with minor neck movements in the absence of any severe trauma.[17]

Environmental risk factors for dissection include recent acute infection (mainly respiratory), use of fluoroquinolone antibiotics, hyperhomocysteinemia, low BMI, low cholesterol, smoking and pulsating tinnitus. Inherited risk factors include arteriopathies such as fibromuscular dysplasia, connective tissue disorders such as Ehlers-Danlos syndrome type IV, and a history of migraine headaches.[18,19]

The patient had risk factors for VAD. He was 27 years old. Most dissections occur in people less than 45 years of age.[17] Evidence of fibromuscular dysplasia was noted on imaging at the ED.

Recent neck or head trauma is also a risk factor for VAD. Medical records document neck pain and

headache began following "dancing in a mosh pit at a rock concert," as opposed to the chiropractor's documentation of simply "dancing at a concert." In contrast to the chiropractic documentation that there was, "No trauma associated," moshing involves violent, high-energy physical contact—including crowd-surfing, shoving, and body-slamming—that is widely recognized as a mechanism of traumatic injury. An analysis of mosh-pit-related emergencies reported that 97% (245 of 253) required basic life support, with one patient necessitating helicopter evacuation,[20] confirming moshing as a significant source of injury and a plausible cause of spontaneous VAD in a susceptible individual.

New, sudden onset, severe suboccipital neck pain and occipital headache are characteristic symptoms of VAD.[21] These symptoms began in immediate temporal proximity to moshing, not to the CSM. The most probable cause of the VAD is the mosh pit neck injury which occurred one week prior to the CSM and the stroke.

The vascular neurologist documented that the initial injury was exacerbated a week later by chiropractic manipulation. It was their opinion that CSM aggravated an existing dissection, it did not cause the dissection. Research supports that patients with neck pain and headache from an existing dissection may seek care from chiropractors.[7–10,22,23]

MOST PROBABLE CAUSE OF STROKE

Causation can be established as more likely than not if plausibility, temporality, and lack of a more probable alternative explanation are present.[14–16] These three criteria are met to establish causation of stroke by CSM. (Table 2)

Table 2: 3-step causation analysis

Criteria	Description
1. Biological plausibility	It is plausible that CSM done in the presence of existing vertebral artery dissection can cause immediate thromboembolic stroke.
2. Temporality	There was an immediate temporal association between cervical spine manipulation and the onset of ischemic symptoms.
3. Lack of a more likely alternative explanation	There is not a more likely alternative explanation for the cause of the immediate post-manipulative stroke in this case.

Criteria 1: Plausibility

It is biologically plausible that CSM performed in the presence of an existing VAD can cause an immediate thromboembolic stroke. Sudden neck movement from CSM can dislodge an existing vertebral artery thrombus which can embolize to the brain causing thromboembolic ischemic stroke. This causal mechanism does not require that CSM cause the dissection.[24]

The plausibility of this mechanism of causation is supported by chiropractic, physical therapy, epidemiology and neurology researchers.[3,7,10,25–40] Supportive studies include case reports, case series, narrative reviews, a biomechanical study, epidemiological studies, and a systematic review.

Due to the life-threatening nature of dissection and stroke, randomized controlled trials (RCTs) to establish causation are unethical.[13] In the absence of RCTs, probabilistic reasoning and inference using the best available evidence to assess risk must be relied on.[14–16]

Allegedly contrary epidemiological studies

Four epidemiological studies on the association of CSM and stroke have been done by chiropractic researchers.[7-10] While these studies suggest low overall risk, they have important limitations when applied to immediate post-manipulative stroke (as noted in the Introduction). These four key studies did not evaluate an immediate exposure period and did not evaluate DC versus PCP visits in the 0–1-day

window.[11,12] They are also less applicable to the population most at risk for vertebral and internal carotid artery dissection (patients under 45 years of age).[8,10] The Cassidy et al. (2008) and Kosloff et al. (2015) studies have been critically reviewed by neurologists and epidemiologists for case misclassification.[38,41,42]

Allegedly contrary canine study

A canine study found CSM did not alter the dimensions of vertebral artery lesions in anesthetized dogs.[43] However, this animal study had substantial limitations. Researchers investigated vertebral artery lesions at the C4 level, not the C0-C2 level, the site of the commonly affected V3 segment in humans. Moreover, the time from the creation of the arterial lesion to the CSM was not documented but appears to be short. Many strokes following CSM occur weeks after the onset of symptoms of VAD, likely because a healing thrombus becomes more loosely adherent and easier to dislodge and embolize to the brain.

Criteria 2: Temporality

There was an immediate temporality between CSM and the onset of ischemic symptoms. In the majority of cases of stroke following CSM, there is an immediate temporality.[2–6] This temporality is dismissed by chiropractic researchers. As noted, the four epidemiological studies led by chiropractic researchers did not evaluate an immediate exposure period.[7–10]

Criteria 3: Lack of a more probable alternative explanation

Several alternative explanations for the cause of the stroke in this case were considered.

Alternative Explanation 1: Protopathic bias (reverse causation)

Protopathic bias is purported to explain the association between CSM and stroke.[9] Protopathic bias occurs when an exposure (CSM) is delivered before a condition (VAD) has been diagnosed.[44] When the condition (VAD) progresses to another condition (stroke) and the two conditions are later diagnosed at the same time, the assumption is that the initial exposure caused them both.

Such protopathic bias, sometimes referred to as reverse causation, would explain the association of CSM and VAD. Rather than CSM causing the dissection, the neck pain and headache from the dissection cause the patient to seek CSM for those symptoms. (Table 3)

Table 3: Protopathic bias/reverse causation in cases of CSM, VAD and stroke

Assumed causal pathway due to bias	CSM → VAD → stroke
Actual causal pathway	VAD → neck pain & headache → CSM → stroke

However, protopathic bias does not explain the association of CSM and immediate stroke. Dissection and stroke are separate medical conditions; they are not one clinical entity. VAD is a tear in the inner lining of a vertebral artery that causes neck pain and headache.[17] Stroke is a loss of blood supply to the brain which causes ischemic symptoms.[45] While dissection can lead downstream to stroke, dissection is not a stroke.

Alternative explanation 2: Toxin release

After the stroke occurred the chiropractor explained, "This happens all the time". Numerous chiropractors believe that patients experience symptoms of dizziness, vertigo, nausea, vomiting, and sweating as the result of detoxification which is a result of spinal manipulation. However, this is no evidence to support this hypothesis.[46]

Alternative explanation 3: Coincidence/ spontaneous event

Another alternative explanation is that it is a coincidence that the stroke occurred immediately after CSM. However, on the day of the stroke, VAD had likely been present for a week. It was only immediately after CSM that ischemic symptoms

began, and VAD evolved into a stroke. It is unlikely that an existing VAD coincidentally evolved into an ischemic stroke immediately after CSM. Smith, et al. noted, "It is highly improbable that a young patient will have a stroke and have had SMT [spinal manipulative therapy] within seconds purely by chance given the relatively low frequency of both events."[31]

Standard of care analysis

Analysis reveals two breaches in the standard of care by the chiropractor that directly contributed to the poor outcome in this case. Both breaches relate to diagnosis. Careful attention to differential diagnosis could have prevented this case.

BREACH 1: FAILURE TO CLINICALLY DIAGNOSE & REFER VERTEBRAL ARTERY DISSECTION

The chiropractor breached the standard of care by failing to formulate a differential diagnosis including VAD and refer the patient to the ED for definitive diagnosis. But for this failure, the VAD could have been diagnosed and treated, likely avoiding the stroke.

Failure to perform an Evaluation & Management service

An Evaluation & Management service should have been performed.[47] The cursory examination documented by the chiropractor does not consist of an Evaluation & Management service. No procedure code for an Evaluation & Management service was documented.

Failure to perform a thorough history

A thorough history taking, especially regarding the time of symptom onset, is the single most crucial factor for detecting symptoms of VAD. Therefore, physicians must perform a thorough history of present illness and physical examination before performing CSM.[18,48–51]

The patient presented with the following symptoms of potential VAD: New, sudden onset, sharp, constant, bilateral suboccipital neck pain (7/10) and dull, intermittent, bilateral occipital headache (3/10) noted bilaterally. Started around a week ago. Getting worse. Sudden onset of pain is characteristic of VAD. Neck pain and headaches that are not resolved with over-the-counter and prescription medications are a risk factor for VAD. The chiropractor took no history of medication use. A headache lasting more than 72 hours is not likely to be a migraine or an episodic tension headache and requires further evaluation prior to performing CSM.[52]

The chiropractor did not perform a thorough history of the time of onset of the patient's symptoms. "Dancing at a concert" is not a thorough description of the onset of symptoms. "Dancing in a mosh pit at a rock concert" is a distinctly different type of symptom onset. During history taking, follow-up of the patient's answers in relation to their new neck pain and headache is extremely important. The physician must insist on detailed information.[18]

Absence of vital signs examination

No vital signs, including blood pressure, were obtained. Blood pressure is clinically indicated when symptoms of potential VAD are present.[18,48,49] Even in the absence of symptoms of potential VAD, chiropractic practice management guidelines for the treatment of neck pain recommend vital signs as part of a physical examination.[47]

Symptoms of potential vertebral artery dissection

There are five distinct symptoms of potential VAD which should warrant referral to the ED. If a patient has two or more of these symptoms, they should be referred for emergency medical treatment.[18] The five symptoms are:

1) Recent head, neck, or thoracic trauma
2) New ipsilateral sub-occipital neck pain
3) Distinct, new, and continued headache
4) Brainstem ischemic symptoms:
    a) Ipsilateral loss of pain and contralateral temperature sensation in the body
    b) Ipsilateral hemiparesis
    c) Nausea
    d) Vomiting
    e) Vertigo

f) Nystagmus
g) Diplopia
h) Dysphagia
i) Dysarthria
j) Dysphonia

5) Cerebellar ischemic symptoms:
   a) Ataxia
   b) Vertigo
   c) Nystagmus

The patient presented with three of these distinct symptoms (symptoms 1, 2 & 3) of potential VAD, and should have been referred to medical emergency without further evaluation or physical testing.[18]

Physical tests for potential vertebral artery dissection

There are four positive physical tests for potential VAD that should warrant immediate referral to the ED. If a patient has two or more of these positive physical tests, they should be referred for emergency medical treatment.[18] The four physical tests are:

1. Evaluation for cervical radiculopathy (C5-C6)
2. Evaluation for a hypertensive blood pressure reading (>140/90)
3. Evaluation for neck swelling
4. Evaluation for midline tenderness suggestive for a fracture

The patient presented with the first of these four positive physical tests for potential vertebral artery dissection. The chiropractor failed to evaluate the other three.

1. The chiropractor documented positive Maximum Compression Test bilaterally, and moderate taut and tender fibers at C5-C6 bilaterally. These are positive physical tests that can indicate C5-C6 cervical radiculopathy. V2 segment VAD can cause cervical radiculopathy if the expanding hematoma compresses the adjacent spinal nerve roots.[53]
2. The chiropractor failed to evaluate for a hypertensive blood pressure reading. The ED documented a blood pressure of 132/99, Stage 2 Hypertension.[54] VAD can cause a hypertensive blood pressure reading through several mechanisms, including pain-induced sympathetic activation, vascular inflammation, or ischemic effects on brainstem centers that regulate blood pressure.[55]
3. The chiropractor failed to evaluate for neck swelling. VAD can cause swelling in the occipital and sub-occipital areas.
4. The chiropractor failed to evaluate for midline tenderness suggestive for a fracture. VAD can cause occipital tenderness over the mid nuchal line.[18,55]

Good outcome with routine clinical treatment

Had the patient been referred to the ED prior to the stroke, the chances of avoiding the stroke were high. Morris et al. found that 98.3% of patients did not develop a stroke in the first 12 weeks following diagnosis of dissection.[56] In general, patients with VAD have relatively good outcomes when treated in routine clinical fashion.[57]

BREACH 2: FAILURE TO CLINICALLY DIAGNOSE & REFER STROKE

The chiropractor breached the standard of care by failing to clinically diagnose symptoms of ischemic stroke and immediately refer the patient to the ED for definitive diagnosis.[18] Immediately after CSM, the patient had the following symptoms of cerebral ischemia: dizziness, dysarthria, diaphoresis, nausea, numbness and ataxia.

With an immediate referral, the patient could have had emergency medical treatment sooner and had a better prognosis from the stroke. There was an ED approximately 700 yards from the office where the stroke occurred.

The two-hour delay caused by the chiropractor decreased the window for tPA treatment after ED admission. If the chiropractor had the patient medically transported to the ED with notification of a possible post-manipulative stroke, CTA neck imaging may have been done earlier within the tPA

treatment window. Unfortunately, CTA neck examination was not performed until after the tPA window.

Other contributing factors

Although the chiropractor's failure to clinically diagnose symptoms of VAD and stroke represents the primary preventable breach in this case, additional delays in the ED also adversely affected the outcome. Despite the patient presenting with acute neurological symptoms immediately following CSM — a known risk factor for VAD and stroke — CTA of the neck was not performed until approximately three hours after arrival. This delay, combined with the earlier two-hour delay attributable to the chiropractor, resulted in the patient falling outside the 4.5-hour window for tPA.

Furthermore, the initial neuroradiology interpretation failed to identify the bilateral VADs, which were only recognized six hours later upon review by the neurology resident. Despite presenting to the ED with multiple symptoms of potential stroke, including bilateral lower extremity paralysis, a Brain MRI was not performed until nine hours after admission. These cumulative delays further diminished the patient's chances of a more favorable recovery.

Exclusion of vertebral artery dissection

There are clinical examination strategies to exclude VAD before performing CSM which could have prevented this unfortunate case and could prevent future cases.[18,48–51] Several case reports document chiropractors making a clinical diagnosis of VAD which was confirmed by advanced imaging.[58–60]

The scope of practice for chiropractors is most often limited to neuromusculoskeletal conditions. Chiropractic education normally lacks residency training and provides limited experience in the clinical diagnosis of vascular conditions. However, since patients experiencing neck pain and headache from dissection may present to chiropractors,[7–10,22,23] more training and experience in the clinical diagnosis of vascular conditions is recommended for chiropractic students and practitioners .[61,62]

Limitations

This is a single-patient case report, as opposed to a case series, which limits generalizability to broader populations. Post-manipulative events were recounted by the patient and a friend, introducing potential recall bias and post-event rationalization.

Conclusion

Forensic causal analysis demonstrates that causation of VAD by CSM cannot be established, as VAD was likely pre-existing due to prior trauma and underlying risk factors. However, causation of the immediate thromboembolic stroke by CSM is established as more likely than not, based on plausibility, temporality, and the absence of more probable alternative explanations. The unfortunate outcome highlights the potential for serious adverse events from CSM in patients with contraindications to spinal manipulation.

Adherence to established standards of care in evaluation and diagnosis could have prevented this negative outcome and can prevent similar occurrences in the future. Training in the clinical diagnosis of VAD is recommended for chiropractors.

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