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Causal analysis of vertebral artery dissection and fatal stroke following chiropractic cervical spine manipulation

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ABSTRACT

A 34-year-old female suffered a fatal stroke 7.5 h after cervical spine manipulation (CSM) performed by a chiropractic physician. Imaging noted vertebral artery dissection (VAD), basilar artery occlusion, and thromboembolic stroke. The medical examiner opined that CSM caused the VAD which embolized to cause the fatal stroke. However, causation of VAD by CSM is not supported by the research.

We utilized an intuitive approach to causation analysis to determine the cause of the VAD and the stroke. Causation of the VAD and the stroke by CSM could not be established as more likely than not. The malpractice case was settled by bringing allegations of misdiagnosis and failure to diagnose and refer the VAD to medical emergency.

We conclude that in the absence of convincing evidence that CSM could cause VAD, forensic professionals should consider VAD as a presenting symptom prior to CSM in such cases. Adherence to the standard of care for the chiropractic profession with attention to differential diagnosis could prevent such cases.

Case presentation

Case information was taken from publicly available documents in the 11/24/2020 Appendix of Declarations and Evidence in Support of Plaintiff's Opposition to Defendant's Motion for Summary Adjudication [1]. These documents include the declaration of the plaintiff chiropractic expert, transcript of the deposition of the defendant, chiropractic records, hospital records, and a certified copy of the autopsy report. Background information was taken from publicly available investigative journalism and media coverage of this case [2,3].

The decedent was a 34-year-old female professional model who presented to a chiropractic physician for diagnosis and treatment on 1/29/2016. Her height was five feet and weight 110 pounds with a body mass index (BMI) of 21.5.

She reported a five-day history of sudden onset, worsening, severe, constant, dull, left suboccipital neck pain and left occipital headache. Pain level 8/10. She also reported nausea. Symptoms were not relieved by anything and affected all her daily activities.

The chiropractor documented that her symptoms began with a neck injury five days earlier on a 1/25/2016 photo shoot. Symptoms occurred when the photographer asked her to hold a pose for a long time that involved arching her back and leaning her neck to the side [2]. The decedent's hair and makeup assistant, personal assistant, and the father of her daughter all confirmed this neck injury. On 1/29/2016 she posted on Twitter that, "Pinched a nerve in my neck on a Photoshoot and got adjusted this morning."

Physical examination findings included cervical spine range of motion (ROM) decreased with pain. Palpation revealed very tender muscle spasm noted in the left cervical spine. Segmental dysfunction noted at C2, C3, C4, C5, and C7. Cervical Distraction relieved the patient's neck pain. Cervical/Jackson Compression was negative. No vital signs or neurological examination were performed. No imaging was ordered or considered. No differential diagnosis was formulated. The chiropractor diagnosed migraine and cervicalgia. Treatment consisted of cervical spine manipulation (CSM), therapeutic ultrasound and therapeutic exercises.

The decedent returned on 2/1/2016. The chiropractor documented a 50 % improvement with increased cervical spine ROM. Pain level was documented as 5/10. Physical examination findings included decreased cervical spine ROM with mild-moderate spasm and mild pain. No vital signs, orthopedic examination, or neurological examination were performed. Diagnosis was again migraine and neck pain. However, an additional diagnosis of torticollis was documented. Treatment consisted of CSM, muscle stimulation, and therapeutic ultrasound.

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The chiropractor did not document the time of the second chiropractic visit. However, the decedent's personal assistant said she talked to the decedent not long after she got home from the chiropractic office around 10:00 am [2]. There was no record of the decedent's activities from 10:00 am to 5:30 pm.

At 5:30 pm, emergency department records document that the decedent suddenly developed slurred speech, vertigo, and left-sided weakness of the arm and leg. She called a friend who transported her to the emergency department.

At the ED, she initially showed significant improvement with IV tissue plasminogen activator (tPA) treatment. At 8:45 pm, Head & Neck CTA (computed tomography angiography) noted bilateral V3 segment VAD and basilar artery occlusion. Prior to DSA (digital subtraction angiography), when she was sat up to evaluate for hemodynamic instability, she started having speech difficulty again. During the DSA procedure, bilateral VADs were noted with left worse than right. She was found to have a total occlusion of the left vertebral artery and basilar artery trunk. Thrombectomy of the left vertebral artery and basilar trunk was successfully performed. However, after the procedure, the decedent exhibited locked-in syndrome from which she never recovered.

On 2/3/2016 the decedent was diagnosed with brainstem death. Brain CT showed acute infarct of the right cerebellar hemisphere, pons, medial right temporal lobe and left occipital lobe.

On 2/4/2016 the decedent was taken off life support and pronounced dead. The medical malpractice lawsuit filed in this case was settled in January of 2022 for $250,000 [3]. Case events are summarized in Table 1.

Autopsy Report

In the autopsy report, the medical examiner opined that CSM caused the VAD which later embolized to cause thromboembolic stroke and death. The following paragraph is the opinion of the medical examiner:

"The cause of death is due to infarction of the brain due to vertebral artery dissection due to blunt force injury of neck. Initial reports are of an injury of the neck resulting in neck pain for which she sought treatment at Back to Health Wellness Center. After a neck adjustment on 2/1/2016 the decedent complained of dizziness, headache, and weakness. Once admitted to Cedars Sinai Hospital, she was diagnosed with bilateral vertebral artery dissections. Despite treatment with tissue plasminogen activator and thrombectomy of the left vertebral artery with Solitaire device (Flow restoration device) the decedent suffered a stroke and was pronounced dead on 2/4/2016 at 1644 h. Bilateral vertebral artery dissection is a rare complication of neck manipulation reported in one per 100,000 to one in 2 million manipulations (South Med J. 2007 Feb; 100(2):201–3) [1]."

To support their opinion that, "bilateral vertebral artery dissection is a rare complication of neck manipulation", the medical examiner referenced one 2007 case report [4] and mischaracterized that study. The 2007 case report cites a 2003 editorial [5] suggesting a rate of stroke in one per 100,00 to one in 2 million manipulations. The 2003 editorial does not mention bilateral VAD. To refer to VAD as a "complication" of neck manipulation is to suggest a causal connection. Although it is accurate to say that VAD has been temporally associated with CSM, there is no convincing evidence of a causal association [6].

Table 1
Case Timeline.

Date	Case Event
1/25/2016	Neck injury with onset of new left neck pain, left headache & nausea
1/29/2016	Cervical spine manipulation
2/01/2016 (AM)	Cervical spine manipulation
2/01/2016 (PM)	Onset of ischemic symptoms 7.5 h after cervical spine manipulation
2/04/2016	Death
1/2022	$250,000 malpractice case settlement

In this 2016 autopsy report, the medical examiner failed to reference the literature which does not support a causal link between CSM and VAD. Multiple biomechanical studies performed prior to 2016 on healthy cadaveric vertebral arteries support that healthy vertebral arteries are very unlikely to suffer intimal tearing due to manipulation [7–10]. More recent 2023 biomechanical research suggests that vertebral arteries do not experience any tensile force during CSM [11]. There is no evidence that CSM is a "blunt force injury of the neck" as the medical examiner claimed.

2008 and 2015 epidemiological studies found that in cases of vertebrobasilar artery stroke following CSM, the patient likely had an existing VAD before the CSM [12,13]. A 2014 literature review found that biomechanical evidence is insufficient to establish the claim that CSM causes VAD in healthy arteries and recommended that practitioners should strongly consider VAD as a presenting symptom. [14]. This study was published on behalf of the American Heart Association Stroke Council and was endorsed by the American Association of Neurological Surgeons and Congress of Neurological Surgeons. In a 2016 systematic literature and meta-analysis found no convincing evidence to support a causal link between CSM and CAD in healthy arteries [6]. This study was authored by a group of six neurosurgeons from Penn State Hershey Medical Center.

Objectives

Thus, as regards the mechanism of causation of VAD, there is a discrepancy between the opinion of the medical examiner and the research. The objectives of this case report were to:

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

Discussion

Methods

As this case was straightforward, we evaluated causation with an intuitive approach [15]. There is only one plausible mechanism of causation of VAD based on the research and the facts of the case. Likewise, there is only one plausible mechanism of causation for the stroke, as well.

Formulation of the causal questions to be investigated

1. What is the most likely cause of VAD in this case?
2. What is the most likely cause of stroke in this case?

Consideration of examination findings, injury/pathophysiologic mechanism, and predictive demographics and history

Most likely cause of left VAD: Spontaneous VAD and can occur with minor neck movements in the absence of any severe trauma [16]. Environmental risk factors for spontaneous VAD include recent acute infection (mainly respiratory), use of fluoroquinolone antibiotics [17], hyperhomocysteinaemia (B6, B9 and B12 vitamin deficiency), low BMI, low cholesterol, smoking and pulsating tinnitus. Inherited risk factors for spontaneous dissection include arteriopathies such as fibromuscular dysplasia and connective tissue disorders such as Ehlers-Danlos syndrome type IV [18].

The decedent had risk factors for spontaneous VAD. She was 34 years old. Most VADs occur in people less than 45 years of age [16]. The decedent had a BMI of 21.5, which is considered to be within the normal range. However, it is significantly less than the average BMI for an adult female in the United States, which is 29.8 [19]. A low BMI is an environmental risk factor for VAD.

It is plausible that the 1/25/2016 modeling pose could cause a spontaneous left VAD. Holding a modeling pose is a minor neck movement that could cause spontaneous VAD in a susceptible individual. New, sudden onset, severe, left sub-occipital neck pain, severe left occipital headache, and nausea are characteristic symptoms of left VAD [20]. These symptoms began in immediate temporal proximity to the 1/25/2016 neck injury, not to the 1/29/2016 or 2/1/2016 CSM. The most likely cause of the left VAD is the 1/25/2016 neck injury.

Most likely cause of right VAD: The immediate consequence of right V3 segment VAD would most likely be right suboccipital neck pain and/or right occipital headache [21]. However, there was no record of these symptoms in this case.

The absence of documented symptoms of right VAD may be due to the poor chiropractic documentation. In deposition, the chiropractor stated that the chart note for the 2/1/2016 date of service was completed four days later, on 2/5/2016, after learning of the 2/4/2016 death of the patient. The chart note states: "This note is done for the treatment performed on February 1st, 2016. I had forgotten to do the note for that day and so I am documenting what I did on that day." The chiropractic documentation is also poor and incomplete in other aspects.

Lee et al. reported two cases of VAD that were asymptomatic at the time of neuroimaging, with the neuroimaging being performed for unrelated reasons [22]. However, Lee et al. did not determine the time of onset, and did not determine that the VAD was asymptomatic at the time of onset. VAD may become asymptomatic as the arterial wall heals, but it is unlikely that a VAD would be asymptomatic at the time of onset [16].

In the absence of any documented symptoms to determine the onset of the right VAD, the age of dissection thrombi can be determined by pathological changes noted during autopsy [23]. In this case, death occurred 10 days after the onset of the symptoms of left VAD. A certified copy of the autopsy report was reviewed. Microscopic review of the left and right vertebral artery thrombi noted the same fibrin deposition with entrapped acute inflammatory cells and red blood cells in both vertebral arteries. The autopsy report did not note that either artery had pathological changes that were more or less chronologically advanced than the other.

Based on the microscopic review of the artery thrombi, it seems plausible that both the left and right VADs were the same age. Therefore, it is likely that both VADs were caused by the 1/25/2016 neck injury. As the left VAD was found to be more severe during the 2/1/2016 DSA procedure, it is plausible that it may have been more painful and overshadowed symptoms from the right VAD.

Most likely cause of stroke: VAD thromboembolism caused by the sudden head and neck movement of CSM would likely be immediate, with ischemic symptoms of stroke occurring within seconds or minutes of CSM [24]. Ischemic symptoms did not begin until approximately 7.5 h later. This makes it highly unlikely that the CSM caused the stroke.

The decedent had the onset of symptoms of VAD one week prior to the 2/1/2016 CSM. The first symptoms of stroke occurred approximately 7.5 h after the CSM. Thus, there is a hazard period of 7.5 h. One week represents 168 h, so the hazard period represents only 4 % of the time during which the stroke may have occurred due to thromboembolism from the VAD. This also makes it highly unlikely that the CSM caused the stroke.

In cases of extracranial VAD, the prognosis is good with complete recovery in 80–90 % of patients [16]. The left and right V3 segment VADs were both extracranial in this case. Thus, there was a very low probability of stroke during the 7.5 h hazard period in the absence of CSM.

The most likely cause of the 2/1/2016 stroke was thromboembolism from the left VAD to the basilar artery 7.5 h after CSM which was unrelated to the CSM.

The question of temporal proximity

Analysis of the causation of stroke raises the question of the temporal proximity between CSM and stroke. "If the sudden head and neck movement from CSM dislodged a loosely adherent thrombus from a VAD, how long would it take for the thrombus to embolize to the brain and impede blood flow in order to cause ischemic symptoms of stroke?" Leading to the following question, "How long is too long from CSM to ischemic symptoms of thromboembolic stroke to establish a temporal relationship as more likely than not?"

There is nothing in the research that addresses this question directly. However, based on the pathophysiology of vertebral artery thromboembolic stroke [25] and published case reports, [26–28] it is likely that ischemic symptoms would occur within seconds or minutes of CSM. A thrombus could embolize from the VAD to the brain in a matter of seconds, although it could take several minutes for the embolus to become positioned in such a way that it impedes blood flow to the brain. A embolus that did not occlude blood flow after several minutes would likely fragment and disappear [23].

Beyond seconds or minutes, it becomes increasingly more likely that there could be a more probable explanation for the cause of the thromboembolism. A loosely adherent thrombus could be dislodged by any minor neck movement [16]. After 7.5 h further neck movements could be a more probable alternative explanation for the cause of vertebral artery thromboembolism. In the case of a sufficiently loosely adherent thrombus, blood flow past the VAD could dislodge the thrombus.

Therefore, there must be a close temporal proximity of the CSM to the onset of ischemic symptoms of stroke to make the causal association plausible. Vertebral artery thromboembolic stroke and CSM both occur with a relatively low frequency. It is highly improbable that a young patient will have a stroke and have had CSM within seconds or minutes purely by chance given the relatively low frequency of both events [29].

The criteria of a close temporal proximity between CSM and ischemic stroke has been used in other causal analyses. In a review study of ten case reports, causation of stroke by CSM was determined to be more likely than not in four case reports where there was a close temporal proximity between CSM and onset of ischemic stroke symptoms [30].

Outcome of the malpractice case

In contrast to the opinion of the medical examiner, the plaintiff did not bring allegations of causation of VAD or stroke by CSM in the malpractice case. The plaintiff brought allegations of misdiagnosis and failure to diagnose and refer VAD to medical emergency which resulted in settlement of the case.

Medicolegal analysis of the standard of care

A medicolegal analysis of the standard of care reveals three breaches in the standard of care for the chiropractic profession, all in the area of diagnosis. Careful attention to differential diagnosis could have prevented this case.

Misdiagnosis of migraine

On 1/29/2016 and 2/1/2016, the chiropractor breached the standard of care for the chiropractic profession when they misdiagnosed the decedent with migraine and failed to diagnose characteristic symptoms of left VAD and refer the patient to medical emergency. But for this failure to diagnose and refer, the decedent could have had emergency medical care which could have prevented the stroke of 2/1/2016.

The chiropractor diagnosed the decedent with migraine on 1/29/

2016. The physician used diagnosis code G43.109, "migraine with aura, not intractable, and without status migrainosus." However, there is no documentation that the decedent had a migraine aura, the pain was intractable (constant for more than 72 h), and if it was a migraine, she did have status migrainosus (constant for more than 72 h).

The headache caused by VAD can resemble migraine headaches with its unilateral pain location. However, migraine headaches are not characterized by pain in the upper cervical region [18]. Headaches lasting longer than 72 h are not likely to be a migraine [31].

There was no documentation that the decedent had any prior history of migraines. Even if the patient did have a migraine, and a history of migraines, migraine is a risk factor for VAD [32]. Therefore, even if migraine was present, VAD should have been included in a differential diagnosis.

Misdiagnosis of torticollis

On 2/1/2016, the chiropractor breached the standard of care for the chiropractic profession when they misdiagnosed the decedent with torticollis and failed to diagnose characteristic symptoms of left VAD and refer the patient to medical emergency. But for this failure to diagnose and refer, the decedent could have had emergency medical care which could have prevented the stroke of 2/1/2016.

The chiropractor diagnosed the decedent with torticollis on 2/1/2016, but not on 1/29/2016. It is not likely that the decedent coincidentally developed torticollis in the 72 h after her last treatment. It is more likely than not that her neck was in an antalgic position due to severe left neck pain from the VAD.

Torticollis is a neurological condition of cervical dystonia; it is not an antalgic neck position due to pain. The standard of care when diagnosing torticollis is that cervical spine x-rays should be ordered to rule out bony abnormality. Cervical spine MRI should be ordered if there is a concern about structural problems or other conditions. In this case, the chiropractor should have had a concern about VAD, as the decedent's history, subjective findings and objective findings all put VAD within her differential diagnosis.

An antalgic neck position severe enough to be misdiagnosed as torticollis was not documented on 1/29/2016. Therefore, it is likely the decedent's condition was worsening three days later on 2/1/2016, not showing a 50 % improvement as the chiropractor documented.

Failure to diagnose and refer left vertebral artery dissection

On 1/29/2016 and 2/1/2016, the chiropractor breached the standard of care for the chiropractic profession when they failed to diagnose characteristic symptoms of left VAD and refer the patient to medical emergency. But for this failure to diagnose and refer, the decedent could have had emergency medical care which could have prevented the stroke of 2/1/2016.

In general, individuals with VAD have relatively good outcomes when treated in a routine clinical fashion [33]. With diagnosis and treatment, VAD rarely progresses into stroke. When VAD is diagnosed and referred for emergency medical care, the chance of avoiding stroke is almost 100 % [34].

Neck pain and/or headache are common presentations to chiropractic offices. However, neck pain and headache from VAD has a characteristic presentation. VAD is characterized by new, sudden onset, suboccipital neck pain and ipsilateral occipital headache. Nausea may also be present due to severe pain or as a symptom of brainstem ischemia (stroke) [18].

The patient presented to the chiropractor with a five-day history of constant, severe, worsening, dull, left suboccipital neck pain, left occipital headache, and nausea that was not relieved by anything, and affected all her daily activities. The pain was of sudden onset and the result of neck injury five days earlier.

VAD should have been considered in a differential diagnosis. However, the chiropractor failed to formulate a differential diagnosis. Research supports that VAD should be considered in the diagnostic assessment of patients presenting with neck pain and headache, even in the absence of other risk factors [33].

Symptoms of potential vertebral artery dissection

There are five distinct symptoms of potential vertebral artery dissection which should warrant referral to the medical emergency department. 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 (weakness on side of the body)
c) Nausea (urge to vomit)
d) Vomiting
e) Vertigo (dizziness)
f) Nystagmus (uncontrolled, repetitive eye movements)
g) Diplopia (double vision)
h) Dysphagia (difficulty swallowing)
i) Dysarthria (difficulty speaking)
j) Dysphonia (abnormal voice)

5) Cerebellar ischemic symptoms:

a) Ataxia (lack of voluntary coordination of muscle movements)
b) Vertigo (dizziness)
c) Nystagmus (uncontrolled, repetitive eye movements)

The decedent had four of these distinct symptoms of potential vertebral artery dissection (1, 2, 3, 4c) and should have been referred for medical emergency treatment prior to any physical testing or treatment.

Recommendations

Chiropractors and other manual therapists who may perform CSM should identify higher risk patients prior to performing CSM. Clinical examination strategies to exclude VAD before performing CSM have been published by researchers from the chiropractic, [18,35] medical, [18] and physical therapy [35,36] professions. Adherence to the standard of care and utilization of these clinical examination strategies would have prevented this tragic case, and could prevent future cases, as well.

Limitations of the analysis

Case information was taken from publicly available court documents [1]. Background information was taken from publicly available investigative journalism and media coverage of this case [2,3]. Any information that has not been made public is not reflected in this analysis.

A certified copy of the autopsy report was reviewed. However, images of the forensic microscopic review of the vertebral arteries were not available for review.

Conclusions

Causal analysis of this case reveals that causation of VAD and stroke by CSM could not be established as more likely than not. The plaintiff settled the case by bringing allegations of misdiagnosis and failure to

diagnose and refer VAD to medical emergency. We conclude that in the absence of convincing evidence that CSM can cause VAD, forensic professionals should consider VAD as a presenting symptom prior to CSM in such cases. Adherence to the standard of care for the chiropractic profession with attention to differential diagnosis could prevent such cases.

CRediT authorship contribution statement

Steven P. Brown: Writing – review & editing, Writing – original draft, Investigation, Conceptualization.

Declaration of Competing Interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: The author provides consultation on medicolegal matters including cervical artery dissection and stroke diagnosed following cervical spine manipulation.

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