The Relationship of Chiropractic Neck Manipulation to Internal Carotid Artery Dissection

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Key terms: cervical manipulation, internal carotid artery, dissection.

Abstract: Forensic experts may be requested to offer an opinion regarding the relationship between cervical chiropractic manipulative therapy (CMT) and subsequent internal carotid artery dissection (ICAD). For the most part, the medical literature contends that cervical CMT may be a cause of ICAD. The proposed mechanism of traumatically induced ICAD involves a sudden, severe stretch of the internal carotid artery (ICA) over the upper cervical spine when the neck is hyperextended and laterally flexed or rotated to the opposite side. However, most ICADs are spontaneous, with no identifiable pre-morbid trauma. In cases where adverse symptoms of ICAD did not occur within a relatively close temporal relationship to the cervical CMT, there will always be doubt about its connection with ICAD. Patients who have an associated primary arterial disease, which is often present in ICAD patients, are at risk when subjected to virtually any sort of head or neck motion.

INTRODUCTION

Forensic experts are occasionally called upon to offer opinions regarding the relationship between chiropractic manipulative therapy (CMT) of the neck and subsequent pathological conditions, the most common associated condition being intervertebral disc herniation (Hurwitz, Aker, Adams, Meeker, & Shekelle, 1996). Another condition that has been suggested to follow CMT is internal carotid artery dissection (ICAD). Indeed, the medical literature commonly contends that cervical CMT may be a cause of ICAD (Lee, Carlini, McCormick, & Albers, 1995; Norris, Beletsky, & Nadareishvili, 2000; Beatty, 1977; Parenti, Orlandi, Bianchi, Renna, Martin, & Murri, 1999). The first report of this relationship was made by Beatty in 1977, who described a patient who was manipulated by a chiropractic physician for neck pain. Symptomatic relief resulted after the CMT, and there were no ill effects associated with the manipulation. Five days later, the patient awoke with right arm weakness and difficulty speaking due to the ischemic effects of ICAD. Beatty attributed the ICAD to the CMT, alleging that because a tear of the arterial intima was found at surgery, there necessarily must have been trauma, and cervical CMT was the only known “trauma” previous to the dissection. In contrast, several authors have more recently pointed out that tearing of the intima is very common, even in cases of spontaneous ICAD where no associated trauma can be found (Stapf, Elkind, & Mohr, 2000; Mokri, Sundt, Houser, & Piepgras, 1986; Terrett, 1996; Deck, 1987).

In our experience, most medical neurologists believe that CMT is an established cause of ICAD. Conversely, after reviewing over 100 papers on this subject, we concluded that the relationship between ICAD and CMT remains tenuous, and a causal relationship is not supported by the literature. CMT is often categorized in the medical literature alongside frank trauma, such as motor vehicle collisions (MVCs) (Mokri, Piepgras, & Houser, 1988; Lucas, Moulin, Deplanque, Tatu, & Chavot, 1998), which suggests that these authors have the perception that CMT is violent and may be injurious. This notion is in stark contrast to studies that have looked at the safety of cervical CMT (e.g., Hurwitz et al., 1996), that estimated the rate of vertebrobasilar accidents or other complications to be one per one million cervical manipulations.
One of the authors of the current paper (ACC) was a panelist on the RAND Corporation study that Hurwitz et al. authored.

MECHANISM OF INJURY

The proposed mechanism of traumatically induced ICAD involves a sudden, severe stretch of the internal carotid artery (ICA) over the upper cervical spine when the neck is hyperextended and laterally flexed or rotated to the opposite side (Stringer & Kelly, 1980; Fabian, Patton, Croce, Minard, Kudsk, & Pritchard, 1996). The above-mentioned neck positioning forces the ICA against the upper cervical vertebrae, allowing it to become fixed in place and stretched, thus becoming susceptible to injury (Lepojarvi, Tarkka, Leinonen & Kallanranta, 1988). This hypothesis of traumatic causation is based upon patient histories, as well as pathological and radiographic observations, although no validating animal or post mortem models have substantiated it.

Upon severe stretch, the intimal lining, being weaker than the media or adventitia, is more vulnerable to developing tears and is the usual site of injury (Stringer & Kelly, 1980; Lee & Jensen, 2000). The leading cause of traumatic ICAD is motor vehicle collisions (Wartridge, Muhlbauer, & Lowery, 1989), with one study reporting that 72% of their group’s traumatically induced ICADs were the result of automobile accidents (Cogbill, Moore, Meissner, Fischer, Hoyt, Morris, et al., 1994). However, most ICADs are spontaneous, with no identifiable pre-morbid trauma. Younger patients appear to be more susceptible to ICA injury leading to dissection because the path of the artery is more convoluted in the elderly. This tortuousness allows the artery to straighten rather than stretch when sudden hyperextension and rotational forces are applied (Davis & Zimmerman, 1983).

Concurrent pathological conditions are often associated with ICAD, and many consider them to be predisposing risk factors (see Table 1). Hypertension was present in 26% and 36% of ICAD patients in two series, which the authors determined was much more prevalent than in the general population (Mokri et al., 1986; Biousse, D’Anglejan-Chatillon, Massiou, & Bousser, 1994). Infection in the weeks prior to ICAD was considered a triggering factor by Constantinescu (2000) and Grau, Brandt, Forsting, Winter, and Hacke (1997). Fibromuscular dysplasia, a nonatherosclerotic and non-inflammatory vascular disorder affecting primarily the ICA and renal arteries, is present in up to 23% of ICAD patients (Luscher, Lie, Stanson, Houser, Hollier, & Sheps, 1987), making it the most frequently reported associated abnormality (Desfontaines & Despland, 1995; Goldstein, Gray, & Hulette, 1995). Barbour et al. (1994) found significant ICA redundancies, kinks, coils, or loops in 62% of their group of 13 ICAD patients, compared to 19% of 108 arteriograms without dissection. Other conditions reported to have been related to ICAD include Marfan’s Syndrome and cystic medial necrosis (Saeed, Shuaib, Al-Sulaiti, & Emery, 2000). Several authors have postulated that there is probably a yet to be identified underlying arteropathy that leads to ICAD (Hart & Easton, 1983; Schievink, Mokri, & Piepgras, 1994).

CLINICAL PICTURE

The most common presenting symptoms of ICAD are headache and neck pain, which are typically severe and of sudden onset. The headache is often described as being unique, or of different character than what had previously been experienced (Silbert, Mokri, & Schievink, 1995). Most commonly, the headache involves the ipsilateral periorbital, frontal, or upper cervical region. The second most common clinical manifestations are ischemic signs and symptoms, which include transient ischemic attacks (TIAs), stroke, or both, and less frequently, transient monocular blindness (Mokri et al., 1986). Another very common sign of ICAD is an incomplete Horner’s Syndrome that includes miosis and ptosis, but not anhydrosis (Anson & Crowell, 1991). Horner’s Syndrome may be painful, and when of sudden onset should at once point to carotid dissection (Biousse et al., 1994; see Table 2).

ICAD can be very difficult to diagnose and is often overlooked, especially in its early stages before ischemic signs are apparent (Treiman, Treiman, Foran, Levin, Cohen, Wagner, & Cossman, 1996). It may go undiagnosed in cases that only cause mild symptoms or are asymptomatic (Mokri, 1990). Often there is a delay between the moment in time that disruption to the intima occurs and the development of ischemic signs, which can span minutes, and has even been reported to span years (Davis & Zimmerman, 1983). Some researchers, when ascribing causation to CMT, have extended the temporal relationship to as long as two months following CMT.

INCIDENCE OF ICAD

The ICAD is the most common cervical artery to undergo dissection (Hart & Easton, 1983). In a study comprising 200 cases of cervicocephalic artery dissection reported by Schievink, Mokri, & O’Fallon, (1994), 74% were ICAD and 24% were vertebral artery dissection (VAD). Epidemiological studies have estimated the incidence of spontaneous ICAD as 2.6 and 2.9 cases per 100,000 population yearly, making the condition uncommon, but not rare (Schievink, Mokri, & Whisnant, 1993; Giroud, Fayolle, Andre, Dumas, Becker, Martin, et al., 1995). Authors of another series of 1,200 first strokes found that 2.5% were due to spontaneous ICAD (Bogousslavsky, Despland, & Regli, 1987). The more conservative

ASSOCIATION OF CMT WITH ICAD
The medical literature was thoroughly searched up to and including the year 2000 to locate reported cases of CMT-related ICAD. We were able to locate 12 cases where ICAD was reported to have occurred subsequent to what was referred to as spinal manipulation or chiropractic manipulation.

The 1977 article by Beatty was typical of what other reports have conveyed concerning ICAD following CMT. In Beatty’s case the patient complained of neck pain, a well-known early symptom of ICAD, to his chiropractic physician. It is possible that the patient was already in the process of developing ICAD prior to the CMT, which progressively developed into characteristic ICAD symptoms. However, the patient noticed symptom relief following CMT, which probably points more toward a musculoskeletal condition, because there is no plausible physiological explanation to explain how spinal manipulation might relieve the symptoms of ICAD. It is also possible that the CMT made an early ICAD worse, but Beatty did not mention any other post-manipulation sequelae occurring prior to the ICAD. As previously mentioned, the Beatty article was cited considerably more than any other when an author was referring to the relationship of CMT to ICAD.

Chiropractic manipulation was mentioned as being related to one of the ICAD cases in a study by Brandt et al. (1998). However, in addition to having CMT up to one month prior to ICAD, the patient was coughing and suffering flu-like symptoms, both regularly-mentioned precursors of ICAD. The patient also had the most severe category of connective tissue abnormalities that any of the group’s other 24 patients displayed, which the authors considered to be a possible indication of a primary arteriopathy.

Peters et al. (1995) described a case of ICAD that followed CMT by three hours. The patient had increasing neck pain and five episodes of vertigo prior to the manipulation. She was also a smoker, took oral contraceptives, and had segmental mediolytic arteriopathy with widespread mucoid degeneration. The authors hypothesized that the mediolytic arteriopathy was a predisposing factor for the dissection that followed CMT.

Hufnagel, Hammers, Schonle, Bohm, and Leonhardt (1999) reported 10 cases of stroke following CMT, with two of them involving the ICA. However, none of the practitioners were chiropractic physicians. More accurately, they mentioned that the manipulations were performed outside their institution by orthopedists in seven patients, a physiotherapist in one, and health practitioners who were not physicians in two. The outcome of these later cases resulted in full recovery in one of the patients, while the other maintained a permanent mild neurological deficit.

Murthy & Naidu (1988) described an ICA aneurysm purportedly following chiropractic spinal manipulation. However, the practitioner was actually a barber. Furthermore, the origin of the aneurysm was at the origin of the ICA, rather than more distally near C1/C2, where the ICA is reported to be fixed during hyperextension and rotation. Any intimal tear and subsequent aneurysm in relation to blunt trauma would be expected to occur at that level. The fact that the patient’s symptoms started so soon after the lay manipulation may have been related to a triggering effect acting on an already existing arterial disorder. The patient had a persistent Horner’s Syndrome as a result of the aneurysm.

Parenti et al. (1999) discussed a moderately hypertensive 50-year-old female patient who developed a sudden onset left occipital headache and a posterior circulation stroke within a few minutes following CMT for neck pain. The authors mentioned that MRI disclosed both vertebral artery (VA) and ICA dissections. However, the VA was intracranial and the ICAD was along its prepetrous tract. The specified location of ICAD described in this case was not in close proximity to the upper cervical vertebrae, which would cause one to question its connection to the manipulation. There is little, if any, evidence to support the notion that intracranial artery dissection can be related to CMT. Indeed, this is the only reported case that we encountered in our review of the literature. Pelkonen, Tikkakoski, Leinonen, Pyhtinen, and Sotaniemi (1998) did not.

Dragon, Saranchak, Lakin, and Strauch (1981) gave an account of an ICAD following manipulation that occurred two days after CMT. The patient had severe left neck pain that was ipsilateral to the ICAD and experienced a brief flashing light in the ipsilateral eye, which we inferred from the article’s context was in progress prior to CMT. The authors did not indicate the type of manipulative practitioner involved. The patient recovered without neurological deficit. This case of dissection may have already been in process at the time CMT was applied.

Lyness and Wagman (1974) described a patient who, within a few moments of being manipulated by an osteopathic physician, became paralyzed in all four extremities and was unable to speak or swallow. A subsequent arteriogram disclosed pathology affecting the basilar, right and left vertebral, and right internal carotid arteries. The authors mentioned the presence of a congenital anomaly affecting the vertebral arteries, which might have been a predisposing factor. In addition, multiple artery dissection may indicate the presence of an underlying arteriopathy in this case.

Lee, Carlini, McCormick, and Albers (1995) reported on a survey of California neurologists concerning strokes and other possible chiropractic related neurologic problems. They reported that three of their stroke cases were due to CMT-attributed ICAD. However, the researchers admitted that the study had many limitations. Some of these limitations were as follows: They did not in any way verify the results of the survey, the survey’s response rate was limited at 36%, only 45% of the cases were verified by angiography, the mechanism was unknown for some of the strokes, and they did not...
indicate if any of the ICAD cases were verified. Indeed, the study did not reliably represent the relationship of CMT to ICAD, Nevertheless, we did count Lee et al.’s three ICAD cases as part of the 12 that were considered.

Norris, Beletsky, and Nadareishvili (2000) reported findings of a series of 74 cervical artery dissections that they indicated were related to spinal manipulation in 28% of the cases. They did not provide sufficient data to determine how many, or if in fact any, of the cases were considered to be ICAD following cervical manipulation. Consequently, we were not able to consider in our tally any of the purported cases of ICAD mentioned by Norris et al.

CMT has been represented in the literature in the same category as frank trauma, such as MVCs, indicating a perception that CMT is violent and injurious (Mokri, Piepgras, & Houser, 1988). This notion is in stark contrast to studies that have looked at the safety of cervical CMT, such as Hurwitz et al. (1996), who estimated the rate of vertebrobasilar accidents or other complications to be one per one million cervical manipulations.

Only about two-thirds of the cases of CMT-attributed ICAD, in which the type of practitioner was identified, have been ascribed to chiropractic physicians, yet they perform 94% of all spinal manipulations (Shekelle, Adams, Chassin, Hurwitz, & Brook, 1992). This finding points to chiropractic’s relative safety, especially when compared to medical and lay manipulators who were involved in a substantial number of the reported ICAD cases, while only performing approximately 6% of all spinal manipulations.

Dubious implication of trivial trauma related to ICAD was evident in several of these papers, where all unusual neck movements were considered in the month preceding ICAD (Brandt et al., 1998; Ricchetti, Becker, and Dulguerov, 1999; Sturzenegger, 1995). A case of VAD described by Mas, Bousser, Hasboun, and Laplane (1987) portrayed a young woman who experienced right-sided neck pain, for which she received manipulation and consequently obtained relief. The author went on to state that the patient felt perfectly fine after the manipulation until the dissection occurred. She suffered the dissection 40 days after CMT, yet neck manipulation was identified as a premorbid trauma.

CONCLUSION

Ostensibly there may be a causal relationship in cases of head or neck pain, with ensuing ischemic signs that immediately follow or develop within hours following CMT, but due to the widespread uncertainty concerning ICAD etiology (Mokri et al., 1988; Cogbill et al., 1994; Giroud et al. 1994), doubt remains. While discussing cervicocephalic arteries in general, Norris et al. (2000) remarked that the “hallmark of dissection” was sudden and often severe neck or occipital pain. Absent some degree of adverse symptoms in relatively close temporal proximity to CMT, there will always be uncertainty about its connection with ICAD. Patients who have an associated primary arterial disease, which is often present in ICAD patients (Lucas et al. 1998), are at risk when subjected to virtually any sort of head or neck motion.

Lansley (1993) noted that there could be another explanation concerning cases of CMT-attributed VAD; the involved patients already had spontaneous dissection, giving rise to symptoms that led them to consult a chiropractor. Mas, Henin, Bousser, Chain, and Hauw (1989) demonstrated pathologically concerning VAD that cervical pain that precedes and motivates chiropractic cervical manipulation may be the first symptom of a hitherto unrecognized spontaneous or traumatic dissection. The same line of reasoning could also apply to some cases of ICAD thought to be linked to CMT.

With the preceding points in mind, the forensic medical expert should carefully evaluate cases involving ICAD that are allegedly attributed to CMT. In order to consider any given CMT as a possible basis for ensuing ICAD, the patient history should reflect a cervical manipulation that involved a sudden, severe stretch of the ICA over the bony segments of the upper cervical spine. More specifically, this mechanism of injury should also involve hyperextension of the neck with either associated lateral flexion or rotation. There ought to be some indication at the time of the CMT that intimal tearing occurred (i.e., neck pain or headache), or ICAD symptoms should develop within 48 hours following CMT.

REFERENCES


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