

CASE REPORTS



Cervical Manipulation to a Patient With a History of Traumatically Induced Dissection of the Internal Carotid Artery: A Case Report and Review of the Literature on Recurrent Dissections

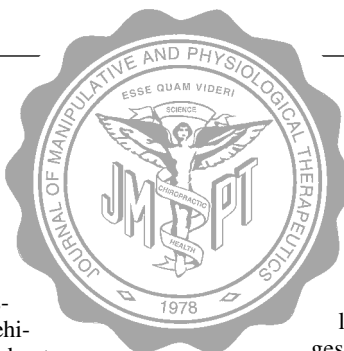
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ABSTRACT

Objective: To describe the use of rotational cervical manipulation in treating a patient who had undergone a traumatically induced dissection of the internal carotid artery and to review the literature on recurrent cervical artery dissections.

Clinical Features: A 21-year-old woman with hemiparesis from an internal carotid artery dissection that occurred as the result of a motor vehicle accident had neck pain and headaches. Moderate range of motion restrictions in the neck were present along with articular restrictions to movement palpation.

Intervention and Outcome: After a year of soft-tissue treatment, we obtained detailed, informed consent from the patient to attempt diversified manipulation to the neck. The patient described greater and more immediate relief and longer pain-



free periods than could be achieved by soft-tissue treatment alone.

Conclusion: Patients with previous cervical artery dissections may present with unrelated neck pain and headaches and request treatment. In selected cases, with complete informed consent, manipulation of the neck may relieve these symptoms. A review of published case reports on recurrent dissections suggests that trauma is not a significant factor in the second dissection. Care must be taken in extrapolating the results from this case to any other patient with a history of cervical artery dissection. (*J Manipulative Physiol Ther* 2001; 24:520-5)

Key Indexing Terms: Manipulation; Carotid Artery; Complications; Dissection; Cervical Artery Dissection; Vertebral Artery

INTRODUCTION

Cervical artery dissection (CAD) involving the vertebral and carotid arteries may follow cervical spine trauma,¹⁻³ be spontaneous without obvious precipitating activity,⁴⁻⁶ be the result of minor or trivial trauma associated with normal daily activities,^{4,7,8} or follow cervical manipulation.⁹⁻¹¹ It is difficult to explain why vertebral artery dissections occur in only 1 case per 1 to 3 million cervical manipulations¹² and why they are rarely reported in the whiplash literature. Although more common than vertebral artery dissection in the general population, CAD is rarely associated with cervical spine manipulation and has only 5 documented cases in the literature.¹³⁻¹⁷ If the primary factor involved in the dissection process was trauma associated with neck movements that stretch the vertebral and carotid arteries, one would expect a much greater number of cases.

The association of these dissections with disorders such as fibromuscular dysplasia, Ehlers-Danlos syndrome types IV and VI, and Marfan's syndrome,⁴ and the random nature of these events has led to the increasingly accepted theory that there is an underlying genetic arteriopathy in the cellular matrix of the arterial connective tissue that predisposes certain individuals to CAD.¹⁸⁻²⁰

Assuming that CADs are due to an underlying arteriopathy, one might expect that a previous arterial dissection would markedly increase the risk of a second dissection, particularly if there was further trauma. Furthermore, it would also be expected that a history of arterial dissection would be a contraindication for cervical manipulation and should alert a patient to be very careful to avoid traumatic neck movements of any kind.

A patient with a history of a post-traumatic carotid artery dissection following a motor vehicle accident recently sought care from one of us (SR), and requested cervical manipulation. The patient, who had benefited from cervical manipulation before her dissection, wished to have further manipulation despite being informed of the potential risks of a recurrent stroke. This precipitated a search of the literature to determine the risk of recurrent dissection in the cervical arteries, and also to determine whether there had been any reported cases of postmanipulation dissections in patients who had previously been diagnosed as having a dissection in any location.

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Table 1. All studies of recurrent dissection identified from the literature

Reference	N*	Number of recurrences		Total number of recurrences (% of N)	Mean follow-up (mo)
		Previously undissected artery	Previously dissected artery		
Case series					
Schievink et al ²⁵	200	16	0	16 (8)	89
Leys et al ²¹	105	0	4	4 (4)	36
Bassetti et al ²²	74	2	1	3 (4)	34
Bogousslavsky et al ²³	30	0	1	1 (3)	38
Pozzatti et al ²⁴	19	0	0	0 (0)†	98
Case reports					
d'Anglejan Chatillon et al ²⁸			1	1	
Goldstein et al ²⁹			1	1	
Youl et al ²⁶		1		1	
Rohr et al ²⁷		1		1	
Grau et al ³¹		1	1	3‡	
Grond-Ginsbach et al ³⁰				2‡	
Total				33	

*Population sample.

†Two patients had recurrent symptoms without verification of a second dissection on echo- or angiography.

‡No detailed data were provided on these additional cases.

CASE REPORT

A 21-year-old woman had a serious auto accident in August 1995 that resulted in considerable head and body trauma, including rib, femur, and mandibular fractures. There were no fractures to the spine or pelvis. As a result of a dissection to the right internal carotid artery (ICA), the patient had a stroke. The diagnosis was confirmed on computed tomographic angiography and Doppler ultrasonography, which revealed damage to the ICA at the bifurcation of the carotid artery, extending superiorly. This resulted in a large infarct in the distribution of the anterior and middle cerebral artery. Neurologic examination shortly after the accident revealed hemiparesis, hyperreflexia, and hypertonicity of the left arm and leg. In addition, she was dysphasic and had a slight facial palsy. After 6 weeks, the patient was transferred from the acute care hospital to a rehabilitation center, where she was confined for a number of months.

Nine months after the accident, the patient sought chiropractic care for residual headaches and pain in her neck and lower back. Before the accident, she had successfully received chiropractic manipulative therapy for the relief of similar symptoms. At the time of presentation she was confined to a wheelchair and moved with great difficulty. Clinical examination revealed residual spastic hemiparesis and facial weakness, moderate to severe decrease in cervical range of motion, moderate to severe articular end-feel restrictions, and extreme hypertonicity of the neck and shoulder girdle muscles. We initially decided to limit treatment to soft-tissue massage and gentle mobilization of the neck and back. This provided her with some relief of her symptoms. This relief, however, was of short duration, lasting from a few days to 1 week. In the ensuing year, the treatment plan was changed very little and was interrupted by various surgical procedures to correct other residual symptoms from the accident. Her neurologic rehabilitation continued, and she gradually became less dependent on her wheelchair and was able to take short walks (± 10 m) under her own power. However, she

continued to complain of recurrent headaches, and motion palpation of the neck revealed segmental blocking in the upper and lower cervical spine that could not be resolved with massage and mobilization.

The patient requested the type of cervical manipulation treatments she had received before the accident. We explained to her that manipulation to the neck might or might not provide more lasting relief of her neck pain and headaches. The patient was also informed of the potential for a recurrent stroke, of our lack of knowledge of the risks involved, and of the probability that her degree of risk after manipulation was likely to be higher than the general population. She was then asked to give a formal consent before the treatment. We explained a second time that it was a potentially risky maneuver and could result in exacerbation of her present symptoms or a second stroke. The patient felt that, in her situation, the benefits outweighed the potential risks.

Two years after her accident, and approximately 1 year after she had begun chiropractic treatment, manipulation of the cervical spine was added to the treatment regimen. The patient has since received more than 30 standard diversified cervical manipulations over a period of 27 months to both the upper and lower cervical regions. These were performed with the patient supine and with contacts taken at the articular pillars. A high-velocity, low-amplitude manipulative thrust was given from posterior to anterior with sufficient rotation to cause joint locking before the thrust.

Outcomes were assessed using the verbal rating scale (VRS). The VRS is an 11-point verbal scale, with "0" representing no pain and "10" representing unbearable pain. The degree of pain was assessed before the treatment and again within a few minutes after the treatment. Treatment with mobilization and massage alone typically resulted in only a 2-point change on the VRS (usually from a value of 6 to a value of 4). However, when manipulation was added, the change in VRS was typically 4 points (commonly from a value of 6 to a value of 2). Of greater significance was the patient's report

Table 2. Percentage of multi-vessel dissections by initial presentation and by recurrent dissection

Reference	Percent multi-vessel dissection (N) by:	
	Initial presentation	Recurrent dissection
Schievink et al ²⁵	28% (56/200)	6% (1/16)
Bassetti et al ²²	11% (7/81)	0% (0/3)
Leys et al ²¹	5% (5/105)	0% (0/4)
Bogousslavsky et al ²³	3% (1/30)	0% (0/1)

that the manipulations provided relief for many weeks, compared with the few days experienced in the beginning of the treatment period. The patient continues to request treatment approximately once per month, and spinal manipulation is the only treatment modality that provides her with immediate and relatively long-term relief of her neck pain and headaches. Four years after her accident, the patient has also made considerable progress in her neurologic function and is no longer dependent on her wheelchair.

DISCUSSION

Case reports and case series were identified by using an on-line search of MEDLINE for the years 1966-1999 with these key words: (recurrent) carotid artery dissection; (recurrent) vertebral artery dissection; (recurrent) cervical artery dissection. Cross-tabulation was performed from the reference section of all articles on the topic and included articles in both English and French.

We identified 3 prospective studies²¹⁻²³ and 2 retrospective studies^{24,25} that presented long-term follow-up of patients who had had CADs. One study²¹ included all cases of dissection, including those resulting from trivial or major trauma, whereas the other studies reported only on those with initial spontaneous dissections. There were an additional 6 papers in which a total of 9 case reports of recurrent dissections were presented.²⁶⁻³¹ This search resulted in a total of 33 cases of recurrent CADs identified in the literature (summarized in Table 1).

Frequency of recurrences. The 5 sequential case series²¹⁻²⁵ followed a total of 428 patients with an initial CAD. The average length of time for the follow-up in these case series was approximately 5 years. The risk for recurrent dissection reported in these articles varied from 0%²⁴ to 8%²⁵ over the period of follow-up, with a median of 4% (Table 1). Leys et al,²¹ however, believes that the study from Pozzati et al²⁴ may not be representative because they report a higher rate of mortality and disability resulting in a lower rate of recurrence. Despite this, Pozzati et al do report recurrent symptoms in 2 patients without verification of a second dissection.

By dividing the number of follow-up months by the total number of recurrences, the likelihood of a second dissection in patients with a previous dissection appears to be in the region of 1% per year with a cumulative risk of 9.5%²¹ to 11.9%²⁵ for the 10 years after the initial dissection.

Arteries of recurrence. Recurrent dissections may occur in a previously dissected or undissected artery. The largest study, by Schievink et al,²⁵ which also had the greatest number of recurrences, did not describe any case where the recurrent dis-

section was in a previously dissected artery. However, recurrent dissections in previously dissected arteries are reported in the other articles (see Table 1). On examination of all case series and case reports (admittedly skewed by the one study), the second dissections seemed to preferentially affect a previously undissected artery (70% of the known cases).

The initial and recurrent dissections, furthermore, need not be in one of the cervical arteries. There are 2 case reports in which a dissection occurred in a renal artery after initial CADs (the initial dissections involved bilateral vertebral artery and ICAs in one case,²⁵ and bilateral ICAs in the other).³² There are also reports of 2 cases of aortic artery dissection many years after an initial ICA dissection.³³

Bilateral dissections. Multiple artery dissections are not an uncommon initial presentation of CAD.^{32,34} In 4 of the 5 case series mentioned, for which data is available, 3% to 28% of patients (mean 12%) initially presented with multiple CADs (Table 2). However, among those with a recurrent dissection, only 1 case resulted in multi-vessel involvement. This case presented with bilateral dissection of the ICA after an initial bilateral dissection of the vertebral artery (VA) 7 years earlier.²⁵

Precipitating factors in recurrent dissections. The issue of recurrent dissection in a previously dissected artery is of particular concern when considering manipulation or the risks of trauma for patients who have experienced 1 episode of arterial dissection. A more detailed analysis of the 9 cases in which the second dissection occurred in a previously dissected artery revealed that 7 dissections were at the level of the previous dissection (Table 3). In the 4 cases in which a detailed medical history was given, connective tissue disease (eg, fibromuscular dysplasia or Ehlers-Danlos disease) was cited as a predisposing factor. In a fifth case, migraine was considered a potential predisposing factor.⁴ In 2 of 3 cases where the initial dissection was spontaneous, the subsequent dissection was also spontaneous. In all of these cases but one, the recurrence occurred many months after the initial dissection.

Risk factors for recurrent dissections. Only 1 study had sufficient numbers of recurrences^{25,35} to be able to identify possible risk factors. The risk factors identified were young age (<45 years) and a family history of arterial dissection. For those younger than 45 years, the cumulative rate of recurrence was identified as 16.8% during the 10 years after the initial dissection. This is much greater than the recurrence rate noted in those patients older than 45 years (6.1%). In those patients with a family history of arterial dissection the rate of recurrence was 50% (n = 5/10), compared with 5.8% (n = 11/190) for those with no family history. Factors that were shown not to have a relationship with recurrent dissection were sex, site of dissection (there was an equal predilection for those with prior VA or ICA dissection), multi-artery dissection, hypertension, smoking, or the use of oral contraceptives.

Trauma and manipulation. In only 2 cases in which a recurrence was described was there any mention of spinal manipulation as a contributing factor. One case involved a patient

Table 3. Reported cases of recurrent cervical artery dissection in a previously dissected artery

Sex, age (y)	Artery involved	Cause of dissection		Possible predisposing factor(s)	Time elapsed between dissections	Reference
		First dissection	Second dissection			
ND	Carotid	ND	ND	ND	32 months	Bogousslavsky et al ²³
F, ND	Carotid	ND	ND	ND	14 years	d'Anglejean ²⁸ Chatillon et al ²⁸
F, 33	Carotid	S (asymptomatic)	S	FMD + habit of smoking + contraceptive use	Possibly 10 years	Goldstein et al ²⁹
F, 41	Carotid	S	S	EDS	4 months	Leys et al ²¹
F, 28	Carotid	Chiropractic maneuver	S	EDS	24 months	Leys et al ²¹
M, 13	Vertebral	Judo	S	FMD	10 months	Leys et al ²¹
F, 47	Carotid	S	While awakening from general anesthesia	Migraine; no other history given	3 years	Basseti et al ²²

ND, No data; S, spontaneous; FMD, fibromuscular dysplasia; EDS, Ehlers-Danlos syndrome.

whose original dissection followed cervical manipulation and who had a second spontaneous dissection 2 years later at the same level within the same artery.²¹ Details concerning the manipulation were not given. In another case, the patient had received manipulation 2 weeks before the second dissection.²⁷ Again, no confirmation of the manipulation itself or any details concerning the nature of the manipulation were given. Both dissections affected the ICA.

In only 2 other cases of recurrence was any trauma involved in the initial dissection. One case occurred following a judo maneuver,²¹ and the second may have been the result of a motor vehicle accident.²⁹ No recurrences were reported to have been the result of trauma, with the possible exception of 1 case where the recurrence occurred while the patient was awakening from anesthesia.

A patient who has had a CAD is often very concerned about the possibility of recurrence and what steps can be taken to avoid such a recurrence. The literature, however, provides meager information that can be conveyed to such a patient.

The most significant information to come from this literature is the fact that recurrences do occur and that the primary risk factors appear to be inherent rather than external. At this time there is very little a patient can do to prevent a recurrence. However, family history and the presence of a previous dissection appear to be important risk factors.

The reported frequency of recurrences is approximately 1% per year, which is not insignificant. The likelihood of recurrence based upon our review appears to be no greater in the previously dissected artery than in the other previously unaffected cervical arteries. Recurrences may also occur in arteries other than cervical vessels, such as the aorta or renal arteries.

A number of facts suggest that a patient's predilection to dissection continues after the initial dissection. The likelihood of dissection in a patient with a previous dissection (1% per year) is considerably greater than in the population as a whole, in which it is estimated that the risk is no more than 2.6 to 2.9 per 100,000 per year for dissections of the ICA.³⁶ The observation that up to 50% of patients in one study had a family history of arterial dissection and that both initial and recurrent dissections are much more com-

mon in younger individuals suggests a genetic predilection. This is supported by recent published biochemical and genetic investigations of patients with a previous dissection. Mayer et al¹⁸ described a point mutation of Type I collagen that might have contributed to vascular fragility in a single patient who had had a spontaneous dissection. Brandt et al,¹⁹ in a more convincing article, performed skin biopsies on 25 patients with spontaneous dissections to evaluate the overall architecture and morphology of the connective tissue. They found that 68% of patients had ultrastructural abnormalities resembling those found in Ehlers-Danlos syndrome. The function of connective tissue is to provide stability and elasticity to the vessel wall, and structural changes in the collagen and elastic fibers could result in functional impairment. These authors concluded that an underlying inherited connective tissue arteriopathy might be the cause of these dissections.¹⁹

Despite the correlation of a family history of dissection with recurrent dissections and the association of heritable connective tissue disorders with spontaneous dissections, it remains difficult to identify who is at risk for an initial dissection. In the study by Brandt et al¹⁹ in which aberrations of connective tissue were found, there was no other history of a heritable connective tissue disorder. Brandt et al stressed that "even assuming an underlying connective tissue disorder, it remains unclear why CAD occurs at a certain point in time and at a distinct location and why recurrence rate and family occurrence are quite low."¹⁹

It would appear that other factors may be necessary for a CAD to occur. The kinking and tortuosity of the VA at the level of the atlas and axis¹¹ and the observation that tractioning and stretching of the internal carotid can occur at the level of the atlas^{37,38} has led to the suggestion that mechanical injury or occlusion may be a precipitating factor in CAD. However, if this was the only mechanism, one would anticipate many more reported cases of dissection to either the ICA or VA, given that over 200 million manipulations are performed each year in the United States alone³⁹ and that most individuals will experience some form of cervical trauma in their lifetime.

Although the literature on initial CADs does suggest a correlation with trauma and manipulation,⁹ the most recent data do not link trauma or spinal manipulation as the precipitating factor for recurrent dissection. Whereas up to 50% of all initial vertebral artery dissections have been related to some form of trauma, in only 2 cases (6%) of recurrent dissections was trauma the precipitating factor in the initial dissection, and in only 1 or 2 cases was there any speculation of trauma as a precipitating cause of the recurrence. This may reflect the fact that the majority of the cases in these reviews concern carotid artery dissections, whereas the majority of trauma-related dissections involve the VA. Regardless, it does not appear that a history of traumatic CAD is a significant factor in the likelihood of recurrence. Trauma also does not appear to be a significant precipitating factor in the risk of recurrences.

A recent study has suggested that there may be a link between CAD and recent generalized (systemic) infection, independent of mechanical or genetic factors.³¹ It found that 58% of consecutive patients who presented with spontaneous CADs had experienced symptoms of infection, compared with the control group of patients with acute cerebral ischemia from other causes (32%). They found that recent infection was independently associated with CAD and not the mechanical factors of coughing, sneezing, or vomiting. They refer to the literature on the effects of inflammation mechanisms of infection or the microbial agents themselves on arterial wall integrity as the factors precipitating the dissection.^{40,41}

To date, there have been just 5 cases reported in which internal carotid dissection occurred after cervical manipulation. This is in stark contrast to the vertebrobasilar system, in which more than 180 such cases have been reported.¹¹ In 2 of the 5 cases of ICA dissection after manipulation there was underlying pathology (atherosclerotic changes or underlying arteriopathy)^{13,14}; a congenital variation of the vertebrobasilar system may have been a predisposing factor in a third case.¹⁵ In the other 2 cases, however, there was insufficient history to make any further comment.^{16,17} In addition, 1 of these cases involved a patient who had a dissection 5 days after the manipulation,¹⁶ raising the issue of whether the manipulation was responsible for the injury. At this time there are insufficient data to make manipulation an independent risk factor for internal artery dissection, although it may precipitate ischemia in a patient who is in the process of dissecting.

CONCLUSION

To our knowledge, there have been no previous case reports of patients with prior CADs being subsequently treated with cervical manipulation. The safety of manipulation for patients with prior dissections has not been adequately discussed in the literature. At present, we do not know the nature of the risk because there are no other reports of patients successfully undergoing cervical manipulation after a dissection. Our patient has undergone multiple cervical manipulations over the past 3 years without any

adverse effects and has found the treatments to be beneficial. This should not be interpreted as meaning that it is safe to manipulate patients with a history of dissection. There are a number of factors that made it reasonable to consider manipulation for this patient: the original dissection was due to major trauma (ie, the forces causing the original injury were much greater than any force that could possibly be generated by a cervical manipulation); there were no other risk factors present, such as history of connective tissue abnormalities or a family history of vascular disease; and the initial dissection was in the carotid artery, which is much less susceptible to manipulation-related trauma than the vertebral artery. Nonetheless, we felt it was important to inform the patient of the perceived risks on at least 2 occasions before attempting manipulation; to try other less traumatic forms of treatment for an extended period before manipulation; and to explain our lack of understanding of the likely outcome of manipulation in this case. Based on her previous experience, this patient felt that the benefits of the treatment outweighed the potential harm. She was willing to accept our lack of understanding and our inability to present information regarding the risks associated with treatment. After receiving the manipulative treatments this patient felt that she had made the correct decision. She achieved greater and quicker relief of symptoms through manipulation than she was able to obtain from other treatment methods or pharmacologic agents.

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