Craniocervical arterial dissections as sequelae of chiropractic manipulation: patterns of injury and management

Clinical article

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Object. Chiropractic manipulation of the cervical spine is a known cause of craniospinal arterial dissections. In this paper, the authors describe the patterns of arterial injury after chiropractic manipulation and their management in the modern endovascular era.

Methods. A prospectively maintained endovascular database was reviewed to identify patients presenting with craniospinal arterial dissections after chiropractic manipulation. Factors assessed included time to symptomatic presentation, location of the injured arterial segment, neurological symptoms, endovascular treatment, surgical treatment, clinical outcome, and radiographic follow-up.

Results. Thirteen patients (8 women and 5 men, mean age 44 years, range 30–73 years) presented with neurological deficits, head and neck pain, or both, typically within hours or days of chiropractic manipulation. Arterial dissections were identified along the entire course of the vertebral artery, including the origin through the V3 segment. Three patients had vertebral artery dissections that continued rostrally to involve the basilar artery. Two patients had dissections of the internal carotid artery (ICA): 1 involved the cervical ICA and 1 involved the petrocavernous ICA. Stenting was performed in 5 cases, and thrombolysis of the basilar artery was performed in 1 case. Three patients underwent emergency cerebellar decompression because of impending herniation. Six patients were treated with medication alone, including either anticoagulation or antiplatelet therapy. Clinical follow-up was obtained in all patients (mean 19 months). Three patients had permanent neurological deficits, and 1 died of a massive cerebellar stroke. The remaining 9 patients recovered completely. Of the 12 patients who survived, radiographic follow-up was obtained in all but 1 of the most recently treated patients (mean 12 months). All stents were widely patent at follow-up.

Conclusions. Chiropractic manipulation of the cervical spine can produce dissections involving the cervical and cranial segments of the vertebral and carotid arteries. These injuries can be severe, requiring endovascular stenting and cranial surgery. In this patient series, a significant percentage (31%, 4/13) of patients were left permanently disabled or died as a result of their arterial injuries. (DOI: 10.3171/2011.8.JNS111212)

Key Words • chiropractic • arterial dissection • manipulation • vascular disorder • internal carotid artery

Chiropractic manipulation of the cervical spine can injure the vessels of the head and neck and produce stroke and other debilitating symptoms. These injuries typically involve the upper cervical segments of the VA, although the intracranial VA and carotid arteries may also be affected. Stroke is produced either by propagation of a thrombus from the dissected arterial segment or by severe dissection-induced arterial stenosis and secondary ischemia. Most patients present within hours to days of cervical manipulation.

The epidemiology of these injuries is almost impossible to ascertain. Studies have suggested that their incidence ranges between 1 in 100,000 and 1 in 6,000,000 manipulations. Given that Americans visit chiropractors more than 250,000,000 times per year and that 30% of these visits involve cervical manipulation, the incidence of arterial dissection is likely to be higher than the lowest estimates. A significant number of dissections may go unreported either because they are mild or asymptomatic.
In 1927, deKleyn and Nieuwenhuys first associated cervical manipulation with stroke. Through cadaveric experimentation, they demonstrated that hyperextension and contralateral rotation of the head significantly reduced VA circulation. In 1947, Pratt-Thomas and Berger reported 3 deaths within hours of chiropractic manipulation, which has been discussed by other investigators. Necroscopic evaluation revealed a thrombus within the BA in 2 patients and spinal cord infarction in the third. In their review of the literature, these authors discovered several similar reports.

Chiropractic manipulation of the cervical spine usually involves 2 types of therapeutic maneuvers. Low-velocity, high-amplitude manipulation consists of a series of gentle, repetitive motions to a specific region of the neck. High-velocity, low-amplitude manipulation is characterized by a sudden thrust delivered to the involved vertebral level. The latter is most often implicated as the cause of cranio cervical dissections. Specifically, this type of manipulation consists of a sudden contralateral rotation of the extended cervical spine. As described above, this type of motion is known to compromise the patency of the cervical VA’s, specifically at the V2 and V3 segments. One can also theorize that this type of maneuver could injure the high cervical and proximal segments of the intracranial ICA because they abut the bony structures of the transverse foramina and skull base.

Previous reports of arterial injury after cervical manipulation have largely advocated anticoagulation therapy with heparin and coumadin as the treatment of choice in symptomatic patients. Given the severity of dissections in a number of our patients, we have often chosen a more aggressive approach using stent placement and craniotomy to relieve brainstem compression. We sought to assess our series of patients for specific factors related to their arterial dissections and, in so doing, better characterize their pattern of injury and optimal treatment strategy.

Methods

We reviewed our prospectively maintained endovascular database to identify patients who experienced cranio cervical arterial dissections after chiropractic manipulation. A history of chiropractic cervical manipulation was obtained through interviews with either the patient or a family member. Specifically, we assessed the interval to symptomatic presentation, the location of the injured arterial segment, neurological symptoms and complaints, endovascular treatment undertaken, surgical treatment, and clinical and radiographic outcomes.

All endovascular procedures were performed by the senior authors (F.C.A. and C.G.M.). Cerebellar decompression was performed by one of the senior authors (F.C.A.) or by other members of our neurosurgical department. Stent deployment was performed after the induction of general anesthesia with electrophysiological monitoring. Heparinization was induced with the goal of an activated clotting time of more than 250 seconds. Antiplatelet therapy was started during the procedure or immediately thereafter unless the patient was believed to require urgent surgical decompression. In this setting of surgical decompression, antiplatelet therapy was deferred until a surgical intervention was either undertaken or deemed unnecessary.

Clinical outcomes were ascertained by the senior authors and compared with the patients’ original symptoms. Radiographic follow-up included one or more of the following: digital subtraction angiography, CT angiography, and MR angiography.

Results

Thirteen patients (8 women and 5 men) presented within hours to days of chiropractic manipulation of the cervical spine (Table 1). Patients were typically young (mean age 44 years, range 30–73 years). Eight patients presented within 2 days of cervical manipulation. Of these 8 patients, 4 presented within hours of chiropractic treatment. The longest interval between manipulation and presentation was 14 days. Of the 13 patients, 12 presented with acute neurological deficits or complaints. The remaining patient complained only of neck and suboccipital pain.

Of the 13 patients, 12 demonstrated VA dissections (Table 1). The dissections most often involved the V2 and V3 segments (9 of 12 patients), but injuries involving all segments of the VA were detected. Three patients had VA dissections that had progressed rostrally to involve the BA. One patient suffered dissections of both cervical ICAs, as well as a dissection of the left V3 and V4 segments of the VA. One patient experienced dissection of only the right petro cavernous portion of the ICA.

Six patients were treated using endovascular interventions (Table 2), which included 5 cases of stent deployment and a single case of BA thrombolysis. Of the 5 stenting procedures, 4 involved the deployment of multiple devices. Overall, 14 stents were deployed in these 5 patients. One patient’s condition deteriorated the day after stent placement and he was found to have in-stent thrombosis. This complication required emergency angioplasty and thrombolysis to restore patency of the affected arterial segment.

Three patients underwent an emergency craniotomy for cerebellar decompression. Two of these patients had undergone stent placement in the hours preceding the craniotomy. The third patient had not required endovascular treatment but demonstrated progressive cerebellar swelling from infarction. After a hematoma developed in the original resection cavity, this patient required a second cerebellar decompression. Six patients were treated using only medication, involving either anticoagulation or antiplatelet therapy. The 2 complications from treatment were the previously noted in-stent thrombosis and the hematoma requiring evacuation.

All patients were followed clinically an average of 19 months (range 0–34 months; Table 3). At presentation, 10 of the 13 patients demonstrated infarctions on diffusion-weighted MR imaging. Of these 10 patients, 3 were left with debilitating neurological deficits and 1 died. The 3 permanently injured but surviving patients were those...
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**TABLE 1: Demographics of 13 patients with arterial dissections**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Days to Presentation After Manipulation</th>
<th>Location of Injury</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>39, F</td>
<td>2</td>
<td>lt V₃ &amp; V₄</td>
<td>neck pain, nausea, vomiting, vertigo, obtundation</td>
</tr>
<tr>
<td>2</td>
<td>33, F</td>
<td>1</td>
<td>lt V₄</td>
<td>neck pain, diplopia, rt hemiparesis, lt hemisensory loss</td>
</tr>
<tr>
<td>3</td>
<td>30, M</td>
<td>2</td>
<td>bilat V₃ &amp; V₄, BA</td>
<td>headache, neck pain, quadriparesis, obtundation</td>
</tr>
<tr>
<td>4</td>
<td>50, F</td>
<td>7</td>
<td>lt V₄, BA thrombosis</td>
<td>rt hemiparesis, lt Horner syndrome, lt hemiataxia</td>
</tr>
<tr>
<td>5</td>
<td>39, F</td>
<td>same day</td>
<td>lt V₄, BA</td>
<td>vertigo, nausea &amp; vomiting, lt hemisensory loss</td>
</tr>
<tr>
<td>6</td>
<td>54, M</td>
<td>1</td>
<td>bilat cervical ICA, lt V₃ &amp; V₄</td>
<td>incoordination, ataxia, expressive aphasia</td>
</tr>
<tr>
<td>7</td>
<td>41, F</td>
<td>3</td>
<td>lt V₃ &amp; V₄</td>
<td>full-body numbness</td>
</tr>
<tr>
<td>8</td>
<td>53, M</td>
<td>7</td>
<td>rt petroccavernous ICA</td>
<td>lt facial droop, aphasia, hemiparesis</td>
</tr>
<tr>
<td>9</td>
<td>73, F</td>
<td>same day</td>
<td>lt V₃ &amp; V₄, BA</td>
<td>lt PICA cerebrovascular accident</td>
</tr>
<tr>
<td>10</td>
<td>38, M</td>
<td>same day</td>
<td>lt V₄</td>
<td>vertigo, obtundation ataxia</td>
</tr>
<tr>
<td>11</td>
<td>34, F</td>
<td>14</td>
<td>lt V₃ &amp; V₄</td>
<td>suboccipital headache, vertigo, rt numbness</td>
</tr>
<tr>
<td>12</td>
<td>48, M</td>
<td>same day</td>
<td>rt V₃</td>
<td>nausea &amp; vomiting, vertigo</td>
</tr>
<tr>
<td>13</td>
<td>39, F</td>
<td>10</td>
<td>lt V₃ &amp; V₄</td>
<td>lt neck pain, nausea &amp; vomiting</td>
</tr>
</tbody>
</table>

who had undergone emergency cerebellar decompression for impending herniation. The remaining 9 (of 13) patients experienced complete resolution of their neurological symptoms and complaints.

Radiographic follow-up was obtained in all but one of the most recently treated survivors. The 5 patients who underwent stent placement all received radiographic follow-up, and all demonstrated normal patency of the affected segment. The patient who had undergone BA thrombolysis also demonstrated normal patency at follow-up. Of the 6 patients who were treated medically and had radiographic follow-up, 4 showed healing of their dissections. One dissection failed to improve but remained stable. Radiographic follow-up ranged from 3 to 26 months (mean 12 months).

**Illustrative Cases**

**Case 3**

This 30-year-old man presented 2 days after chiropractic manipulation with quadriparesis and progressive obtundation. After cervical manipulation he had immediately noted the onset of a suboccipital headache and neck pain. Only after complaining to his wife of diffuse weakness 2 days later was he taken to the hospital as an emergency. Computed tomographic angiography showed dissections of both V₃ and V₄ segments of the VA, as well as a near-occlusive dissection of the proximal BA. Magnetic resonance imaging demonstrated a right cerebellar infarction with mild mass effect, as well as patency of the fourth ventricle (Fig. 1A and B). He was immediately started on intravenous heparin and was taken to the anesthetic suite after becoming progressively more obtunded.

Angiography confirmed the dissections (Fig. 1C–G). Because the right VA appeared dominant and maintained continuity with the BA, we elected to place the stents from the midbasilar artery into the right V₃ segment in a telescoping, descending fashion. Four stents were immediately deployed and restored near normal patency of the right vertebral and basilar arteries (Fig. 1H–J). The patient remained intubated and on heparin and was returned to the neurosurgical intensive care unit in stable condition.

The next morning he awakened and could move all 4 extremities on command. By early afternoon, his condition deteriorated again and he was rushed to the angiographic suite. A right VA injection demonstrated thrombosis of the V₄ and BA stents (Fig. 1K). Angioplasty and intraarterial thrombolysis with tissue plasminogen activator and abciximab restored near-normal patency of these segments (Fig. 1L).

The patient failed to awaken from anesthesia. A second MR image demonstrated progression of his cerebral hemorrhage.

**TABLE 2: Treatment of 13 patients with arterial dissections**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Endovascular Tx</th>
<th>Surgical Tx</th>
<th>Tx Complications</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>lt V₃ &amp; V₄ stenting (3 total)</td>
<td>cerebellar decompression</td>
<td>none</td>
</tr>
<tr>
<td>2</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>rt V₄ &amp; BA stenting (4 total)</td>
<td>cerebellar decompression</td>
<td>in-stent thrombosis</td>
</tr>
<tr>
<td>4</td>
<td>BA thrombolysis</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>lt V₃ &amp; V₄ stenting (3 total)</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>rt ICA stenting (3 total)</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>10</td>
<td>none</td>
<td>cerebellar decompression</td>
<td>recurrent ICH</td>
</tr>
<tr>
<td>11</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>12</td>
<td>none</td>
<td>none</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>lt V₄ stent</td>
<td>none</td>
<td>none</td>
</tr>
</tbody>
</table>

* ICH = intracranial hemorrhage.
operative room for emergency cerebellar decompression. Heparin, transfused platelets, and took the patient to the (Fig. 1M). As a life-saving measure, we discontinued the cerebellar infarction and effacement of his fourth ventricle normal patency of his stents (Fig. 1P and Q).

After a prolonged hospital stay and rehabilitation, the patient remains hemiparetic but ambulatory with a left facial droop and dysarthria. He is unable to work and depends on his wife for at-home care. Four-month follow-up demonstrated embolic infarcts of the left thalamus and left facial palsy. Hemostasis was achieved after 4 hours, with a resulting estimated blood loss of 4.5 L. Within 24 hours, the patient emerged from anesthesia. He followed commands but was noted to have left hemiparesis. After a CT scan confirmed adequate decompression and hemostasis within the surgical cavity, his dual-antiplatelet therapy was restarted (Fig. 1N and O).

After a prolonged hospital stay and rehabilitation, the patient remains hemiparetic but ambulatory with a left facial droop and dysarthria. He is unable to work and depends on his wife for at-home care. Four-month follow-up angiography and 24-month CT angiography confirmed normal patency of his stents (Fig. 1P and Q).

**Case 6**

This 54-year-old man presented 1 day after cervical manipulation complaining of suboccipital headache, incoordination, and ataxia. He was noted to have expressive aphasia on neurological examination. Computed tomographic angiography revealed severe dissections of both cervical ICAs extending rostrally to the petrous segment, as well as a dissection of the left V3 through V1 (Fig. 2A–D). The left VA was thrombosed just proximal to the BA (Fig. 2E and F). Nonetheless, the right VA appeared normal and filled the BA. Magnetic resonance imaging demonstrated embolic infarcts of the left thalamus and cerebellar hemisphere. A CT perfusion study showed attenuation of flow in the right cerebral hemisphere (Fig. 2G).

Given the CT perfusion findings, we elected to address the right ICA dissection. After administering heparin, we placed 3 stents in a descending, telescoping fashion from the right petrous segment down to the proximal right ICA (Fig. 2H and I). Postoperatively, we discontinued the heparin and the patient was maintained on dual-antiplatelet therapy. After a short stay at our inpatient rehabilitation facility, the patient recovered completely.

Two months after treatment, the patient underwent a second CT perfusion that demonstrated symmetric perfusion of both hemispheres (Fig. 2J). Follow-up CT angiography 8 months after treatment revealed wide patency of the right ICA stents, remodeling but persistence of the left ICA dissection, and continued occlusion of the left V3 segment (Fig. 2K and L). He continues to take a single adult aspirin per day as his treatment.

### Discussion

Chiropractic manipulation of the cervical spine can produce dissections of the cranial and cervical arteries. These dissections may be severe, mandating aggressive endovascular and open surgical intervention. The upper VA (V1) is most commonly affected, although the entire vertebrobasilar system can be injured. Although rare, the high cervical and proximal ICA can also be injured. After dissection, infarction commonly occurs through either thromboembolic or ischemic mechanisms. In our series, 10 patients presented with abnormalities on diffusion-weighted MR imaging; of these 10 patients, 3 were left permanently disabled and 1 died.

Historically, treatment in such cases has consisted of anticoagulation therapy and observation. Because of the severity of many of the dissections in our patient series, we often pursued a more aggressive therapeutic approach. This approach consisted of stent placement in 5 patients, transarterial thrombolysis in 1 patient, and cerebellar decompression in 3 patients. Treatment was believed necessary either because of the radiographic severity of the arterial injury or because of a decline in neurological function. Often, the arterial injury proved lengthy, requiring the deployment of multiple stent devic-

### TABLE 3: Clinical and angiographic follow-up of 13 patients with arterial dissections

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Clinical Follow-Up</th>
<th>Angiographic Follow-Up</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mos</td>
<td>Sx</td>
</tr>
<tr>
<td>1</td>
<td>24</td>
<td>It hemiataxia &amp; incoordination, rt homonymous hemianopsia</td>
</tr>
<tr>
<td>2</td>
<td>27</td>
<td>none</td>
</tr>
<tr>
<td>3</td>
<td>24</td>
<td>It facial droop, lt hemiataxia, dysarthria, lt hemiparesis</td>
</tr>
<tr>
<td>4</td>
<td>34</td>
<td>none</td>
</tr>
<tr>
<td>5</td>
<td>22</td>
<td>none</td>
</tr>
<tr>
<td>6</td>
<td>30</td>
<td>none</td>
</tr>
<tr>
<td>7</td>
<td>31</td>
<td>none</td>
</tr>
<tr>
<td>8</td>
<td>16</td>
<td>none</td>
</tr>
<tr>
<td>9</td>
<td>0</td>
<td>deceased</td>
</tr>
<tr>
<td>10</td>
<td>4</td>
<td>ataxia</td>
</tr>
<tr>
<td>11</td>
<td>6</td>
<td>headache</td>
</tr>
<tr>
<td>12</td>
<td>4</td>
<td>none</td>
</tr>
<tr>
<td>13</td>
<td>5</td>
<td>none</td>
</tr>
</tbody>
</table>
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In total, 14 stents were deployed in 5 patients. All 12 of the surviving patients demonstrated some degree of neurological improvement at clinical follow-up. Nine patients recovered completely. Although permanently disabled, 3 patients improved from the nadir of their neurological deterioration. Six patients were treated medically with either anticoagulation or antiplatelet therapy, because of the relative lack of severity of their dissections or because of their clinical neurological stability.

That chiropractic manipulation can produce symptomatic dissections of the craniocervical vessels has been the subject of debate in both the chiropractic and neurological literature. Patients often visit the chiropractor complaining of head or neck pain, and a certain percentage may have preexisting arterial dissections. Nonetheless, as demonstrated in this series, patients can present within hours to days of chiropractic manipulation with new objective neurological deficits or more severe neurological complaints. This temporal relationship suggests that either the arterial injury was produced de...
novo or made worse as a result of manipulation. Worsening of the patient’s complaints or the manifestation of a new neurological deficit after cervical manipulation should prompt urgent medical evaluation. 56

In 2003, Smith et al. 50 determined that “cervical manipulation was independently associated with VA dissection even after controlling for neck pain.” Using a nested case-control study design, the authors found that patients with dissections were nearly 5 times as likely to have undergone manipulative therapy within 30 days. The authors concluded that patients undergoing chiropractic cervical manipulation should be informed of, and sign consent forms for, the risk of stroke or vascular injury. 50,54

Interestingly, the Association of Chiropractic Colleges does not mandate informed consent for manipulative therapy; it only suggests it as a guideline. 2,34

The frequency of arterial injury after cervical manipulation is almost impossible to ascertain. In 2001, Rothwell et al. 46 examined patients who had suffered verteobasilar strokes and who had visited a chiropractor for cervical manipulation within a year before their ictus. 11 Through a comparison with a larger control group that had not suffered posterior circulation strokes, the authors made several observations. 46 Most importantly, younger patients (< 45 years of age) suffering a verteobasilar stroke were 5 times more likely than controls to have undergone cervical manipulation within a week of their presentation. Similarly, in this same age group, patients with posterior circulation strokes were 5 times as likely to have had 3 or more chiropractic visits in the month before their clinical deterioration.

Rothwell et al. 46 found no association between chiropractic manipulation and stroke in older patients (> 45 years of age). Their finding may reflect several factors. Chiropractors may manipulate the cervical spines of younger patients more aggressively, thereby raising the likelihood of arterial injury. Furthermore, if patients have a propensity for cervical arterial dissection, it may manifest at an earlier age. 19 Finally, younger, more active patients are likely to suffer from cervical discomfort and more often seek chiropractic adjustments.

Another factor that fuels the debate over the risk of stroke after chiropractic manipulation is referral bias. In 2002, Haldeman et al. 16 assessed the clinical perceptions of chiropractors and neurologists about the risk of vertebral dissection after manipulation. Through surveys and extrapolations based on data retrieved from the Canadian Chiropractic Protective Association, the malpractice insurance carrier representing the majority of Canada’s chiropractors, the authors estimated that 23 vertebral dissections had occurred after treatment during the 10-year period spanning 1988 to 1997. Based on a survey
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of a subset of chiropractors, the authors estimated that 134,466,765 cervical manipulations had been performed during this period. These numbers produce a rate of 1 VA dissection associated with every 5,846,381 cervical manipulations.

The authors then sought to discover how many neurologists had been made aware of vascular complications after cervical chiropractic manipulation. Whereas for each VA dissection a single chiropractor (the practitioner) was aware of the complication, the authors discovered that multiple neurologists, typically 3 or 4 on average, had consulted on or treated each affected patient. Therefore, they were able to conclude that 1 of every 48 chiropractors and 1 of every 2 neurologists in Canada were aware of a VA dissection resulting from cervical manipulation during the course of their lifelong practices. This extreme disparity in the perceived risk of dissection between these practitioners distorts the debate over the actual incidence of this complication.15,27

Since 2007 we have treated 13 cases of cranio cervical arterial dissections as a result of chiropractic manipulation of the cervical spine. This number alone argues in favor of a much higher incidence of arterial injury after manipulation than that reported by Haldeman et al.15,16 (1 in nearly 6 million). In all likelihood, a substantial number of cases are not reported either because the injury is not severe or is asymptomatic. Given that the natural history of minor arterial dissections is endoluminal remodeling and healing, most of these cases probably never reach clinical attention. Therefore, it is impossible to predict the risk of arterial dissection after chiropractic manipulation of the cervical spine.

The severity of the arterial injuries in our patient cohort was also dramatic. Not only were these lesions often lengthy, their neurological sequelae were frequently severe. Three patients were left permanently debilitated by stroke and 1 died, producing an adverse outcome rate of 31% (4 of 13 patients). In a subset of patients, the radiographic and clinical severity of these dissections prompted an aggressive therapeutic approach, including cerebellar decompression and the deployment of multiple stents. Although prior studies advocated anticoagulation as the treatment of choice in this setting, modern endovascular and surgical techniques may be required to restore vessel patency and to preserve neurological function.

Conclusions

Chiropractic manipulation of the cervical spine can produce dissections of the cranial and cervical segments of the vertebral and carotid arteries. Given the popularity of chiropractic treatment, the incidence of this phenomenon is most likely underreported. These arterial injuries can be lengthy and severe, producing adverse neurological sequelae and even death (including 31% of patients in this series). Consequently, aggressive endovascular and surgical techniques may be required to restore vessel patency and to preserve neurological function.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following. Acquisition of data: Albuquerque, Hu, Dashti, Alkire, McDougall. Analysis and interpretation of data: Albuquerque. Drafting the article: Albuquerque, Hu, Abla, Clark. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: Albuquerque. Administrative/technical/material support: all authors. Study supervision: all authors.

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