Endovascular Treatments of Traumatic Carotid and Vertebral Vascular Injuries

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Objective: The purpose of this report is to determine the safety and efficacy of endovascular therapy in the management of craniocervical vascular injuries.

Methods: Fifteen patients with traumatic carotid and vertebral lesions were treated using therapeutic endovascular methods. In 13 patients with blunt trauma, five patients had a carotid-cavernous fistula, 8 had a dissecting pseudoaneurysm or arterial dissection. One of two penetrating patients had complete transection of the vertebral artery, and the other had an internal carotid artery-internal jugular vein fistula with two pseudoaneurysms. Endovascular therapy was accomplished by implanting the balloons, porous or polytetrafluoroethylene covered stent, and/or embolic materials including coils or glue.

Results: All fistulas and pseudoaneurysms were successfully embolized with coils, glue, or stents. Most of all parent arteries except two patients were preserved. Sacrifice of the parent artery was inevitable in cases of thrombus formation due to coil migration into the parent artery and the existing transaction of the parent artery. No additional surgical procedures for vascular lesions were required. There were no delayed neurological or vascular complications. Additionally, no lesions recurred during follow-up periods (mean 26.9 months).

Conclusion: From the author's experiences, the endovascular therapy using stents, balloons, and coils is both feasible and safe in treatment of the traumatic vascular injuries. Long-term follow-up review of these repairs will be requisite to provide a full evaluation of the safety and efficacy of these devices.

KEY WORDS: Carotid artery injuries · Vertebral artery injuries · Embolization therapeutic.

Introduction

Traumatic injury of the craniocervical arteries is probably an underdiagnosed condition. The frequent coexistence of traumatic brain injuries seems to obscure its presentation. Because of concurrent systemic injuries, the management of these patients is somewhat challenging from the prospective of both diagnosis and treatment.

Those lesions located near the skull base are particularly hazardous to approach and difficult to repair surgically. Extensive exposure to achieve adequate proximal and distal vessel control may result in significant surgical mortality and morbidity. If the injured vessel segment is inaccessible, proximal occlusion or segmental isolation is the most common treatment options.

Over the last two decades, endovascular therapies have had a significant impact on the treatment options for various diseases affecting the extracranial and intracranial vasculature. These minimally invasive techniques are now considered to be the preferred treatment modality in a number of conditions. Recently, several reports have contained descriptions of endovascular procedures in which stents and coils have been used to treat symptomatic dissections of the extracranial vessels having pseudoaneurysms and/or fistula, with parent artery preservation.

These devices permit minimally invasive arterial repairs to be performed from easily accessible sites, remote from the area of arterial trauma. These factors make such a therapeutic strategy extremely attractive for arterial injuries of the extracranial or intracranial carotid and vertebral lesions.

In this report we describe our preliminary clinical experience with extracranial or intracranial traumatic vascular lesions, which were treated using endovascular methods.
Materials and Methods

Patient Population
Between January 1997 and July 2003, fifteen consecutive patients with traumatic carotid and vertebral lesions were treated using therapeutic endovascular methods. The clinical characteristics in 15 patients with traumatic vascular lesions treated listed in Table 1. There were eleven men and four women with a mean age of 40.8 years (range 25-56 years). Most of patients except two (case 5 and 15) of the incidental detection on the magnetic resonance imaging (MRI) screening were symptomatic. Clinical diagnosis in each case was supported by objective imaging, including computed tomography, MRI, and arteriography. Most of patients presented with symptoms related to the positive finding of imaging studies. Three patients experienced with ischemic attack in the territories of the involved vessels. Five patients (case 1, 3, 5, 8, and 15) presented with the ambiguous symptoms and signs, and two (case 3 and 5) were associated with cervical spinal fracture and two (case 7 and 10) had multiple systemic injuries. Patients with carotid-cavernous fistula (CCF) presented with clinical symptoms of pulsating exophthalmos, chemosis, retroorbital bruirt, ophtalmoplegia, and/or diminution of visual acuity. One vegetative patient with CCF presented with intracranial hematoma (case 7).

Two of the lesions were the result of penetrating injuries, one affecting on the distal extracranial internal carotid artery (ICA) and the other on the vertebral artery (VA). Thirteen were the one of blunt traumas, five affecting the intracranial ICA, two the extracranial VA, two the intracranial VA, two the extracranial ICA, one the extracranial and intracranial VA, and one the external carotid artery (ECA).

Five patients had CCFs, 8 had dissecting pseudoaneurysms or arterial dissections. One of two penetrating patients had complete transection of the VA, and the other had an ICA-interna jugular vein (IJV) fistula with two pseudoaneurysms.

The guiding principle of vital arteries repair is occlusion of the arterial injury site from within the arterial lumen without compromising arterial flow.

Table 1. Clinical characteristics in 15 patients with traumatic vascular lesions treated using endovascular methods

<table>
<thead>
<tr>
<th>Case</th>
<th>Age/Sex</th>
<th>Clinical presentation</th>
<th>Clinical diagnosis</th>
<th>Vascular lesion</th>
<th>Affected vessel</th>
<th>Injury mechanism</th>
<th>Associated injuries</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>42/M</td>
<td>hoarseness, dysphagia</td>
<td>acute EDH</td>
<td>PA</td>
<td>IMA of ECA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>32/M</td>
<td>pulsatile swelling, limb weakness</td>
<td>MCA infarction</td>
<td>PA, AVF</td>
<td>cervical ICA and UV</td>
<td>penetrating</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>49/M</td>
<td>stab wound in midface</td>
<td>penetrating injury</td>
<td>transection</td>
<td>VA</td>
<td>penetrating</td>
<td>C1 arch fracture</td>
</tr>
<tr>
<td>4</td>
<td>36/M</td>
<td>dysphagia, hoarseness, sensory change, ataxia</td>
<td>PICA infarction</td>
<td>PA and dissection</td>
<td>intracranial and extracranial VA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>37/M</td>
<td>posterior neck pain</td>
<td>Hangman’s fracture,</td>
<td>PA and dissection</td>
<td>extracranial VA</td>
<td>blunt, flexion</td>
<td>Hangman’s fracture</td>
</tr>
<tr>
<td>6</td>
<td>46/M</td>
<td>diplopia, exophthalmos, conjunctival injection</td>
<td>basal skull fracture</td>
<td>CCF</td>
<td>cavernous ICA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>45/M</td>
<td>ptosis, decreased mental status</td>
<td>traumatic ICH, DAI</td>
<td>CCF</td>
<td>cavernous ICA</td>
<td>blunt</td>
<td>pelvic bone fracture</td>
</tr>
<tr>
<td>8</td>
<td>40/M</td>
<td>headache, dysphagia</td>
<td>traumatic SAH</td>
<td>PA</td>
<td>intracranial VA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>52/F</td>
<td>suboccipital headache</td>
<td>basal skull fracture</td>
<td>PA</td>
<td>extracranial VA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>10</td>
<td>56/F</td>
<td>decreased mental status</td>
<td>traumatic SAH</td>
<td>PA</td>
<td>intracranial VA</td>
<td>blunt</td>
<td>hemithorax</td>
</tr>
<tr>
<td>11</td>
<td>25/M</td>
<td>limb weakness</td>
<td>lacunar infarction</td>
<td>PA</td>
<td>cervical and petrosal ICA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>35/M</td>
<td>diplopia, bruit</td>
<td>basal skull fracture</td>
<td>CCF</td>
<td>cavernous ICA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>13</td>
<td>49/F</td>
<td>diplopia, exophthalmos</td>
<td>minor head injury</td>
<td>CCF</td>
<td>cavernous ICA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>14</td>
<td>31/M</td>
<td>diplopia, bruit</td>
<td>basal skull fracture</td>
<td>CCF</td>
<td>cavernous ICA</td>
<td>blunt</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>38/M</td>
<td>incidental finding</td>
<td>DAI</td>
<td>PA</td>
<td>petrosal ICA</td>
<td>blunt</td>
<td></td>
</tr>
</tbody>
</table>

This is not essential to the traumatic lesions involving nonessential vessels. These can be effectively treated by catheter-directed arterial embolization. This was accomplished by implanting the balloons, porous or polytetrafluoroethylene (PTFE)-covered stent, and/or embolic materials including coils or glue.

As all procedures were similar to one another, all adhered to the basic principle of preservation of patency in essential vessels and total occlusion of nonessential arteries.

Endovascular Procedures

The endovascular treatments in 15 patients with traumatic vascular lesions listed in Table 2.

Transarterial Embolization by Detachable Balloons (Fig. 1) or Combined Endovascular Treatment with Coils and Stents for CCF (Fig. 2)

All patients with CCF were treated initially by transarterial detachable balloon embolization therapy under the general anesthesia. From a transarterial approach, a 7-French catheter was placed into the ICA. Five thousand units of heparin was then given intravenously to prevent thrombus formation in the catheter and balloon system. The detachable balloon system used for most cases was the Balloon Goldvalve Detachable Balloon System (Nycomed, Paris, France). Often more than one balloon may be used to occlude large compartments of the cavernous sinus. Three of five patients with CCF were treated with two or three balloons. The balloon was attached to a 2/3-French coaxial polyethylene catheter and the set was placed through the larger 7-French introducing catheter. The balloon was then partially inflated and was flow-directed to the fistula orifice. Once the balloon was in the correct position, a second arteriogram was obtained to ensure that the fistula was completely occluded and the parent artery was patent. The patient was reexamined neurologically, and if stable, the balloon was detached by gentle retraction. Usually because of the high flow of the fistula, the balloon could easily transverse the tear of the vessel. However, in some instances where the fistula was small/large or on unfavorable curve of the vessel, balloons embolization was failed. Alternatively, coils embolization was attempted with stent placement into the ICA for inhibiting coil protrusion into the parent artery. An 8-French MPA guiding catheter and the set was placed through the larger 7-French introducing catheter. The balloon was then partially inflated and was flow-directed to the fistula orifice. Once the balloon was in the correct position, a second arteriogram was obtained to ensure that the fistula was completely occluded and the parent artery was patent. The patient was reexamined neurologically, and if stable, the balloon was detached by gentle retraction. Usually because of the high flow of the fistula, the balloon could easily transverse the tear of the vessel. However, in some instances where the fistula was small/large or on unfavorable curve of the vessel, balloons embolization was failed. Alternatively, coils embolization was attempted with stent placement into the ICA for inhibiting coil protrusion into the parent artery. An 8-French MPA guiding

Table 2. Angiographic and clinical outcome in 15 patients with traumatic vascular lesions treated using endovascular methods

<table>
<thead>
<tr>
<th>Case</th>
<th>Endovascular treatment</th>
<th>Result on angiogram</th>
<th>Parent artery preservation</th>
<th>Clinical outcome</th>
<th>Follow-up period (mos)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>platinum coils and glue embolization</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>resolution of symptoms</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>PTFE-covered stent</td>
<td>aneurysm occluded, fistula closed</td>
<td>Yes</td>
<td>elimination of swelling and bruise</td>
<td>8</td>
</tr>
<tr>
<td>3</td>
<td>GDC coils embolization</td>
<td>embolization &quot;sandwich&quot; above and below the level of transection</td>
<td>No</td>
<td>hemodynamically stable</td>
<td>34</td>
</tr>
<tr>
<td>4</td>
<td>three porous stents</td>
<td>aneurysm disappeared, resolution of stenosis</td>
<td>Yes</td>
<td>gradual improvement of symptoms and neurological defects</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>wall stent</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>no new neurological symptoms</td>
<td>55</td>
</tr>
<tr>
<td>6</td>
<td>porous stents with GDCs embolization</td>
<td>fistula closed</td>
<td>Yes</td>
<td>resolution of symptoms</td>
<td>33</td>
</tr>
<tr>
<td>7</td>
<td>three balloons embolization</td>
<td>fistula closed</td>
<td>Yes</td>
<td>no new neurological symptoms</td>
<td>27</td>
</tr>
<tr>
<td>8</td>
<td>stent-assisted GDCs embolization</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>gradual improvement of symptoms</td>
<td>16</td>
</tr>
<tr>
<td>9</td>
<td>stent insertion</td>
<td>decreased aneurysmal dilatation</td>
<td>Yes</td>
<td>no new neurological symptoms</td>
<td>39</td>
</tr>
<tr>
<td>10</td>
<td>stent-assisted GDCs embolization</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>resolution of symptoms</td>
<td>36</td>
</tr>
<tr>
<td>11</td>
<td>stent insertion</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>no new neurological symptoms</td>
<td>18</td>
</tr>
<tr>
<td>12</td>
<td>two balloons embolization</td>
<td>fistula closed</td>
<td>Yes</td>
<td>resolution of symptoms</td>
<td>22</td>
</tr>
<tr>
<td>13</td>
<td>two balloons embolization</td>
<td>fistula closed</td>
<td>Yes</td>
<td>resolution of symptoms</td>
<td>15</td>
</tr>
<tr>
<td>14</td>
<td>porous stent with GDCs embolization</td>
<td>fistula closed</td>
<td>No</td>
<td>resolution of symptoms</td>
<td>13</td>
</tr>
<tr>
<td>15</td>
<td>stent-assisted GDCs embolization</td>
<td>aneurysm occluded</td>
<td>Yes</td>
<td>no new neurological symptoms</td>
<td>7</td>
</tr>
</tbody>
</table>
catheter (Cordis Endovascular Systems, Inc., Miami, FL) was positioned in the distal cervical segment of the ICA. The fistulous portion of the cavernous ICA crossed with a 0.014-inch, hydrophilic-coated wire (Wizdom; Cordis Endovascular Systems, Inc., Miami, FL). Then, a 4 18-mm S670 coronary stent (Arterial Vascular Engineering, Inc., Santa Rosa, CA) was advanced over the wire and deployed by inflating the balloon to 6-8 atm gradually. After stent placement, the microwire was navigated with roadmapping techniques through the fistula into the cavernous sinus. Selective angiograms were obtained through the guiding catheter once it was positioned in the cavernous sinus. After the catheter tip was secured in the cavernous sinus, standard and/or fibered GDCs (Target Therapeutics, Fremont, CA) were introduced through the stent pore into the cavernous sinus. Repeated angiograms confirmed complete fistula closure with preservation of parent artery.

Combined Anterograde and Retrograde Coils Embolization of a Transected Vertebral Artery (Fig. 3)

Under the local anesthesia, the patient underwent GDCs embolization for the complete isolation of the injured segment. We undertook a contralateral approach to the distal portion of the injured segment of the left VA, using a 8-French MPA guide (Cordis Endovascular, Miami Lakes, FL) into the right VA and a 3.2-French Rapid Transit microcatheter (Cordis Endovascular, Miami Lakes, FL) after the systemic heparinization with a 5000 units bolus followed by hourly bolus infusion of 2500 units and monitoring of the activated clotting time. Microcatheter had to be passed the right VA to the basilar artery and down the segment of distal left VA. After the selective catheterization of the distal portion of the left VA, GDCs were deployed within the left VA. Therefore, GDCs were deployed within the left VA. The final angiography demonstrated a complete occlusion of the injured segment of the left VA.

Stent–Graft Placement of an ICA–IVJ Fistula and Pseudoaneurysm (Fig. 4)

The stent-graft device (JOSTENT, JOMED, Helsingborg, Sweden) consisted of a thin-walled expandable PTFE placed between two flexible stainless steel stents. Under systemic heparinization, a 7-French guiding catheter was positioned in the common carotid artery (CCA). A 6–12 × 38-mm peripheral graft stent was mounted across the fistula on a 6 40-mm ultra-thin diamond balloon (Medi-tech/Boston Scientific Corp, Natick, MA) through a 0.035-inch guide wire (Terumo Corp, Tokyo, Japan). Stent was deployed by inflating the balloon to 8 atm with

Stent placement for a dissecting aneurysm of the intracranial vertebral artery. A preoperative angiogram shows dissecting aneurysm of the left vertebral artery (A). After stent placement, the size of aneurysm is decreased (B).
optimal angiographic results. The fistula was immediately closed. The postdeployment angiogram demonstrated complete resolution of the pseudoaneurysm and restoration of the normal luminal diameter of the ICA.

Stent Placement with or without Coils Embolization of the Dissecting Aneurysms (Fig. 5) or Arterial Dissections (Fig. 6)

A balloon occlusion tolerance test of the injured vessel was performed before undertaking the placement procedure in cases of 4, 9, 10, and 11. These procedures were performed during a separate diagnostic session. Symptoms of cerebral ischemia did not develop in any patient. All stent placement procedures were performed via a transfemoral approach while the patient was in a state of general anesthesia. Six patients with a dissecting aneurysm of the ICA and VA were treated using a porous stent [extracranial ; wall stent (Boston Scientific, Bulach, Switzerland), intracranial ; S-670 coronary stent]. Because of persistence of the pseudoaneurysm in two patients, an additional procedure was required to occlude the aneurysm by inserting GDCs.

Following completion of the stenting and coiling procedure, control angiograms of the intracranial circulation were obtained to exclude thromboembolic occlusion.

Heparinization was continued for 24 hours and then allowed to taper physiologically. Oral administration of aspirin (100mg daily) or clopidogrel (75 mg daily) was continued for 6 weeks.

Transarterial Coils and Glue Embolization of a Pseudoaneurysm of the ECA (Fig. 7)

Selective angiograms were obtained through the guiding catheter once it was positioned in the internal maxillary artery. After the catheter tip was secured in the internal maxillary artery, standard and/or fibered GDCs (Target Therapeutics, Fremont, CA) were introduced into the aneurysmal sac. After the coils embolization under the general anesthesia, 0.5ml glue (B. Braun, Tuttingen, Germany) mixed with lipiodol was packed into the remnant of the aneurysmal sac. Repeated angiograms demonstrated complete occlusion of the aneurysmal sac.

**Results**

All fistulas and pseudoaneurysms were successfully embolized with coils, glue, and stents. There was no infection, episode of distal embolization, vessel rupture, or puncture site complication. The parent arteries of all patients except two patients (case 3 and 14) were preserved. Thrombus formation due to coil migration into the ICA resulted in sacrifice of the ICA (case 14), but there was no neurological complication. One patient with pseudoaneurysm treated by stenting (case 9) was decreased in size, but not completely occluded. Follow-up examinations included conventional angiography (14 patients) and MR angiography (1 patient) performed 4 to 12 months postoperatively, and clinical assessment ranging from 7 to 55 months in duration (mean 26.9 months). The presenting symptoms and signs were gradually improved. Especially in patients with fistula, the neurological symptoms and signs were dramatically improved. Preexisting neurological deficits due to embolic infarction was also gradually improved. No additional surgical procedures for vascular lesions were required. There were no delayed neurological or vascular complications and no lesions recurred during follow-up periods. The angiographic and clinical outcome of each patient is summarized in Table 2.

**Discussion**

Traumatic carotid and vertebral artery lesions often present in an acute setting with life-threatening consequences.
Traumatic Carotid and Vertebral Lesions

Unfortunately, not all vascular injuries associated with head and neck trauma are so readily recognized, and they are not associated with definitive therapeutic guidelines. Superimposition of severe brain injury renders major vascular disruptions prone to delayed or missed diagnosis. There are certain clinical features associated with an increased likelihood of the patient’s suffering a traumatic cerebrovascular injury, including a closed-head injury, facial fractures, and cervical spine fractures. In our experience, five patients presented with focal neurologic findings not otherwise explained, and the diagnosis for vascular injuries was delayed. Two patients were associated with cervical spinal fracture and two patients had multiple systemic injuries. A high index of suspicion and a fast notice of the presenting signs and symptoms of cerebrovascular insufficiency associated with head trauma are needed to result in a high detection rate of occult major intracranial and extracranial vascular injuries.

The mechanism of vascular injuries has significant bearing on presentation and natural history. These injuries result from both blunt and penetrating mechanisms. However, injuries from blunt force trauma more commonly present situations. The incidence of carotid and vertebral injury is unclear. Traditionally, the incidence of traumatic injury to the carotid artery from blunt trauma has been reported to be less than 0.1%. Although clinically evident vertebral injury has been reported to occur in less than 1% of all victims of blunt trauma, one study shows a 46% incidence of vertebral injury in patients who have midcervical spine fractures or subluxations. Penetrating injuries to the carotid and vertebral arteries account for only 3% and 0.5%, respectively, of arterial injuries treated in civilian trauma centers. In the blunt and penetrating mechanisms, a spectrum of vascular injuries may result including spasm, thrombosis, arteriovenous fistulas, mural arterial dissections, dissecting aneurysm and rarely, complete transection.

In the population of patients who have multiple injuries, it is not unusual to find traumatic injuries of the extracranial cerebral arteries incidentally during imaging for other purposes. Even in those who have symptomatic dissection, approximately half of them have been shown to have a normal neurologic examination at presentation. Patients with aneurysms resulting from traumatic dissections or penetrating injuries are more likely than those with spontaneous dissections to display significant neurological deficits. Traumatic dissections are more often associated with aneurysms or stenosis and are less likely to resolve without intervention. With spontaneous ICA dissections, angiographically evident improvement occurs in 60% of aneurysms and 80 to 90% of stenoses. With traumatic ICA dissections, however, angiographically demonstrated resolution occurs in only 20% of aneurysms and 50% of stenoses. All patients in this harbored symptomatic vascular lesions resulting from penetrating trauma or adjacent fractures, which we believed justified aggressive therapy.

Vascular lesions resulting from blunt or penetrating cranio cervical injuries are difficult to manage. Increasing experience with arterial trauma has taught us that some locations these lesions are dangerous to approach and difficult to repair. Large incisions and wide exposure are often required to achieve the proximal control and distal exposure required to close vascular defects and restore arterial and venous continuity. Recently, there has been remarkable progress in the development of porous and covered stents, which have been used primarily in the coronary and peripheral circulations. Neurovascular applications for stent use have more recently emerged. Stenting, both extra and intracranially, has gained popularity as a form of treatment for atherosclerotic lesions of the cerebral vasculature. There have been several case reports of the use of endovascular techniques, including stenting to correct traumatic dissections of the carotid and vertebral arteries. Little has been published, however, regarding covered stent placement for neurovascular disease. Experience in the coronary and peripheral circulations with the use of PTFE-covered stents has been particularly favorable, with extremely low rates of neointimal hyperplasia, indicating a potential antiproliferative effect of sealing the vessel wall. In our studies, eight cases was used the porous and PTFE-covered stents, and was successfully achieved the preservation of the parent artery. From the view of the parent artery patency, this result of stenting for the parent artery preservation is better than one of other treatment options, including the surgical approach and parent artery-sacrificing embolization.

Endovascular treatment has several advantages: (1) They can be performed under local anesthesia, thereby allowing continuous neurologic monitoring of the patient’s condition. This is particularly important if parent vessel occlusion is necessary. (2) In the acutely injured patient, time is of the essence, and if life-threatening hemorrhage, airway compromise, or cerebral ischemia has developed, then therapy can be rendered immediately after adequate diagnostic angiography. (3) Recovery time is faster and there are fewer traumas of adjacent cranial nerves and vessels.

A rare but important complications associated with stent use is the development of subacute thrombotic occlusion shortly after implantation. A number of variables appear to correlate with a higher of thrombosis, including small vessel size, proximal and distal dissection, and underdilation of the stent. These complications rate are declining due to preoperative and postoperative a variety of pharmaceutical manipulations. Clinical trials comparing various antithrombotic regimens following coronary artery stenting after angioplasty have demonstrated that treatment with
either ticlopidine and aspirin or clopidogrel and aspirin is more effective in reducing stent thrombosis than treatment with aspirin alone or aspirin and warfarin\(^{20,26}\).

### Conclusion

In the patients who have multiple injuries, it is difficult to find traumatic injuries of the extracranial cerebral arteries. Sometimes traumatic vascular injuries are found incidentally, are asymptomatic, or present with ambiguous neurologic symptoms and signs. Favorable outcome is guaranteed by early diagnosis and treatment. Screening for vascular injuries should be carried out in (1) any patient who has focal neurologic findings not otherwise explained; (2) any patient who has any two of the following three: closed-head injury, facial fractures, or cervical spine injury.

The goal of endovascular therapy is the selective elimination of the fistulous communication, aneurysm or stenosis with preservation of functional patency of the cerebral arteries. From our experiences, the endovascular therapy using stents, balloons, and coils is both feasible and safe in treatment of the traumatic vascular injuries. Long-term follow-up review of these repairs will be necessary to provide a full evaluation of the safety and efficacy of these devices.

### References


