

## Case Report

## Bilateral Occipital Infarctions Associated With Carotid Stenosis in a Patient With Persistent Trigeminal Artery

Andrew P. Gasecki, MD; Allan J. Fox, MD; Louise H. Lebrun, MD; Nicole Daneault, MD;  
for the Collaborators of the North American Carotid Endarterectomy Trial (NASCET)

**Background** Embolization via a persistent trigeminal artery, one of the embryonic vascular connections that may persist between the carotid and basilar arteries, is an unusual occurrence.

**Case Description** We describe a 76-year-old man with bilateral occipital infarctions presumably related to an ulcerated left carotid stenosis. Clinically, a left inferior homonymous quadrantanopia was present.

**Conclusions** Anomalous connections between the carotid and the usual posterior circulation territory should be considered in evaluating patients with vertebrobasilar stroke. When they are

identified, patients with symptoms attributable to the pontine vertebrobasilar territory supplied by the anomaly may be considered for carotid endarterectomy in the presence of concomitant severe carotid stenosis detected angiographically. Proper identification and treatment of such cases would be expected to prevent recurrence of disabling strokes in the vertebrobasilar circulation. These anomalies will likely be overlooked by ultrasound techniques and depend on good intracranial arteriographic images. (*Stroke*. 1994;25:1520-1523.)

**Key Words** • carotid artery diseases • ultrasonics • vertebrobasilar circulation

A persistent trigeminal artery is a rare vascular anastomosis between the carotid and basilar territories but is the most common of the primitive persistent carotid-basilar connections. Ulcerative atheromatous changes at the carotid bifurcation have been implicated as a potential source for microemboli passing through the trigeminal artery to the basilar artery and resulting in symptoms in the posterior circulation territory. Previously reported cases of transient vertebrobasilar symptoms related to carotid disease in patients with trigeminal arteries were determined on a clinical basis<sup>1-6</sup>; none were confirmed radiologically. This report documents occipital infarctions due to presumed emboli from a severe extracranial carotid lesion through a persistent trigeminal artery. Clinical implications for prevention of stroke in the distal basilar circulation are discussed.

### Case Report

A 76-year-old right-handed white man with a history of hypertension suddenly developed a left-sided headache and left lower field visual loss that persisted. Two months later he had a similar headache but with transient (15 to 20 minutes) right hemifield bright lights, during which he was unable to read the end of lines. He had no focal motor, sensory, or coordination symptoms

with these episodes. Subsequently, the patient was placed on aspirin and referred to the Hopital Saint-Luc for further investigations. His blood pressure was well controlled on antihypertensive medications.

Physical examination revealed a left inferior homonymous quadrantanopia confirmed by visual field charting. The remainder of the neurological examination was normal. The blood pressure was normal (120/80 mm Hg in both arms), the heart rate regular (68 beats per minute), and the patient had no carotid, supraclavicular, or orbital bruit or cardiac murmur.

A computed tomographic scan showed right parieto-occipital and left occipital infarction (Fig 1). Echocardiography, done without bubble contrast via a subxiphoid approach, and electrocardiogram were within normal limits. Selective carotid angiogram revealed 14% right and 47% left internal carotid stenosis. The left trigeminal artery (Fig 2), supplying both posterior cerebral arteries, was rising from the left internal carotid artery. Vertebral angiography confirmed that the proximal basilar artery was hypoplastic compared with the basilar artery beyond the trigeminal. There was no proximal atherosclerotic lesion related to the vertebrobasilar system or the aortic arch, which showed only minimal atherosclerotic irregularities without any stenosis of the vessels rising from it. The right vertebral artery was hypoplastic. Ultrasound revealed antegrade flow in both vertebral arteries.

The patient was maintained on warfarin for 8 months and subsequently on ticlopidine for 8 months. After 16 months of follow-up, he developed recurrent episodes of left-hand weakness. A second angiogram showed a 73% right internal carotid stenosis (Fig 3, left panel) and a 70% left internal carotid stenosis (Fig 3, right panel). Because the results of the North American Symptomatic Carotid Endarterectomy Trial had deter-

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From the Departments of Clinical Neurological Sciences (A.P.G., A.J.F.) and Diagnostic Radiology (A.J.F.), University Hospital, London, Ontario, and the Department of Neurology (L.H.L., N.D.), Hopital Saint-Luc, Montreal, Quebec, Canada.

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Correspondence to Allan J. Fox, MD, University Hospital, Department of Diagnostic Radiology, 339 Windermere Rd, London, Ontario N6A 5A5, Canada.

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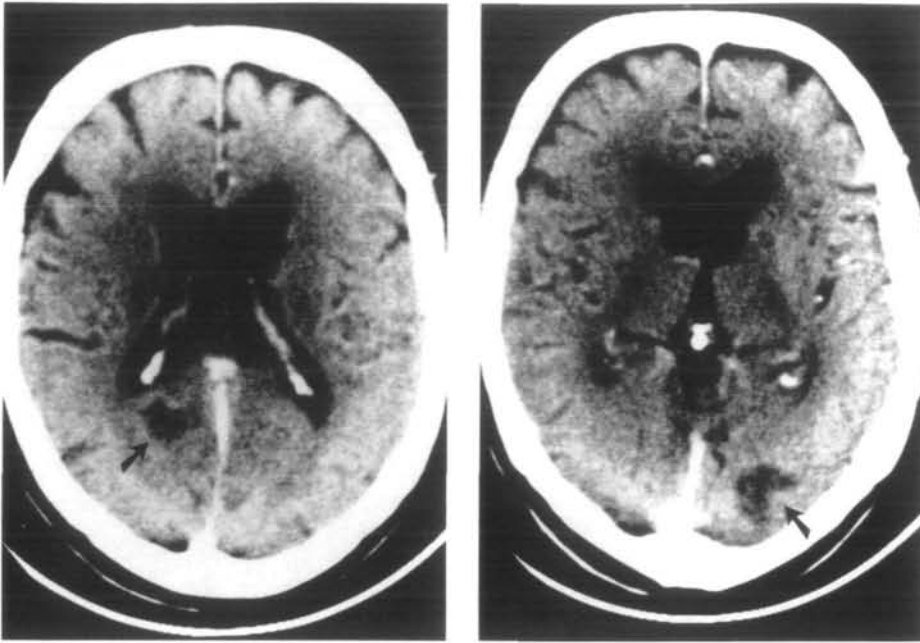


FIG 1. Computed tomographic scans without contrast show a right parieto-occipital infarction (left panel) and a left occipital infarction (right panel).

mined benefit for symptomatic patients with severe (>70%) stenosis, he underwent right carotid endarterectomy and, a month later, left carotid surgery, both without complications. Intraluminal shunts were used during both surgeries.

### Discussion

Occipital ischemia due to microembolization is usually caused by vertebrobasilar anomalies. Occasionally it has been described in conjunction with a fetal origin of the carotid portion of the posterior cerebral artery.<sup>7,8</sup> In our patient, the bilaterality of infarcts documented on the computed tomographic scan of the brain was explainable by probable embolization from the extracranial

carotid artery via a persistent trigeminal artery. A direct connection between the carotid and basilar arteries through the trigeminal artery effectively replaced the terminal territory of supply of the basilar artery through the posterior cerebral arteries to both occipital lobes.

The trigeminal artery is the most common persistent carotid-basilar anastomosis observed in adult life. The reported angiographic frequency of the trigeminal artery is estimated at between 0.02% and 1.25%<sup>9-16</sup> but may be as high as 4% in mentally retarded patients.<sup>17</sup> The trigeminal artery has been associated with aneurysms,<sup>11,13,18-20</sup> arteriovenous malformations,<sup>19</sup> cavernous sinus fistula,<sup>21,22</sup> moyamoya disease,<sup>23</sup> anomalies of origin of the cerebellar arteries,<sup>24</sup> and agenesis or occlusion of the internal carotid artery.<sup>25</sup>

Few reports relate the symptoms of vertebrobasilar ischemia to atherosclerotic changes at the carotid bifurcation, suggesting a link between carotid disease, vertebrobasilar symptoms, and the trigeminal artery. Waller et al<sup>4</sup> first implicated extracranial carotid disease as a possible source of emboli to the posterior circulation through the persistent trigeminal artery. Internuclear ophthalmoplegia,<sup>26</sup> transient bilateral cortical blindness,<sup>1,2</sup> recurrent pontine hemorrhages,<sup>27</sup> recurrent infarctions of the brain stem,<sup>28,29</sup> and recurrent transient ischemic attacks in the vertebrobasilar territory<sup>3-6</sup> have been reported to be associated with persistent trigeminal artery.

The trigeminal artery appears in the 3-mm embryo, rises from the internal carotid artery, and supplies the precursors of the basilar artery (longitudinal neural arteries). Therefore, it forms the major blood supply to the posterior circulation structures until the 14-mm fetal stage, when it regresses. The posterior cerebral and posterior communicating arteries take over its function to carry the blood flowing from the distal end of the basilar artery. If it persists into adult life, the trigeminal artery is a somewhat tortuous artery coming off the cavernous portion of the internal carotid artery. It courses posteriorly, beneath or above the abducens

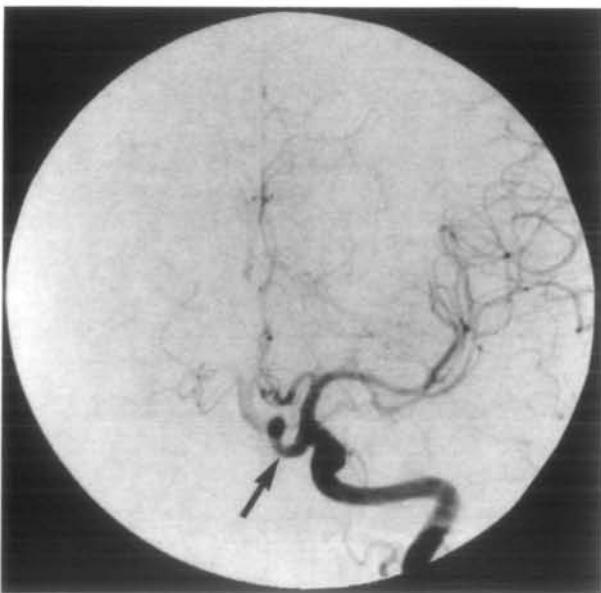


FIG 2. Selective carotid angiogram (left carotid injection, anterior oblique projection). The left trigeminal artery (arrow), coming off the left internal carotid artery, supplies the terminal portion of the basilar artery and both posterior cerebral arteries.

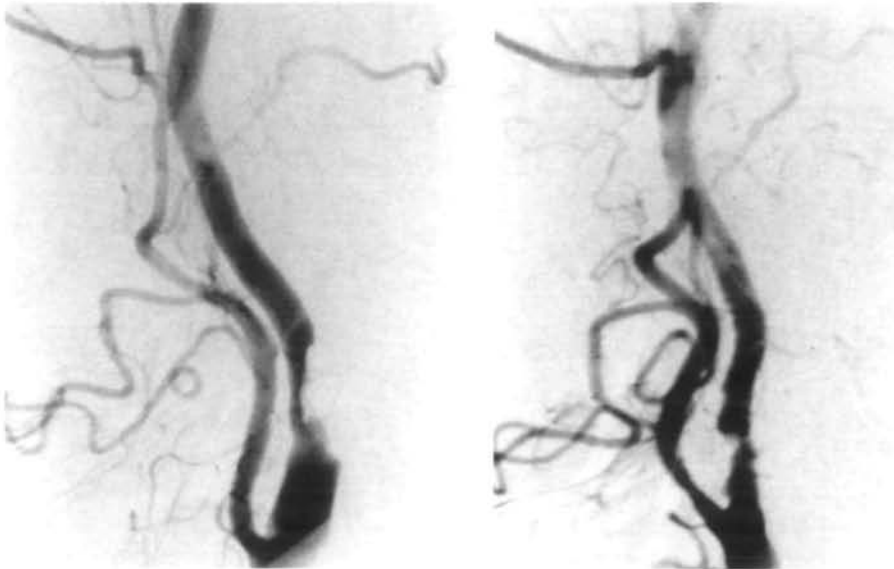


FIG 3. Selective carotid angiograms (right and left carotid injections, lateral projections) reveal ulcerated 73% stenosis of the right internal carotid stenosis (left panel) and 70% stenosis of the left internal carotid stenosis (right panel).

nerve, and then medially to the trigeminal ganglion to join the basilar artery between the superior and anterior inferior cerebellar arteries. There is no clinical syndrome specifically correlated to the existence of a trigeminal artery. The close proximity to the cavernous sinus structures may result in signs of cranial nerve dysfunction (oculomotor,<sup>30</sup> trigeminal,<sup>4</sup> trochlear,<sup>31</sup> and abducens palsies<sup>32</sup>). This vessel may be at risk during gasserian ganglion surgery<sup>32,33</sup> and has been implicated as a possible cause of trigeminal neuralgia.<sup>34,35</sup> The only reported case of inadvertent occlusion of the trigeminal artery resulted in transient ophthalmoplegia and facial hypalgesia and permanent sixth nerve paresis, which were related in part to intraoperative manipulation and bleeding.<sup>36</sup> Symptomatic moderate carotid stenosis, with which this patient initially presented, is currently under close scrutiny. Benefit of endarterectomy was shown only for severe carotid stenosis,<sup>37</sup> and our patient underwent bilateral endarterectomies when the stenoses, associated with bilateral symptoms, reached severe degrees (70% and 73%).

Anomalous anastomoses between the carotid and the usual posterior territory usually can be well shown on both conventional and magnetic resonance angiography and should be considered in the differential diagnosis of vertebrobasilar stroke. A trigeminal artery would be overlooked by carotid ultrasound techniques. Magnetic resonance angiography was shown by Schuierer et al<sup>38</sup> to identify the trigeminal artery in a noninvasive manner. This technology, when fully developed and available, will become the investigation of choice for this anomaly. In the meantime, patients with vertebrobasilar stroke should undergo adequate intracranial imaging by conventional arteriography so as not to overlook the presence of a persistent trigeminal artery. While anomalies such as this are uncommon, the results of carotid endarterectomy for severe stenosis in preventing future stroke are compelling and far more successful than available therapies for vertebrobasilar stroke. Identification of this lesion in the presence of severe carotid stenosis points to the need for carotid endarterectomy to prevent stroke in the distal basilar circulation. The risk of serious stroke as a result of overlooking severe

carotid stenosis in the possible presence of this anomaly justifies conventional intracranial arteriography. The benefit obtained from surgery in patients with severe ( $\geq 70\%$ ) stenosis outweighs the 0.5% to 1% risk of arteriography.<sup>37</sup>

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