Vertebral Artery Dissection and Cerebellar Infarct Triggered by Anger: A Case Report

J F Harrison, R A Vega, A M Martino

Citation

J F Harrison, R A Vega, A M Martino. *Vertebral Artery Dissection and Cerebellar Infarct Triggered by Anger: A Case Report*. The Internet Journal of Neurosurgery. 2013 Volume 9 Number 1.

Abstract

Vertebral artery dissection is an important cause of posterior circulation infarcts. There have been numerous reports of associated risk factors for vertebral artery dissection, however high levels of anger and hostility have not been reported. We report a case of anger precipitating an ischemic stroke in a 40-year-old male with no known vascular risk factors. A 40-year-old male experienced an acute onset of severe headache, dizziness and vomiting after becoming violently angry. CT scan revealed a right cerebellar lesion with obstructive hydrocephaly. An external ventricular drain was placed, and MR angiogram subsequently identified a right 2 cm vertebral artery dissection. The drain was removed 72 hours after admission, and the patient was discharged on day nine following the ischemic event to an inpatient rehab facility. To our knowledge, this is the first report of anger as a sole risk/precipitating factor for stroke in a young person without additional risk factors. Management of trait anger may be an important adjunctive therapy for reducing stroke risk as well as avoiding triggers for ischemic events.

INTRODUCTION

Vascular risk factors associated with stroke are well documented in the literature. However, much less understood and perhaps of significant importance are precipitating factors that may lead to an acute stroke. Anger has been associated with an increased risk of stroke and may be an important precipitating element in some patients (4). To our knowledge, this is the first report of anger as a sole risk/precipitating factor for stroke in a relatively young, previously healthy person without any known additional contributing factors.

CASE REPORT

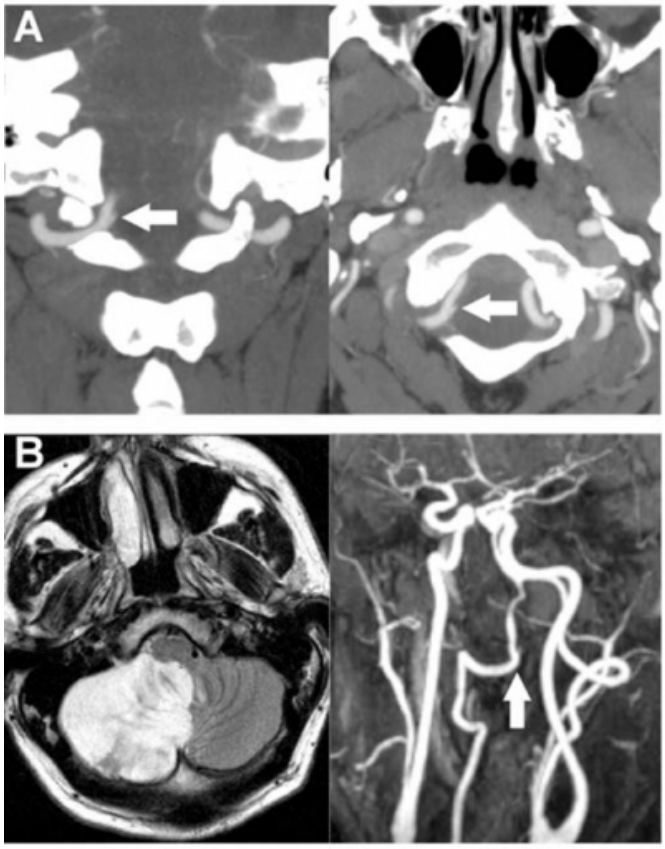
A 40-year-old male became angry with one of his employees in January of 2009. Immediately following a tirade, the patient experienced an acute onset of severe headache and vomiting. In the months leading up to this incident, the patient and his family had noticed that he had become increasingly prone to sudden bouts of anger. The patient and his family attributed his worsening anger to increased stress at work due to the recent recession in the economy. The patient reported no significant past medical or family history of cardio or cerebrovascular disease. The patient denied the use of tobacco or illicit drugs and reported no recent history of trauma.

Forty-eight hours following the initial onset of symptoms, the patient arrived in the ED complaining of severe headache and dizziness. On admission, the patient's vital signs and neurological exam were normal with the exception of a mild upper extremity dysmetria noted to be worse on the right side. Additionally, upon standing, the patient was very unsteady. Echocardiogram and laboratory tests including a urine drug screen were all normal. CT scan revealed a right cerebellar mass defect with fourth ventricular compromise and obstructive hydrocephaly. An external ventricular drain was placed, and he was admitted to the ICU for management. MRI and CT angiography confirmed a right cerebellar infarct and revealed a right vertebral artery defect (Figure 1). 3D MR angiogram identified a right 2 cm vertebral artery dissection with a narrow distal lumen. The decision was then made to manage the patient conservatively with intravenous fluids and Aspirin therapy. Therapeutic anticoagulation with Heparin was not commenced due to the risk of causing hemorrhage into the infarct.

During the course of his hospital stay, the patient's headache and vomiting resolved. The external ventricular drain was successfully weaned off and removed 72 hours after admission. However, he continued to exhibit appendicular ataxia and was discharged nine days following the onset of symptoms to an inpatient rehabilitation facility

Figure 1

CT angio axial and coronal sections through the C1-occipital junction (A). T2 weighted MRI shows increased signal in the right cerebellum with leftward shift of the fourth ventricle, and 3D MR with gadolinium enhanced angiogram showing a right 2 cm vertebral artery dissection with a narrow distal lumen (B). Arrows point towards the right vertebral artery defect.



DISCUSSION

It has long been postulated that extreme expressions of emotion can precipitate catastrophic vascular events, and there is compelling evidence linking anger to increased morbidity and mortality associated with cardiovascular disease (5). Given the epidemiologic data relating to heart disease, it stands to reason that similar cerebral vascular reactivity in response to anger and hostility may also lead to ischemic stroke. Several studies have associated stroke with a preceding episode of anger (1), and findings from the Framingham Heart Study show the incidence of stroke was significantly higher in persons with high levels of anger/hostility (3). A prospective epidemiological study based on the Kuopio Ischemic Heart Disease Study revealed that men who are prone to frequent outward expressions of anger had twice the risk for stroke and that an exaggerated sympathetic arousal may play a role in precipitating acute ischemic events (4). More recently, Williams et al (8) examined the relationship between trait anger and incident stroke risk in middle-aged men and women and found that participants who were 60 years of age or younger with high trait anger were at 3 times greater risk for ischemic stroke compared to participants with low trait anger.

A triggering hypothesis has emerged that views anger as a physiologic modifier of hemodynamics that may contribute to an acute ischemic event (5). Evidence suggests that exaggerated discharge of catecholamines can acutely injure blood vessel walls via transient increases in pressure and flow velocity (7). In addition to hemodynamics and vasoconstriction, catecholamines also have prothrombotic effects (2). Therefore, excessive sympathetic arousal and associated neuroendocrine activation in response to stress and frequent bouts of anger may underlie the relationship between anger and ischemic stroke.

This case report documents an example of cerebellar infarct secondary to vertebral artery dissection and is one example of a possible temporal relationship between a bout of rage and an ischemic stroke. Due in part to tethering of the vessels within the transverse foramen, sudden lateral movements of the head have been reported to stretch the vertebral arteries resulting in dissection (6). If during this incident or sometime previously the patient had injured the intimal lining of his right vertebral artery, then it is possible that the additive physiologic modifiers described above in combination with arterial dissection may have triggered this ischemic event.

In conclusion, control of precipitating events that may trigger an ischemic stroke such as anger may be an effective means for lowering stroke risk. Therefore, in addition to traditional treatments and lifestyle modifications, management of trait anger may be an important adjunctive therapy for reducing stroke risk as well as avoiding psychological triggers of stroke

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Author Information

Jason F. Harrison, M.D., Ph.D.
Department of Neurosurgery, Virginia Commonwealth University
Richmond, VA, USA
jharrison@mcvh-vcu.edu

Rafael A. Vega, M.D., Ph.D.
Department of Neurosurgery, Virginia Commonwealth University
Richmond, VA, USA
ravega@mcvh-vcu.edu

Anthony M. Martino, M.D.
Department of Neurosurgery, University of South Alabama
Mobile, AL, USA
amartino@usouthal.edu