

**Exercise Induced Transient Neurological Deficit in a Patient with
Cerebellar Developmental Venous Anomaly**

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Abstract

Background—The association of venous angiomas or developmental venous anomalies (DVA) with transient neurological deficit is rare. We present a rare case of a cerebellar developmental venous anomaly resulting in transient neurological deficits.

Case Description—A 58-year-old man with recurrent left sided facial dysesthesia, hemiparesis, and mild difficulty ambulating after exercise. A similar episode was experienced six months earlier under the same circumstances. Computed tomographic (CT) scan demonstrated an ill-defined hyperdensity in the right cerebellar hemisphere. Cerebral angiography demonstrated a venous angioma that dilated with valsalva maneuver.

Conclusion—We report the first case of a cerebellar venous angioma causing exercise induced transient neurologically deficits. Limiting strenuous exercise may be needed to avoid symptom recurrence. Further research is warranted on the hemodynamic effects of intracranial DVA's.

Keywords

Venous angioma; transient neurological deficit; developmental venous anomaly; angiography

Introduction

Venous angiomas or developmental venous anomalies (DVA) are a congenital malformation of the normal venous drainage of the brain. They are usually asymptomatic and follow a benign course but may cause seizures, headaches, and rarely bleeding. We present a case of recurrent transient neurological deficit associated with a DVA.

Case Report

A 58-year-old man, with past medical history significant for dyslipidemia, suddenly developed a warm sensation on the left side of his face after jogging for approximately one mile at his local community center. Subsequently, he developed left hemiparesis and mild difficulty while ambulating. He reported resolution of symptoms upon discontinuation of physical activity. He underwent a computed tomographic (CT) scan that demonstrated an ill-defined mixed hyperdensity within the right cerebellar hemisphere (Fig. 1). He was subsequently transferred to our medical center for further



Figure 1. Noncontrast computed tomographic (CT) scan image at the level of the 4th ventricle demonstrating hyperdensity within the right cerebellar hemisphere with multiple small surrounding calcifications.(arrow)

evaluation. He denied any history of vertigo, presyncope, or headache. On further questioning, he noted an identical episode that had occurred six months prior while he was jogging that also resolved upon resting.

On physical examination, his blood pressure was 147/88 mmHg, and he had no neurological deficits. Magnetic resonance imaging (MRI), diffusion-weighted imaging (DWI) sequence at this level of the fourth ventricle revealed a focal hypointensity consistent with a flow void (Fig. 2). Fluid-attenuated inversion recovery (FLAIR) at this level demonstrated focal hyperintensity in the right cerebellar hemisphere with radially oriented smaller hyperintensities consistent with developmental venous anomaly (Fig. 3). Gradient echo sequence at the same location showed focal hyperintensity in a typical “caput medusae” pattern representative of a DVA (Fig. 4). MRI with contrast demonstrated a DVA of the inferior aspect of the right cerebellum (Fig. 5). He underwent a diagnostic cerebral angiogram (Fig. 6) to evaluate for the presence of any arteriovenous malformation (AVM) in the vicinity of cerebellar hyperdensity. Cerebral angiography was significant for a 13.8 mm × 7.4 mm dilated vein fed from circumferentially radiating small veins filling during the venous phase that became more prominent during valsalva maneuver (Fig. 7).

The patient remained asymptomatic throughout his hospitalization and was reassured and discharged home with instruction to minimize strenuous exercise. He was seen in clinic four weeks later and remained asymptomatic.

Discussion

DVAs are generally caused by arrested development of venous structures early in fetal development and may also undergo evolutionary changes later in life.[1]. Previously thought to be rare with radiological imaging dating back to 1967, these anomalies are now considered quite common, occurring in almost 2% of the general population due to better visualization by contrast-enhanced CT and contrast-enhanced MRI (which is also the preferred imaging for diagnosis) [1,2]. DVAs are described as multiple radially oriented dilated medullary veins that drain into a transparenchymal venous stem, described as the “caput medusa” of the cerebral venous drainage [1]. These DVAs usually drain into larger collector veins that can range from small portions of the brain to large portions of hemispheric and even entire hemispheres.

There are several locations in the brain where DVA occur. A study published in 1991 with 100 patients



Figure 2. Magnetic resonance imaging using diffusion-weighted imaging (DWI) at the level of the fourth ventricle image (a) There is a focal hypointensity within the right cerebellar hemisphere consistent with a flow void.



Figure 3. Magnetic resonance fluid-attenuated inversion recovery (FLAIR) at the level of the fourth ventricle image demonstrating Focal hyperintensity in the right cerebellar hemisphere with radially oriented smaller hyperintensities consistent with DVA (arrow).

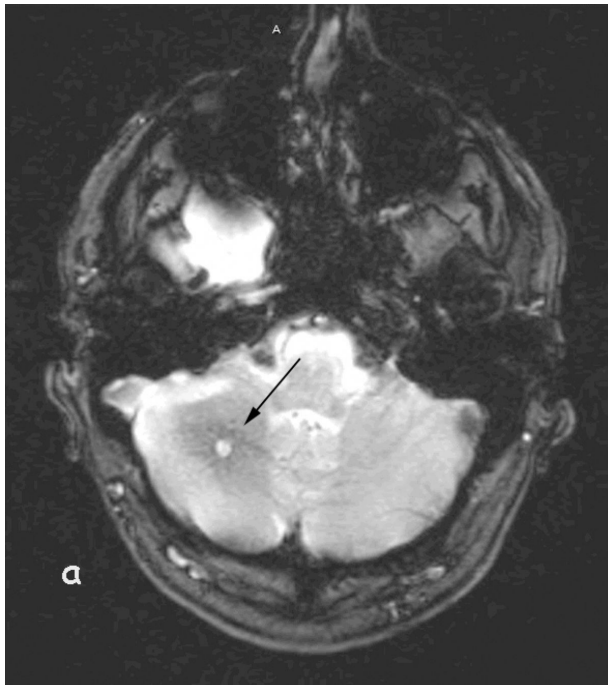


Figure 4. Magnetic resonance imaging using gradient recalled echo [MR GRE] at the level of the fourth ventricle image (a) Focal hyperintensity with surrounding blooming artifact that can be secondary to a remote hemorrhage (arrow).

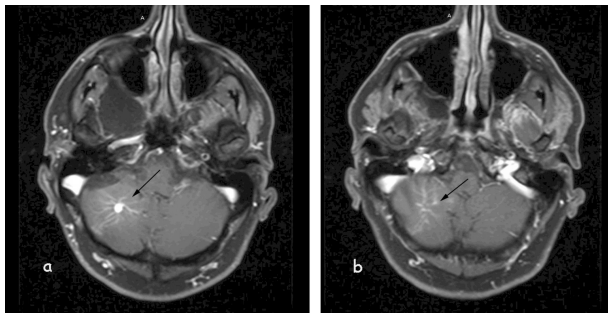


Figure 5. Magnetic resonance imaging with contrast at level of the fourth ventricle image (a) and (b) prominent central vein with radially oriented smaller draining veins in a classic “caput medusa” pattern consistent with cerebellar Dva.

showed that there were 42 cases involving frontal lobe, 24 cases involving parietal lobe, 4 cases involving occipital, 2 cases of temporal, 11 cases of basal or ventricular, 14 cases of cerebellar, and 3 cases with DVA in the brain stem [3]. This study demonstrated that majority of DVAs are found in the frontoparietal (36%–64%) region draining toward the frontal horn of the lateral ventricle

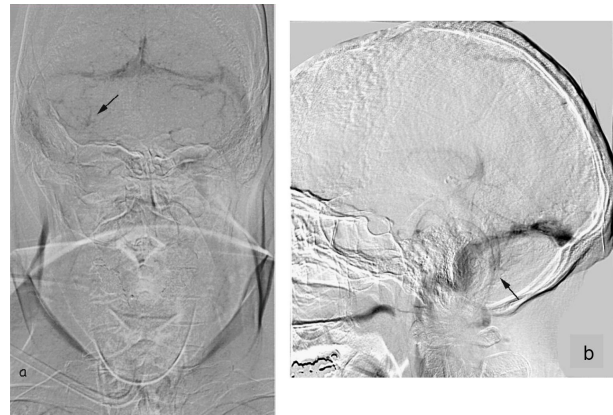


Figure 6. (a) Anteroposterior view (AP view) image (b) lateral view of left vertebral artery injection during midvenous phase demonstrating right cerebellar hemisphere venous prominence before valsalva maneuver with smaller relatively inconspicuous draining veins.



Figure 7. Angiographic image (a) Anteroposterior view (AP view) image (b) lateral view of left vertebral artery injection during midvenous phase demonstrating right cerebellar hemisphere venous prominence during valsalva maneuver with multiple smaller draining veins in a burst pattern consistent with a DVA.

and cerebellar hemisphere (14%–27%) draining toward the fourth ventricle [4].

Patients with DVAs usually remain asymptomatic and the anomaly remains dormant throughout life. Usually, treatment is not indicated, except in events such as persistent headache, dizziness, ataxia, seizures, or in rare cases hemorrhage. Hemorrhage is usually associated with other related vascular malformations, namely cavernous angiomas, mixed vascular malformations, or rarely blue rubber bleb nevus syndrome. This warrants further evaluation with contrast-enhanced MRI in patients with DVA associated with intraparenchymal

hemorrhage [5,6]. Surgical decompression is usually performed due to mass effect of hematoma as opposed to resecting a potential source of bleeding.

Another very rare presentation of DVA is a transient neurological deficit. In review of literature, to the best of our knowledge, there was only one other case of a 59-year-old woman hospitalized for transient neurological deficit and was found to have a subcortical venous angioma of the left parietal lobe. The low prevalence may have resulted from a previous putaminal hemorrhage causing decreases in blood circulation in the left cerebrum. The authors speculated that the neurological deficits were caused by either transient ischemic thrombus or a steal phenomenon due to decrease in blood circulation [6]. Our patient has a right cerebellar DVA and his symptoms localized to the left cerebellar hemisphere. However, the DVA becomes more prominent on angiography during valsalva maneuver suggests hemodynamic shunting of blood toward the lesion away from adjacent areas resulting in a potential source of ischemia.

Conclusion

Intracranial DVAs are common cerebrovascular malformations that may be associated with transient neurologi-

cal deficits. Such occurrences are under-recognized given the low prevalence of DVAs in the general population. Further cerebrovascular flow studies of intracranial DVAs are warranted to better understand their hemodynamic effects on adjacent areas.

References

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