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CASE REPORT Open Access

Rare cause of bilateral basal ganglia calcifications: hemispheric AVM

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Abstract

Background Intracranial calcifications may occur physiologically or pathologically for many reasons. In arteriovenous malformations (AVMs), calcification is not uncommon and is usually detected in the lesion vessel walls and surrounding parenchyma. However, rarely calcifications can also be seen in bilateral basal ganglia and especially in watershed areas, which are far from the lesion.

Case presentation In this article, we present a 47-year-old case of hemispheric AVM accompanied by bilateral basal ganglia calcification.

Conclusions Since the direct diagnosis of AVM in non-contrast brain-computed tomography (CT) is difficult, the detection of calcification in these regions requires the presence of AVM in the differential diagnosis.

Keywords Hemispheric AVM, Basal ganglia calcifications, MR imaging

Introduction

Arteriovenous malformations (AVM) are the abnormal connections between the normal arteries and veins of the brain without resistance vessels. In the classical brain AVM, this connection occurs through a nidus. If there is a direct connection without this stucture, it is also called arteriovenous fistula (AVF) (1–3). They are thought to be congenital, but are usually diagnosed at the mean age of 31 with signs of intracranial hemorrhage and seizures (4, 5). AVM can be divided into two main types according to nidus form; compact (glomerular) nidus and diffuse (proliferative) nidus. There is no normal brain tissue between abnormal blood vessels in the compact or glomerular type. Proliferative or diffuse type is rare and is characterized by the interspersed of normal brain tissue between abnormal vessels. Usually, one lobe or whole brain hemisphere is affected. This type constitutes 2–4% of all brain AVMs and is more common in females (1, 6). Calcifications in the AVM, if any, are usually present in the vascular walls of the AVM and around of this area that gliotic parenchyma (7). Bilateral basal ganglion calcification and subcortical calcifications far from the lesion have rarely been reported. In this article, we aimed to present the imaging features of a patient with hemispheric proliferative type AVM with bilateral basal ganglia and subcortical calcification.

Case presentation

A 47-year-old female patient was admitted to our clinic with blurred consciousness and seizures. Neurological examination revealed right hemiparesis and her glasgow coma score (GCS) was 14. Initially, non-contrast brain-computed tomography (CT with Definition Flash, Siemens Healthcare) was performed. CT scans showed relatively indeterminate hypodensities extending from the left hemisphere to the brain stem. Left lateral ventricle was under compression and from left to right shift was present. Mildly hyperdense tubular areas were noted in the left frontal-parietal region, which may be compatible with enlarged vascular structures. Diffuse

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calcifications were also observed in bilateral basal ganglia and both hemispheres, more on the left. (Fig. 1). Brain magnetic resonance imaging (MR) was performed with a 3 T device (Magnetom Skyra; Siemens Healthcare, Erlangen, Germany) with a preliminary diagnosis of mass and vascular malformation due to the presence of marked oedema and tubular densities. Tortuous tubular structures showing signal void that started at the left parietal lobe and spread to the entire hemisphere were evaluated as AVM. Post-contrast MR angiography showed that the feeding arteries of the AVM were left anterior cerebral artery, left medial cerebral artery, and posterior cerebral artery together (Fig. 2). Drainage veins were superior sagittal sinus especially through dural veins at the vertex level. There was also drainage to the left sinus rectus and cavernous sinus. On T1-weighted images, increased signals in the subcortical areas at the lesion side and in bilateral putamen and thalamus were evaluated in favor of calcification. Susceptibility weighted imaging (SWI) sequence showed no bleeding in the AVM neighborhood and SWI and phase images confirmed the presence of the calcifications (Fig. 3). Post-contrast dynamic susceptibility contrast (DSC) T2* perfusion images showed hyperperfusion in the AVM and hypoperfusion in the adjacent gliotic area. On tractography, it was observed that the association and projection fibers were destroyed in the affected area (Fig. 4). After cerebral MR angiography confirmed that the lesion was AVM. The patient was not considered for surgical treatment in our center, but was discharged with GCS: 15 by symptomatic treatment.

Discussion

Intracranial calcifications are not uncommon and can be seen physiologically and pathologically for many reasons. Basal ganglion calcification, which is more common in elderly patients, occurs in approximately 1% of all brain CTs. Although it is considered to be more idiopathic in the elderly, it should be considered pathological in individuals younger than 40 years. The differential diagnosis includes toxic, infectious, metabolic, and hereditary causes. Subcortical calcifications are also nonspecific findings and may occur due to many reasons such as Fahr's disease, tuberosclerosis, Sturge-Weber syndrome and metabolic diseases (8, 9). Arteriovenous malformations are one of the common cerebral vascular malformations caused by abnormal vascular connections. Calcifications in AVM usually occur on the walls of abnormal vessels but neighbor parenchyma and basal ganglia calcifications have been rarely described in the literature (1, 7). Its mechanism is not fully understood but 'cerebral steal' phenomenon is considered to be the most likely cause in the etiology. Cerebral blood flow studies have shown that AVMs have three times more blood flow than normal. Therefore, it is believed that dystrophic calcifications occur in hypoperfused areas. In contrast to the other diseases mentioned, calcifications in AVMs are symmetrically

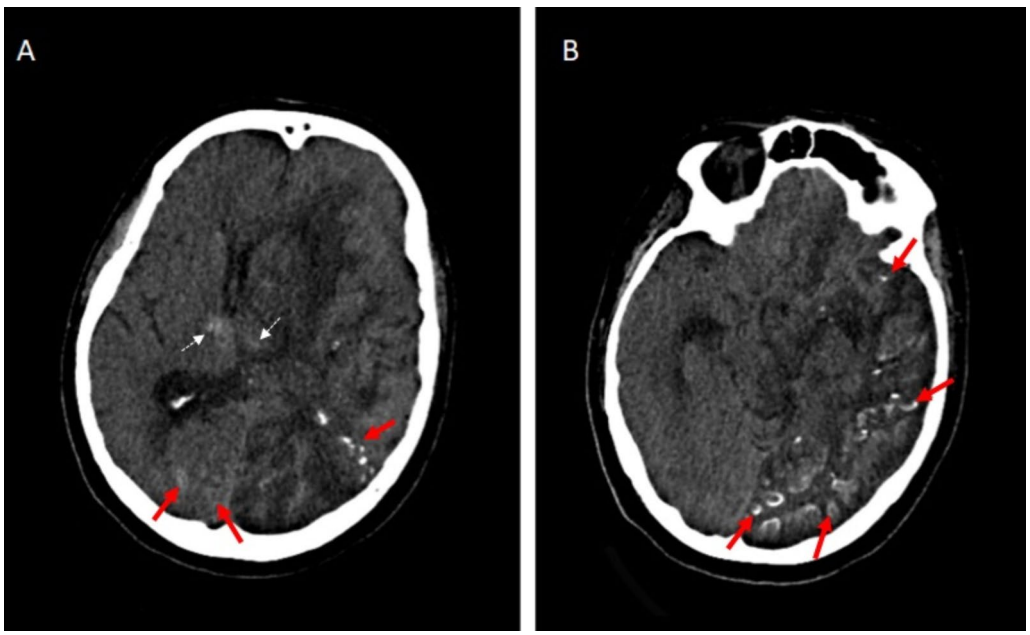

Fig. 1 Non-contrast axial soft tissue windowed brain CT scan shows bilateral symmetrical basal ganglia calcification (**A**, white arrows) and diffuse calcifications (red arrows) in both hemispheres, prominent on the left

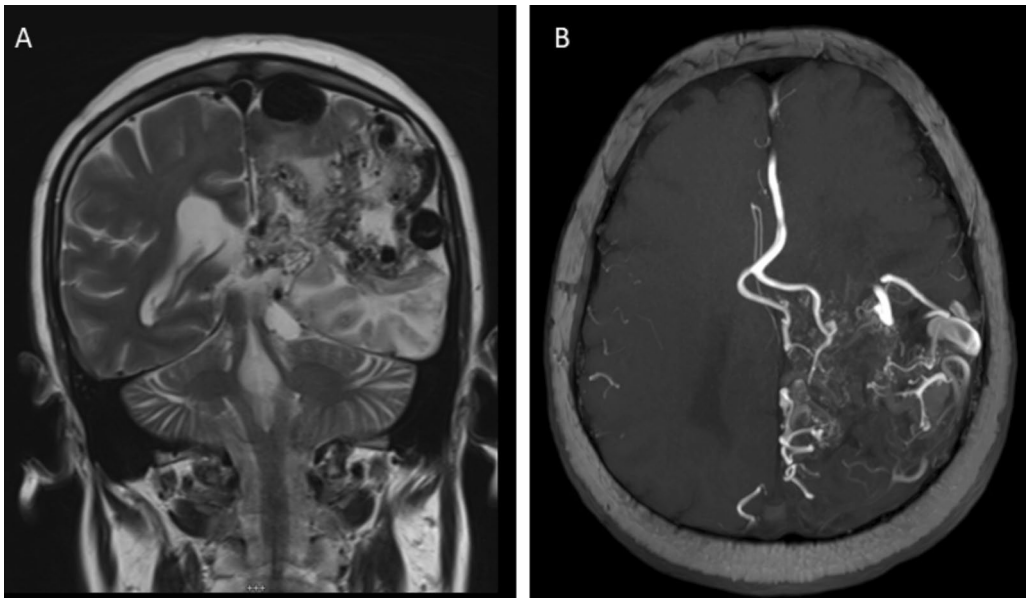

Fig. 2 T2-weighted coronal MR image (**A**) show the tortuous tubular signal void areas in the left hemisphere of the AVM. In post-contrast MR angiography images (**B**), it is seen that the AVM feeds from the anterior, middle and posterior cerebral artery

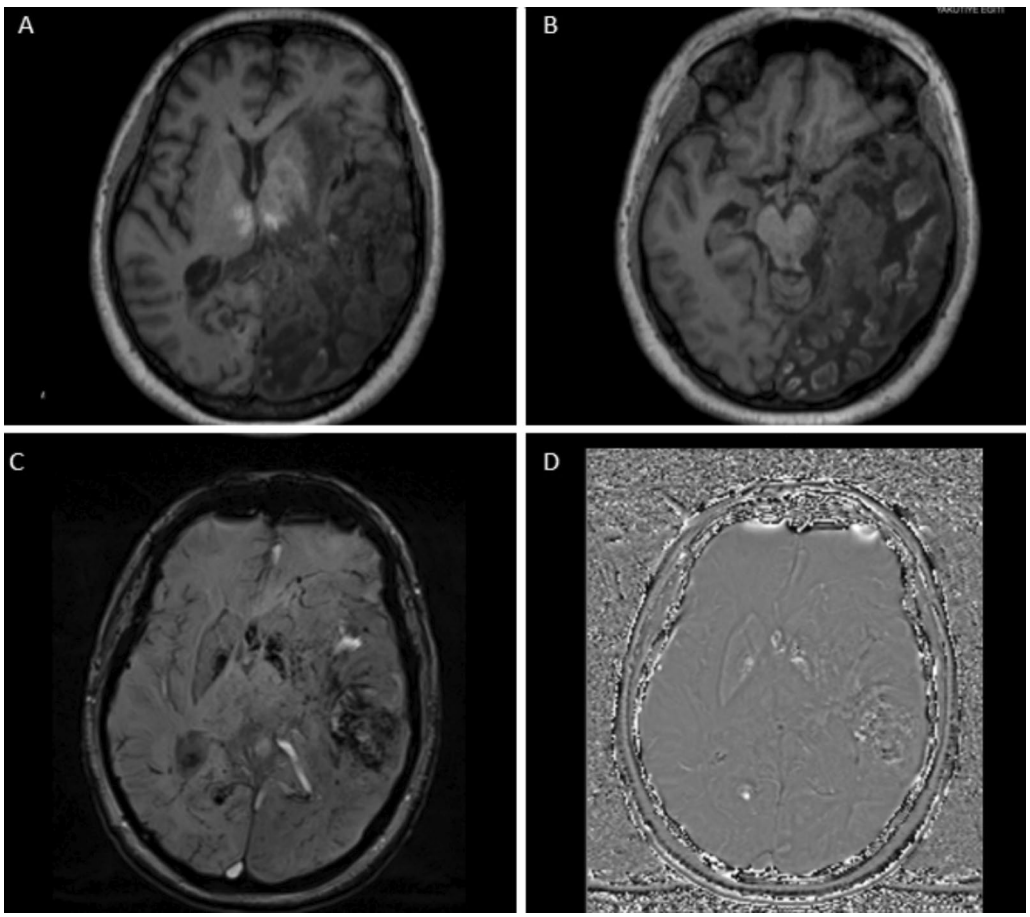

Fig. 3 Hyperintensities of calcifications in basal ganglia and subcortical areas are observed on T1-weighted axial images (**A**, **B**). The same areas are seen as hypointense in the SWI sequence (**C**), and hyperintense in phase images (**D**)

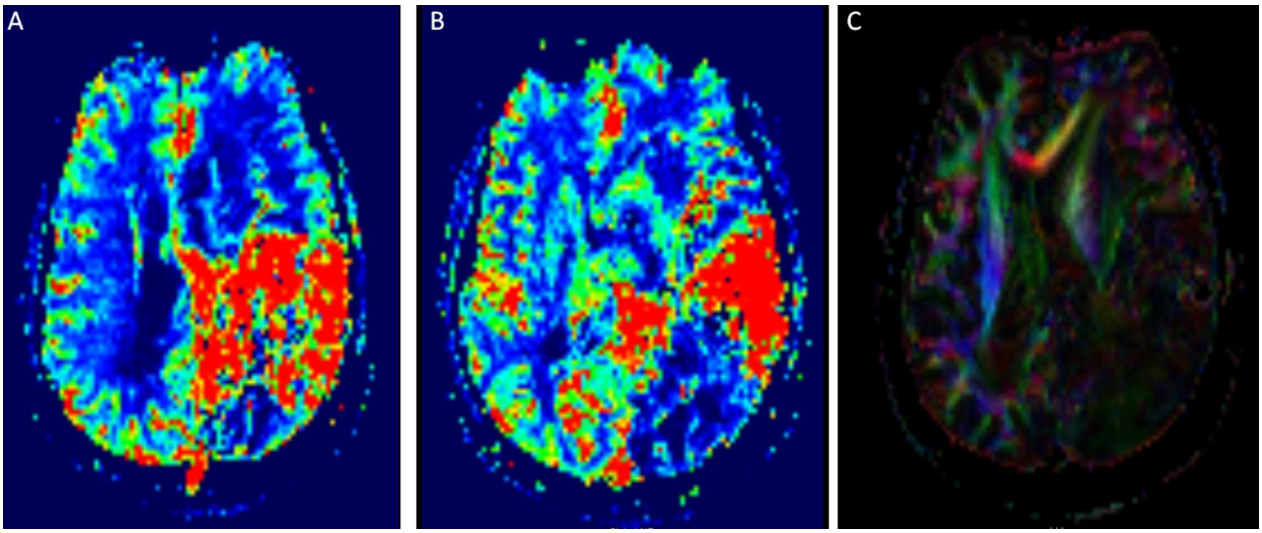

Fig. 4 Post-contrast DSC T2* perfusion image cerebral blood flow maps (**A**, **B**) show hyperperfusion in the AVM and hypoperfusion in the adjacent gliotic area. Tractography (**C**) shows destruction of the association and projection fibers at the left lobe

subcortical and no other calcification foci are observed (7–11).

Conclusions

AVM may not be diagnosed directly on non-contrast brain CT. Therefore, AVM should be kept in mind when calcification is detected, even if it is rare. Although cerebral angiography is the gold standard in the diagnosis of AVM, MR and MR angiography play an important role in the diagnosis and detection of accompanying anomalies.

Abbreviations

AVM	Arteriovenous malformation
CT	Computed tomography
AVF	Arteriovenous fistula
GCS	Glasgow coma score
MR	Magnetic resonance
SWI	Susceptibility weighted imaging
DSC	Dynamic susceptibility contrast

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Author contributions

EG—conceptualization, data collection, and writing—initial draft. HO—writing—initial draft, editing, and supervision. All authors read and approved the final manuscript.

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Availability of data and materials

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Declarations

Ethics approval and consent to participate

Ethics committee approval was not considered necessary because it was a case report. Informed consent was obtained from the patient for this study.

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Competing interests

The authors declare that they have no competing interests.

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