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Case Report

Appearance of an unusual ring enhancing brain capillary telangiectasia on 3.0T MRI with dynamic susceptibility contrast perfusion

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ABSTRACT

We present the appearance of brain capillary telangiectasia on 3.0T magnetic resonance imaging (MRI) perfusion. A 42-year-old female presented with intermittent left arm weakness and paresthesia. Initial 1.5T MRI obtained 2 months after presentation demonstrated a 6 mm right caudate head lesion with ring-like enhancement, and no significant surrounding edema or mass effect. On gradient echo there was mild associated susceptibility artifact. Follow-up 3.0T MRI demonstrated increased blooming on 3.0T imaging relative to prior 1.5T imaging. The lesion also demonstrated increased blood volume on dynamic susceptibility contrast perfusion. Given these imaging findings and interval stability, a definitive imaging diagnosis of capillary telangiectasia was made. Recognition of the MRI findings of capillary telangiectasia is imperative to avoid misdiagnosis and prevent unnecessary intervention.

Introduction

Brain capillary telangiectasias are slow flow vascular malformations, which are classically located in the pons [1]. These vascular lesions are typically benign and incidental [2]. However, capillary telangiectasia can be challenging to image as they are typically occult on computed tomography (CT) and angiographic imaging [1]. Similarly, they can be subtle on non-contrast magnetic resonance imaging (MRI) and demonstrate only faint enhancement on post contrast sequences [1,3]. These lesions are more apparent on susceptibility-weighted imaging (SWI) and gradient echo (GRE) sequences, likely secondary to perceivable increased deoxyhemoglobin through slow flowing capillaries [1,3]. Additionally, it is noted that high field MRI may exaggerate findings of susceptibility in slow flow cerebral vascular malformations, as seen with cavernous malformations [4].

Recognition of MRI findings of capillary telangiectasia is imperative to avoid misdiagnosis and prevent unnecessary intervention. An unusual appearance or location of a capillary telangiectasia can be misdiagnosed as malignancy, demyelination, or subacute infarction [3].

In this case report, we present the MRI appearance of an unusual capillary telangiectasia with ring-like enhancement

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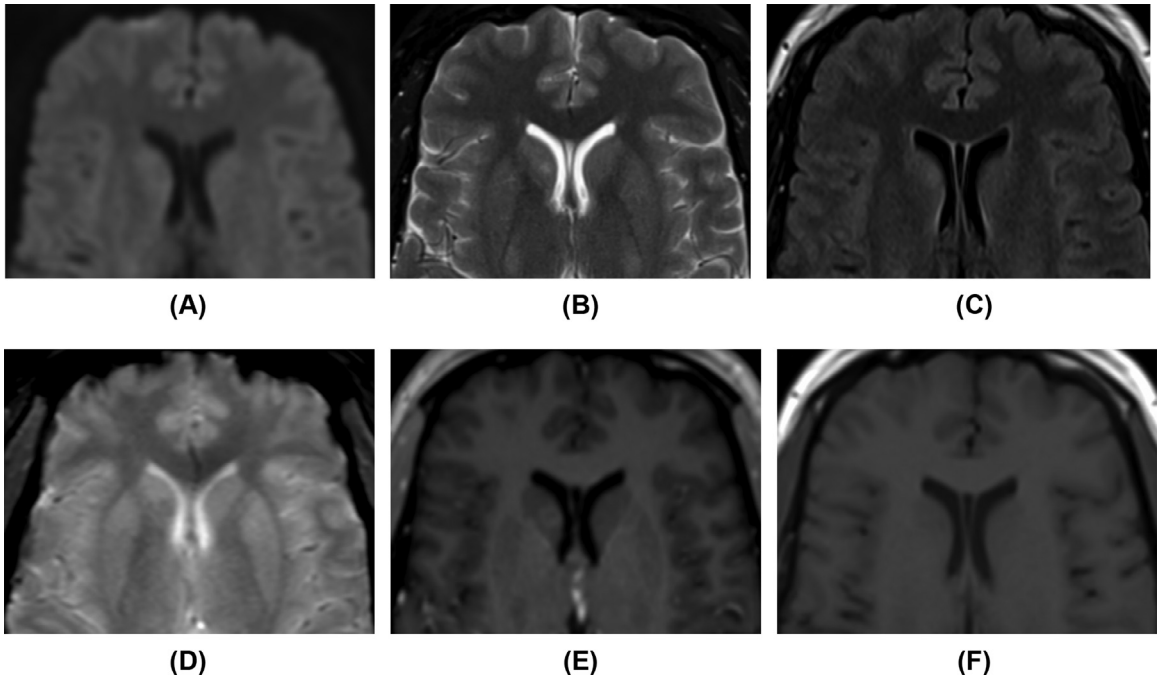

Fig. 1 – Initial 1.5T MRI obtained 2 months after presentation demonstrate an enhancing 6 mm right caudate head lesion with (A) no restricted diffusion. The lesion is minimally (B) T2 and (C) T2-FLAIR hyperintense without surrounding edema or mass effect. (D) On GRE sequence there was mild associated signal loss. (E) The lesion demonstrates rim-enhancement on post contrast T1-weighted imaging, compared to (F) isointense to minimally hypointense on pre-contrast imaging. One-month follow-up imaging on a 1.5T MRI was essential unhanged (not shown).

in the caudate. The lesion was conclusively diagnosed using high-field 3.0T MRI with perfusion imaging.

Case report

A 42-year-old female with a history of fibromyalgia and a left rotator cuff tear presented with intermittent left arm weakness, left shoulder pain, and left upper extremity paresthesia. She also reported to a lesser extent occasional left-sided weakness and paresthesia in a nonspecific distribution, as well as occasional blurry vision and headaches. Neurological exam examination was normal without focal defects. Follow-up electromyography and sensory nerve conduction studies of the left upper extremity were negative. Laboratory test results indicated low vitamin D, 25-hydroxy levels (16 ng/mL), but otherwise normal c-reactive protein (3.2 mg/L), sedimentation rate (22 mm/h), and vitamin B12 levels (586 pg/mL). The patient had a prior normal MRI of her cervical spine, but otherwise never had previous neuroimaging. A brain MRI was obtained to rule out the possibility of a demyelinating process.

Two months after her initial presentation and without interval treatment, a 1.5T MRI was performed, which revealed a 6 mm right caudate head lesion with rim enhancement (Fig. 1). The lesion demonstrated mild T2 and fluid-attenuated inversion recovery (FLAIR) hyperintense signal without restricted diffusion or surrounding edema. On GRE sequences (slice thickness 5 mm; SWI not typically obtained on adults at our institution), there was evidence of mild susceptibility artifact within the lesion. Images were scrutinized for the presence of additional lesions; however, no additional lesions or abnormal enhancement were identified. Differential diagnosis at the time included capillary telangiectasia, but given the ring-like enhancement, additional considerations included multiple sclerosis, metastatic disease, subacute infarction, infection, and other inflammatory processes such as sarcoidosis. Subsequent one-month follow-up imaging on a 1.5T MRI showed no interval change.

Additional follow-up 3.0T MRI (Fig. 2) with perfusion sequences was obtained seven months after initial presentation, and at five months after initial MRI to ensure stability of the lesion. Dynamic susceptibility contrast perfusion images were obtained for further diagnostic work-up for the patient's lesion. These scans remonstrated the unchanged right caudate head rim-enhancing lesion without mass effect, surrounding edema, or interval change. However, with regard to GRE imaging, there was increased blooming on 3.0T imaging relative to prior 1.5T imaging. Dynamic susceptibility perfusion imaging was also performed on this study, which demonstrated intralesional increased cerebral blood volume (CBV). A head CT was not obtained at our institution to rule out underlying calcifications within the lesion.

Given the lack of clinical/laboratory correlation, imaging stability, no significant mass effect or surrounding edema, increased susceptibility and blooming on 3T imaging, and perfusion findings, a definitive imaging diagnosis of capillary telangiectasia was made for the right caudate lesion.

Of note, as part of a thorough clinical work-up, the patient underwent concurrent orthopedic evaluation. The patient's

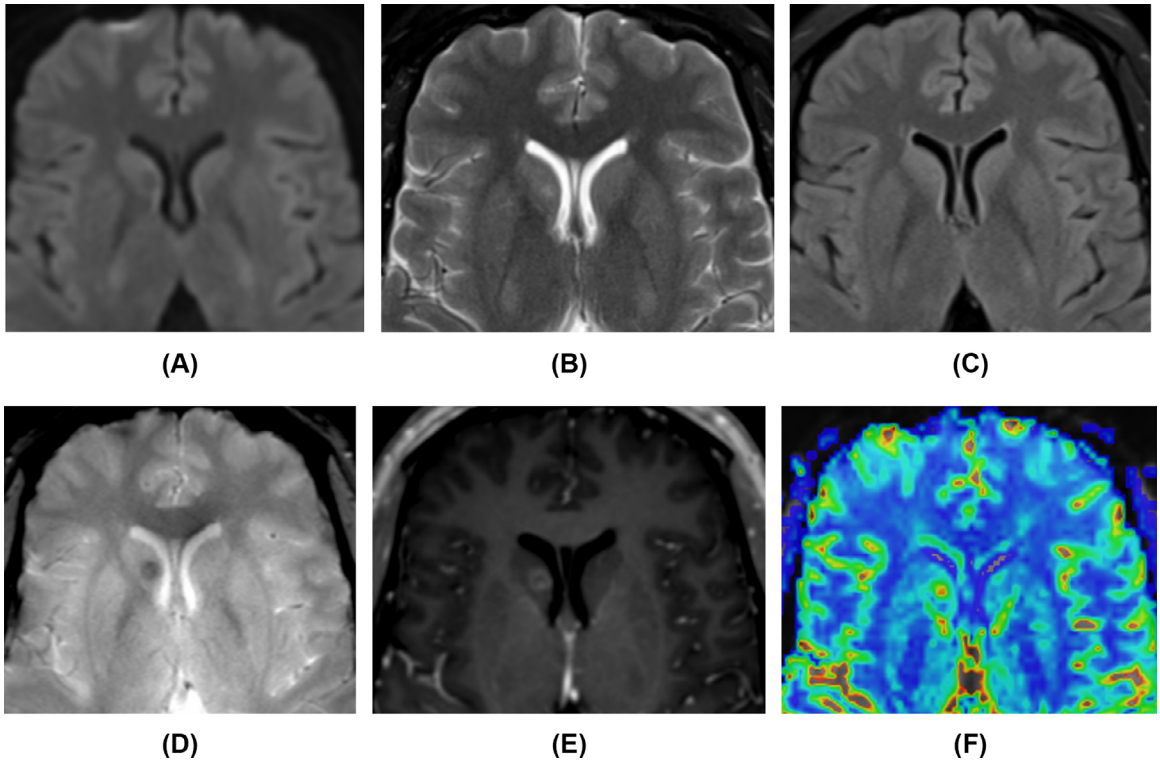

Fig. 2 – Follow-up 3.0T MRI obtained five months after initial MRI remonstrates the unchanged right caudate head lesion with (A) no restricted diffusion. The lesion remains mildly (B) T2 and (C) T2-FLAIR hyperintense without significant edema. (D) On GRE sequences there is increased blooming on 3.0T imaging relative to prior 1.5T imaging. (E) There is unchanged subtle rim-enhancement on postcontrast T1-weighted imaging. (F) The lesion demonstrated increased CBV on perfusion sequences.

intermittent left upper extremity weakness and left shoulder pain were attributed to left shoulder biceps tendonitis with superior labrum anterior and posterior tear, subacromial impingement, partial rotator cuff tear, and bursitis. The patient underwent a left should arthroscopy, sub-acromial decompression, and debridement for biceps tendon tendinosis five months after her initial presentation, with reported clinical improvement. Patient was also diagnosed with migraines 9 months after initial presentation and treated conservatively. These diagnoses may be the underlying etiology of the patient's intermittent left upper extremity weakness and occasional blurry vision.

Discussion

Brain capillary telangiectasias are small slow flow vascular malformations composed of dilated capillary-like vessels [1,5]. On histopathologic microscopic evaluation, the vessel walls are made of a single layer of endothelial cells, and surrounding brain tissue is normal without gliosis [5]. Calcification, hemorrhage, and hemosiderin laden macrophages are not associated with this type of malformation [5]. Additionally, when multiple telangiectasias are present, the possibility of hereditary hemorrhagic telangiectasia should be strongly considered [5]. No additional lesions were identified on more sensitive 3.0T GRE sequences,[4] or post-contrast imaging making hereditary hemorrhagic telangiectasia unlikely in this case. Lesions are typically small, asymptomatic, and incidental, although larger lesions may potentially be symptomatic depending on their location [2,5,6]. In our case, the patient's symptoms were likely unrelated to the caudate lesion given intermittent pattern and normal neurological exam, as well as the patient's improvement following orthopedic surgery.

Classically, capillary telangiectasia is found in the pons with faint stippled enhancement [1,5]. As with the current case, an abnormal appearance or location of a lesion may confound diagnosis. The differential for capillary telangiectasia includes malignancy, demyelination, and subacute infarction [3]. It is imperative to properly diagnose capillary telangiectasia as a patient may undergo unnecessary and potentially harmful invasive procedures, such as a biopsy, if the lesion is misdiagnosed on imaging [5]. The malformation in this case was in an atypical location and masqueraded as a solitary ring enhancing lesion. The differential for a ring enhancing lesion includes metastasis, abscess, glial neoplasm, subacute infarction, demyelinating disease, radiation necrosis, and lymphoma [7]. Given the interval stability of the lesion and the lack of surrounding edema and mass effect, a more aggressive process was able to be effectively excluded. Although no head CT was obtained for confirmation, a calcified mass was

considered unlikely given the overall signal characteristics including the increased blood volume on perfusion. Atypical presentation of unusual infectious lesions, such as solitary neurocysticercosis and intracranial tuberculous granulomas, was also deemed less likely given interval stability and no significant supporting infectious clinical history.

Knowing key imaging features of capillary telangiectasia on various MRI sequences at varying field strength may help radiologists avoid misdiagnosing a capillary telangiectasia. This lesion can be challenging to image as they are typically occult on CT, angiographic imaging, and some MRI sequences [1,3]. These lesions are typically iso- to mildly hypointense on T1WI, and iso- to mildly hyperintense on T2WI and FLAIR [1,8]. They also classically demonstrate blush-like stippled enhancement on post contrast imaging, and signal loss on SWI and GRE sequences [1,8].

Generally, capillary telangiectasia are more apparent on GRE and SWI sequences secondary to increased deoxyhemoglobin through slow flowing and stagnant capillaries [1,3]. Given the mild paramagnetic effects of deoxyhemoglobin, these lesions demonstrate signal loss on GRE but not on T2WI [8]. Studies have demonstrated capillary telangiectasia may be more apparent on SWI compared to GRE, as this sequence is more sensitive to susceptibility changes [8,9]. High-field MRI may also intensify findings of susceptibility in slow flow cerebral vascular malformations, such a cavernous malformations [4]. The lesion in this case demonstrated GRE signal loss that was exaggerated on high field 3.0T MRI compared to 1.5T.

Because capillary telangiectasia are small and slow flow lesions, they are typically occult on angiographic imaging [1,5]. Furthermore, bleeding and thrombus within a capillary telangiectasia can obscure the vascular malformation on angiography, although some lesions may be visualized on delayed imaging [5]. On perfusion MRI sequences, vascular malformations demonstrate hyper-perfusion, although not commonly used [10,11]. Additionally, a previous case report indicated increased “nidal” CBV in capillary angioectasia, which was associated with a vein of Galen malformation [12]. However, in our case of an isolated capillary telangiectasia, the lesion demonstrated expected increased CBV on MRI perfusion. Although tumors can also demonstrate increased perfusion, resultant mass effect and interval change from a lesion on follow-up imaging can help narrow the differential, as in our case.

Conclusion

Although typically benign and incidental, an unusual appearance or location of a capillary telangiectasia may require additional follow-up MRI evaluation [1,3,4]. In this case, we presented the high field MRI GRE and perfusion appearance of an atypical ring enhancing lesion without significant edema. On 3.0T MRI the capillary telangiectasia demonstrated increased signal loss relative to 1.5T imaging. Increased CBV was also identified on perfusion sequences, which is consistent with a slow flow vascular malformation. Given the imaging findings and stability overtime, a definitive imaging diagnosis of capillary telangiectasia was made. Knowing key imaging features of capillary telangiectasia on various MRI sequences at varying field strength may help radiologists avoid misdiagnosing a capillary telangiectasia and consequently avoid recommending unnecessary invasive procedures.

Declarations of interest

None.

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