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A Lobar Hematoma Mimicking a Brain Tumor in a Patient With Two Mechanical Heart Valves: The Value of Serial Magnetic Resonance Imaging

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Abstract

Subacute intracerebral hemorrhage can resemble a neoplasm on imaging, particularly when it is heterogeneous, surrounded by edema, and lacks a typical hyperacute appearance. We describe the case of a 76-year-old woman with mechanical aortic and mitral prostheses on warfarin who presented with right hemiparesis and an international normalized ratio (INR) of 4.5. Brain computed tomography showed a 2.3 cm heterogeneous left frontal parasagittal lesion with surrounding edema, without contrast enhancement. Magnetic resonance imaging (MRI) revealed a 24 × 23 × 15 mm lesion with peripheral T1 hyperintensity, a cystic/necrotic center, marked susceptibility artifact, and finger-like perilesional edema, along with scattered cerebral and cerebellar microbleeds. Primary or metastatic tumor, infection, septic embolism from prosthetic endocarditis, and cerebral amyloid angiopathy were considered. Thoraco-abdominopelvic computed tomography, infectious serologies, blood cultures, and transesophageal echocardiography did not support neoplastic or infectious causes. Serial MRI at six weeks showed near-complete resolution of edema with signal changes typical of late subacute blood products, and at seven months, a small residual hemosiderin-lined cavity without mass effect. The patient recovered independent gait. After a complete systemic workup and neurosurgical referral, serial MRI showing the expected evolution of blood products established the diagnosis before a biopsy was required, which was particularly relevant in a patient at high thrombotic and bleeding risk.

Categories: Neurology, Radiology, Internal Medicine
Keywords: anticoagulation, brain tumor mimic, intracerebral hemorrhage, magnetic resonance imaging, mechanical heart valve

Introduction

Intracerebral hemorrhage (ICH) is a feared complication of vitamin K antagonist therapy, and supratherapeutic anticoagulation is associated with larger hematomas and worse outcomes [1]. Rapid reversal is a cornerstone of management, and prothrombin complex concentrate normalizes the international normalized ratio (INR) faster than fresh frozen plasma [2]. In the hyperacute phase, ICH is easily recognized on computed tomography (CT), and a fluid-fluid level within the hematoma strongly suggests an underlying coagulopathy [3]. In the subacute phase, however, a hematoma may appear heterogeneous, with a cystic or necrotic-looking center, a peripheral rim, and vasogenic edema, features shared with hemorrhagic primary or metastatic neoplasms [4,5].

Lobar location in an older adult raises the possibility of cerebral amyloid angiopathy (CAA), which is currently diagnosed using magnetic resonance imaging (MRI)-based criteria that combine hemorrhagic and non-hemorrhagic markers [6]. Cerebral microbleeds, a marker of both CAA and hypertensive arteriopathy, are also associated with a higher risk of ICH in anticoagulated patients [7]. When an underlying tumor cannot be excluded, systemic workup and tissue diagnosis are usually pursued, and follow-up imaging documents the evolution of the lesion in the meantime. In patients with mechanical heart valves, vitamin K antagonists remain the only recommended long-term oral anticoagulants [8], and the interruption of anticoagulation required for a brain biopsy carries substantial thrombotic risk.

We report a lobar hematoma that mimicked a neoplasm on initial CT and MRI and was ultimately characterized by its evolution on serial MRI.

Case Presentation

A 76-year-old woman presented to the emergency department with right-sided weakness and urinary incontinence that began the previous evening. Her history included mechanical aortic (2003) and mitral (2013) valve replacements, with pericardial surgery two months before admission for a large organized pericardial effusion. She also had permanent atrial fibrillation, hypertension, dyslipidemia, hypothyroidism, asthma, chronic anemia with a hemolytic component attributed to a paravalvular leak, and an ischemic stroke in 2010 without sequelae. She was on warfarin with a previously therapeutic INR. At baseline, she was independent and cognitively intact.

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On admission, blood pressure was 159/57 mmHg, heart rate 39 beats per minute, and oxygen saturation 97% on room air. She was alert and oriented, without aphasia, dysarthria, or visual field defects. There was right hemiparesis (Medical Research Council grade 3/5) [9] with very mild right central facial palsy. Admission laboratory results, including a supratherapeutic INR of 4.5, are shown in Table *1*.

Parameter	Admission	After reversal	Reference range
Hemoglobin (g/dL)	14.1	-	11.5-16.5
Platelets (×10⁹/L)	205	-	150-500
Prothrombin time (seconds)	52.9	13.1	11.7-15.3
International normalized ratio	4.5	1.1	<1.0
Activated partial thromboplastin time (seconds)	64.0	34.6	24.8-34.4
C-reactive protein (mg/dL)	1.06	-	<0.5

TABLE 1: Laboratory results on admission and after reversal of anticoagulation.

Non-contrast head CT showed a rounded, heterogeneous left frontal parasagittal cortico-subcortical lesion of 2.3 cm, with a fluid-fluid level (dense dependent component) and surrounding edema effacing adjacent sulci (Figure *1*). A subsequent contrast-enhanced CT showed no abnormal enhancement. Given the INR of 4.5 and the possibly hemorrhagic content of the lesion, anticoagulation was reversed after discussion with Transfusion Medicine (which runs our anticoagulation clinic), with 2000 IU of prothrombin complex concentrate and 5 mg of phytomenadione, and the INR decreased to 1.1 on repeat testing the same day (Table *1*), consistent with adequate reversal [2]. After discussion with the regional Neurosurgery center, surgery was not indicated, and MRI was recommended to characterize the lesion and look for an underlying cause. After weighing the risk of hematoma expansion against the very high thrombotic risk of two mechanical valves after recent mitral surgery, Internal Medicine, Cardiology, and Transfusion Medicine jointly decided to maintain anticoagulation, and therapeutic enoxaparin (60 mg twice daily) was started on day 2.

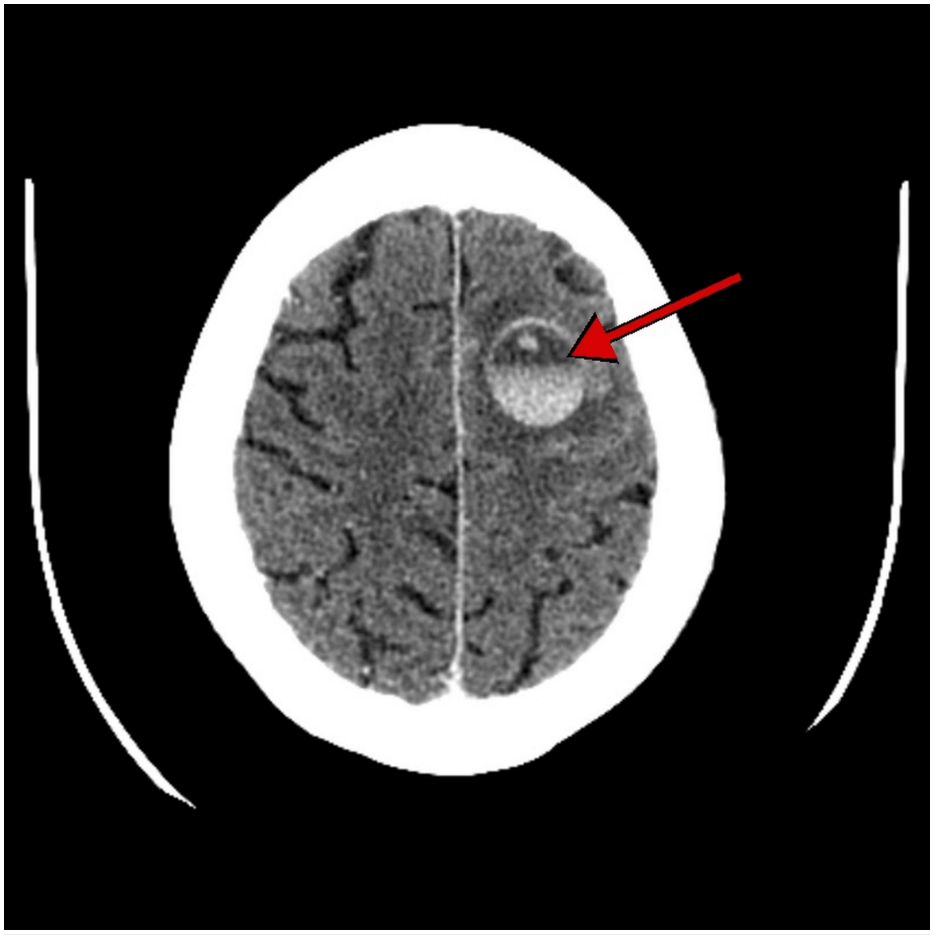


FIGURE 1: Non-contrast head computed tomography at presentation.

Axial image showing a rounded, 2.3 cm left frontal parasagittal lesion with a fluid-fluid level (arrow) and perilesional edema.

Brain MRI, performed five days later, showed a 24 × 23 × 15 mm heterogeneous lesion in the left centrum semiovale and high convexity, with peripheral T1 hyperintensity, a cystic/necrotic center, marked hypointensity on susceptibility-weighted imaging (SWI), and finger-like perilesional edema, without midline shift (Figure *2*). SWI also showed multiple microbleeds in both cerebral and cerebellar hemispheres, together with periventricular and subcortical leukoencephalopathy and old lacunes. These findings favored CAA-related lobar hemorrhage, although a primary or secondary tumor could not be excluded. After the MRI, Neurology was consulted and, given the marked perilesional edema, recommended dexamethasone (10 mg bolus, then 4 mg three times daily) and prophylactic levetiracetam (500 mg twice daily).

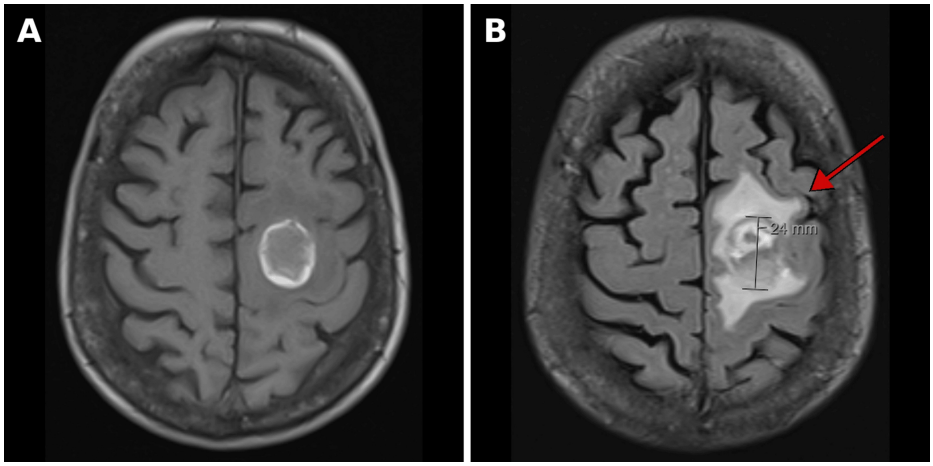


FIGURE 2: Brain magnetic resonance imaging five days after the initial computed tomography.

(A) Axial T1-weighted image showing peripheral spontaneous hyperintensity of the lesion. (B) Axial T2-weighted image showing a heterogeneous lesion with a cystic/necrotic-appearing center and extensive finger-like perilesional edema (arrow).

The differential diagnosis included hemorrhagic primary or metastatic neoplasm, infection, septic embolism from prosthetic valve endocarditis, and CAA. Thoraco-abdominopelvic CT showed no lesion suspicious for malignancy. Mammography and breast ultrasound were unremarkable. Serologies were consistent with past immunity to cytomegalovirus, toxoplasma, rubella, Epstein-Barr virus, herpes simplex virus 1, varicella-zoster virus, and parvovirus B19; Venereal Disease Research Laboratory (VDRL), human immunodeficiency virus (HIV), and viral hepatitis tests were negative, and cryptococcal antigen was negative. Blood cultures were sterile. Transesophageal echocardiography showed normally functioning mechanical prostheses without vegetations, but a severely dilated left atrium with dense spontaneous echo contrast and a non-organized left atrial appendage thrombus, a further indication for anticoagulation.

Neurology noted that the blood products could still mask an underlying lesion and suggested neurosurgical evaluation and repeat MRI within three months. The case was referred to Neurosurgery, but no surgical intervention was performed. Cardiology advised that anticoagulation could not be withheld because of the risk of prosthetic valve thrombosis, so warfarin was resumed on day 14 (target INR 2.5-3.5). She was discharged home on day 17 with severe right hemiparesis and home care support, on warfarin with enoxaparin bridging until the INR reached 2.5.

Follow-up MRI six weeks after presentation showed subtotal regression of vasogenic edema, a hyperintense center on T1-weighted and fluid-attenuated inversion recovery (FLAIR) images, and a regular hypointense rim on gradient-echo/SWI, consistent with extracellular methemoglobin and peripheral hemosiderin deposition. These findings favored a lobar hematoma over an expansile lesion (Figure *3A*, *3B*). Because some heterogeneity persisted at the upper pole of the lesion, further MRI follow-up was recommended, and corticosteroids were gradually tapered. Seven months after presentation, MRI showed marked regression to a well-defined 13 × 7 × 22 mm residual area with T1 hyperintensity and gradient-echo hypointensity, complete resolution of edema, and retraction of the adjacent ventricle, consistent with the residue of a benign hemorrhagic process (Figure *3C*, *3D*). At that follow-up visit, she was walking independently, with mild right central facial palsy, grade 5/5 strength in the right arm and 4/5 in the right leg, and remained on warfarin. Corticosteroids were stopped, and she was discharged from the outpatient clinic.

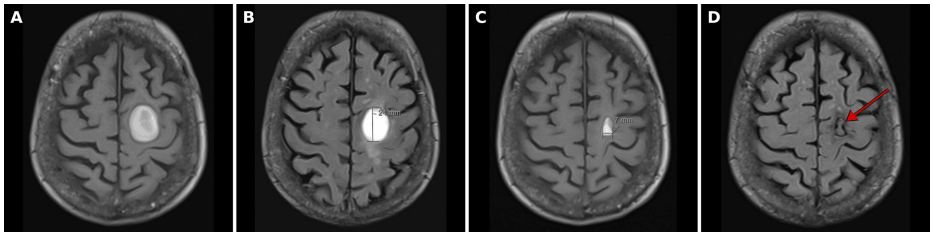


FIGURE 3: Follow-up magnetic resonance imaging.

Six weeks after presentation, axial T1-weighted (A) and T2-weighted (B) images show diffuse hyperintensity of the hematoma, consistent with extracellular methemoglobin, with unchanged dimensions and near-complete regression of edema. Seven months after presentation, axial T1-weighted (C) and T2-weighted (D) images show a small residual lesion with a hypointense hemosiderin rim (arrow), without edema or mass effect.

Discussion

This case shows that a lobar hematoma can reproduce most imaging features of a hemorrhagic tumor and that serial MRI, by showing how the lesion evolves over time, can be the decisive diagnostic test. The diagnostic question was not whether the lesion was hemorrhagic, but whether the hemorrhage was primary or secondary to an underlying lesion, as hemorrhagic neoplasms can closely resemble primary hematomas [4].

Several features suggested a neoplasm: a rounded heterogeneous lesion, a cystic/necrotic-looking center, a peripheral rim, and disproportionate finger-like edema. Hemorrhagic tumors, however, typically show heterogeneous and delayed evolution of blood products, an incomplete or absent hemosiderin rim, and persistent edema [4]. Subacute hematomas mimicking brain tumors have been described in similar terms [10]. Benign hematomas follow a predictable sequence: deoxyhemoglobin, then intracellular and extracellular methemoglobin from the periphery inward, and finally a complete hemosiderin rim with progressive shrinkage [5]. In our patient, the six-week MRI showed exactly this sequence, with a regular hemosiderin rim and regression of edema, and the seven-month study confirmed a small residual cavity with retraction of the adjacent ventricle, which is incompatible with an expansile lesion. Because a hemosiderin rim alone does not exclude an underlying tumor [4], the shrinkage of the lesion and the resolution of edema over time were essential to the diagnosis.

The absence of enhancement on CT was reassuring but not conclusive, and the peripheral T1 hyperintensity precluded reliable assessment of enhancement on MRI. A further caveat is that the patient received dexamethasone, which can induce radiological regression of primary central nervous system lymphoma and other brain tumors [11]. Corticosteroids are not indicated for ICH itself [12]; in this case, dexamethasone was started because a neoplasm with vasogenic edema had not yet been excluded. For this reason, the regression of edema and the reduction in lesion size were not taken as evidence of a benign process on their own. Instead, it was the MRI markers, namely the expected signal evolution of blood products, the formation of a regular hemosiderin rim, and the retraction of the adjacent ventricle, that supported a benign hemorrhagic etiology.

The cause of the hemorrhage was probably multifactorial. Supratherapeutic anticoagulation (INR 4.5) increases the risk and volume of ICH [1], and the fluid-fluid level on the admission CT was consistent with a coagulopathy-related hemorrhage [3]. Lobar location, age, and scattered microbleeds raised the possibility of CAA [6]. However, the patient also had hypertension, cerebellar microbleeds, and old lacunes in the pons and striatum, a pattern that suggests coexisting hypertensive arteriopathy (mixed small vessel disease); a definite diagnosis of CAA requires neuropathology and was not pursued.

Biopsy was considered, and the patient was referred to Neurosurgery, but several factors favored waiting for follow-up MRI. First, the patient had two mechanical valves, atrial fibrillation, and a left atrial appendage thrombus, so the interruption of anticoagulation needed for surgery carried a high thromboembolic risk. Second, in the acute and subacute phases, blood products can obscure an underlying lesion and lower the diagnostic yield of a biopsy, so repeat imaging after hematoma resorption is a reasonable first step; in tumor-related hemorrhage, the underlying lesion may only become evident on follow-up MRI [13]. Third, the systemic workup for malignancy and infection was negative. Guidelines support resuming anticoagulation after ICH in patients with strong indications, weighing timing against the risk of recurrence [14]. In a multicenter cohort of 137 patients with mechanical heart valves and anticoagulation-associated ICH, restarting therapeutic anticoagulation before day 13 was associated with more hemorrhagic complications, and day 6 was proposed as the earliest reasonable time in patients at high thromboembolic risk [15]. Our patient received therapeutic enoxaparin from day 2, earlier than this threshold, because of the very high thrombotic risk of two mechanical valves after recent mitral surgery; a left atrial appendage thrombus was later found on transesophageal echocardiography. The day 6 MRI, obtained four days after enoxaparin was started, showed no enlargement compared with the admission CT, and the lesion dimensions

were unchanged at six weeks. While the neurosurgical evaluation was underway, the follow-up MRI showed the typical evolution of a hematoma, so biopsy was no longer necessary, and the diagnosis rests on this imaging evolution and on clinical follow-up.

Maintaining anticoagulation in a patient with possible CAA requires particular justification. Lobar location and microbleeds are associated with a higher risk of ICH, which weighs against long-term anticoagulation [7,14], and in patients with atrial fibrillation, left atrial appendage closure may be considered as an alternative [14,16]. In our patient, however, this would not have addressed the thrombotic risk of two mechanical valves. In patients with mechanical valves, early resumption of anticoagulation after ICH is considered reasonable [14]. The left atrial appendage thrombus was itself an indication for anticoagulation, managed with low-molecular-weight heparin followed by warfarin. Furthermore, CAA was possible rather than definite, and the old lacunes and cerebellar microbleeds suggested a contribution from hypertensive arteriopathy. In this context, the multidisciplinary team considered that the thromboembolic risk outweighed the risk of recurrent hemorrhage.

Conclusions

A subacute lobar hematoma can mimic a hemorrhagic neoplasm on CT and MRI, particularly in an anticoagulated patient. When systemic workup is negative, serial MRI that documents the expected evolution of blood products, a complete hemosiderin rim, resolution of edema, and lesion shrinkage can establish the diagnosis, sometimes before tissue sampling is needed. This is particularly relevant in patients with mechanical heart valves, in whom interrupting anticoagulation for a biopsy carries a high thrombotic risk.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Clara Pinto, Ana Sofia Pimenta, Ana S. Martins, Carolina M. Marçal, Alexandre Castro Lopes

Acquisition, analysis, or interpretation of data: Clara Pinto, Ana Sofia Pimenta, Ana S. Martins, Carolina M. Marçal, Alexandre Castro Lopes

Drafting of the manuscript: Clara Pinto, Ana Sofia Pimenta, Ana S. Martins, Carolina M. Marçal, Alexandre Castro Lopes

Critical review of the manuscript for important intellectual content: Clara Pinto, Ana Sofia Pimenta, Ana S. Martins, Carolina M. Marçal, Alexandre Castro Lopes

Supervision: Clara Pinto, Ana S. Martins

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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