restricted diffusion. There is typically no gadolinium enhancement. After glycemic correction, similarly to the clinical findings, such regions tend to return to normal signal intensity.

It is important to highlight the role of susceptibility-weighted imaging (SWI) in differentiating between changes seen in HCHB and areas of calcification or hemorrhage, which represent the most common differential diagnoses. Calcium and blood deposits both generally manifest as hyperintensities on T1-weighted images with corresponding hypointensities on T2*-weighted images and SWI; conversely, HCHB changes tend to present as unilateral hyperintensities on T1-weighted images with no matching changes on T2*-weighted images or SWI[7,8].

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Anterior cerebral artery aneurysm rupture presenting as hemorrhage in the splenium of the corpus callosum

Dear Editor,

A 43-year-old, right-handed male presented with a three-day history of severe, holocranial headache. Three weeks prior, he had experienced another series of severe, pulsatile headaches accompanied by fever, malaise, and paresthesia of the second and third digits of the left hand. The neurologic examination revealed apraxia of the left hand and constructional apraxia of the right hand, without sensorimotor or cerebellar deficits, consistent with callosal disconnection syndrome.

Non-contrast computed tomography and magnetic resonance imaging demonstrated a hematoma in the splenium of the corpus callosum (Figure 1). No subarachnoid blood was visualized. Cerebral angiography revealed evidence of recent aneurysm rupture at the junction of the A1 and A2 segments of the right anterior cerebral artery (ACA) and vasospasm of the distal right ACA (Figure 2A). The decision was made to embolize the aneurysm with detachable coils (Figure 2B). At the conclusion of the procedure, there was complete embolization of the aneurysm sac, without disruption of the integrity of the intracranial arteries or defect in the brain parenchyma. The remainder of the hospital stay was uneventful, and the patient was discharged on post-admission day 11 with a prescription for a 6-day tapered course of nimodipine. Angiography performed at 6 months of follow-up demonstrated that the coils remained in place within the aneurysm sac (i.e., the aneurysm sac continued to be occluded).

Reports of remote intraparenchymal hemorrhage as a presenting finding of aneurysm rupture are rare[1]. For example, in a group of 460 patients with subarachnoid hemorrhage, Abbed et al.[2] reported 116 cases of intraparenchymal hematoma formation, none of which appeared to be proximal to the site of aneurysm rupture. In fact, our search of the literature revealed only isolated cases of remote focal hemorrhage. In 2002, Friedman et al.[3] described a ruptured anterior communicating artery aneurysm associated with a perisylvian frontotemporal hematoma. Also in 2002, Lee et al.[4] described the case of a patient with ruptured saccular ACA aneurysm that evolved to hemorrhage of the left putamen. In 2005, Paus et al.[5] reported an even more perplexing case of anterior communicating artery aneurysm rupture, with adjacent subarachnoid hemorrhage and focal hematoma in the

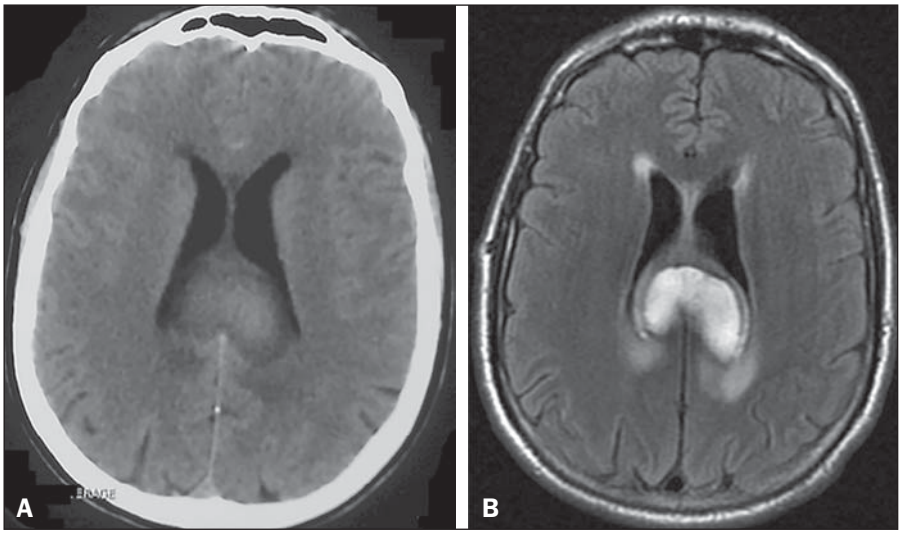

Figure 1. Non-contrast computed tomography (**A**) and T2-weighted fluid attenuated inversion recovery magnetic resonance imaging (**B**) demonstrating a large, heterogeneously enhancing mass in the splenium of the corpus callosum, consistent with a focal collection of intraparenchymal blood. No evidence of subarachnoid hemorrhage is apparent.

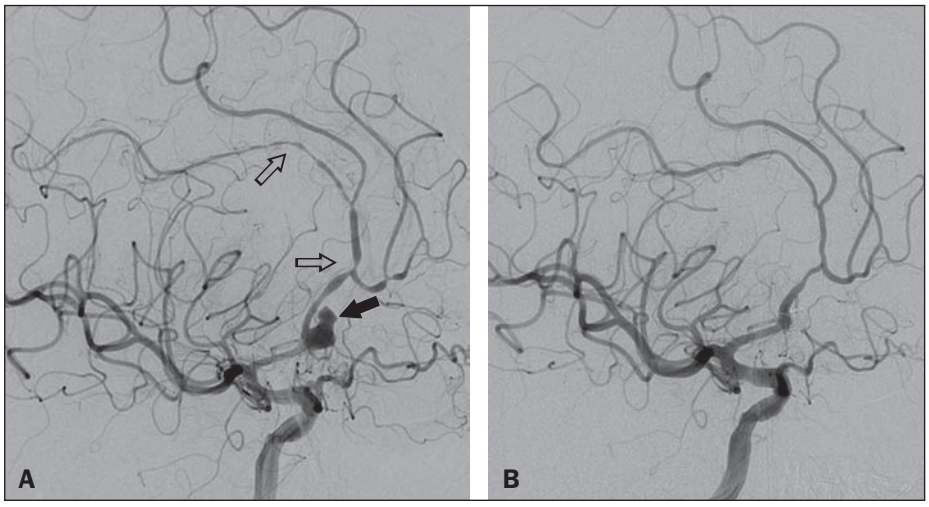

Figure 2. A: Digital subtraction angiography of the cerebral vessels, demonstrating an aneurysm (black arrow) at the junction of the A1 and A2 segments of the right ACA. The aneurysm is irregular in appearance, with a rupture sac and Murphy's test suggestive of recent rupture. The right A2 segment is characterized by an irregular caliber and a beaded appearance (open arrows), consistent with arterial vasospasm. **B:** Complete embolization of the aneurysm sac after coiling.

left posterior temporal lobe that was distant from the aneurysm and from any subarachnoid cisterns.

The case presented here is important because it establishes a mechanism for remote bleeding. In previous reports, a variety of explanations for distant hemorrhage have been proposed, including hypertensive crisis, the formation of jets through subarachnoid cisterns, venous infarction, intraluminal thrombosis, hemorrhagic infarction secondary to vasospasm, and occult vascular anomaly. However, none of those reports provided direct evidence to support any of the proposed mechanisms. In contrast, in our case, we observed definite angiographic evidence of vasospasm in the vessels between the aneurysm and the site of hemorrhage. That constitutes a strong indication that vasospasm-associated hemorrhagic infarction is a mechanism of remote hematoma formation following cerebral aneurysm rupture.

In conclusion, we have described a case of ACA aneurysm rupture presenting as remote intraparenchymal hemorrhage in the splenium of the corpus callosum and have demonstrated that vasospasm-induced hemorrhagic infarction is a plausible mechanism for distant bleeding. Neuroradiologists and neurosurgeons should be aware of this rare phenomenon in order to reduce the likelihood of inappropriate treatment.

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Fat necrosis associated with the use of oral anticoagulant therapy: atypical mammographic findings

Dear Editor

We report the case of a 54-year-old female with systemic lupus erythematosus, lupus nephritis, antiphospholipid syndrome, and deep vein thrombosis, who was being treated with an oral anticoagulant and a corticosteroid, as well as receiving immunosuppressive therapy. Her international normalized ratio was between 2 and 3, and she presented with recurrent spontaneous hematomas. She had been diagnosed 20 months prior with miliary pulmonary tuberculosis, which had been treated for 12 months. After the patient had undergone mammography (Figure 1), we reviewed the clinical data: she reported a recent spontaneous left-sided hematoma, with palpable nodules and ecchymosis, in the superolateral quadrant. As can be seen in Figure 2, ultrasound with Doppler flow imaging showed correspondence between this findings and an irregular hypoechoic nodule with indistinct margins without vascularization, measuring 6.0 × 3.0 cm, associated with architectural distortion, in the superolateral quadrant—together with images suggestive of lipid cysts. Initially undetermined, the lesion was considered likely benign, suggestive of fat necrosis, probably associated with anticoagulant use and hematoma formation. To avoid biopsy, we opted for a strategy of observation only.

Fat necrosis is often silent, appearing only as an abnormal mammographic finding. In rare cases, it can manifest as a palpable mass without associated mammographic findings. It is typically secondary to incidental or iatrogenic trauma and can occur in patients who are using anticoagulants or even in those without a relevant history.

Mammography is the most important test in the assessment of fat necrosis. Depending on the stage and the amount of fibrosis, it can manifest as a lipid cyst or as features that simulate malignancy: spiculated hyperdense areas; nodules accompanied by