

Visual Diagnosis in Emergency Medicine



GYRAL CALCIFICATION IN AN ADULT MASQUERADING AS SUBARACHNOID HEMORRHAGE

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CASE REPORT

An 85-year-old female with a medical history significant for coronary artery disease, status post pacemaker placement, and a prior stroke presented to an outside emergency department (ED) with complaints of lethargy, diaphoresis, dysarthria, and an episode of transient loss of consciousness at her nursing home. There was no history of trauma and she was not on any anti-coagulants. A computed tomography (CT) scan was performed and read by the ED physician as subarachnoid hemorrhage (SAH) in the left sylvian fissure, and the patient was emergently transferred to our hospital for further management with a tentative diagnosis of SAH. Upon arrival to our intensive care unit, she was following commands, oriented to self, dysarthric but with fluent speech. Cranial nerve examination was within normal limits except for the presence of right-sided upper motor neuron–type facial nerve palsy. Careful evaluation of the CT scan showed the presence of a serpentine hyperdensity along the left perisylvian cortex consistent with dystrophic calcification from prior ischemic insult rather than an SAH, along with chronic microvascular ischemic changes, old lacunar infarcts, and age-appropriate cerebral parenchymal volume loss (Figure 1). CT angiography performed as part of a

stroke work-up showed left perisylvian gyral enhancement compatible with a subacute infarct with no obvious structural vascular lesion (Figure 2). She was subsequently evaluated and treated by the stroke neurologist secondary to the diagnosis of subacute infarct rather than SAH.

DISCUSSION

Intracranial calcification refers to calcification within the cranial cavity and generally represents calcification within the parenchyma of the brain or its vasculature. Calcification within the brain can be classified as physiologic or pathologic and metastatic or dystrophic. Physiologic calcification is defined as normal calcification that occurs in tissues over time with aging, and pathologic calcification is abnormal, resulting from some kind of disease process, including infectious, metabolic, vascular, neoplastic, and congenital causes (1). Although metastatic calcification results from systemic disorders of calcium metabolism, calcification occurring secondary to pathologies within the brain itself is referred to as dystrophic (2). In addition to distinguishing physiologic from pathologic calcification, calcification needs to be differentiated from other pathologies that manifest as hyperdensities on CT, including

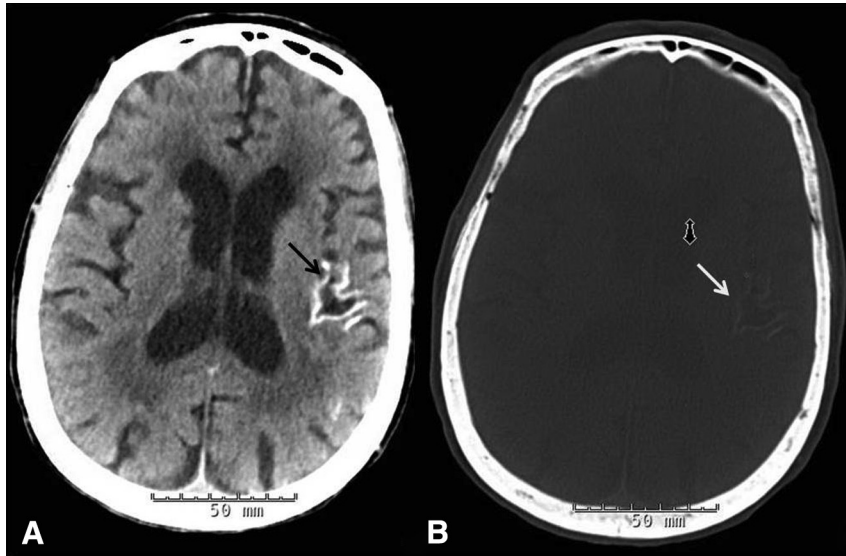


Figure 1. (A) Axial noncontrast computed tomography (CT) of the head demonstrating presence of serpentine hyperdensity (arrow) along left perisylvian cortex consistent with dystrophic calcification. (B) Bone window of the CT confirming the hyperdensity (arrow) to be calcification rather than hemorrhage.

hemorrhage (1). This is especially important in the ED, as a rapid and correct diagnosis is essential to ensure that the patient receives appropriate therapy and medical care. Incorrect diagnosis of intracranial calcification as a



Figure 2. Axial contrast computed tomography (CT) scan done during CT angiography showing left perisylvian gyral enhancement (arrow) compatible with a subacute infarct.

hemorrhage, an absolute contraindication for thrombolysis, can result in withholding of intravenous thrombolytic therapy in a patient with an acute ischemic stroke.

The most common site for physiologic calcification in the brain is the pineal gland, with choroid plexus, habenula, basal ganglia, falx, tentorium, petroclinoid ligaments, and sagittal sinus not infrequent sites for intracranial calcification (1). Although recognition of calcifications at these sites might not be difficult, dystrophic calcification at unusual locations can pose a diagnostic challenge. A pathologic calcification is smaller in size, asymmetric, and involves a location other than the common sites of physiologic calcification (1,3). Calcification in a gyriform distribution is typically seen in patients with Sturge-Weber syndrome, and can also occur as a result of meningitis (3). Although the diagnosis of gyriform calcification is straightforward when it involves characteristic locations such as parieto-occipital region, as seen in Sturge-Weber syndrome, or in patients with a known history of clinical condition predisposing to calcification, it can be confused for hemorrhage when it follows a typical subarachnoid location, such as sylvian fissure, as described in the present case (1,2,4). Careful evaluation of the CT scan in such cases will reveal the hyperdensity along the gyrus rather than true subarachnoid space.

Use of different CT windows can be helpful in differentiating between hemorrhage and calcification. Various tissues have different density on CT, as measured in Hounsfield units (HU), and can vary from -1000 (air) to $+1000$ HU (bone). Acute blood is bright on CT and measures $+55$ to $+75$ HU as compared with calcification, which is denser than blood

and generally measures in the low 100s. Although a missed diagnosis of SAH can be catastrophic, physicians need to be aware of the potential masqueraders of SAH. If underlying hemorrhage is excluded in an efficient and timely manner, life-saving thrombolytic therapy can be administered to eligible patients with an ischemic stroke. Emergency physicians and radiologists should be aware of this unusual presentation of intracranial gyriform calcification to direct appropriate clinical care.

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