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Imaging suggestive, but symptoms atypical

A 66-year-old man with chronic obstructive pulmonary disease (COPD) was brought to the emergency department with a 2-week history of progressive dyspnea followed by altered mental status. He had no history of diabetes mellitus, hypertension, or drug abuse.

On physical examination, he was stuporous. He had no fever or hypotension, but his pulse and breathing were rapid, and he had central cyanosis, bilateral conjunctival congestion, a puffy face, generalized wheezing, basilar crackles in both lungs, and leg edema.

Laboratory testing showed hypoxia and severe hypercarbia. His hematocrit was 65% (reference range 39–51) and his hemoglobin level was 21.5 g/dL (13–17).

The patient was diagnosed with an exacerbation of COPD. He was intubated, placed on mechanical ventilation, and admitted to the intensive care unit.

Computed tomography (CT) performed because of his decreased level of consciousness (**Figure 1**) showed increased attenuation in the ambient cistern and the lateral aspect of the lateral cerebral fissure, suggesting subarachnoid hemorrhage. The attenuation value in these areas was 89 Hounsfield units (typical values for brain tissue are in the 20s to 30s, and for blood in the 30s to 40s). To further evaluate for subarachnoid hemorrhage, lumbar puncture was performed, but analysis of the fluid sample showed normal protein and glucose levels and no cells.

Based on the results of cerebrospinal fluid evaluation and on the CT attenuation value, a diagnosis of pseudosubarachnoid hemorrhage due to polycythemia was made.

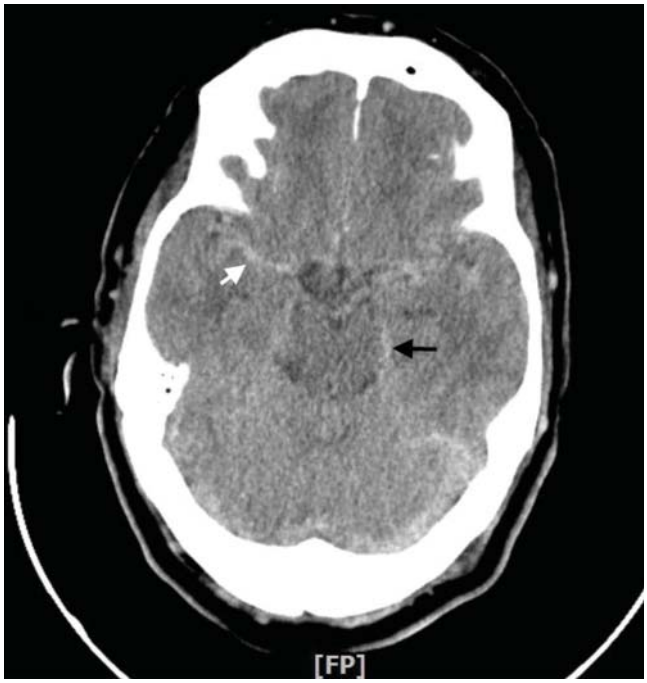

FIGURE 1. Computed tomography showed increased attenuation in the ambient cistern (black arrow) and cortical sulci (white arrow).

SUBARACHNOID VS PSEUDOSUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage typically begins with a "thunder-clap" headache (beginning suddenly and described by patients as "the worst headache ever.") While not all patients have this presentation, if imaging suggests subarachnoid hemorrhage but the patient has atypical signs and symptoms (eg, other than headache), then pseudosubarachnoid hemorrhage should be considered.

Brain CT is one of the most reliable tools for diagnosing subarachnoid hemorrhage in the emergency department. Done within 6 hours of symptom onset, it has a sensitivity of 98.7% and a specificity of 99.9%.[1] Magnetic resonance imaging can also visualize subarachnoid hemorrhage within the first 12 hours, typically as a hyperintensity in the subarachnoid space on fluid-attenuated inversion-recovery sequences[2] and on susceptibility-weighted sequences.

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Lumbar puncture is also an important diagnostic tool but carries a risk of brain herniation in patients with brain edema.

Pseudosubarachnoid hemorrhage is an artifact of CT imaging. It is rare, and its prevalence is unknown.[3] However, it may be seen in up to 20% of patients after resuscitation, as a result of diffuse cerebral edema that lowers the attenuation of brain tissue on CT, making the vessels relatively conspicuous. It can also be seen in purulent meningitis (due to proteinaceous influx after blood-brain barrier disruption),[4] in meningeal leukemia (due to increased cellularity in the leptomeninges), and in severe polycythemia (from a higher concentration of blood and hemoglobin in the vessels).[3,5–7]

Although the level of attenuation on CT may help distinguish subarachnoid from pseudosubarachnoid hemorrhage, its accuracy has not been defined. Inspecting the CT images may clarify whether areas with high attenuation look like blood vessels vs subarachnoid hemorrhage.

Our patient recovered and had an uneventful follow-up. The cause of his elevated hematocrit was likely chronic hypoxia from COPD.

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