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The Hemorrhage that Wasn't: Polycythemia Presenting as a Pseudointracranial Hemorrhage in Pedestrian vs Automobile Trauma Alert

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ABSTRACT

Introduction: Although polycythemia vera is rarely seen, absolute polycythemia is seen more frequently and can drastically change imagine interpretation when taken out of context of the patient's chief complaint.

Case Report: We report the case of a 21-year-old male without any known medical history who presented as a trauma patient initially diagnosed with acute subarachnoid and subdural hemorrhages. Further examination of the imaging and later serum laboratory findings demonstrated a primary diagnosis of absolute polycythemia, which was an incidental diagnosis that resulted in the patient's care team and disposition being changed.

Conclusion: Absolute polycythemia is not uncommon, and its causes vary from congenital and myeloproliferative disorders to chronic obstructive pulmonary disease and sleep apnea. It can cause pseudoenhancement in noncontrast computed tomography (CT) scans because of the increased protein level in the blood, and emergency physicians should consider all differential diagnoses of pseudoenhancement.

Keywords: Polycythemia, trauma, erythrocytosis, false positive interpretation

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Introduction

Polycythemia is a condition in which there is an increased hemoglobin level, which is a result of either decreased plasma volume or increased red cell mass (RCM). Increased RCM is referred to as absolute polycythemia (also referred to as erythrocytosis in some studies) and is seen in approximately 50 per 100,000 people in the US population, making it a condition that an emergency physician is likely to see (1). The most common primary cause of absolute polycythemia is myeloproliferative conditions such as polycythemia vera and erythropoietin receptor mutations. All secondary causes stem from an increase in erythropoietin levels and include hypoxemic lung disease (e.g., COPD and obstructive sleep apnea), erythropoietin-producing malignancies (e.g., renal cell carcinoma and hepatocellular carcinoma), hereditary high oxygen affinity hemoglobin, and environmental factors including living at a high altitude or chronic exposure to carbon monoxide (2).

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Case Report

A 21-year-old male with no past medical history presented as a trauma alert at an academic level 1 trauma center for pedestrian vs automobile at 30 mph with loss of consciousness (LOC) for 30 seconds. His initial Glasgow Coma Scale (GCS) score was 14 (confusion); his pupils were equal, round, and reactive; he had no visible external head or neck trauma.

A stat noncontrast computed tomography (CT) scan of the head was preliminarily read as being a diffuse subarachnoid hemorrhage

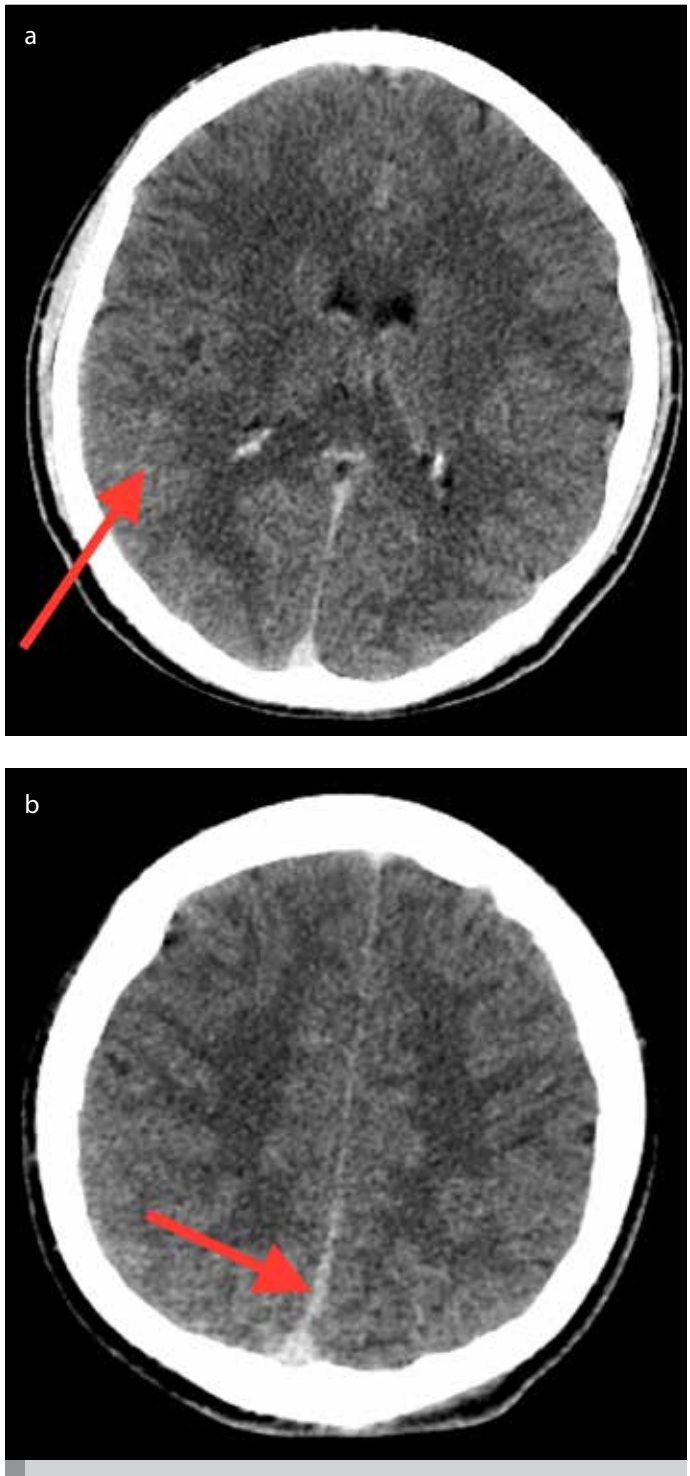

FIGURE 1. a, b. (a) Axial noncontrast CT of the head image shows false positive subarachnoid hemorrhage. (b) Axial noncontrast CT of the head image shows false positive falcine subdural hemorrhage on noncontrast CT of the head.

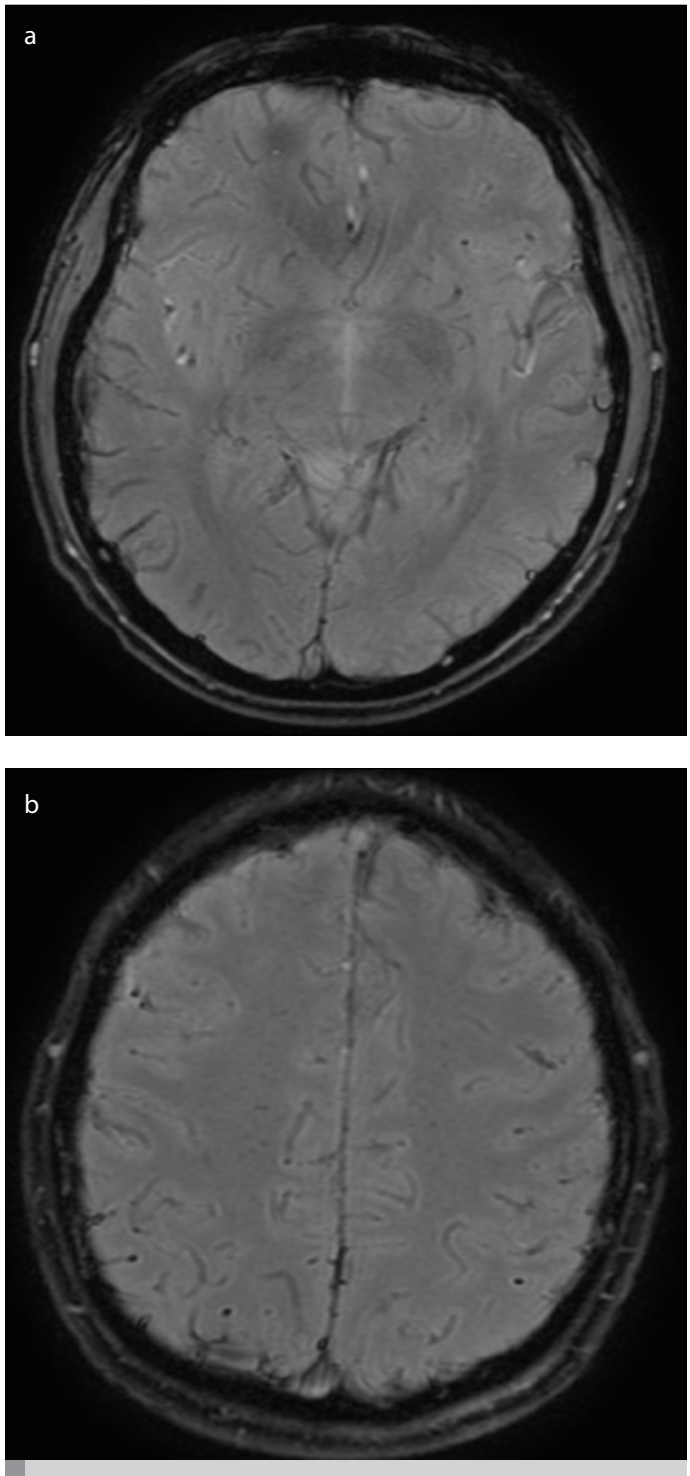

FIGURE 2. a, b. Axial gradient echo MRI images matching the anatomical locations of Figures 1a and 1b, respectively, show normal subarachnoid space and vasculature.

and falcine subdural hemorrhage (Figure 1a and 1b). Neurosurgery was consulted and agreed with the diagnosis of intracranial hemorrhage; the patient was assigned to an intensive care unit bed for hourly neurological monitoring. Soon thereafter, the scan was read by a more senior radiologist as being inconclusive because of the presence of contrast in the vessels, making it insensitive for an intracranial hemorrhage. However, the patient had not received any contrast prior to the CT scan of the head. A review of the patient's laboratory findings demonstrated a hemoglobin level of 23.5 g/dL and a hematocrit of 72.1%. In this context, the noncontrast CT findings of the head were favored to represent an artifact due to the patient's new diagnosis of erythrocytosis, rather than an acute intracranial hemorrhage. A STAT magnetic resonance imaging (MRI) of the brain confirmed that the high density locations on the noncontrast head CT were normal appearing vessels.

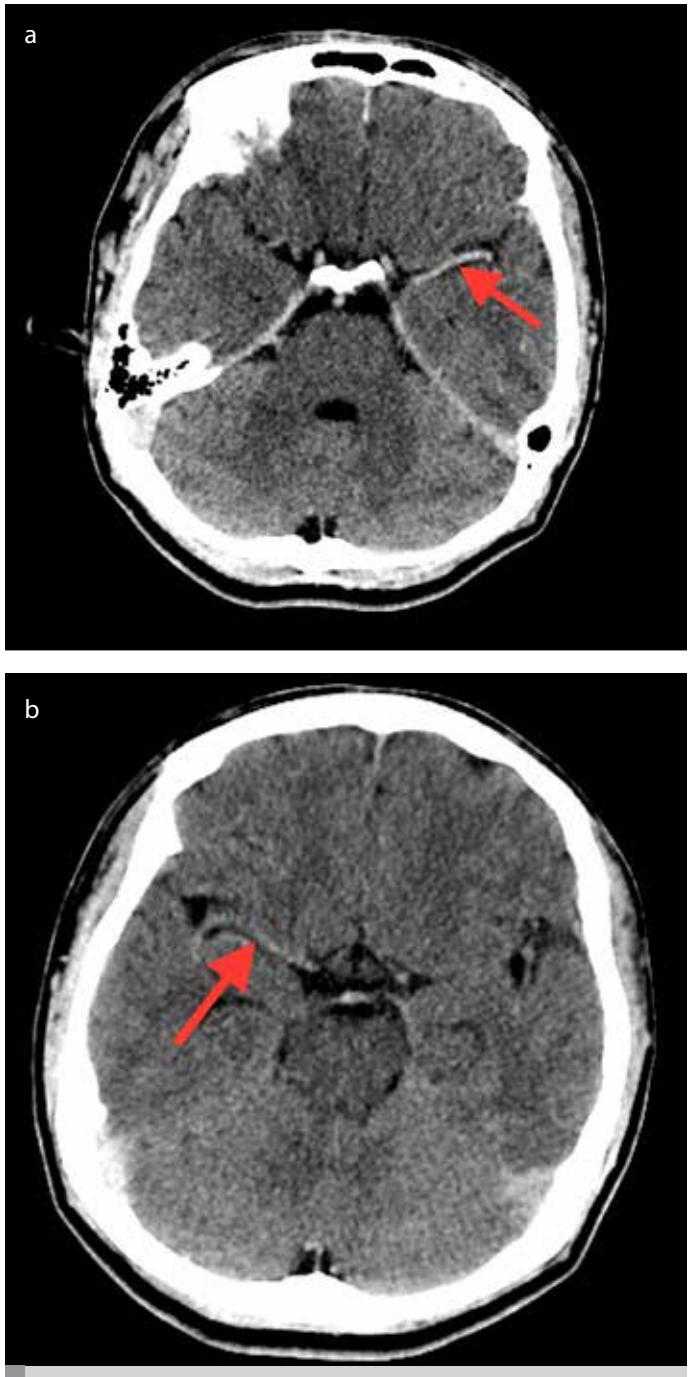

FIGURE 3. a, b.Axial noncontrast CT of the head images show bilateral dense middle cerebral arteries, which favors a diagnosis of erythrocytosis over intracranial hemorrhage

The patient was admitted to the surgical ward (rather than intensive care unit) and later transferred to the hematology service during which an extensive workup found no evidence of primary polycythemia or a conventional cause for secondary polycythemia. He underwent therapeutic phlebotomy, remained asymptomatic, and was discharged the following day with conservative management.

Discussion

Polycythemia can be asymptomatic and therefore undiagnosed for many years, leading to its discovery as an incidental finding (1). Patients in whom polycythemia is incidentally discovered are usually appropriate for an urgent outpatient evaluation by a hematology specialist. However, if a patient describes symptoms consistent with hyperviscosity as a cause, such as fatigue, headache, blurred vision, transient loss of vision, paresthesias, or mental status changes, admission for performing limited phlebotomy should be considered.

The added context of the patient's high hematocrit was essential to interpret the head CT findings and make the final correct diagnosis of new erythrocytosis with artefactual vessel hyperdensity on imaging. Subtle differences between a subarachnoid hemorrhage and enhanced vasculature can be seen by comparing the noncontrast head CT images in Figure 1a and 1b to the MRI images in Figure 2a and 2b. The presence of dense middle cerebral arteries bilaterally in the absence of prior contrast can aid in the identification of erythrocytosis (Figure 3). Although Hounsfield units (HU) are often helpful to differentiate tissue densities, measuring absolute HU in noncontrast head CT's to differentiate between a pseudoenhancement and a true hemorrhage is not reported in the radiology literature because acute hemorrhage in noncontrast CT is diagnosed by a greater density than the patient's white or grey matter. In other words, there is not a single density that defines acute hemorrhage; it is the difference in densities that is important. There is no defined degree of increased density to diagnose a true hemorrhage. Even if an estimate existed, it would vary based on the hemoglobin level and would not be sufficiently reliable to distinguish a real hemorrhage from a pseudohemorrhage (3).

It is also important to note that erythrocytosis can present with a subarachnoid hemorrhage (nontraumatic by case reports) and dural venous sinus thrombosis (4); therefore, suspicion of erythrocytosis should be confirmed by MRI.

Erythrocytosis causing pseudoenhancement has been rarely reported in a nontraumatic context (5, 6). Other differential diagnoses for pseudosubarachnoid hemorrhage findings in CTs scan include diffuse cerebral edema, purulent meningitis, large parenchymal infarction with associated edema, mass effect from a subdural hemor-

rhage, contrast extravasation, status epilepticus, spontaneous intracranial hypotension, and after myelography (7).

Conclusion

Differential diagnoses for trauma patients should always include potential medical causes in addition to traumatic causes. A classic example is a patient who experiences syncope and presents with what can initially appear to be a primary trauma problem. Carefully evaluating all possible etiologies of enhancement on noncontrast CT of the head, particularly in trauma patients, can prevent unnecessary interventions and provide the correct treatment and disposition.

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