Severe Anemia Associated With Transient Neurological Deficits
Amir Shahar, MD, and Menachem Sadeh, MD

In two patients with carotid artery stenosis and anemia, neurological deficits appeared whenever the hemoglobin level fell below a critical level of 5-6 g/dl and resolved with correction of the anemia. Profound anemia should be considered as a cause of focal neurological deficit, especially if there is evidence of cerebral atherosclerosis. (Stroke 1991;22:1201-1202)

Ischemia is the most common cause of transient neurological deficit. Other causes include migraine, epilepsy,1 hypoglycemia,2 and subdural hematoma.3 Because anemia is not routinely considered in the differential diagnosis of transient neurological deficit, we report this association in two patients and discuss the circumstances that brought about this rare occurrence.

Case Reports

Case 1
A 67-year-old man was admitted because of upper gastrointestinal tract bleeding. He had a 20-year history of gout and hypertension and suffered from mild renal failure. Several days before admission, noninvasive investigations of the carotid arteries were performed because of asymptomatic bruits. Ninety percent narrowing of the left carotid artery and 85% of the right were revealed, and aspirin was prescribed. He was pale, but otherwise appeared to be in good general health. Blood pressure was 170/90 mm Hg and pulse 80. He had a cardiac (2/6) systolic murmur. Laboratory studies showed the following results: hemoglobin was 8.3 g/dl, creatinine 2.7 mg/dl, urea 170 mg/dl, and uric acid 9.5 mg/dl. Electrocardiography showed left ventricular hypertrophy and strain, and chest x-rays showed the heart to be enlarged. Echocardiography demonstrated good ventricular contractions with no evidence of valvular disease or mural thrombus. Endoscopy of the stomach revealed superficial erosions. Aspirin was discontinued, and the patient was discharged after packed cell transfusion.

Two weeks later he was readmitted because of generalized fatigue and nonfluent expressive aphasia. The aphasia appeared a few hours before his readmission. The neurological examination was otherwise normal. Hemoglobin was 5.6 g/dl, and the feces analysis showed bleeding. The anemia was corrected by blood transfusion, after which the aphasia resolved. The neurological deficit lasted approximately 10 hours. A computed tomography (CT) scan of the brain was normal. Endoscopy of stomach and duodenum showed no source of bleeding. He was discharged on cimetidine therapy.

During the following months the patient was hospitalized several times because of gastrointestinal bleeding. Whenever the hemoglobin dropped below 5 g/dl, he developed aphasia. Repeated CT scans did not show infarcts. Occasionally he reported to the hospital because of "loss of speech," aware of the necessity for blood transfusion. The aphasia was always resolved soon after transfusion.

The source of bleeding was eventually found to be angiodysplasia of the duodenum, which was resected with part of the jejunum. Bleeding stopped and hemoglobin levels have since been found to be always above 10 g/dl. No episodes of transient aphasia have recurred, and he has remained free of any neurological deficit.

Case 2
A 60-year-old woman with chronic idiopathic thrombocytopenic purpura and non-insulin-dependent diabetes mellitus presented with melena. Twelve years previously she had undergone splenectomy. A year before admission, transient right hemiparesis led to the discovery of bilateral carotid artery stenosis.

She appeared in good general health, although pale. Blood pressure was 130/90 mm Hg and pulse 96. No orthostatic hypotension was detected. Bilateral carotid bruits were heard. Hemoglobin was 8.4 g/dl and thrombocyte count 6,000/mm³. All other blood tests were normal. Endoscopy of the stomach...
and duodenum revealed fresh blood and several gastric erosions, but no ulcer. Treatment with prednisone (80 mg daily) and cimetidine and transfusions of blood and thrombocytes were started. However, she continued to bleed, and when hemoglobin levels dropped below 6 g/dl, right hemiparesis and aphasia appeared. A CT scan of the brain was normal. The anemia was corrected, and the neurological deficit, which lasted for about 16 hours, resolved rapidly. Carotid artery duplex studies showed 90% narrowing of the left artery and 80% of the right.

Despite the continued presence of melena and blood loss of approximately 1 l/day, the source of bleeding was not detected on endoscopy. Whenever hemoglobin level dropped below 6 g/dl, right hemiparesis appeared, occasionally accompanied by aphasia. Symptoms occurred when the patient was lying or sitting in bed (where she had spent most of the time). Blood pressure was normal during these episodes, and no orthostatic hypotension was detected. Repeated CT scans were also normal. The neurological signs gradually disappeared during the blood transfusion, and the anemia was corrected in six or seven such episodes. After it was determined that the hemiparesis was induced by the anemia, blood was given prophylactically, and she had no further neurological events. A diagnosis of intestinal angiodysplasia was entertained, but because of the thrombocytopenia, mesenteric angiography could not be carried out. She was therefore treated with interferon (3,000,000 units every second day) and estrogen. After 3 weeks the bleeding ceased, although there was no improvement in the thrombocyte count.

She was discharged, neurologically symptom-free, on estrogen and prednisone medication. Hemoglobin levels remained stable, and 6 months of follow-up were uneventful.

Discussion

Anemic hypoxia is the result of reduced hemoglobin content with normal oxygen tension or saturation. Anemic hypoxia due to blood loss or hemolysis has not been considered as a cause of neuronal dysfunction, because even with profound anemia the brain oxygen requirement is being met. We have found only one case report of transient ischemic attack caused by anemia in a 2-year-old girl who presented with left hemiparesis and a hemoglobin level of 4 g/dl. Our patients had two reasons for brain hypoxia: reduced cerebral blood flow due to 90% obstruction of the left carotid arteries, and severe anemia. Neither of these factors alone produced neurological impairment; however, their combination critically reduced the amount of oxygen available for brain tissue requirements, which resulted in focal neurological deficit. Neurological deficit always appeared in the course of active bleeding, soon after hemoglobin levels dropped below a critical level. This proved to be a reversible dysfunction that resolved during blood transfusion to raise hemoglobin levels. No other hemodynamic factors, such as arterial hypotension or cardiac arrhythmia, that might have reduced flow below threshold levels beyond the stenotic lesion were present.

Tissue dysfunction induced by combined anemia and arterial stenosis, although not described for the brain, is well known for the heart. Angina pectoris is often aggravated and occasionally made manifest by anemia, especially if the latter develops rapidly.

The satisfactory outcome in these two patients would seem to warrant heightened awareness of the coexistence of anemia and carotid artery insufficiency as a determining factor in the etiology of transient neurological syndromes.

References


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A Shahar and M Sadeh

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