A baby with hiatal hernia presenting with severe iron-deficiency anemia

Ağır demir eksikliği anemisi ile başvuran hiatal hernili bir bebek

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Dear Editor,

Hiatal hernia (HH) is a condition that develops as a result of a developmental defect of the diaphragm, occurs rarely in the childhood age group, and is frequently asymptomatic (1). Iron deficiency anemia is observed frequently in children and results from factors that increase the need for iron including insufficient intake, malabsorption or bleeding in the gastrointestinal system (GIS) (2). Here, we report a baby who was followed up with a diagnosis of iron deficiency anemia for a long period, suspected because of abnormal air density in the lower right-middle mediastinal region on lung radiography obtained because of respiratory tract infection, and was diagnosed as having HH with barium esophagastroduodenal (EGD) imaging.

A 17-month-old female patient who had been receiving treatment because of iron deficiency anemia detected in an external center, but did not respond sufficiently to treatment, was hospitalized in our pediatric ward with a diagnosis of anemia and acute bronchiolitis when she was found to have symptoms of cough and wheezing and a hemoglobin value of 6.8 g/dL. In a physical examination, her general status was reported as moderate-good, pale appearance; consciousness: clear; respiratory rate: 32/min; cardiac heart beat: 162/min, blood pressure: 90/50 mm Hg; and body temperature: 36.7°C. She had a body weight of 9.7 kg (10-25p) and a height of 78 cm (10-25p). The head-neck examination revealed pale conjunctiva, hyperemic oropharynx, hypertrophic tonsillae. She had no cervical lymphadenopathy. The respiratory sounds were rough bilaterally and rhonchi were heard in places. In a cardiovascular system examination, the heart was rhythmic, no additional sounds were heard and a 1/6 systolic murmur was heard in the mesocardiac area. An abdominal examination revealed no organomegaly. Her neuromotor development was compatible with her peers and no pathologic findings were observed in a neurologic examination. In the laboratory tests, urea, creatinine, aspartate aminotransferase, alanine aminotransferase, lactate dehydrogenase, uric acid, C-reactive protein, and serum electrolyte values were found as normal. The complete blood count was as follows: hemoglobin 6.8 g/dL, white blood cells (WBC=12,600/mm³), erythrocytes 3.56 M/uL, mean corpuscular volume (MCV) 54.1 (normal: 72–88) fl, red cell distribution width (RDW) 18.2%, platelet count 386,000 K/µL. In the tests performed to elucidate the cause of anemia, a direct Coombs test was found as negative, occult blood in stool was found as positive, the reticulocyte value was found as 2.2%, the serum ferritin level was 2.6 (normal: 10–60) ng/mL, transferrin saturation was found as 374% (normal: >16%) µg/dL, and the erythrocytic series on peripheral smear was found to be hypochromic microcytic. Other findings were found as normal. Echocardiography performed because of the cardiac murmur was found as normal. In the follow-up, an appropriate erythrocyte suspension was transfused (15 cc/kg) to the patient who developed severe anemia and tachycardia. Respiratory syncytial virus-A was found in a viral respiratory panel multiplex polymerase chain reaction test, which was performed because of the symptoms of cough and wheezing, and symptomatic treatment was administered. An abnormal air shadow was noted in the lower-middle mediastinal region on lung imaging obtained because of the respiratory tract symptoms. On thoracic computed tomography, HH was found and one-third of the stomach was observed to be located in the posteromedial part of the right hemithorax. Barium esophagoduodenoscopic imaging confirmed HH (Fig. 1a, b). Anti-reflux treatment was initiated and the patient was followed up. After a short period, an open partial fundoplication procedure was performed. In the postoperative sixth month, it was observed that his clinical status was good and her signs and symptoms had recovered completely.
The most common type of anemia in childhood is iron deficiency anemia and blood loss in the GIS must be evaluated in these children (2). In the literature, it has been reported that iron deficiency anemia may rarely develop due to bleeding in the GIS secondary to gastric ulcer, gastritis or incarcerated hernia pouch erosion (Cameron ulcer) (3–5).

In conclusion, HH should be considered in the differential diagnosis in patients with bleeding in the GIS who are found to have severe and treatment-resistant iron deficiency anemia, and HH must be excluded in those with abnormal air density in the middle/lower mediastinal region on lung imaging.

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Figure 1. Abnormal air density in the lower right-middle mediastinal region on postero-anterior lung graphy (white arrow, a). A wide lesion in the paramediastinal region (appearance of hiatal hernia) on barium esophagastroduodenal graphy (white arrow, b)