staff.

The medical profession has played an important role in childhood advocacy and the protection of children from battering and abuse. This will continue to be a high priority for all physicians who deal with children. However, it is important to be cognizant of conditions that can masquerade as battered child syndrome. Familiarity with alternative diagnoses is critical in order to avoid the multidimensional trauma of inaccurately labeling a child as battered. This report illustrates that children with unexplained and abrupt onset of bruising may have a medical condition, erythema multiforme, which can be confused with child battering.

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Noninvasive Diagnosis of Pulmonary Hemorrhage in Rheumatoid Arthritis

The noninvasive diagnosis of pulmonary hemorrhage usually relies upon the presence of hemoptysis or hemosiderin-laden macrophages in the sputum. Respiratory tract secretions, however, may be contaminated with blood during passage through the large airways or upper respiratory tract, and hemosiderin-laden macrophages may be found in the sputum of patients with other diseases such as pulmonary edema. In addition, pulmonary hemorrhage may occur without associated hemoptysis or significant changes in the chest roentgenogram. We have used serial measurements of the pulmonary uptake of carbon monoxide combined with M-mode echocardiography to monitor left atrial size in order to document pulmonary hemorrhage in an 11-year-old girl with rheumatoid arthritis.

CASE REPORT

S.F. had been well until 8 years of age when she developed a seropositive chronic arthritis of both large and small joints in the upper and lower extremities. The clinical, radiologic, and laboratory findings satisfied the criteria for the diagnosis of juvenile rheumatoid arthritis. Initially, there was a satisfactory response to salicylate therapy. However, over the subsequent year there were several exacerbations of her arthritic symptoms, and rheumatoid nodules developed on her hands, elbows, and ankles. At 9 years of age, she developed a progressive anemia with the hemoglobin concentration ultimately decreasing to 5.5 g/dL. There was no history of blood loss, and blood was not detected in her stools. Results of investigations for her anemia were as follows: reticulocyte count (19), bilirubin, 0.8 mg/dL, ferritin, 20 μg/L (normal, 18 to 300 μg/L), serum iron, 34 μg/dL, total iron binding capacity, 420 μg/dL. Heinz bodies were not seen, and no hemosiderin or hemopexin could be detected in the urine. Results of Coombs' tests were negative, and roentgenographic assessment of the chest, stomach, and duodenum was normal. Peripheral erythrocytes were hypochromic and microcytic, and a bone marrow aspiration revealed erythroid hyperplasia with a predominance of pronormoblasts and a myeloid-erythroid ratio of 1.5:1.0. No stainable iron was seen. A blood transfusion was administered, and oral iron therapy was instituted. Over the subsequent 18 months, the arthritic symptoms were satisfactorily controlled with corticosteroid and salicylate therapy and her hemoglobin concentration ranged from 9 to 12.9 g/dL.

When the patient was 10 years old, a diagnosis of bronchitis was made. Although her respiratory symptoms slowly resolved, she was hospitalized 1 month later because her hemoglobin level had decreased to 6.7 g/dL. A reticulocyte count of 11.8% suggested blood loss or hemolysis, but no site of bleeding could be found and a hemolytic process could not be confirmed. A study utilizing 51Cr-labeled erythrocytes demonstrated a shortened

REFERENCES


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significant increase in pulmonary uptake of CO (see Fig 1).

One month later, S.F. was admitted to the intensive care unit with a 12-hour history of severe chest pain, dyspnea, and orthopnea, combined with a cough productive of blood stained mucus. Results of clinical and laboratory investigations revealed congestive heart failure, bilateral pulmonary infiltrates, and anemia (hemoglobin, 5.6 g/dL). The ECG was characterized by widened bizarre ventricular complexes indicative of left bundle branch block and, possibly, associated ischemia. M-mode and two-dimensional ECG revealed an enlarged left atrium and a dilated poorly contracting left ventricle with no associated pericardial effusion. Levels of SGOT, creatinine phosphokinase, and lactic acid dehydrogenase were mildly elevated. The patient’s condition slowly improved with therapy consisting of oxygen, digoxin, diuretics, corticosteroids, and a blood transfusion. Over the following week, the pulmonary infiltrates cleared, but the cardiomegaly persisted. Seven weeks later, when clinically stable the patient expectorated approximately 30 mL of bright red stained mucus and complained of some shortness of breath. Coexistent with this hemoptysis, a right perihilar infiltrate appeared; it had not been present on the chest roentgenogram taken three days previously (Fig 1). In addition, there was a marked increase in the uptake of CO by the lung with no change in the hemoglobin concentration or left atrial dimensions as assessed by M-mode echocardiography (Fig 2). Pulmonary uptake of CO was measured by a modification of the method of Ogilvie and co-workers2 and O’Brodovich et al. This single-breath diffusing capacity (DCOsb) was corrected for lung volume by dividing the DCOsb by the simultaneously measured alveolar volume (VA), thus obtaining the Kco. Further investigations at this time revealed pigmented macrophages in her sputum and normal urinalysis and circulating complement levels, whereas circulating immune complexes, antiliglomerular basement membrane antibody, and milk precipitants could not be detected. A diagnosis of pulmonary hemorrhage, probably due to a vasculitis, was made and the patient was placed on a regimen of 3 mg/kg of azothioprine in addition to continuing corticosteroid therapy. Although the Kco value decreased shortly after the hemoptysis, it did not return to the previous normal values for 5 weeks (Fig 2). This prolonged mild elevation of the Kco value may have resulted from an ongoing vasculitis and subclinical pulmonary hemorrhage. Since discharge from the hospital, she has remained well with only mild joint symptoms but she continues to have significant cardiomegaly, impaired left ventricular function, and unchanged abnormal ECG.

**DISCUSSION**

Chronic arthritis is the dominant clinical finding in rheumatoid arthritis. Less commonly, other tissues, such as the myocardium, may be involved in the inflammatory process.4,5 Our patient had at least three episodes of anemia that could not be attributed to her chronic disease, and the documentation of pulmonary hemorrhage suggests that the lungs were the site of the bleeding. Pulmonary hemorrhage in rheumatoid arthritis has not previously been reported: the diagnosis requires a high index of suspicion that can be strengthened by the performance of these noninvasive techniques.

We have used the pulmonary uptake of carbon monoxide to noninvasively confirm the diagnosis of pulmonary hemorrhage in a young girl with rheumatoid arthritis and unexplained episodes of severe anemia. Hemoglobin has a high affinity for CO and, therefore, blood within the alveoli will persistently elevate...
enhance the uptake of CO. Normally the amount of CO uptake by the lung is dependent primarily on the volume of hemoglobin exposed to the gas exchanging areas of the lung, and is minimally, if at all, affected by the diffusion characteristics of the alveolar-capillary membrane. Pulmonary uptake of CO is, therefore, primarily influenced by the volume of ventilated lung (VA), the perfused capillary surface area (Vc) within that lung volume, and the hemoglobin concentration of the blood within the lung. Thus, to diagnose pulmonary hemorrhage by measuring the pulmonary uptake of CO, all three of these variables must be assessed as bleeding within the lung may decrease VA, result in cardiac dysfunction and engorgement of pulmonary vessels (increase Vc), or lower hemoglobin concentration. When the pulmonary uptake of CO is measured during a single 10-second breath hold, the lung volume into which the gas is distributed (VA) may be determined because the inert gas helium is added

\[ K_{co} = \frac{\text{ml/min}}{\text{mmHg/L}} \]

Fig 2. Uptake of CO by lung (Kco) immediately prior to and following episode of hemoptysis and appearance of pulmonary infiltrate (arrow). Kco values for S.F. (open circle) and for 95% confidence limits for normal subjects (stippled area) are corrected to hemoglobin value of 15 g/dL using correction equation derived in children. Inasmuch as the patient's hemoglobin concentration ranged from 7.5 to 8.5 g/dL and left atrial dimension (solid triangle) did not change, abrupt increase in Kco likely resulted from bleeding within lung. Five weeks later, corrected Kco returned to normal value (6.7 mL/min/mm Hg/L).
to the inspired gas mixture. An uptake of CO per unit of lung volume (Kco) can therefore be calculated: \( Kco = \frac{DCOsb}{VA} \). Although the pulmonary capillary blood volume cannot be measured directly, when left atrial pressure increases, recruitment and distension of the pulmonary vessels occur. Therefore, we indirectly assessed pulmonary vascular engorgement in our patient by measuring left atrial dimensions using M-mode echocardiography. As neither left atrial dimension nor hemoglobin concentration changed when the patient produced blood-tinged sputum, we concluded that the marked increase in Kco value must have resulted from an increase in the amount of blood within the gas exchanging portions of the lung. Previous investigations have demonstrated that pulmonary edema or chronic left atrial hypertension, such as that seen in mitral stenosis, usually result in a low or normal values for Kco.

Ewan et al have previously used an increase in CO uptake by the lung to detect pulmonary hemorrhage in patients with Goodpasture's syndrome. To determine whether increases in Kco value were due to an increase in intravascular or extravascular blood, they used the radioactive isotope C\(^{15}\)O. They demonstrated that clearance of this isotope from the lung is delayed when extravascular blood is present. Regrettably, a cyclotron is required for production of this extremely short-lived isotope (half-life, two minutes), thus limiting the usefulness of their technique. Their data suggest that only fresh bleeding will increase the Kco, and less than 200 mL can be detected.

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**Note Added in Proof.** Following acceptance of this manuscript for publication, S.F. died of congestive heart failure. She was 11½ years old. Post-mortem examination revealed severe pulmonary hemosiderosis and a nonspecific cardiomyopathy. The heart was dilated and hypertrophied, and extensive scarring was present in the endocardium and myocardium.

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**REFERENCES**

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