CASE RECORDS of the MASSACHUSETTS GENERAL HOSPITAL

Case 6-2014: A 35-Day-Old Boy with Fever, Vomiting, Mottled Skin, and Severe Anemia

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PRESENTATION OF CASE

Dr. Anna L. Cook (Pediatrics): A 35-day-old boy was admitted to the pediatric intensive care unit (ICU) of this hospital because of fever, vomiting, mottled skin, and severe anemia.

The patient was born at another hospital, after an uncomplicated 37.5-week gestation, by cesarean section for breech presentation with a fraternal twin. His mother had received prenatal care, and prenatal screening tests for gonorrhea, chlamydia, syphilis, group B streptococcus, human immunodeficiency virus, and hepatitis B virus had been negative; screening for immunity to rubella virus was positive. Placental membranes had ruptured less than 24 hours before birth, and antibiotic drugs were administered before delivery. The 1-minute and 5-minute Apgar scores were both 9. The first hepatitis B immunization was administered to the patient before he was discharged. Two days before presentation, at a routine 1-month examination by his pediatrician, he was well, and the second hepatitis B immunization was administered.

The day before admission, the patient's mother noted increased fussiness, with pale and warm skin; one episode of vomiting occurred. On the morning of admission, tachycardia was noted and vomiting increased. On examination at the pediatrician's office, the patient was pale, with mottled skin; the temperature was 38.6°C. He was referred immediately to the emergency department of a second hospital.

On examination, the weight was 3.61 kg, the rectal temperature 38.0°C, the blood pressure 113/63 mm Hg, the pulse 172 beats per minute, the respiratory rate 64 breaths per minute, and the oxygen saturation 97% while he was breathing ambient air. He was pale, listless, and fussy, with mild suprasternal and intercostal retractions, without grunting or nasal flaring. The remainder of the examination was normal. A lumbar puncture was performed. Samples of the blood, urine, and cerebrospinal fluid were obtained for culture. Blood levels of electrolytes, glucose, calcium, magnesium, alkaline phosphatase, and alanine aminotransferase were normal, as were the results of renal-function tests; other test results are shown in Table 1. Urinalysis revealed dark red, cloudy urine, with a pH of 7.0, a specific gravity of 1.015, a large amount of blood, 100 mg per deciliter of protein, and trace leukocyte esterase by dipstick, as well as 0 to 5 red cells and white cells per high-

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power field and a small amount of bacteria, a moderate number of epithelial cells, and 15 to 20 granular casts per low-power field. Ceftriaxone, ampicillin, and a bolus of normal saline were administered intravenously, and a transfusion of leukocyte-depleted, packed red cells (type O, Rh-negative) was begun. Less than 4 hours after presentation, the patient was transferred to this hospital; en route, clindamycin, crystalloid solution, and the remainder of the red-cell transfusion were administered intravenously, and oxygen was given through a nasal cannula.

The patient had previously been well, consuming both breast milk and formula. He lived with his parents, his twin, and an older sibling in a suburban, forested neighborhood, where the mother frequently walked. Approximately 2.5 months before the admission, the parents had vacationed on an island off the coast of Massachusetts; years earlier, they had visited the Caribbean, which has been their only international travel. The parents reported that they recently had upper respiratory tract symptoms; the family had a pet dog.

Table 1. Laboratory Data.*

Variable	Reference Range, Age-Adjusted†	Other Hospital	This Hospital, on Admission
Blood			
Hematocrit (%)	31.0–55.0	16.9 (ref 26.7–36.8)	20.9
Hemoglobin (g/dl)	10.0–18.0	6.1 (ref 9.1–12.1)	7.6‡
White-cell count (per mm³)	5000–19,500	8000	6500
Differential count (%)			
Neutrophils	20–46	6 (ref 14–40)	9
Band forms	0–10	11 (ref 0–14)	2
Lymphocytes	50–85	68 (ref 40–70)	79
Reactive lymphocytes		8 (ref 0–10)	
Monocytes	4–11	6 (ref 0–14)	10
Eosinophils	0–8	1 (ref 0–6)	0
Myelocytes		1 (ref 0)	
Nucleated red-cell count (per 100 white cells)	0		4
Platelet count (per mm³)	150,000–400,000	23,000 (ref 140,000–450,000)	36,000
Mean corpuscular volume (μm³)	85–123	98.0 (ref 86–97)	88
Erythrocyte count (per mm³)	3,000,000–5,400,000		2,390,000
Smear description		Moderate stippling and polychromasia, 35–40% erythrocytes with multiple ring forms	1+ polychromasia, 1+ anisocytosis, 18% erythrocytes with intracellular parasites including multiple ring forms
ABO blood type		O, Rh-positive; antibody screen negative	
D-Dimer (ng/ml)	<500		5906
Bilirubin (mg/dl)			
Total	2.0–15.0	1.3	1.5
Direct	0.0–0.4	0.5‡	0.5
Protein (g/dl)			
Total	6.0–8.3	4.7	5.0
Albumin	3.3–5.0	2.9	2.9
Globulin	2.3–4.1	1.8	2.1
Aspartate aminotransferase (U/liter)	9–80	121	134
Lactate dehydrogenase (U/liter)	110–210		2254
Lipase (U/liter)	13–60		11
Amylase (U/liter)	3–100		<3

Table 1. (Continued.)

Variable	Reference Range, Age-Adjusted†	Other Hospital	This Hospital, on Admission
Cerebrospinal fluid			
Color		Colorless	
Turbidity		Clear	
Red-cell count (per mm^3)			
Tube 1		32	
Tube 4		54	
White-cell count (per mm^3)			
Tube 1		11	
Tube 4		11	
Differential count (%)			
Neutrophils		0	
Lymphocytes		44	
Monocytes		56	
Protein (mg/dl)		36 (ref 30–100)	
Glucose (mg/dl)		57 (ref 40–80)	
Gram's stain		Very few white cells, no organisms	
Wright's stain		Intraerythrocytic ring forms	
Venous blood gases			
Inspired oxygen (liter/min)			1 (through a nasal cannula)
Base excess (mmol/liter)			2.2
pH	7.30–7.40		7.34
Partial pressure of carbon dioxide (mm Hg)	38–50		55
Partial pressure of oxygen (mm Hg)	35–50		32
Bicarbonate (mmol/liter)	22–27		28

* The abbreviation ref denotes the reference range at the other hospital. To convert the values for bilirubin to micromoles per liter, multiply by 17.1. To convert the values for phosphorus to millimoles per liter, multiply by 0.3229. To convert the values for glucose to millimoles per liter, multiply by 0.05551.

† Reference values are affected by many variables, including the patient population and the laboratory methods used. The ranges used at Massachusetts General Hospital are age-adjusted for patients who are not pregnant and do not have medical conditions that could affect the results. They may therefore not be appropriate for all patients.

On examination, the patient was crying vigorously and had pale and mottled skin. The temperature was 39.1°C, the blood pressure 101/69 mm Hg, and the pulse 176 beats per minute; the respiratory rate was 68 breaths per minute, with supraclavicular, intercostal, and subcostal retractions, and the oxygen saturation was 97% while the patient was breathing 1 liter of oxygen through a nasal cannula. The anterior fontanelle was soft and flat, and there was no scleral icterus. A holosystolic-flow murmur, grade 2/6, was heard. The abdomen was soft, without tenderness or distention, and the spleen was palpated 2 to 3 cm below the costal margin. The arms and legs were mottled and slightly cool. The remainder of the examination was normal. The blood levels of electrolytes, glucose, phosphorus, ionized calcium, and creatine kinase were normal, as were the results of coagulation tests; other test results are shown in Table 1. A chest radiograph showed diffuse bilateral pulmonary infiltrates, a finding consistent with pulmonary edema, and a hazy opacity in the left upper abdomen, a finding consistent with splenomegaly (Fig. 1). Results of ultrasonography of the head were normal. A transthoracic echocardiogram was normal for the patient's age, with a patent foramen ovale and left-to-right shunting.

A diagnosis was made.

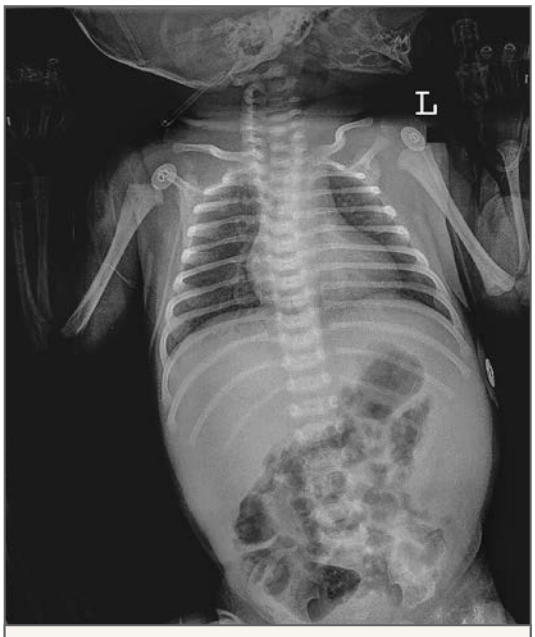

Figure 1. Radiograph of the Chest and Abdomen.
An anteroposterior chest and abdominal radiograph obtained on admission shows perihilar vascular congestion, a finding consistent with mild pulmonary edema. The cardiac and mediastinal silhouette is normal. The bowel-gas pattern in the abdomen is normal, and a homogeneous haziness overlying the left upper abdomen is consistent with splenomegaly.

DIFFERENTIAL DIAGNOSIS

Dr. Lynn M. Luginbuhl: I am aware of the diagnosis in this case. This previously healthy 35-day-old twin presented with acute onset of fever, irritability, and vomiting, as well as with signs of respiratory distress, tachycardia, poor perfusion, and splenomegaly and with laboratory findings of hemolytic anemia, thrombocytopenia, and an elevated level of aspartate aminotransferase. Noninfectious causes of this patient's illness, such as congenital heart disease and metabolic disorders, are unlikely because of the normal echocardiogram, normal electrolyte levels, and lack of acidosis. In this age group, bacterial sepsis is a leading cause of this constellation of signs and symptoms, and patients with bacterial sepsis typically present with fever and abnormalities of the respiratory, gastrointestinal, and neurologic systems.[1] Thus, evaluation in the emergency department with cultures of blood, urine, and cerebrospinal fluid, followed by antibiotic therapy, is appropriate. Bacteremia in young infants is most commonly due to *Escherichia coli* or group B streptococci.[2,3] *Staphylococcus aureus*, particularly community-acquired methicillin-resistant *S. aureus*, is becoming a common pathogen but is usually associated with focal infections in bones, joints, or skin.[4] Infection with *Listeria monocytogenes*, other enteric and nonenteric gram-negative bacilli, or viridans group streptococci occasionally causes sepsis in this age group. Results of examination of the patient's cerebrospinal fluid are not suggestive of meningitis because of the normal levels of glucose and protein and the absence of notable pleocytosis for a 35-day-old infant.

BABESIOSIS

It is always prudent to rule out a bacterial infection in a 35-day-old infant with fever. However, by the time the patient arrived at this hospital, we had learned from the other hospital that he had profound anemia and that examination of a peripheral-blood smear revealed that 35 to 40% of the erythrocytes contained multiple ring forms. These intraerythrocytic ring forms in combination with fever, splenomegaly, hemolytic anemia, thrombocytopenia, vomiting, respiratory distress, dark urine, and elevated liver levels of aminotransferases are highly suggestive of infection with *Babesia microti*.[5-9]

Infection with *B. microti* is usually mild or asymptomatic but can be severe in infants, the elderly, asplenic patients, and immunocompromised hosts. Although it seems unlikely that a 35-day-old infant would contract this infection, the usual incubation period for an acquired tickborne infection is 1 to 6 weeks, and congenital infections manifest at 4 to 5 weeks of age.[5] It is important to remember that plasmodium species are also intraerythrocytic pathogens, but malaria is unlikely in this case because neither the patient nor his mother had traveled to an area where malaria is endemic.

COINFECTION WITH OTHER TICKBORNE PATHOGENS

Although *B. microti* infection is the leading diagnosis in this case, it is important to consider the possibility of coinfection with other tickborne pathogens. Could this patient also have Lyme disease? *Ixodes scapularis* ticks may transmit *Borrelia burgdorferi* (the agent of Lyme disease) and *Anaplasma phagocytophilum* (the cause of human granulocytic anaplasmosis), in addition to *B. microti*. Of all patients with babesiosis, 12 to 53% have a history of Lyme disease or have positive tests for antibodies against *B. burgdorferi*,[10-12] because a

high percentage of ticks infected with babesia are coinfected with *B. burgdorferi*.[13] Therefore, concomitant Lyme disease in this patient is possible. Coinfection with *B. microti* and *A. phagocytophilum* is probably less likely.[13,14] There are no clinical findings suggestive of Lyme disease in this case. In particular, there is no erythema chronicum migrans, a rash that is present in almost 90% of cases of pediatric Lyme disease. *B. burgdorferi* may be transmitted perinatally, but there is no proven congenital syndrome associated with Lyme disease. The clinical presentation of patients with human granulocytic anaplasmosis overlaps with that of babesiosis, except that neutropenia and leukopenia are more prolonged in human granulocytic anaplasmosis. The neutropenia in this patient raises concern for anaplasmosis. Coinfection with *Ehrlichia chaffeensis* (the agent of human monocytic ehrlichiosis) or *E. ewingii* (the agent of canine granulocytic ehrlichiosis) is unlikely, since these infections are rare in New England and the tick vector is *Amblyomma americanum* rather than *I. scapularis*, which is the usual vector in *B. microti* infection. Patients with Rocky Mountain spotted fever (due to *Rickettsia rickettsii*) can present with a nonspecific, febrile illness, with vomiting, thrombocytopenia, and elevated aminotransferase levels, and the petechial rash may not be present early in the illness. However, Rocky Mountain spotted fever is uncommon in Massachusetts, and the tick vector for this disease in the northeastern United States is *Dermacentor variabilis* (the American dog tick), rather than *I. scapularis*.

In this case, the preponderance of evidence points toward a diagnosis of babesiosis. However, to be comfortable with this diagnosis, we need to postulate how a 35-day-old infant would acquire this infection. This infant could have acquired babesiosis either congenitally or through a tick bite. The babesiosis could have been acquired congenitally if the mother had been infected with *B. microti* at 32 weeks' gestation while visiting an island off the coast of Massachusetts. The absence of a tick bite or illness in the mother's history does not rule out this possibility, because most adults infected with *B. microti* are asymptomatic and do not have a known history of a tick bite.[15] Parasitemia may persist for months or years, allowing ample time for perinatal transmission. The congenitally infected infant typically becomes ill at 4 to 5 weeks of age, and this patient's age is within that range. The babesiosis could have been acquired through a tick bite if a tick had bitten the patient during one of the family's walks outside, or the patient could have been bitten by a tick that was carried into the house by the family dog. The incidence of babesiosis is almost 100 times as high on the islands off the coast of Massachusetts as on the mainland; thus, it is far more likely that the mother was infected during pregnancy than that the patient was infected after birth.[16] I believe that this is a case of probable congenital babesiosis.

DR. LYNN M. LUGINBUHL'S DIAGNOSIS

Congenital or acquired *Babesia microti* infection.

PATHOLOGICAL DISCUSSION

Dr. John P. Dekker: Thick and thin peripheral-blood smears were examined for parasites (Fig. 2). Numerous intraerythrocytic and occasional extraerythrocytic structures of different sizes and with different morphologic features were present. Many erythrocytes contained one or two large ring forms, features that in conjunction with the epidemiologic history were consistent with *B. microti*. A number of erythrocytes contained four ring forms, which probably represented the stages that occur after the breakdown of the tetrad ("Maltese cross") configuration, a feature characteristic of babesia.

In addition, further blood testing was performed to rule out infection with other tick-borne pathogens. A blood culture was negative, as were polymerase-chain-reaction (PCR)–based tests for anaplasma and ehrlichia species and serologic tests for IgM and IgG antibodies against borrelia.

MANAGEMENT

Dr. Phoebe H. Yager: When we received the call from the other hospital requesting a transfer of this patient to this pediatric ICU, I was concerned that the infant was showing signs of septic shock, and I wondered whether he had received adequate fluid resuscitation and appropriate broad-spectrum antimicrobial therapy. We were told that he was tachycardic and tachypneic but had a normal blood pressure for his age and a brisk capillary refill. Also, he had responded vigorously during the evaluation for sepsis, although

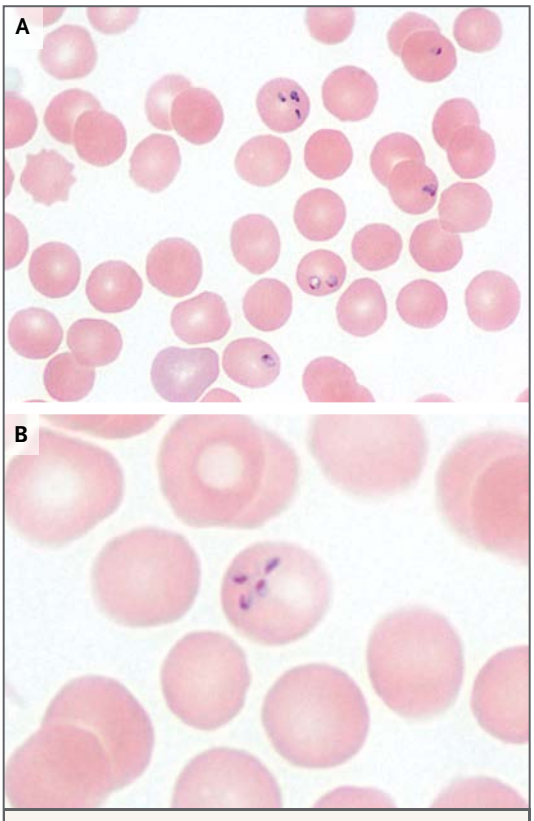

Figure 2. Peripheral-Blood Smear (Giemsa).
A thin peripheral-blood smear shows erythrocytes containing organisms characterized by a ring (stained pale purple) with central clearing and a peripheral purple chromatin dot (Panel A). Several erythrocytes contain four organisms (Panel B), which represent the stages that occur after the breakdown of the classic tetrad ("Maltese cross") configuration, a feature characteristic of babesia.

he quickly fell asleep when not bothered. Given that sepsis seemed to be a likely diagnosis, we agreed with continuing the administration of ceftriaxone and recommended the addition of ampicillin for broader antibacterial coverage and clindamycin for treatment of suspected babesiosis.

The patient was severely anemic, with signs of respiratory distress and tachycardia. We recommended a slow transfusion of packed red cells and the administration of supplemental oxygen through a nasal cannula. Although the patient was tachypneic, with a respiratory rate of 60 breaths per minute and some retractions, we were told that he had had no apneic episodes or desaturation events. Therefore, we thought the risk of respiratory failure was low, and we did not recommend intubation. Placement of a second peripheral intravenous catheter was requested for the transfusion and for use in the event of hemodynamic decompensation. The transfusion of packed red cells was started at the other hospital, and the decision was made to allow the transfusion to continue while the patient was en route to this hospital, although the risk of a transfusion reaction was kept in mind.

On the patient's arrival in the pediatric ICU, we further broadened the antimicrobial treatment by adding quinine to augment therapy for babesiosis and doxycycline to target other tick-borne pathogens. We performed a post-transfusion complete blood count, which revealed persistent anemia, neutropenia, and thrombocytopenia. The prothrombin time and international normalized ratio were normal, although the elevated D-dimer level, borderline fibrinogen level, and thrombocytopenia suggested the presence of at least some component of compensated disseminated intravascular coagulation. The patient showed evidence of ongoing hemolysis, including an elevated level of lactate dehydrogenase, hemoglobinuria, and splenomegaly. He had mildly elevated aminotransferase levels but no evidence of acidosis, renal dysfunction, or other metabolic derangements.

This patient met the criteria for severe sepsis, according to the consensus definitions of sepsis and its related syndromes put forth by the American College of Chest Physicians and the Society of Critical Care Medicine.[17] He had evidence of the systemic inflammatory response syndrome (i.e., fever, tachycardia, and tachypnea) that was probably due to infection, as well as evidence of multiorgan dysfunction. He did not meet the criteria for septic shock because he did not have fluid refractory hypotension. We believed that the presence of severe, ongoing hemolytic anemia, thrombocytopenia, altered mental status, and respiratory distress in a patient with an immature immune system and a high level of parasitemia represented fulminant disease with a clinically significant risk of progression to multiorgan failure and possibly death.[18] Given this concern, we placed an arterial catheter for hemodynamic monitoring and in anticipation of the need for frequent blood tests and exchange transfusion.

EXCHANGE TRANSFUSION

The use of exchange transfusion as an adjunct therapy for babesia infection is controversial be-

cause, to our knowledge, no randomized, controlled studies have shown a survival benefit. However, exchange transfusion is considered a potentially lifesaving intervention for patients with severe disease.[19] The Centers for Disease Control and Prevention (CDC) recommends that exchange transfusion be considered in persons with *Plasmodium falciparum* infection, a parasitemia level of at least 10% parasitized erythrocytes, coma, renal failure, or the acute respiratory distress syndrome (regardless of the parasitemia level).[20,21] Suggestions for the use of exchange transfusion to treat babesia infection have been extrapolated from these recommendations.[22,23] Exchange transfusion is thought to have three benefits. First, exchange transfusion reduces the level of parasitized red cells, thereby leading to a decrease in hemolysis, in hypoxia, and in parasite replication. Second, it removes proinflammatory cytokines, tumor necrosis factor, and interleukin-1, which contribute to fevers, hemodynamic instability, and the development of the acute respiratory distress syndrome and multiorgan failure. Third, it is thought to improve rheologic properties of the blood, an effect that may improve end-organ perfusion.[23]

Although automated systems are used to perform exchange transfusions in adults, these systems cannot be used in infants because of the infants' small size and the risk of hemodynamic instability and life-threatening metabolic derangements that could occur during an exchange that is too rapid. Therefore, a manual technique, which requires at least two dedicated providers for the duration of the procedure, is necessary (Fig. 3). The exchange transfusion was associated with an acceptable rate of adverse events in this patient, and his condition seemed to improve during the procedure, although he required calcium repletion once for hypocalcemia. The infusion catheter stopped functioning midway through the planned double-volume exchange transfusion, but because the patient's condition was stable, we decided to await the results of a repeat thin peripheral-blood smear to assess his parasitemia level and to proceed with the procedure only if the parasitemia level exceeded 10%. He had clinical improvement and a parasitemia level of 5.2%, and so the full exchange transfusion was not completed.

The parasitemia level returned to 10% on hospital day 2, but the decision was made not to perform further exchange transfusion because of the patient's continued clinical improvement. The patient received a second platelet transfusion because of a low platelet count, of 24,000 per cubic millimeter. The administration of ampicillin and ceftriaxone was discontinued when urine, blood, and cerebrospinal fluid cultures that had been performed at the other hospital reportedly showed no growth. However, owing to recurrent fevers that occurred later that evening and persistent neutropenia, a repeat blood culture was performed, and an empirical, short course of cefepime was initiated, pending the results of the blood culture.

On hospital day 6, after having been afebrile for 72 hours, the patient no longer required intensive monitoring and was transferred to the general pediatric service. He was still anemic and neutropenic but had signs of adequate end-organ perfusion and resolution of his respiratory distress. His skin was no longer mottled, and a murmur was no longer detected. He was feeding well by mouth and was more alert and no longer fussy. His medications at the time of transfer were quinine, clindamycin, and iron supplements. Doxycycline had been discontinued when test results for anaplasma came back normal.

Daily peripheral-blood smears continued to show a drop in parasitemia levels, and by hospital day 8, only a single ring form was seen on the thin smear. Twenty-one days after the initiation of treatment, thick and thin peripheral-blood smears were negative for babesia.

Dr. Cook: After the patient was transferred to the pediatric service, he did not require any further transfusions. He no longer had symptoms of anemia, and he was feeding well. His medications were switched to azithromycin and atovaquone on discharge. The parasite load at discharge was undetectable. The patient was seen at a follow-up visit 1 week later. He had completed the course of antibiotics, the hematocrit had improved, and testing for babesia was negative. He was thriving and developing normally. The only remaining question in this case was how this 35-day-old infant acquired babesiosis: Was the infection transmitted congenitally or acquired after birth?

Dr. Dekker: To determine whether this infection was acquired in utero or after delivery, additional testing of samples collected from the patient's twin and mother was performed at the

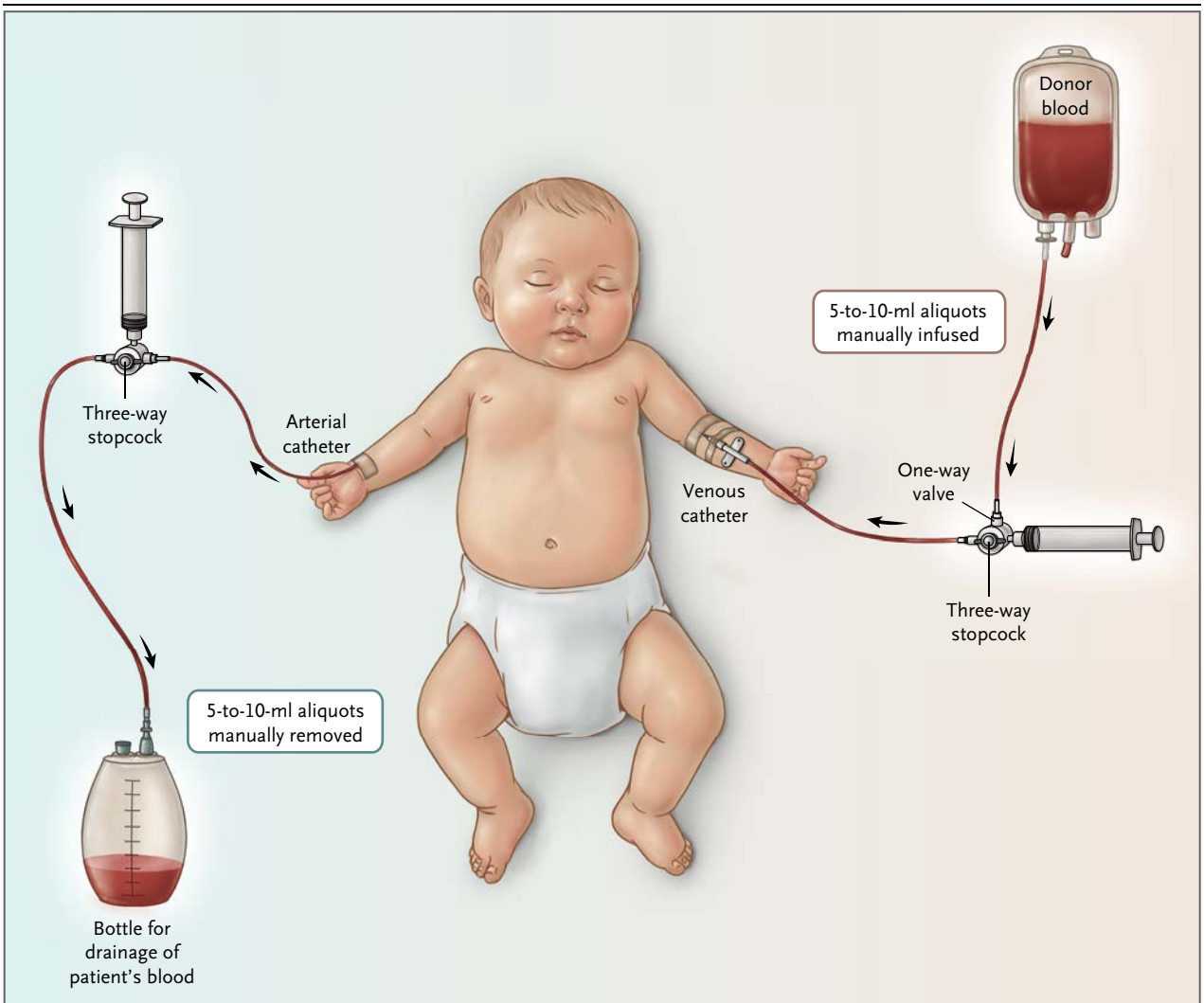

Figure 3. Exchange Transfusion in an Infant.

To avoid clinically significant hemodynamic shifts and metabolic abnormalities, small (5 to 10 ml) aliquots of donor blood are manually infused into the patient's venous circulation while equal aliquots of the patient's blood are manually withdrawn through an arterial catheter.

CDC (Table 2). Thick and thin peripheral-blood smears, prepared from samples collected from the twin and mother at the time of the patient's diagnosis, were examined, and no morphologic evidence of babesia was found. Also, PCR tests of blood samples collected from the twin and mother at the time of diagnosis were negative for babesia. However, antibodies against *B. microti* were detected in blood samples from both the twin and the mother at a high titer of 1:4096, and antibodies against *B. microti* were present at a titer of 1:1024 in a blood sample from the patient that was drawn after the exchange transfusion.

Because of the rarity of congenital transmission of babesia, we collaborated with colleagues at the CDC in an academic investigation to determine whether babesia could be detected in placental tissue and in dried blood spots collected during neonatal screening at the time of delivery. Formalin-fixed, paraffin-embedded placental tissue was sent to the CDC for immunohistochemical staining and PCR testing (Table 2); both methods failed to detect babesia in the placental tissue. PCR testing of the dried blood spots, also performed at the CDC, was negative for babesia. Although PCR testing did not detect nucleic acids from babesia, the presence of high-titer antibodies against *B. microti* in the patient, his twin,

Table 2. Additional Test Results.*

Sample	Test	Result
Patient		
Blood collected after partial exchange transfusion	*Babesia microti* antibody titer	Positive at 1:1024
Blood	PCR for *Anaplasma phagocytophilum*	Negative
Blood	PCR for ehrlichia	Negative
Blood	IgM and IgG antibodies against borrelia	Negative
Dried blood spot collected at birth	PCR for babesia	Negative
Placental tissue collected at birth	Immunohistochemical staining for babesia	Negative
Placental tissue collected at birth	PCR for babesia	Negative
Patient's twin		
Blood	*B. microti* antibody titer	Positive at 1:4096
Blood collected at the time of the patient's diagnosis	PCR for babesia	Negative
Dried blood spot collected at birth	PCR for babesia	Negative
Patient's mother		
Blood	*B. microti* antibody titer	Positive at 1:4096
Blood collected at the time of the patient's diagnosis	PCR for babesia	Negative

* PCR denotes polymerase chain reaction.

and his mother suggests that the mother became infected during pregnancy and the patient acquired the infection in utero.[5,7-9]

A Physician: Should the patient's twin receive treatment for babesiosis?

Dr. Luginbuhl: It is not recommended to treat asymptomatic persons who have no evidence of active disease on peripheral-blood smears or PCR tests. The patient's twin was completely well, with a negative peripheral-blood smear and a negative PCR test. Obviously, there was concern about babesiosis, but the plan was to monitor the twin very closely.

A Physician: Was this mother breast-feeding the patient? Could the parasite be transmitted through breast milk?

Dr. Luginbuhl: I believe the patient's mother was breast-feeding, but there is no evidence that babesia can be transmitted through breast milk.

FINAL DIAGNOSIS

Babesiosis, most likely congenitally acquired.

This case was presented at Pediatric Grand Rounds.

No potential conflict of interest relevant to this article was reported.

Disclosure forms provided by the authors are available with the full text of this article at NEJM.org.

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