

Case Records of the Massachusetts General Hospital



Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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Case 15-1997

PRESENTATION OF CASE

A newborn boy was admitted to an intensive care unit because of respiratory depression.

The infant was born to a 28-year-old woman (gravida 2) at 38 weeks' gestation, after an uneventful pregnancy. The parents were from the Cape Verde Islands. The mother's blood group was O, Rh-positive. A test for antirubella antibodies was positive; tests for antibodies to hepatitis B surface antigen and the human immunodeficiency virus, a serologic test for syphilis, and vaginal cultures for chlamydia and gonococci were negative. A healthy daughter had been delivered three years earlier. The mother had eight siblings and seven half-siblings, one of whom had had a cardiac-valve replacement. The baby's maternal grandmother had died in childbirth of "a heart attack and hemorrhage from the mouth" at 43 years of age.

The baby was delivered by cesarean section after an attempt at vaginal birth resulted in fetal distress, with deceleration of the heart rate to 40 beats per minute. The membranes had ruptured 11 hours before delivery, with clear amniotic fluid. The infant cried on delivery and then became apneic and hypotonic but had a normal heart rate and normal perfusion. The Apgar scores were 3 at one minute and 5 at five minutes. Stimulation of the infant failed to alleviate intermittent apnea, and bag-valve-mask ventilation produced minimal improvement in muscle tone and respiratory effort, with grunting and nasal flaring and retraction. The trachea was intubated 10 minutes after birth, and the infant was taken to the newborn intensive care unit.

The temperature was 37.2°C, the pulse was 138, and the respirations were 30. The blood pressure

TABLE 1. HEMATOLOGIC LABORATORY VALUES.

| VARIABLE | ON ADMISSION | 11 HOURS AFTER BIRTH |
|---|--|----------------------|
| Hematocrit (%) | 45.4 | 45 |
| Nucleated red cells (per 100 white cells) | 14 | |
| White-cell count (per mm ³) | 13,500 | 13,500 |
| Differential count (%) | | |
| Neutrophils | 54 | 74 |
| Lymphocytes | 38 | 14 |
| Monocytes | 6 | 12 |
| Eosinophils | 2 | |
| Platelet count (per mm ³) | 267,000 | |
| Blood-smear findings | A few microcytes, macrocytes, acanthocytes, burr cells, and a few oval cells; rare spherocytes | |

TABLE 2. BLOOD CHEMICAL VALUES.*

| VARIABLE | ON ADMISSION | 11 HOURS AFTER BIRTH |
|-----------------------|--------------|----------------------|
| Urea nitrogen (mg/dl) | — | 11 |
| Creatinine (mg/dl) | — | 1.1 |
| Glucose (mg/dl) | 68 | 60 |
| Calcium (mg/dl) | 9.1 | 9.2 |
| Bilirubin (mg/dl) | | |
| Total | — | 4.5 |
| Conjugated | — | <0.5 |

*To convert the value for urea nitrogen to millimoles per liter, multiply by 0.357. To convert the value for creatinine to micromoles per liter, multiply by 88.4. To convert the values for glucose to millimoles per liter, multiply by 0.05551. To convert the values for calcium to millimoles per liter, multiply by 0.250. To convert the values for bilirubin to micromoles per liter, multiply by 17.1.

was 70/55 mm Hg. The infant was 52 cm long, with a weight of 3500 g and a head circumference of 33 cm.

On examination, the infant was limp. His head showed considerable molding; the anterior fontanelle was open and flat. His eyes were open and staring. No dysmorphic facial features were observed. Breath sounds were coarse bilaterally, with air entry greater in the left lung. The heart sounds were normal. The femoral pulses were ++, and the capillary-refill time was two seconds at the chest. The arms and legs were cyanotic but improved during ventilation with oxygen. A grasp reflex was present bilaterally.

TABLE 3. ARTERIAL-BLOOD GAS VALUES ON ADMISSION.

| VARIABLE | VALUE* |
|--|--------|
| Partial pressure of oxygen (mm Hg) | 85 |
| Partial pressure of carbon dioxide (mm Hg) | 22 |
| pH | 7.51 |
| Bicarbonate (mmol/liter) | 17 |

*During ventilation with 30 percent oxygen.

TABLE 4. FINDINGS ON LUMBAR PUNCTURE 11 HOURS AFTER BIRTH.

| VARIABLE | FINDING |
|---|------------------------------|
| Amount of fluid (ml) | 4 |
| Appearance of fluid | Blood-tinged (did not clear) |
| Red cells (per mm ³) | 11,800 |
| White cells (per mm ³) | 32 |
| Differential count (%) | |
| Neutrophils | 79 |
| Lymphocytes | 8 |
| Monocytes | 12 |
| Eosinophils | 1 |
| Nucleated red cells (per 100 white cells) | 3 |
| Glucose (mg/dl)* | 48 |
| Total protein (mg/dl) | 100 |

*To convert the value to millimoles per liter, multiply by 0.05551.

ally; muscle tone was poor in the arms and legs, and the Moro reflex was absent.

Laboratory tests were performed (Tables 1, 2, and 3). An anteroposterior radiograph of the chest showed a right medial basilar pneumothorax. A blood specimen was obtained for culture, and ampicillin, gentamicin, glucose, and water were administered intravenously. Oral intake was withheld. The infant was extubated 10 hours after birth and subsequently breathed room air comfortably, with an oxygen saturation of 100 percent. He remained afebrile. Eleven hours after birth, a seizure began in the left arm and spread to the left leg and subsequently to the right leg and arm, with grunting, increased oral secretions, and questionable mouth movements. No apneic episodes were observed. Blow-by oxygen was given, with no change in the oxygen saturation or respiratory effort. The seizure ended within five minutes after the intravenous administration of lorazepam and phenobarbital. Examination at that time revealed a normal anterior fontanelle and normal heart and lungs. The arms and legs moved symmetrically, with normal muscle tone. Moro, grasp, and patellar reflexes were present. Laboratory tests were performed

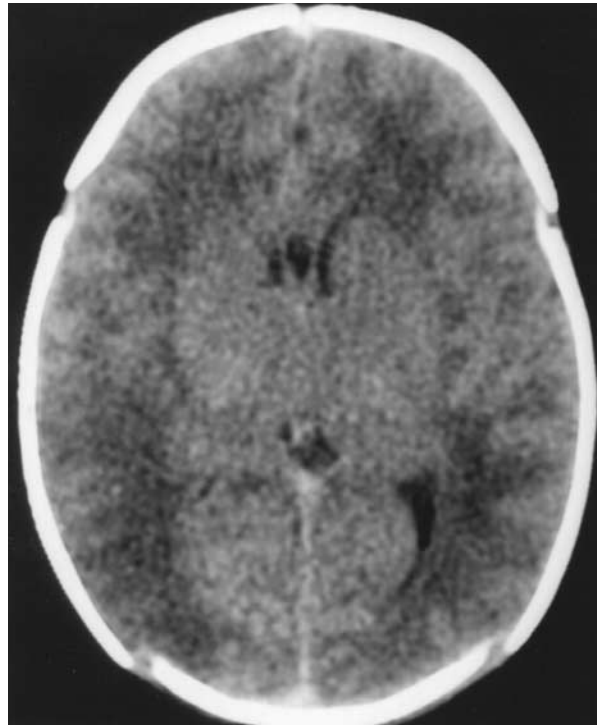


Figure 1. CT Scan of the Brain without Intravenous Contrast Material, Showing an Area of Low Attenuation within the Right Posterior Parietal, Posterior Frontal, and Temporal Lobes — Findings Consistent with an Area of Infarction.

(Tables 1 and 2). The blood culture was negative. A lumbar puncture was performed (Table 4).

A neurologist found that the baby was sedated, with his eyes closed and no crying on stimulation. Movements and muscle tone on the left side were diminished, and ankle jerks on both sides were hypoaffective. A computed tomographic (CT) scan of the brain (Fig. 1), obtained without the administration of contrast material, showed an area of low attenuation in the right posterior parietal lobe and portions of the posterior frontal and temporal lobes — findings consistent with infarction.

A diagnostic report was received.

DIFFERENTIAL DIAGNOSIS

DR. N. PAUL ROSMAN*: This case has four defining features: perinatal distress, postnatal respiratory depression, early left-sided seizures with a right cerebral infarct, and an intriguing family history of heart disease.

During the attempted vaginal delivery, there was fetal distress, with a striking deceleration of the heart

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rate to 40 beats per minute. Early deceleration is thought to reflect compression of the fetal head; late deceleration has been attributed to fetal hypoxemia, and variable decelerations (slowing of the fetal heart-beat with uterine contractions) to compression of the umbilical cord. Despite delivery by cesarean section, this child was hypotonic and apneic after birth. The considerable molding of his head was undoubtedly related to the failed attempt at vaginal delivery. The intracranial pressure was apparently not elevated, because the anterior fontanelle was flat. The absence of dysmorphic changes is evidence against a malformation of the brain, which might otherwise account for the difficulties at birth. The pneumothorax was probably caused by the resuscitation after delivery. The 14 nucleated red cells in the blood indicated subacute or chronic intrauterine bleeding or some other type of intrauterine stress. There was no sign of postnatal bleeding, except for the red cells in the cerebrospinal fluid. The left-sided weakness observed during the last reported neurologic examination, in contrast to the symmetric movements observed immediately after the seizure, may indicate that a second left-sided seizure had occurred, causing diminished left-sided movements or postictal (Todd's) paralysis. This weakness was probably not caused by edema around the brain infarct. It is possible, however, that the left-sided weakness had been missed on the earlier examination. May we review the radiologic studies?

DR. SUSAN A. CONNOLLY: The CT scan (Fig. 1) shows an extensive area of low attenuation within the right parietal lobe, involving portions of the posterior frontal and temporal lobes and suggesting infarction in a middle cerebral arterial distribution.

DR. ROSMAN: I shall first comment on the possibility of central nervous system infection in this case. Almost none of the findings suggest bacterial meningitis. There had not been prolonged rupture of the membranes, the amniotic fluid was clear, there was no evidence of systemic illness, blood cultures were negative, and the cerebrospinal fluid glucose level was normal. The disproportionately large number of white cells in relation to the increased number of red cells (a ratio of 1 white cell to 369 red cells) in cerebrospinal fluid from a presumably nontraumatic lumbar puncture was probably caused by the presence of red cells in the fluid. The slightly elevated protein level can be accounted for by the brain infarct, with the 11,800 red cells per cubic millimeter making a small contribution.

Meningoencephalitis is much more likely than bacterial meningitis, with herpes simplex encephalitis, a frequently devastating disorder in the newborn, a leading consideration. Seizures often occur early in neonatal herpes simplex encephalitis, and the cerebrospinal fluid findings in this case are compatible with that disorder, even though the red-cell count was higher than is usually seen. However, there is lit-

tle other evidence of encephalitis. Although the presence of genital herpes in the mother is frequently unsuspected, skin lesions are usually seen in the child, typically on the scalp and face with a cephalic presentation,¹ and none were reported in this infant. Also, the brain lesions seen on CT scanning in infants with herpes simplex encephalitis are characteristically in the inferior frontal and anterior temporal lobes, unlike the findings in this infant.

Could this child's difficulties at birth have resulted in a hypoxic-ischemic insult sufficiently severe to have caused the cerebral infarct? In a series of 175 cases of focal cerebral infarction in newborns described by Volpe,² no cause was identified in 52 percent of the cases, and perinatal asphyxia was implicated in 33 percent; none of the 12 other causes accounted for more than 2 percent of the cases. Perinatal hypoxic-ischemic brain injury can also be asymmetric,³ as in this case. Possible causes of the asymmetry include an asymmetry of the vessels supplying the two cerebral hemispheres; the asymmetric reactivity of those vessels to a variety of influences, such as the presence of subarachnoid blood, which causes vasoconstriction; and metabolic differences between the two sides of the brain, affecting their susceptibility to ischemia. Although there was subarachnoid hemorrhage in this case, the brain infarct was nonhemorrhagic. However, isolated subarachnoid hemorrhage has been reported in association with perinatal asphyxia.⁴ In a series of 62 asphyxiated full-term infants, 18 had evidence of intracranial hemorrhage on CT scans, with the blood limited to the subarachnoid space in 3 of the infants.⁵ Such subarachnoid hemorrhage has been attributed to the tearing of primitive arteries that overlie the cerebral hemispheres and undergo involution as the fetus approaches full term,⁶ as well as to circulatory failure, with stasis in the superficial circulation, cortical venous infarction, and hemorrhage.⁷ The main factor that is not consistent with hypoxic-ischemic encephalopathy as the primary cause of the cerebral infarct in this case is the absence of any previous problems in the pregnancy that could have caused hypoxic-ischemic encephalopathy. Also, the number of red cells in the cerebrospinal fluid, although compatible with the presence of hypoxic-ischemic encephalopathy, is probably excessive, but I could find no study in which the findings on the CT scan and in the cerebrospinal fluid were compared in such cases. In view of the large amount of molding, the child very likely experienced considerable head trauma before birth, which was probably the main cause of the increased number of red cells in the cerebrospinal fluid. The infarct may have been another factor, for although it did not appear hemorrhagic on the CT scan, the resolution may not have been high enough to show a small hemorrhage. Also, although the unilaterality of the brain lesion, apparently located within a particu-

lar vascular territory, is compatible with the presence of hypoxic-ischemic encephalopathy, this finding does not support the diagnosis. A final reason to rule out perinatal asphyxia as the cause of the infarct in this case is the family history, to which I shall return.

If the infarct was caused by a vascular occlusion, was it venous or arterial? The diagnosis of intracranial venous thrombosis is supported by the presence of blood in the cerebrospinal fluid, since brain infarction complicating venous thrombosis is typically hemorrhagic. Brain hemorrhage without infarction can also result from intracranial venous thrombosis.^{8,9} In this case, the description of the maternal grandmother's death in childbirth is more suggestive of pulmonary embolism with hemoptysis than myocardial infarction. If pulmonary embolism occurred, it probably resulted from deep-vein thrombosis, a leading cause of maternal death during the third trimester of pregnancy. What is the evidence against neonatal intracranial venous thrombosis? First, although seizures are a frequent presenting feature in such cases and can occur on the first day of life, they usually occur later. In 17 cases of neonatal dural-sinus thrombosis, seizures began on the 1st to the 19th day, with a mean age of 4.4 days at their onset.⁸ In addition, 85 percent of the thrombi affect the superior sagittal sinus²; in that circumstance, one would expect to find bilateral rather than unilateral cerebral involvement on CT scans. Also, the CT scan in this case showed no evidence of high signal intensity in the region of the superior sagittal sinus or any other intracranial venous structure — a finding that would have supported a diagnosis of venous thrombosis. Finally, in cases of neonatal intracranial venous thrombosis, CT scans typically do not show parenchymal injury, particularly during the first few days; brain edema is much more characteristic.²

What data favor arterial occlusion as the cause of this child's stroke? First, neonatal cerebral arterial occlusion is not rare. In autopsies on 592 newborn infants, cerebral arterial infarcts were found in 5.4 percent of the infants and in 15 percent of those who were delivered at term.¹⁰ Also, neonatal cerebral arterial infarction is commonly heralded by early seizures; in a series of 50 full-term newborn infants with seizures, it was the second most common cause.¹¹ In addition, more than three fourths of cerebral arterial infarcts in newborn infants are unilateral, and most of the infarcts involve the territory supplied by the middle cerebral artery,¹² as in this case. Although most infarcts are left-sided, I believe the right cerebral involvement in this case is an example of the important minority of infarcts that are right-sided.

Many factors that confer a predisposition to arterial thrombosis in newborn infants were apparently absent in this case, including sepsis, disseminated intravascular coagulation, dehydration, meningitis, homocystinuria, and insertion of central venous and

arterial lines. Congenital heart disorders, such as transposition of the great vessels¹³ and cardiac valvular abnormalities, are important causes of neonatal stroke. Since the findings on an electrocardiogram or an echocardiogram are not mentioned in the case history, I assume that either the findings were normal or the studies were not performed. Occlusion of the middle cerebral artery has rarely been attributed to birth trauma. Although this child doubtlessly had head trauma during the attempted vaginal delivery, the degree of trauma required to cause occlusion of the middle cerebral artery is substantial, as in a case of high-forceps delivery during which "unusual forces were exerted on the head and neck."¹⁴ Maternal cocaine use should also be considered as a cause of this child's stroke, and urine testing for cocaine and its metabolites could have been the diagnostic test in this case. Maternal cocaine use has been linked to small body size, including head size, in newborn infants, as well as to neonatal stroke.¹⁵ Although this infant's head was smaller than average, the child was not small for his gestational age, he was not jittery, and his neonatal reflexes were not overactive. Also, a recent study of 39 newborn infants who had been exposed to cocaine in utero showed that the cranial ultrasonographic findings in these infants did not differ from those in controls, and there were no cases of neonatal stroke.¹⁶

What is the possible relation between the respiratory depression and the brain infarction in this case? I believe that the infarction occurred in utero, followed by postnatal respiratory depression, which is a frequent sequence. In a large clinical and radiologic study of congenital and childhood stroke, resuscitation at birth was required in six of seven infants with infarction from occlusion of a single artery and seizures.¹⁷ Also, the CT findings in this case provide much stronger support for infarction in utero than for perinatal infarction, since decreased attenuation in a vascular distribution becomes apparent on a cranial CT scan only several days after an arterial stroke in a newborn infant.¹⁸ Thus, this child's perinatal respiratory depression was probably caused by an intrauterine stroke due to vascular occlusion involving the territory supplied by the right middle cerebral artery.

What caused the occlusion? Most such occlusions appear to be embolic, as reported in one series of 32 neonatal cerebral arterial infarcts studied at autopsy.¹⁰ Nonetheless, the evidence for embolic occlusion is inferential rather than direct in many, if not most, reported cases. The findings on the CT scan in this case support a diagnosis of embolism, because although there was involvement in much of the territory supplied by the right middle cerebral artery, the anterior parietal region was spared. The findings suggest that an embolus to the right middle cerebral artery may have broken up, with fragments occluding more distal portions of the arterial tree.

There are several potential sources of emboli in this case. In the absence of clinical, electrocardiographic, or echocardiographic evidence of heart disease, a cardiac source is unlikely. The ductus arteriosus is another potential source of emboli in any case of in utero stroke. In this case, however, it is more likely that emboli originated from thrombosed veins within the placenta.^{19,20} Such emboli would enter the umbilical vein, flow to the right side of the heart, pass through a patent foramen ovale into the left side of the heart, enter the aortic arch, innominate artery, and right common carotid and internal carotid artery, and then proceed to obstruct the right middle cerebral artery. Why should venous thrombosis within the placenta, the putative source of emboli, have occurred in this case? I again turn to the family history.

The child's maternal grandmother probably died from a pulmonary embolus that arose from deep-vein thrombosis. Was she particularly susceptible to such a complication? During the past several decades, a growing number of disorders, both genetic and acquired, that lead to hypercoagulability of blood and vascular thromboses have been recognized. The disorders most frequently linked to venous and arterial occlusions in children are deficiencies of antithrombin III, protein C, and protein S and the presence of antiphospholipid antibodies (lupus anticoagulant or anticardiolipin antibodies).²¹⁻³⁰ Recently discovered and seemingly most important, however, is a mutation in the factor V gene that causes an inherited resistance to the anticoagulant function of activated protein C.³⁰ When these disorders of coagulation are genetic, they have an autosomal dominant pattern of inheritance. All of them are associated with deep-vein thrombosis and thromboembolic disease, but their manifestation from generation to generation may be extremely variable. Thus, one may find deep-vein thrombosis with pulmonary embolism in a grandparent, no thrombotic disorder in a parent, and intracranial vascular occlusion in a child. An inherited resistance to activated protein C has been found in 2 to 5 percent of the general population and in 40 to 50 percent of patients with thromboembolic disease. This latter frequency is at least 10 times that of antithrombin III, protein C, or protein S deficiency.³⁰

It is almost impossible to make clinical distinctions among the various disorders resulting in hypercoagulability. Even using the Internet, I could not find a connection with the Cape Verdean origin of this child's parents. Not only are these disorders very similar clinically, but also two or more of them often coexist. In addition, blood levels of protein C and other coagulation inhibitors can normally be low in the newborn child, increasing the difficulty of establishing a diagnosis.

A genetic hypercoagulable state can account for

all the features of this case. Such a disorder could have caused thromboembolism in the child's grandmother and intracranial vascular thrombosis in the child. It is perhaps even more likely that such a disorder could have favored the development of placental-vein thrombosis, which then served as a source of cerebral embolism in the child.

The diagnostic report received in this case could have been a pathological study of the placenta, showing the presence of venous thrombi, from which one could infer that emboli formed and caused the child's stroke. Placental vascular thrombosis could also explain the increased number of nucleated red cells in the child's peripheral blood. Thrombosis of placental vessels is a common incidental finding, although if present in this case, it was probably pathogenetically important. My second choice for the diagnostic report is the result of a blood assay, indicating a disorder conferring a predisposition to hypercoagulability, such as a deficiency of protein C, protein S, or antithrombin III in the infant or the presence of antiphospholipid antibodies in the infant, his mother, or both.

DR. ROBERT M. INSOF: We also favored a diagnosis of an embolic event, most likely occurring in utero. When no evidence of infectious cause was found, we tested the infant for factors associated with hypercoagulation. Protein S, protein C, and all the coagulation factors tested were normal.

CLINICAL DIAGNOSIS

Intrauterine stroke.

DR. N. PAUL ROSMAN'S DIAGNOSIS

Intrauterine stroke due to occlusion of the right middle cerebral artery, probably caused by emboli from placental veins.

PATHOLOGICAL DISCUSSION

DR. DRUCILLA J. ROBERTS: The pathological findings in the placenta made up the diagnostic report. Gross examination showed multiple yellow-white, soft, irregular lesions, 5 to 10 mm in diameter. Histologic examination revealed abscesses (Fig. 2), in which the intervillous (maternal) space was filled with a predominantly neutrophilic infiltrate mixed with debris and fibrin. Inflammatory cells were also present within the villous stroma and its vessel walls (Fig. 3). Some vessel walls were destroyed. Unattached and organizing thrombi were both present within the chorionic-villus vessels (Fig. 4 and 5). The uninvolved villi appeared normal. There was no acute chorioamnionitis.

The differential diagnosis of acute villitis with intervillitis includes infections, which are usually bacterial. These infections reach the placenta through the maternal circulation or the cervix. The classic placental infection associated with microabscesses and

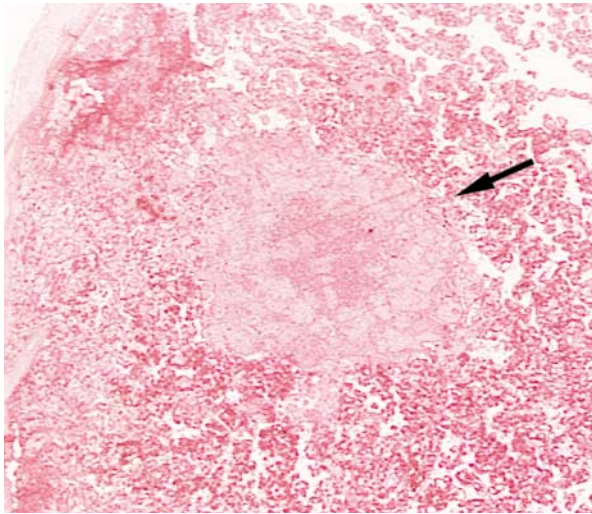


Figure 2. Placental Microabscess (Arrow) (Hematoxylin and Eosin, $\times 55$).

acute villitis is due to *Listeria monocytogenes*, although it is usually accompanied by acute chorioamnionitis.^{31,32} Campylobacter and streptococcal infections have been reported to result in similar histologic findings.^{33,34} Special stains, including immunohistochemical stains, were used to identify the organisms in this case. *Listeria* is often difficult to identify in tissue with Gram's stains and in this case was not identified with the use of the Brown and Hopps stain. Dieterle's stain, however, showed numerous small, rod-shaped and curved organisms (Fig. 6). These organisms were not present within the chorionic villi, villous thrombi, or villous vessels. The morphologic changes strongly supported the diagnosis of listeria infection.

Until recently, there was no method to identify such organisms except culture. Investigators at Yale University School of Medicine have evaluated a polyclonal antibody that reacts with the O antigen of listeria serotypes 1 and 4 and can be used on paraffin-embedded tissues.³⁵ The antibody does not appear to cross-react with campylobacter or streptococcal species in tissue sections. In this case, immunohistochemical staining of the organisms was positive for listeria (Fig. 7).

Listeria is a ubiquitous saprophytic organism that grows at a wide range of temperatures and even in the refrigerator. It infects humans and a variety of animals. Fecal carriage is common in humans, but infection is very rare and usually restricted to immunocompromised persons or pregnant women. The infection is generally traced to contaminated foodstuffs, primarily unpasteurized milk and milk products and unwashed vegetables fertilized with raw manure.^{36,37} Infections in pregnant women are most often sporadic, but epidemics have occurred.^{38,39} Preg-

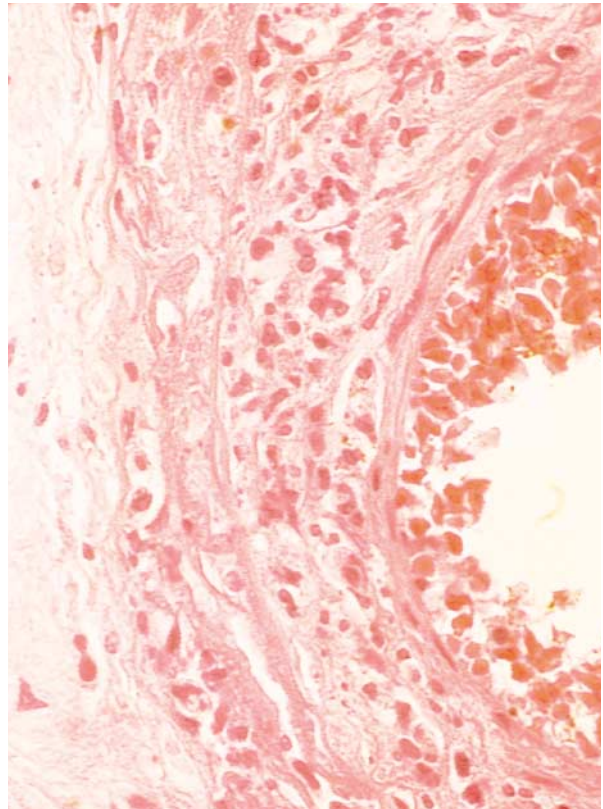


Figure 3. Acute Inflammation of the Vessel Wall within a Chorionic Villus (Hematoxylin and Eosin, $\times 240$).

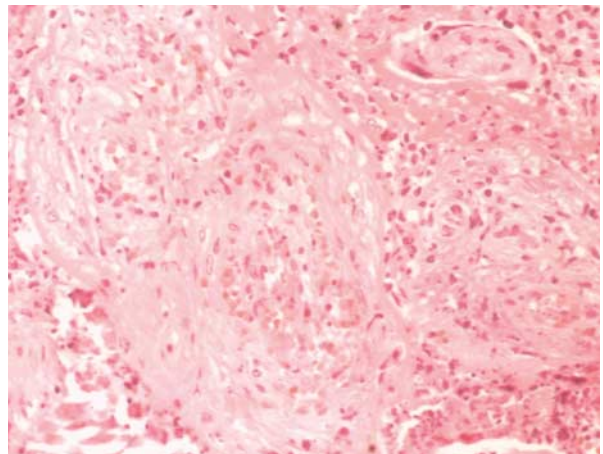


Figure 4. Organizing Intravascular Thrombus in a Stem Villus (Hematoxylin and Eosin, $\times 90$).

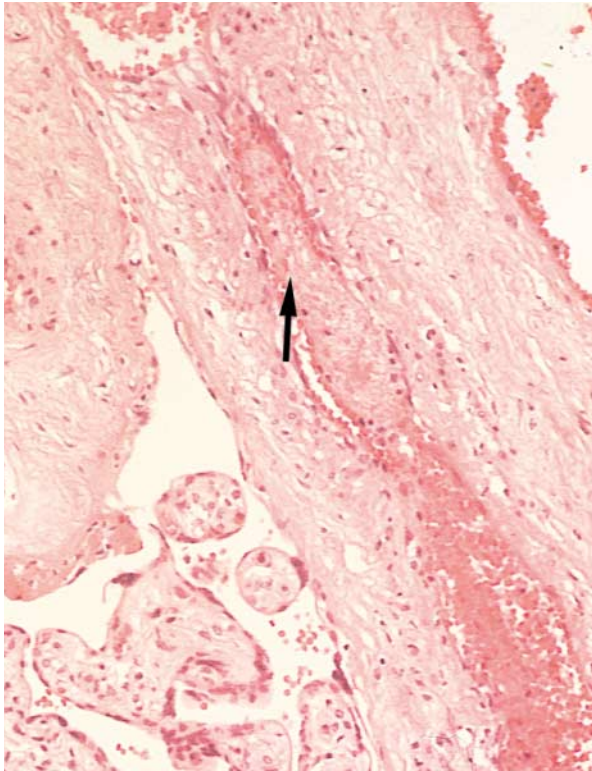


Figure 5. Unattached Thrombus in a Stem Villus (Arrow) (Hematoxylin and Eosin, $\times 70$).

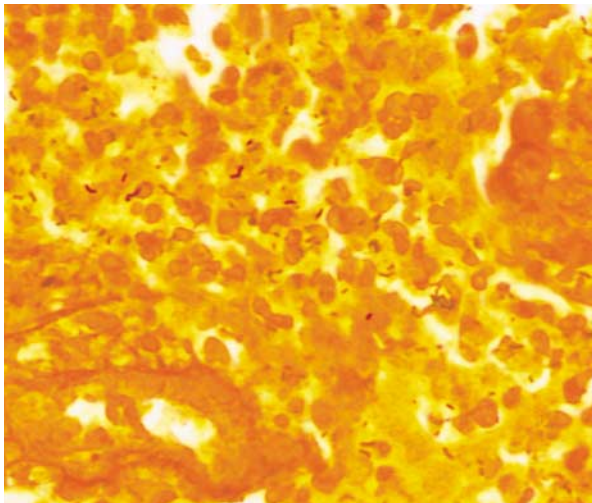


Figure 6. Darkly Stained *Listeria* Organisms in Placental Abscess (Dieterle's Stain, $\times 220$).

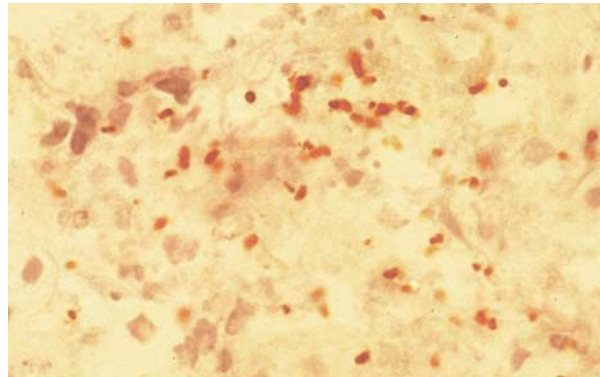


Figure 7. Whole and Fragmented Red-Stained *Listeria* Organisms in Abscess (Hematoxylin and Eosin, $\times 355$).

Immunohistochemical staining was performed with a polyclonal antibody directed against listeriolysin O antigen.

nant women are often asymptomatic, but they may be febrile. The organism has been isolated from stool, vaginal, and blood specimens, indicating that the route of placental infection can be hematogenous or can ascend from the vagina. Pregnancy-associated listeriosis is fatal in 27 percent of infected infants, but not all placental infections result in congenital infections.^{39,40} Even among infants who are not infected, there is a very high rate of neurologic compromise.⁴⁰

Placental chorionic vascular thrombi are rare but probably underdiagnosed.⁴¹ They have been reported in association with maternal coagulopathy,^{42,43} but fetal inheritance of the coagulopathy has not been demonstrated. Placental chorionic vascular thrombosis may have severe neurologic sequelae.^{41,44,45}

We have concluded that the congenital stroke in this infant was probably due to an embolic event that originated in the placental vessels as a result of their destruction by listerial microabscesses. Since organisms were not found in the thrombi and there was no clinical evidence of sepsis in the infant, the thromboembolus was probably not septic.

DR. INSOF: The baby was treated with a full course of antibiotics, even though cultures showed no evidence of any infection. He was discharged while receiving phenobarbital and has had no recurrent seizure activity. His neurologic status is normal.

DR. ROSMAN: The last case I discussed at one of these exercises was that of a progressive brain-stem syndrome in a child. Given the information that the CT findings were normal, I did not review the images before presenting my differential diagnosis. When the CT scan was subsequently shown, an enlarged pons was visible, which was found at autopsy to have resulted from a pontine astrocytoma. Thus, when I was asked to discuss this case, I decided to review the films beforehand; regrettably, although

the findings were reported correctly, they did not help me establish the diagnosis. If I am ever asked to discuss another case, the question of whether to review the radiographs will remain.

ANATOMICAL DIAGNOSES

Placental vascular thrombosis due to listeria infection.
(Cerebral embolism and infarction.)

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