mal jaundice, coupled with normal liver enzyme values, and the gradual disappearance of antibody levels in our study indicate that passive transfer of hepatitis B and C antibodies occurred after IVIG infusion. The negative HCV RNA result in two infants also was reassuring that no active HCV infection was present.

For best interpretation of serologic results for certain infectious agents, administration of blood products, including IVIG, should be considered. Because IVIG contains IgG predominantly, assays that evaluate other classes of antibodies, such as IgM, may be useful in the evaluation of patients for acute onset of an infectious disease.

REFERENCES


Prenatal gastrointestinal bleeding caused by esophagitis and gastritis

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We report a case of a prenatal esophagitis and gastritis revealed at 33 weeks of gestation by the presence of bloody amniotic fluid and dilated intestinal loops that was confirmed by endoscopy at birth. Complete recovery occurred after treatment with ranitidine. (J PEDIATR 1994;125:465-7)
report a case in which gastrointestinal bleeding occurred in a fetus 2 weeks before birth.

CASE REPORT

A premature boy was born to a 26-year-old healthy Tunisian mother (gravida 2, para 2). The pregnancy was uneventful until 33 weeks of gestation. There was no evidence of maternal stress, and no drug therapy (especially nonsteroidal antiinflammatory agents or corticosteroids) was given during the pregnancy.

At 33 weeks of gestation, fetal ultrasonographic scanning revealed dilated intestinal loops with polyhydramnios and echogenic amniotic fluid. Amniotic puncture yielded brown bloody fluid (the Kleihauer test was not performed).

Two weeks later, delivery was induced because of a suspected intestinal abnormality and polyhydramnios. A boy weighing 2080 gm was born after normal cephalic presentation. The Apgar scores were 9 at 1 minute and 10 at 5 minutes. Tracheal and gastric aspirates obtained brown, bloody fluid. The result of clinical examination at 2 hours of life was normal but stools were bloody. The heart rate was 117 beats/min, and the blood pressure was 54/36 mm Hg. Vitamin K1, 10 mg, was administered intravenously.

Laboratory studies showed the following: severe anemia (hemoglobin, 114 g/L [11.4 g/dl]); hematocrit, 0.35; erythrocyte count, \(17 \times 10^6/\)L (17,000/mm³); reticulocyte count, \(164 \times 10^6/\)L (164,000/mm³); and platelet count, \(177 \times 10^9/\)L (177,000/mm³). The infant’s hemostasis status was normal (fibrinogen, 3.9 gm/L; factor II, 60%; factor VII + X, 73%; and factor V, 164%). Results of sequential studies for bacterial sepsis were negative, and stool examination by agglutination of dry spot latex (Diarlex; Orion Diagnostica, Espoo, Finland) did not detect rotavirus and adenovirus. The Kleihauer test, performed on a sample of amniotic fluid at birth, showed that 98% of the erythrocytes were of infant origin. The chest X-ray study showed patchy areas of alveolar consolidation with focal overinflation. Results of the abdominal x-ray study and ultrasonographic examination were normal.

A 40 ml transfusion of packed erythrocytes was given to the infant, and feeding was withheld until endoscopy was performed at 2 days of life (Olympus GIF XP 20). Diffuse hemorrhagic gastritis and esophagitis of the lower third of the esophagus were found, with no ulceration. Intravenous treatment with ranitidine (10 mg/kg per day) and cisapride (0.8 mg/kg per day) was then started. Hematemeses and bloody stools stopped within 2 days. Feeding with a hydrolysate formula (Alfaré) was started at day 3 and was well tolerated.

When the infant was 21 days of age, findings of follow-up endoscopy were normal. Ranitidine therapy was stopped at the age of 30 days, and 24-hour pH monitoring revealed acid reflux, with pH <4 for 17% of the total time. On day 36 the serum gastrin level (148 pg/ml) was normal for age.4,5

DISCUSSION

Upper digestive tract hemorrhage is not uncommon in the early neonatal period.1,2 In the infant we describe, gastrointestinal bleeding was caused by diffuse hemorrhagic gastritis associated with esophagitis. Prenatal bleeding was suspected because of the ultrasonographic findings at 33 weeks of gestation and was confirmed by amniotic fluid examination. Other maternal or placental causes of bloody amniotic fluid (placenta previa, abruption placenta, hematoma of the placenta, and rupture of the umbilical cord) were ruled out by ultrasonographic and clinical findings.

Fetal hemorrhage of this type has rarely been reported.3,7 Wen et al.3 reported a case of fetal gastric ulcer diagnosed during delivery because of bloody amniotic fluid. The endoscopic appearance of the gastritis and esophagitis in our patient was not different from that usually described in the newborn infant with gastrointestinal bleeding.8

Cytomegalovirus infection cannot be ruled out in our patient because blood, urine, and gastric fluid cultures were not done in the neonatal period.9 Gastric biopsy to detect Helicobacter pylori was not performed, but the presence of this agent is very unlikely, because it is predominantly transmitted by the oral route.10 Results of testing for rotavirus, adenovirus, and bacterial pathogens were negative.

Cow milk protein allergy has been suggested as a factor in the pathogenesis of neonatal gastritis,8 but prenatal bleeding cannot be explained in this way, even though transplacental passage of native cow milk protein has been proposed as a possible mechanism in neonatal colitis.11

Gastroesophageal reflux was studied by 24-hour pH monitoring after antisecretory treatment and yielded an abnormal acid reflux index.12 Prenatal peptic esophagitis seems unlikely, because of continuous fetal swallowing of amniotic fluid (which has a neutral pH). However, alkaline duodenogastric reflux cannot be ruled out. Various studies have shown the existence of physiologic hypergastrinemia in the first few days of life,4,5 which may be responsible for hyperacidity in the esophagus and stomach.13 In our patient, the serum gastrin level, measured only at the age of 38 days because of prior ranitidine therapy, was normal. However, fetal hypergastrinemia could have affected the prenatal gastritis. Litchenberger et al.14 demonstrated that hypergastrinemia can be related to high concentrations of volatile amines in the amniotic fluid.

To our knowledge, this is the first reported case of proven prenatal severe gastritis and esophagitis. Further investigations are required to determine the cause.

REFERENCES

Maternal mild hyperphenylalaninemia: Results of treated and untreated pregnancies in two sisters

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The outcomes of mild hyperphenylalaninemia (MHP) in three children of two sisters were compared. The IQ of the child from an untreated pregnancy was 105; the developmental quotients of the two infant offspring from treated and untreated pregnancies were 122 and 114, respectively. The IQ of the sister with untreated MHP was 101; that of the sister who received dietary treatment for MHP during infancy was 90. Thus MHP and maternal MHP appear to have been clinically inconsequential in this family. (J Pediatr 1994;125:467-9)

Maternal phenylketonuria has severe teratogenic implications, including microcephaly, mental retardation, congenital heart disease, and intrauterine growth retardation. Treatment with a phenylalanine-restricted diet that controls the maternal phenylalanine levels during pregnancy, particularly if begun before conception, seems to modify and may prevent the fetopathy. Thus an increased phenylalanine concentration in the mother, even more concentrated in the fetus, or a phenylalanine metabolite, seems to be the teratogenic factor. However, many of those with persistently elevated blood...