Polycythemia as a cause of necrotizing enterocolitis

To the editor: Many conditions have been reported to be causally related to the occurrence of necrotizing enterocolitis in neonates. Recently we treated a baby girl with necrotizing enterocolitis caused by polycythemia.

The infant was born to a primiparous 26-year-old woman whose uneventful pregnancy terminated at the 39th week. The infant weighed 2098 g. She was the second and smaller twin and was delivered vaginally as a breech presentation. At birth she was flaccid and blue, requiring resuscitation with oxygen by bag and mask. Her length was 46 cm and her head circumference, 31 cm; both measurements were, like her birth weight, below the 3rd percentile.

At 4 hours of age she was transferred to the neonatal intensive care unit of the Montreal Children's Hospital. On arrival she was plethoric, jittery and hypertonic. Her blood glucose value was 29 mg/dL. Intravenous administration of 10% glucose in water resulted in substantial improvement of her symptoms. Her hemoglobin value was 21.5 g/dL and the venous hematocrit, 76%. Early oral feedings with formula supplying 20 Cal/oz (3 kJ/mL) were well tolerated, and 10 feeds had been given by 34 hours of age, when she began to vomit. Four hours later she was lethargic and had slight periumbilical erythema. Her abdomen was soft and not distended; bile-stained fluid was obtained by suctioning the stomach. Abdominal radiographs showed pneumoperitoneum with pneumatosis intestinalis. There was by now obvious cellulitis of the left abdominal wall. The hematocrit was now 85%; the leukocyte count, 3.3 x 10^9/L; and the platelet count, 92.0 x 10^9/L.

Laparotomy disclosed multiple perforations of the necrotic hepatic and splenic flexures of the colon. Subtotal colectomy was performed. Culture of peritoneal fluid produced a growth of *Enterococci.*

Her postoperative course was in keeping with generalized abdominal infection and ascending cholangitis. On the 2nd postoperative day focal seizures involving the right arm occurred. These were controlled with phenobarbital. Multifocal epileptogenic abnormalities were evident on electroencephalograms. Subsequent examinations showed gradual improvement in this manifestation.

At 16 weeks of age a colocolic anastomosis was performed. Parenteral therapy with gentamicin and ampicillin was continued for a total of 20 weeks; three attempts to discontinue antibiotic therapy during this period were followed by fever and clinical deterioration. At 6 months of age the child's growth was catching up.

Pathologic examination of the surgical specimen revealed several perforations of the colon and many areas of ulceration and erosion of the mucosa. Microscopic examination showed that some areas were completely necrotic; in others the mucosa was infiltrated with polymorphs. In the submucosa, areas of gas infiltration were seen. Many vessels in the muscular layers were partially or completely obliterated by recent thrombi.

In the neonate an increased hematocrit is the most important factor causing increased blood viscosity. When the hematocrit rises above 72% the increase in viscosity is exponential.[1] Blood viscosity increases as the rate of shear decreases; hence, in the microvasculature sludging occurs when there is slow flow, with resulting impairment of tissue oxygenation and a tendency to microthrombus formation.[2] In our patient the extreme polycythemia may have been related to the infant's being small for gestational age. The neonatal asphyxia, which perhaps contributed to the initial ischemic insult to the bowel, may also have resulted in increased polycythemia.[3]

If any of the symptoms and signs reported as being associated with polycythemia in the neonate, such as plethora, cyanosis, hypoglycemia, cardiomegaly, respiratory distress,[4] or central nervous system manifestations of lethargy, jitteriness or seizures, had persisted, we would have treated our patient by partial exchange transfusion.

This experience suggests that, even if symptoms are not present, early partial exchange transfusion with plasma or an appropriate electrolyte solution is advisable in newborn infants with polycythemia to avoid the serious complications of hyperviscosity.[5]

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References

1. GROSS GP, HATHAWAY WE, MCGAUGHAY HR: Hyperviscosity in the neonate. *J Pediatr* 82: 1004, 1973
2. DIETERFASS L: Blood viscosity, internal fluidity of the red cell dynamic coagulation and the critical capillary radius as factors in the physiology and pathology of the circulation and micro-circulation. *Med J Aust* 1: 688, 1968
3. PHILIP AGS, YEE AB, ROSY M, et al: Placental transfusion as an intra-uterine phenomenon in deliveries complicated by foetal distress. *Br Med J* 2: 11, 1969
4. GATTI RA, MUSTER AJ, COLE RB, et al: Neonatal polycythemia with transient cyanosis and cardiorespiratory abnormalities. *J Pediatr* 69: 1063, 1966
5. LEAKE RD, THANOPOULOS B, NEIBER R: Hyperviscosity syndrome associated with necrotizing enterocolitis. *Am J Dis Child* 129: 1192, 1975

Oropharyngeal gonorrhea: disseminated gonococcal disease

To the editor: Weisner and colleagues[1] described a population of heterosexual males with disseminated gonococcal disease (DGD) in whom only cultures from oropharyngeal swabs were positive for *Neisseria gonorrhoeae.* This suggests that, on occasion, the throat may serve as the source of disseminated disease. We add to their observations the results of a study of the incidence of oropharyngeal gonorrhea in our clinic population and a review of all cases of DGD in our teaching hospitals in the past 3 years.

During a recent investigation of the effectiveness of an oral probenecid–amoxicillin suspension in the treatment of uncomplicated gonorrhea, throat swabs were taken routinely before treatment in an attempt to isolate *N. gonorrhoeae.* One heterosexual male had positive cultures from both urethral and throat swabs. Two urethral swabs obtained after treatment gave negative cultures; unfortunately swabs of the throat were not taken on this occasion. Two weeks later the patient presented with the classic stigmata of DGD[2] but admitted no sexual re-exposure. A urethral swab was obtained and the patient given probenecid and procaine penicillin intramuscularly, followed by a 7-day course of ampicillin given orally. Cultures of the pretreatment urethral swab as well as the post-treatment urethral and throat swabs were negative for *N. gonorrhoeae.*

Review of inpatient charts at Victoria, University and St. Joseph's hospitals, London, Ont. revealed 12 cases of DGD — 9 in females and 3 in males. Throat swabs had been taken prior to therapy in seven cases; cultures were positive for *N. gonorrhoeae* in three — two heterosexual males and one female. In the female, cultures of all material tested (blood, joint aspirate, and endocervical and throat secretions) were positive; in one male both blood and throat secretions, and in the other only throat secretions yielded positive cultures.

This is a small series; nevertheless it is impressive that in three of four heterosexual males seen locally the throat was the probable source for DGD. In conjunction with data from the study by Weisner and associates[1] this suggests that the throat may indeed be a major source for DGD, as it is for disseminated meningococcal disease, and indicates the importance in any suspected case of DGD of taking a throat swab before treatment to attempt to culture *N. gonorrhoeae.*

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References

1. WEISNER PJ, TRONCA E, BONIN P, et al: Clinical spectrum of pharyngeal gonococcal infection. *N Engl J Med* 288: 181, 1973
2. HOLMES KK, COUNTS GW, BEATY NM: Disseminated gonococcal infection. *Ann Intern Med* 74: 919, 1971