Method for Preventing Neonatal Septicemia, the Leading Cause of Death in the Neonatal Foal

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Septicemia remains the leading cause of death in neonatal foals. The open gut is considered the likely mechanism for bacterial exposure. Methods that allow early gut closure may be as important or more important than serum IgG in preventing neonatal bacterial infection. Author’s address: Dept. of Medicine and Epidemiology, School of Veterinary Medicine, University of California, Davis, CA 95616. © 1997 AAEP.

1. Introduction

After operating the neonatal intensive care unit at the University of California for over 10 years, I have observed a large number of septic foals. As with field studies of causes of illness and death in foals less than 7 days of age, the leading cause of illness upon admission in our neonatal unit is bacterial infection (septicemia that is predominantly due to gram-negative organisms). This is despite a number of studies indicating that passive transfer failure and low serum IgG are the cause of most of these neonatal deaths and subsequent efforts to raise IgG levels in newborn foals. However, I now do not believe that low IgG is the cause of this problem, nor have our efforts to raise IgG by various means seemed to have made a great deal of difference in the incidence of septicemia over the past 10 years based on presentation of cases to our hospital.

For years field veterinarians have been indicating that good management is more important than absolute amounts of IgG. The purpose of this paper is to define what components of good management result in a lower incidence of infections, even with low IgG in foals on some farms. This paper discusses the early research on the immunology of the newborn and observations from field epidemiologic studies and colostrum deprivation studies as they related to the route of infection and a method for the prevention of septicemia.

2. Materials and Methods

A. Immunology Studies

Let’s take a look at the pioneering research on passive transfer done over 20 years ago by Jeffcott.10,11 His research demonstrated the indiscriminate active absorption of large molecules (including IgG) by the open gut shortly after birth. Unfortunately this avenue for the major route of exposure of the foal to pathogens was underestimated or ignored. Dr. Jeffcott demonstrated that cells that line the newborn gut will nonspecifically ingest various large molecular weight compounds (via pinocytosis) and not just immunoglobulin. Additionally, the lack of tight junctions between gut barrier cells allowed >70,000 molecular weight molecules to pass freely into the lymphatics and circulate among cells. When these cells were used up, the gut assumed
its normal structure and no further absorption of large molecules could take place. Dr. Jeffcott failed to address the potential absorption of bacteria across this open gut and its role in acquiring bacterial bloodborne infection. Twenty years later Dr. Jeffcott remained puzzled by the industry confusion among farm management regarding failure of passive transfer and the need to measure IgG in foals. Several studies have indicated foals with only 200 mg/dl IgG at 24 h of age don’t get sick on some farms. The reason farm management does not always treat a low IgG level could be that conditions that favor the foal’s rapidly ingesting colostrum or milk of any kind have seemed more important than a specific IgG level in the foal.

B. Field Studies
Several years ago I was involved with an outbreak of neonatal salmonellosis on a large Thoroughbred farm. The foals were born healthy and had >800 mg/dl IgG (often >2000 mg/dl). However, they still became infected by 24–48 h when foaling in a clean barn, with clean feed and water and sterile personnel. We performed 2860 Salmonella cultures during the course of that outbreak. Mares were found to be asymptptomatically shedding low numbers of Salmonella Ohio obtained from contaminated feed of a brood mare mix. When the mares defecated during stage two labor they contaminated the afterbirth, which contaminated the perineum. During udder seeking the foals ingested the Salmonella prior to obtaining any colostrum and S. Ohio bacteremia resulted. This was clue number one regarding the open gut as an immediate access for bacteria despite IgG levels.

Clue number two came from colostrum deprivation models in foals where, despite rigorous hygiene, the foals became bacteremic and most died despite therapy. The foals were allowed 30–60 min of bonding, which consisted of udder seeking, perineum licking, and so on. They were then removed from the mares and promptly became bactereemic with the usual organisms such as Escherichia coli. Many other colostrum deprivation studies have had similar results, and lack of IgG has been inappropriately implicated as the sole cause. Exposure of the foals to pathogenic bacteria during udder seeking appeared to be the principal route of infection and demonstrated the magnitude of bacterial exposure during this udder seeking activity.

Clue number three came on a trip to the International Conference on Equine Infectious Disease in Tokyo. During a tour of a facility used for Rhodococcus equi studies, I was brought to a stable where colostrum deprived foals were born. For the past 2 years, 12 foals per year were raised with no illness. I inquired as to the method and found that the mares foaled in dirt floor, straw bedded barns. I inquired as to antibiotic use: one shot of procaine penicillin and disinfection of the navel (betadine); in addition, milk replacer was used for 24 h (cow’s milk purchased at a market).

None of these procedures would have an influence on gram-negative bacteremia. (I had just finished a study indicating betadine was worse than nothing at disinfecting the navel.) Upon further questioning I found that immediately following delivery the foal was moved to the next stall adjacent to the mare. No mare contact occurred. Prior to rising, the foal was fed as much cow’s milk as it wanted from a bottle and continued to be fed upon demand for 24 h, whereupon the foal was returned to the mare that had been milked out of colostrum. In my opinion, the procedure that was effective in this system was the rapid closure of the open gut and lack of udder seeking and the associated bacterial absorption across the open gut. In the other two scenarios of the Salmonella outbreak and colostrum deprivation, the foals ingested pathogenic organisms during udder seeking, which crossed the open gut directly into the blood stream to produce septicemia. The effectiveness of rapid gut closure even without IgG is demonstrated in that model.

Clue number four came with a lecture on foal septicemia in England. At a farm with good management and routine use of antibiotics for 72 h after birth, the incidence of septicemia was 0.3%. Routine use of antibiotics had been in place for 20 years on this farm. This demonstrated the relative safety of routine antibiotics as well as an extremely effective program at preventing septicemia.

3. Discussion and Hypothesis
My hypothesis is that the early administration or ingestion of colostrum is associated with reduced illness in foals largely because of early (rapid) gut closure and the prevention of absorption of bacteria across the gut wall. Conditions leading to delayed gut closure allow bacteria to cross the open gut, resulting in septicemia in many cases. Likewise, exposure to pathogenic bacteria during prolonged udder seeking, or environmental licking or ingestion of bacteria by the foal as a result of delayed feeding or nursing, are additional methods for bacterial presentation to the open gut. It is suggested that this is the principal route of infection for neonatal septicemia in foals and other farm animals that are dependent on an open gut for passive transfer.

This means that conditions that may be associated with delayed gut closure, such as neonatal maladjustment syndrome (birth hypoxic encephalopathy), prematurity, dystocia, musculoskeletal problems, being weak at birth or being a twin, and so on, would lead to significant incidences of septicemia, which they certainly do. Thus a foal with high IgG could be a marker for well being based on rapid and early feeding prior to bacterial access to the foal across the open gut. Additionally this would explain healthy foals that stood and nursed vigorously but did not become ill despite low serum IgG. Delayed nursing and early exposure to pathogens (prior to any colos-
trum) are the key factors in risk of infection in this hypothesis. If correct it will allow therapy to prevent the leading cause of death in foals less than 7 days of age.

From these hypotheses and the study from England regarding short-term antibiotics and control of the Salmonella outbreak described above, it is my opinion that a short course of parenteral broad-spectrum antibiotics beginning at 6–12 h of age is a prudent and potentially effective means to treat exposure from bacteria across the gut wall in the neonate. It is suggested that foals that do not have an observed birth be considered exposed to pathogens and receive a 48–72 h course of antibiotic therapy.

4. Method for Preventing Septicemia

1. Keep the mare in the facilities in which the foaling will take place to allow production of antibodies in the mare to pathogens within the local area. Clean the foaling stalls twice daily and disinfect the stalls prior to use. Wash the mare daily to reduce bacterial buildup on the hair coat and perineum from stall housing.

2. Immediately following delivery, prevent the foal from contacting the mare until steps 3 and 4 are completed.

3. Wash the mare after foaling with large volumes of soap and water to remove bacteria around the perineum and udder and rear quarters where the foal may contact fecal bacteria during udder seeking. Dry the mare.

4. Milk 2–4 oz of colostrum (preferably greater than 1060 specific gravity) from the mare's cleaned mammary gland and bottle feed the foal prior to the foal rising and upon obtaining a suck reflex. Use colostrum from a colostrum bank if necessary.

5. If the foal is weak, tube feed the foal within 1 h of birth with 6–8 oz of colostrum, or if none is available use mare milk replacer; if none of that is available, use cow's milk. In orphan foals, continue feeding from a bottle or pan until 10% of body weight is fed. Feed when the foal is hungry.

6. In any foals without an observed birth and all the above precautions, begin parenteral antibiotic therapy within 6–8 h of birth and treat for 48–72 h only. Longer treatment may produce antibiotic resistance and should be reserved for ill foals. The choice of antibiotic therapy will vary with the area. If you have nothing to go on in your area, try using procaine penicillin 20,000 units/kg IM and gentocin 6.6 mg/kg IM. Both of these are given once daily. (Procaine penicillin given once daily gives 16 h of penicillin blood levels, which is adequate for most gram-positive organisms in these circumstances.)

7. This year when you get a call about a weak newborn foal and it will be hours before you see it, make arrangements for your staff to get the antibiotics to the owner immediately if there is likely to be any delayed gut closure from delayed nursing. Then proceed and establish your doctor–client–patient relationship and work the foal up while it is on antibiotics.

For those of you afraid of aminoglycoside antibiotics in foals, pull a serum creatinine before and after administration if you so desire. I have administered aminoglycosides to over 500 neonatal foals in our neonatal unit to date, with two aminoglycoside toxicities. We keep the ill foals hydrated and that is most important. In a bright, alert foal receiving short-term antibiotics, this should be considered a relatively safe treatment.

5. Conclusions

It appears that good management for preventing infections are clean stalls, clean mares, factors that aid early ingestion of colostrum, and short-term postbirth parenteral antibiotics in the newborn.

References