1981

Passive Immunity in the Neonatal Calf

Carl G. Rischen

Iowa State University

Follow this and additional works at: https://lib.dr.iastate.edu/iowastate_veterinarian

Part of the Large or Food Animal and Equine Medicine Commons, and the Veterinary Microbiology and Immunobiology Commons

Recommended Citation

This Article is brought to you for free and open access by the Journals at Iowa State University Digital Repository. It has been accepted for inclusion in Iowa State University Veterinarian by an authorized editor of Iowa State University Digital Repository. For more information, please contact digirep@iastate.edu.
Passive Immunity in the Neonatal Calf

by Carl G. Rischen*

Introduction

The purpose of this paper is to review a subject that is of interest to the student of immunology as well as being of interest and practical importance to the practicing veterinarian and to the owner and manager of any type of cow-calf operation. The subject is that of passive immunity transferred from the dam to the calf via colostrum and the problem of an acquired immunodeficiency in the neonatal calf due to an inadequate intake of colostrum or due to ingestion of colostrum after intestinal permeability to intact immunoglobulin has ceased.

The normal neonatal calf is fully capable of mounting a full immune response to antigenic stimulation. This response, however, is a primary response, and involves a somewhat prolonged lag period. Plus it may result in a rather low concentration of antibodies. In other words, it is a case of too little too late. With time, the colostrum-deprived calf will produce immunoglobulins in response to normal antigenic stimuli from the environment. The result of this production is a serum level of immunoglobulin that is equal to the immunoglobulin serum levels of a normal calf within eight weeks.1

The problem during this period of time is that the calf is extremely susceptible to a variety of infectious processes, the most notable being enteric and septicemic infections with pathogenic strains of *Escherichia coli* and respiratory infections.

Colostrum: Secretion and Characteristics

In all mammals, immunological assistance is provided for a period of time by passive immunity transferred to the neonate from the dam. In the calf, this transfer occurs postpartum by means of neonatal ingestion of maternal colostrum.

Bovine colostrum consists of an accumulation of mammary secretions from the last few weeks of pregnancy along with proteins transferred from the blood under the influence of estrogens and progesterone near parturition. These serum proteins consist largely of gammaglobulins (Table 1).

| TABLE 1 Immunoglobulin concentrations in bovine colostrum and milk (mg/100 ml) |
|-------------------------------|-----------------|-----------------|
| IgA                           | IgM             | IgG             |
| colostrum                     | 100-700         | 500-1300        | 3400-3900 |
| milk                          | 10-50           | 10-20           | 50-750   |

Upon examination, it has been observed that the concentration of immunoglobulins in colostrum at parturition is higher than the maternal serum levels.2 These levels may be as much as five times the maternal serum levels.4

We also know that there is a certain degree of secretory selectivity in the mammary gland, thus, the IgG secreted in the colostrum is only of the IgG-1 group, or electrophoretically fast IgG.11.14 Electrophoretically slow IgG, or IgG-2, is not transferred from maternal serum to colostrum, thus it does not appear in the serum of the suckling calf.4 The IgA secreted in the colostrum is accompanied by a secretory piece7 and is absorbed and resecreted by the neonatal intestine.16

The character of the colostrum changes rapidly, with a decrease in total protein and a decrease in total immunoglobulin2 (see Table 2). The concentration of specific immunoglobulins tends toward those of mature milk, listed in Table 1.

---

*Mr. Rischen is a third year student in the College of Veterinary Medicine, ISU.
As this change involves a major decrease in immunoglobulins and occurs rapidly, it is obvious that it will be advantageous for the neonate to feed as soon as possible postpartum. It is also obvious that the first milk should go to the neonate, although the correlation between immunoglobulin concentration in the colostrum and resulting serum immunoglobulin level in the neonate is a point of controversy.²

**Absorption of Colostral Immunoglobulin by the Neonate**

A factor of greater importance is getting colostrum to the neonate while the colostral immunoglobulins can still be absorbed intact by the neonatal intestine. The decrease in intestinal permeability is commonly thought to occur over the first twenty-four hours postpartum, but intestinal impermeability to colostral immunoglobulins has been observed as early as four to six hours postpartum.⁹

The mechanism of immunoglobulin absorption is actually quite simple. The low proteolytic activity in the neonatal digestive tract and the presence of trypsin inhibitors in the colostrum enable colostral immunoglobulins to reach the ileum intact. There they are taken up by pinocytosis, are transported into the lacteals and into the intestinal capillaries. The IgA fraction is resecreted into the lumen while the IgG and IgM fractions enter the systemic circulation. This process of immunoglobulin absorption appears to be non-selective, although the degree of uptake of IgG and IgM seems to be related, appearing in the neonate's serum in a predictable ratio independent of colostral concentrations.⁴ Overall, the process seems to be quite efficient, as there is a rapid increase in neonatal serum IgM and IgG concentrations within twenty-four hours, reaching a peak within forty-eight hours.⁴,² The level of serum immunoglobulins, however, is still less than the immunoglobulin serum level of the dam.²

After absorption ceases and immunoglobulin levels reach a maximum level, they begin to decline as a result of normal catabolic processes. Under normal circumstances the rate of decline depends on the immunoglobulin class involved, with the time required to reach unprotective levels dependent on the initial concentrations.

**Action of Colostral Immunoglobulins**

Colostral immunoglobulins provide a double line of passive immunological defense for the calf, providing protection against both septicemia and enteric disease. Defense against enteric infection appears to be predominantly a local effect involving IgA as the principle immunoglobulin acting at the paramucosal surface. This localized protection may involve other immunoglobulins as well. Penhale¹² suggests, for example, that there is a small but significant influence of the IgM colostial fraction within the gastrointestinal tract, contributing directly to the control of enteric disease.

In experiments in which calves were given IgM intravenously, colostral whey orally, and the two routes of administration employed simultaneously, there were strong indications that the colostral whey provided enough local protection to delay the onset of enteric disease and to reduce the severity of the resulting diarrhea.⁵,⁶

Immunoglobulins IgM and IgG in the neonatal serum are generally regarded primarily as protective against septicemia, especially against *Escherichia coli*. IgM probably constitutes the primary defense in this case. Evidence suggests that antibodies to the lipopolysaccharide O-antigen of gram negative enteric bacteria are primarily IgM in nature. Also, work with *Salmonella typhimurium* indicates that the IgM antibodies are more efficient in complement-mediated bacteriolysis and as opsonins than IgG.⁴,¹² That this protection is primarily systemic is supported by the experiments of Logan and Penhale⁵,⁶ in which intravenous IgM prevented detectable septicemia while colostral whey alone had no effect on the time of onset or on the severity of colibacillosis.

Putting these actions into a more real life perspective, we find that we must consider the comprehensive nature of the passive immunity provided by colostrum. There are several distinct mechanisms involved, each adapted to provide protection in a particular body.
compartment. We must also consider the possibility that colostral whey may contain protective factors in addition to immunoglobulins and that there may be a combined activity of different immunoglobulin classes that is absent in purified fractions.\(^5,6\)

One important and interesting immunological interaction that occurs involves the effect of successful enteric protection on the effectiveness of systemic protection. Evidence suggests that calves with diarrhea demonstrate an increased loss of immunoglobulins already in circulation and a loss of significant amounts of all serum proteins.\(^8\) This loss of circulating immunoglobulins may be of great enough magnitude that a calf with previously normal serum levels will tend toward hypogammaglobulinemia and a subsequent predisposition to septicemia.

The overall protective effect of passively transferred immunity may offer another type of protection as well. Thomas \(et al.\)^{15} suggest that colostral immunoglobulin protection of the calf against respiratory infection may not be due directly to a specific passive antibody, but it may be an indirect result due to reducing the animal's susceptibility to colibacillosis and thus reducing the overall stress on the animal.

**Causes of Hypogammaglobulinemia**

Problems begin to arise when a calf begins to suffer from hypogammaglobulinemia. Perhaps the most common cause of neonatal hypogammaglobulinemia is the failure of the calf to ingest colostrum during the period of intestinal permeability to intact immunoglobulins. There is, however, a variety of other possibilities. One problem that relates in some respects to the time of ingestion is the concentration of immunoglobulins in the colostrum and the volume of colostrum consumed. As illustrated in Table 2, the level of immunoglobulins in colostrum declines rapidly postpartum and it is important that the calf is able to feed immediately.

Another interesting management consideration that will affect the immunoglobulin levels of the calf is the method of ingestion. It has been observed that calves that suckle their dams nearly always attain higher immunoglobulin levels than calves that are bucket-fed colostrum.\(^{14}\)

Seasonal factors may play a role also, as a relationship between season and the incidence of hypogammaglobulinemia is sometimes observed. Although no mechanism has been suggested, low levels of immunoglobulins in neonatal calves have been observed during the winter months.\(^5\)

Familial or breed characteristics are also involved in serum immunoglobulin levels reached by the calf. Tennant \(et al.\)^{13} observed that Jersey calves absorb more gammaglobulins than Holstein-Friesian calves during the same period. After a given period of time, the gammaglobulin levels of the two breeds return to similar levels. Proposed mechanisms for this difference are that the Jersey system might be more efficient due to differences intrinsic in the intestinal system or due to differences in the amount of exogenous factors in the colostrum that facilitate intestinal absorption of gammaglobulin.

A final problem which is encountered is that some calves do not absorb colostral immunoglobulins. The defect here is obscure, considering the “simplicity” of the system. Klaus \(et al.\)^{4} suggests the possibility of premature development of the intestinal enzyme system and a premature closure of the permeability period.

**Summary and Concluding Remarks**

Overall, we find that the process of colostral transfer of immunoglobulins from the dam to the calf and the subsequent passive immunity of the calf is really quite complex. It involves a variety of physiological mechanisms in the dam and in the calf, and the outcome of the overall process is of extreme importance to the survival of the calf.

In comparisons of hypogammaglobulinemic calves to calves with normal serum immunoglobulin levels, we see higher mortality rates, higher incidences of septicemia, higher incidences of acute diarrhea, and higher incidences of acute respiratory tract infections in the hypogammaglobulinemic calves.\(^{14}\) Although dietary immunoglobulin replacement or the use of normal adult serum is often advocated in treatment of hypogammaglobulinemic calves,\(^{16}\) experiments demonstrate that this practice may be ineffective.\(^5,6\) McBeath \(et al.\)^{9} suggests that good husbandry and antibiotic therapy may be the best methods of altering mortality rate while the calves are building up their own immune system.
We must also maintain a proper perspective on the actual level of effectiveness of the passive immunity transferred to the calf. One consideration is that the passive immunity only protects the calf against serotypes to which the dam is immune. Also, the general health of the calf and a variety of environmental factors will be of importance.

One last consideration here is that immunoglobulin levels alone do not always give an accurate measure of neonatal resistance to infection. High immunoglobulin levels may inhibit the ability of the neonate to respond immunologically to a variety of antigens. Here, familial or breed factors may play a role. A prime example of this is the fact that Jersey calves suffer higher morbidity and mortality rates associated with neonatal disease than Holstein-Friesian calves in spite of higher immunoglobulin levels. Really, the only conclusion that we can draw at this time is that we have an understanding of some of the basic mechanisms involved, but there are many mechanisms and interactions that involve far more than just serum levels of immunoglobulin.

REFERENCES