A systematic approach to calf Gastroenteric disease

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ABSTRACT: Gastrointestinal disease represents one of the most significant problems in calf health accounting for up to 50% of the mortality in pre-weaned dairy heifer calves. The multifactorial nature of disease process necessitates a systematic approach to on farm investigations to ensure correct diagnosis and enable the implementation of appropriate management changes. This article describes a stepwise approach in which the disease process is characterised and its pattern described. Having next identified factors impacting on mucosal health and immunity and potential bio security breakdowns; targeted diagnostics testing can be performed allowing therapeutic and management recommendations to be made. DOI: 10.1111/j.2044-3870.2010.00022.x

INTRODUCTION

Calf diarrhoea is one of a small number of instances in which a clinical sign is universally considered, classified, or referred to as a disease. The problem with classifying calf diarrhoea as a disease is the subsequent inclination of the vet or farmer towards a pathogen-oriented clinical approach. By considering diarrhoea as an indicator of altered gastrointestinal function, the clinician is more likely to adopt a systematic approach to diagnostics, therapeutics and prophylaxis, and is more apt to recognize the contribution of non-infectious factors to the clinical problem. This is not to say that enteric pathogens are irrelevant in neonatal gastrointestinal disease. In the US, neonatal enteric disease has been associated with approximately 50% of the mortality observed in pre-weaned dairy heifer calves (Barrington *et al*., 2002), and enteric pathogens have been implicated in the death of 25% of the annual calf crop (Barrington *et al*., 2002). It is clear from epidemiological studies that the risk of neonatal enteric disease is not restricted to any one particular husbandry system or to a specific environment. This means that a 'one size fits all' approach to farms with neonatal enteric disease is unlikely to be successful. The aim of this paper is to furnish the shrewd clinician with an appreciation of the enteric disease causality web along with an awareness of gastrointestinal pathophysiology. In combination, these broad concepts can be used to structure a systematic approach to the investigation and management of any bovine neonatal enteric disease outbreak.

CAUSALITY WEB

Neonatal gastroenteric disease is associated with a complex interaction of multiple factors. In order to facilitate a structured clinical approach, these factors can be classified under two abbreviated mechanistic categories, those affecting mucosal health and immunity (MHI) and those affecting exposure to infectious agents (i.e. biosecurity). Since all other key determinants can be considered as contributing to disease susceptibility via one or both of these mechanisms (Fig. 1), a comprehensive investigation should incorporate:

a. identification and quantification of any breakdown(s) of MHI or biosecurity, and
b. proof of a link between this breakdown and the pattern of enteric disease.

As discussed in a previous article, the association of key determinants with risk and causality on a particular farm is a key step in implementing effective therapeutic and prophylactic strategies (Gay, 2006, and Potter & Aldridge 2010).

OVERVIEW OF SYSTEMATIC APPROACH

A systematic approach should include the following steps:

- characterise the disease
- define the pattern of disease
- identify (and where possible quantify) breakdowns in MHI and biosecurity
- identify key determinants related to reduced MHI and biosecurity*
- determine spatial and temporal relationships between key determinants and pattern of disease
- determine need for diagnostics
- select and perform diagnostics
- initiate appropriate therapeutics
- institute prophylaxis.

DISEASE CHARACTERISATION

This is an important first step in the process of problem solving a group outbreak of enteric disease, and yet is often only superficially addressed. The tendency to skim over this step can be explained by the phenomenon described earlier, whereby calf

* an experienced clinician commonly evaluates key determinants continually in the process of understanding the strengths and weaknesses of a particular farm enterprise .

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diarrhoea is mistakenly viewed as a single disease. An accurate characterization of the clinical entity, of which the diarrhoea is one aspect, is foundational to planning the subsequent stages of investigation on a particular farm. The process of disease characterization should be performed by a careful and detailed evaluation of one or two representative calves from the group. It is beyond the scope of this article to describe the subtle differences in presentation (clinical history, physical exam, clinical biochemistry) of calves with enteric disease, but it is most useful to look for clues as to the underlying pathophysiological processes rather than focussing on the identification of a particular pathogen. Some examples of clinical signs that may help the clinician differentiate different enteric pathology are shown in Table 1.

Clinical tip: when carried out judiciously this step will provide important information that can be used to:

- ascertain age propensities and potential transmission routes
- identify appropriate individuals for diagnostic testing
- design treatment triage schemes
- devise biosecurity/biocontainment strategies
- estimate welfare and economic impact.

PATTERN OF DISEASE

Once the disease existent in a particular farm unit has been characterized, it is time for some farmyard epidemiology! Remember, when examining groups, the individual animal can be considered a sentinel

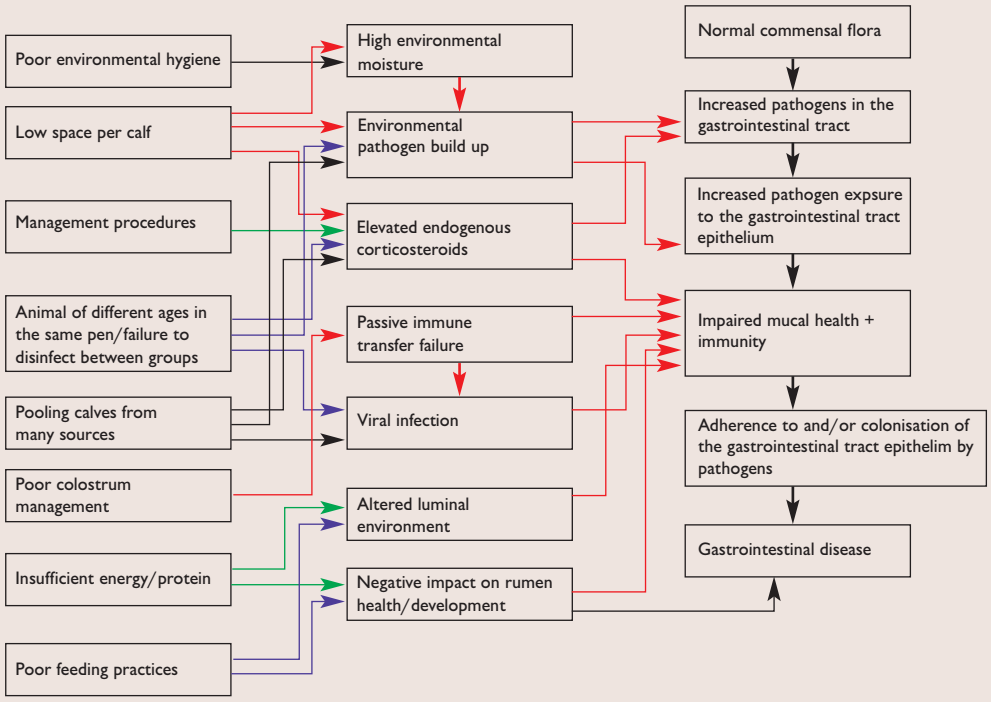

Fig. 1: *Causal web for risk factors associated with neonatal gastrointestinal disease.*

TABLE 1: Examples of clinical signs that can be used as identifiers of gastroenteric dysfunction and pathology

Clinical sign	Pathophysiological processes
Fever	Helps differentiate inflammatory and non-inflammatory gastrointestinal disease
Change in abdominal contour	Indicator of forestomach or enteric dysfunction; this is often subtle; additional information can be gained from succussion
Diarrhoea	Colour, consistency, presence of blood are clues as to anatomical location of disease, and whether the GI dysfunction is primary or secondary.
Weight loss	The ability of the GI tract to digest and absorb nutrients is only deranged in specific gastroenteric disease entities
Inflammation in another body system (e.g. umbilicus)	Provides clues as to pathogen identity and highlights the possibility of a compromise in immunological health
Ultrasonography	Can assist in localizing the disease within the GI tract; e.g. abomasal vs enteric disease
Clinical biochemistry	With the advent of portable and affordable blood chemistry analyzers, this becomes a very practical step in defining the disease entity; e.g. identification of a hypersecretion

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for events occurring at a population level. The objective of this activity is to identify the spectrum of disease present in the population. This information will be used in combination with the key determinants as evidence of causality. This step involves constructing a graphic representation of the clinical disease across the farm enterprise and, as discussed in a previous paper (Potter & Aldridge, 2010), is best achieved using a composite scoring system. The excellent calf respiratory health scoring system from the University of Wisconsin can serve as a template that can be modified to include the parameters identified as important in the disease characterization step. For instance, in neonatal gastroenteric disease it would be useful to include faecal scoring, umbilical examination and hydration in the scheme.

KEY DETERMINANTS AND PATTERN OF DISEASE

MHI and biosecurity

The practice of initially combining all key determinants into one of two broad categories encompassing host (MHI) and pathogen (biosecurity/biocontainment) facilitates the identification of potential hazards with risk and causality. The key inquiry in designing preventative programs is the identity of the controllable hazards contributing to the disease in that specific population of interest. One of the most common novice errors in preventative medicine is the temptation to coerce farmers into a husbandry system based on our general knowledge of disease epidemiology. An expert clinician is able to identify and weight the hazards contributing to specific risk and use this to design a programme to minimize those factors with the greatest impact and to achieve maximum health and economic benefit.

In order to select those factors with the greatest impact, the clinician must provide evidence of causality. For instance, if mucosal health is not an issue, then changing determinants that affect mucosal health is unlikely to provide much benefit. Examples of indicators that could help identify and differentiate between impaired mucosal health and immunity and disruption of biosecurity and biocontainment are shown in Table 2. Since most recognized pathogens are endemic on the great majority of farms (e.g. coronavirus in adult cow faeces), biocontainment is the more appropriate risk term. In some instances it may not be possible to differentiate MHI or biosecurity disturbances from one another, and on occasion one may mimic the other. For example, in some instances of poor hygiene the pathogen load becomes so large as to overwhelm even high levels of acquired colostral immunity. In these instances, the disease pattern can resemble that of reduced mucosal immunity when in fact mucosal immunity is adequate.

Key determinants

There are three broad areas of key determinants in neonatal gastrointestinal disease, those affecting the calf, those affecting the pathogen, and those affecting both (e.g. the environment) (Barrington *et al.*, 2002). Factors affecting the calf consist mainly of hazards that impact MHI, such as maternal nutrition during pregnancy, the acquisition of maternal immunity through colostrums and nutritional management. Failure of passive transfer (FPT) is usually associated with low immunoglobulin concentration in dairy calves and late ingestion in beef calves (Barrington *et al.*, 2002). Nutritional practices such as the quality and composition and quality of milk replacer and feeding practices that effect ruminal development are also important in mucosal health. Risk factors impinging on contribution of the pathogen include virulence factors, pathogen load and the presence of other microbes. Pathogen load can be affected both qualitatively and quantitatively by the source of origin (e.g. markets) and by on farm practices such as injudicious antimicrobial administration. The physical environment of the calf can also present certain hazards, but these invariably provoke effect

TABLE 2: Examples of indicators of abnormal mucosal health/immunity and suboptimal biosecurity or biocontainment

Indicators of abnormal mucosal health	Indicators of reduced mucosal immunity	Indicators of suboptimal biosecurity or biocontainment
Abnormal forestomach activity (e.g. distension; inappropriate motility)	Disease outbreak may involve animals with no direct contact between each other	Often point source outbreaks with spread between in contact animals
Inconsistent faecal consistency	Infections affecting animals of similar immunological age	Multiple age groups showing infectious disease problems
Failure to thrive; poor growth	Mild and non-specific mucosal infections (e.g. conjunctivitis, URT infections)	Pattern of disease usually associated with recognized pathogens
Poor hair coat	Problems with multiple pathogens	Problems with multiple pathogens
Disease outbreaks related to changes in feeding practices	Poor response to antimicrobials and vaccination	Disease outbreaks often related to poor environmental hygiene
	Infection disproportionate to microbial challenge (e.g. with optimal environmental hygiene)	Disease outbreaks often related to contact between calves of different ages

through the host or pathogen. For instance ventilation and humidity impact pathogen load while environmental temperature and handling strategies influence immunity. The major key determinants in neonatal gastroenteric disease are summarized in Fig. 1.

Clinical Tip: develop a system for quantifying key determinants e.g. practical measures for environmental moisture (how wet is the bedding?); a scoring template for biosecurity. These quantitative measures will not only be useful for substantiating causality, but also for performing intra- and inter-farm comparisons, and for use as a farmer incentive and to gauge improvement.

Relationships between key determinants and pattern of disease

Once the pattern of disease and the key determinants have been established for a particular population of animals, as farmyard epidemiologists the next step as is to substantiate causality. This is achieved by demonstrating a relationship between the key determinants and the pattern of disease. For example, if environmental hygiene is an essential risk factor then the occurrence of clinical cases will be higher where hygiene is measurably worse. Similarly, if colostral immunity is an important predisposing factor then the calves with clinical signs should have low serum immunoglobulin levels. It is important not to overlook the importance of this step of the investigation. If you cannot substantiate causality, your intervention measures will be speculative and therefore at risk of failing. As we all know, failed intervention measures can be a substantial waste of financial and labour resources, and a source of discouragement to both the farmer and the vet.

DIAGNOSTICS

As mentioned previously, the common clinical approach to neonatal gastroenteric disease is focused on the identification of causal pathogens. There are a wide range of calf enteric pathogens including viruses, bacteria and protozoa, and a plethora of commercially available packages on offer to assist the attending veterinarian in identifying pathogens in diarrhoeic calves. There is also an increasing interest in patient-side, point of care diagnostics to optimize the speed and cost effectiveness of pathogen identification. In view of the importance of microbes in bovine neonatal gastroenteric disease, knowledge of the pathogens involved in the disease outbreak is an essential aspect of the investigation. However, there are several principles that should inform our selection of diagnostic tests:

- most neonatal enteric pathogens are ubiquitous on UK dairy and beef farms
- the 'iceberg principle' in which the ratio between clinically infected and sub-clinically infected calves is commonly between 1:5 to 1:20
- a number of enteric pathogens are present in normal adult cow faeces
- most enteric agents survive well in the environmental conditions present on UK farms.

All of the above will affect the reliability of our diagnostic tests results by generating a tendency for either false positive or false negative results. The negative impact of these can be minimized by strategic sampling and test selection.

Clinical Tip: the following can help optimize your diagnostic sampling productivity

- Select animals with key clinical signs for sampling
- Use cheaper tests with a low tendency for false negatives as a screening test
- Follow up positives from this screening test with a more expensive test that has a low tendency for false positives.

There are also a wide range of non-microbiological diagnostic tests that can assist in the management and prevention of neonatal enteric diseases, both at an individual and a population level. These include total serum protein levels (for mucosal health and immunity), rumen fluid analysis (for suspected feeding practice irregularities), and serum electrolytes (to identify hypersecretory disorders).

THERAPEUTICS

It is beyond the scope of this article to discuss the details of successful therapy for calves with

gastroenteric disease. Suffice to say that the leading cause of mortality in affected calves is dehydration and electrolyte disturbances and, when present, the infectious component of neonatal gastrointestinal disease is invariably self-limiting. It follows that the backbone of routine therapeutics should be fluid and electrolyte replacement that is custom designed on the clinical signs exhibited by a particular affected individual. Antimicrobials and anti-protozoals are best used sparingly, and only when there is evidence that the pathogen burden is problematic (Barrington *et al.*, 2002).

PROPHYLAXIS

By adopting a structured approach to the investigation of an outbreak of neonatal gastroenteric disease, the astute clinician will have gathered all of the information needed for a systematic prevention programme. To summarize, the specific characteristics of the disease will have been identified as well as the pattern of disease in the population. A web of causality will have been constructed by isolating the effect of the key determinants on mucosal health and immunity and/or pathogen biosecurity/biocontainment in the enterprise. Finally the relationship of key determinants (host, pathogen or environmental) with the pattern of disease in the population will have been determined in support of the causality web, and the ability to modify or eliminate these determinants established.

The prevention programme is simply the systematic modification and elimination of the rogue determinants in such a way that disease is reduced or removed, and optimal productivity and profitability restored. In completing this process it is important to undergo an impact study in which a cost-benefit analysis is used to determine the most profitable intervention strategy.

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