Liver Abscesses in Beef Cattle: Potential for Dairy Monitoring?

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Introduction

The prevalence and economic impact of liver abscesses in feedlot cattle are well documented.[7] Abscessed livers in slaughtered feedlot cattle generally result from an aggressive feeding program, with incidence averaging from 12% to 32% in most feedlots.[3] Because liver abscesses are secondary to ruminal acidosis and rumenitis, such feeding practices as rapid increase in energy intake and poor or inconsistent bunk management – characterized by irregular feeding (both amounts and intervals) – promote a higher incidence of liver abscesses.[4] While abscesses are the leading cause of liver condemnation, the greatest economic impact of liver abscesses is from reduced animal performance and carcass yield. Reported effects of abscesses on animal performance have ranged from no effect to a depression in daily gain as great as 11% and a decrease in feed efficiency as much as 9.7%.[3] Cattle with multiple and/or large liver abscesses also may require more carcass trimming because of adhesion of abscesses to the diaphragm and surrounding organs. In some instances, condemnation of the entire viscera is necessary, contributing to significant economic loss.

Etiology and Pathogenesis

Almost all studies on the bacterial flora of liver abscesses have concluded that *Fusobacterium necrophorum* (biotypes A and B) is the primary etiologic agent.[7] Of the two biotypes, biotype A is more virulent and is more frequently encountered than biotype B in liver abscesses of cattle. In most situations, *Actinomyces (Arcanobacterium) pyogenes* is the second most frequent pathogen isolated from liver abscesses (Figure 1). *Fusobacterium necrophorum,* an anaerobe, is a normal inhabitant of the rumen. The ruminal wall appears to be the niche for *A. pyogenes,* an aerobe.[9] Ruminal wall provides an aerobic microenvironment in the otherwise anaerobic environment of the rumen.

Liver abscesses are secondary to the primary foci of infection in the ruminal wall. Because of the high correlation between liver abscesses and ruminal lesions,

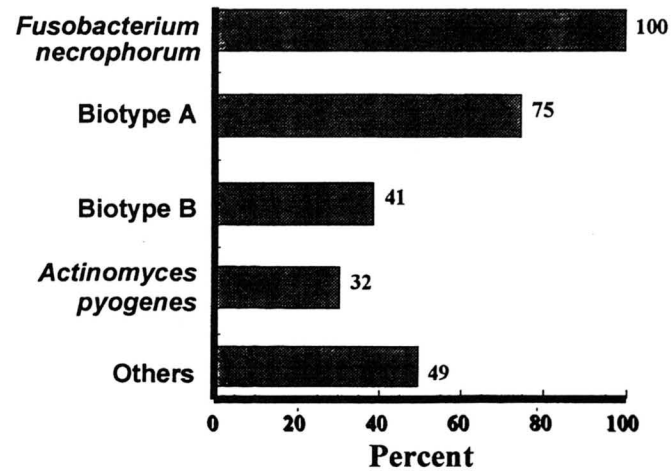

Figure 1. Bacterial flora of liver abscesses (152 abscesses from 103 livers) from feedlot cattle.

the term "rumenitis - liver abscess complex" is commonly used. The ruminal wall that is damaged from acidity or penetration of foreign objects (sharp feed particles, hair, etc.) becomes susceptible to invasion and colonization by *F. necrophorum*, a normal inhabitant of the rumen. Once colonization has occurred, *F. necrophorum* can gain entry into the blood or cause ruminal wall abscesses and subsequently shed bacterial emboli to the portal circulation (Figure 2).

More direct evidence for the pathogenesis of liver abscesses was obtained by restriction fragment length polymorphism analysis of rRNA genes (ribotyping) of *F. necrophorum* and *A. pyogenes* isolates from the rumen

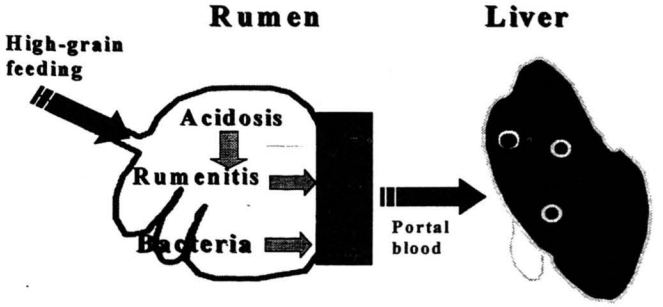

Figure 2. Pathogenesis of liver abscesses in cattle fed a high-grain diet.

and liver abscesses of the same animal (Table 1). The genetic similarity between the isolates from liver abscesses and ruminal walls supported the hypothesis that *F. necrophorum* and *A. pyogenes* isolates of liver abscesses originated from the rumen.[8,9]

Undoubtedly, the virulence factors of *F. necrophorum* and *A. pyogenes* play a critical role in the colonization of the ruminal epithelium and entry and establishment of infection in the liver. The liver is highly vascular and therefore richly oxygenated, and is a highly defended organ because of its numerous phagocytic cells (leukocytes and Kupffer cells). The leukotoxin of *F. necrophorum* is considered to be the major virulence factor, and synergism with *A. pyogenes* may contribute to the establishment of infection within the ruminal wall and liver.

Dairy cows

Prevalence of liver abscesses in dairy cows has not been documented, other than the anecdotal observations that liver abscesses are not uncommon in slaughtered heifers and cows. Liver abscessess as high as 40% to 60% have been claimed in packing plants that routinely slaughter cows. Most of the cows slaughtered are culled animals removed as a routine strategy to improve herd productivity, and low productivity is often related to disease. Liver abscesses are detected only at the time of slaughter because cattle – even those that carry hundreds of small abscesses or several large abscesses without extensive adhesions – seldom exhibit any clinical signs.

Generally, hematology and liver function tests have not proved to be good indicators of liver abscesses. Ultrasonography is a useful technique for detecting liver abscesses.[1,6] However, ultrasonographic scanning cannot visualize the whole liver, particularly the left side facing the internal organs. Moreover, other organs such as the lungs and kidneys cover parts of the lobes.

Abscesses in the liver occur as the result of entry and establishment of pyogenic bacteria. Routes by which these bacteria can gain access to the liver include the portal vein, hepatic artery, umbilical vein (in the newborn), bile duct system, and direct extension. Entry via the hepatic artery (following an episode of general septicemia) or the bile ducts (usually due to obstruction or ascendance of the infection from the duodenum) is a rare occurrence. In dairy cows, liver abscesses are most likely to occur by entry via the portal vein or direct extension of infection from adjacent tissues and organs, usually of traumatic origin. An example would be direct puncture of the liver by a foreign body lodged in the reticulum.

Dairy cows are predisposed to acidosis and rumenitis because of increased energy density and rapid dietary changes. Cows are generally switched from high forage diet prepartum to a high-energy, low forage diet postpartum in order to meet the energy demands of lactation. The change also typically includes a switch from hay to silage or pelleted forages. Cows are most prone to ruminal acidosis during the first 30 to 35 days postpartum. Subclinical acidosis is the most prevalent form. Ruminal damage in subacute acidosis happens more gradually and is due to increased ruminal concentration of volatile fatty acids. The inflamed and damaged ruminal epithelium facilitates bacterial invasion and entry into the portal circulation.

Traumatic reticuloperitonitis, caused by metallic objects lodged in the reticulum and perforating through the reticular wall – rarely involving ruminal wall – is often a predisposing factor for liver abscesses in dairy cows. Local peritonitis resulting from perforation could extend into the liver to set up abscesses. Adult dairy cows are more likely to be affected with traumatic reticuloperitonitis because of more frequent exposure to metallic objects. Routine use of magnets and prudent pasture and feed bunk management should lessen the incidence of liver abscesses of traumatic origin.

Sequelae

Septic cardiac and pulmonary emboli are also associated with liver abscesses in feedlot and dairy cattle. Generally, the condition starts as phlebitis caused

Table 1. Comparison of isolates from the rumen and liver abscesses by restriction fragment length polymorphism of rRNA genes (ribotyping)[a]

	Fusobacterium necrophorum		*Actinomyces pyogenes*	
Comparison	Number of paired isolates tested	Match	Number of paired isolates tested	Match
Liver abscesses vs. ruminal wall	9	8	6	2
Liver abscesses vs. ruminal contents	6	0	2	1
Ruminal wall vs. ruminal contents	6	0	2	2

[a]Narayanan *et al*[8,9]

by the extension of liver abscesses involving caudal vena cava. The phlebitis leads to thrombus formation anywhere between the liver and right atrium, but most often it is found at the point of entry of caudal vena cava into the diaphragm. The clinical syndrome and extent of lesions observed depend on the degree of thrombosis and types of organisms involved. The syndrome can range from death because of rupture of caudal vena cava to various degrees of pulmonary embolism, pneumonia, infarction, endocarditis, hemoptysis, and epistaxis. Collectively, these lesions are categorized under caudal vena cava thrombosis syndrome.[2,5,11,12]

Prevention

Control of liver abscesses in feedlot cattle generally has depended on use of antimicrobial compounds. The five antibiotics approved for prevention of liver abscesses in feedlot cattle are bacitracin methylene disalicylate, chlortetracycline, oxytetracycline, tylosin, and virginiamycin.[7] These antibiotics vary in their inhibitory effect on *F. necrophorum* and *A. pyogenes* and their effectiveness in preventing liver abscesses. Bacitracin is the least effective and tylosin is the most effective. In addition to reduction in liver abscesses, feeding of antimicrobial compounds will improve weight gain and feed efficiency.[10,13] Besides inclusion of antimicrobial compounds in the feed, proper bunk management to minimize ruminal imbalance is well accepted as a key factor for effective control of liver abscesses.

An effective vaccine would be highly desirable, because while antimicrobial compounds reduce the incidence of liver abscesses they do not eliminate the problem. Moreover, the antimicrobial feed additives are not approved for use in lactating dairy cows. A vaccine approach also would alleviate public health concerns associated with the use of subtherapeutic levels of antibiotics in the feed. Because leukotoxin is considered to be the primary virulence factor involved in the onset of liver abscesses, immunity directed against leukotoxin may be related to protection. Leukotoxin of *F. necrophorum*, an exotoxin, is a high-molecular weight (>300,000) protein and therefore, is strongly immunogenic.[14] Cell-free culture supernatant of a high leukotoxins producing strain of *F. necrophorum*, mixed with a suitable adjuvant, has been shown to elicit a high antileukotoxin antibody titer when injected in steers. It provided significant protection to experimentally induced liver abscesses (Table 2).[7] Further studies involving commercial feedlot cattle are needed to assess the benefit of a leukotoxoid vaccine to control liver abscesses.

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Table 2. Efficacy of *Fusobacterium necrophorum* leukotoxoid vaccine against experimentally induced liver abscesses in steers[a,b]

Experiment	Treatment	No. of steers	Incidence of liver abscesses	
			No. of steers positive	%
1	Control	5	5	100
	Vaccinated	5	0	0
2	Control	5	3	60
	Vaccinated	25	8	32
3	Control	5	0	100
	Vaccinated	20	5	25

[a]Vaccine consisted of culture supernatant from a high-leukotoxin producing strain of *F. necrophorum* containing inactivated leukotoxin mixed with an adjuvant. Two injections were given subcutaneously at 3 wk intervals.
[b]Liver abscesses were induced by ultrasound-guided, intraportal injection of *F. necrophorum*.
[c]Adapted from Nagaraja and Chengappa[9]

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