Epidemiologic Investigation of Vesicular Stomatitis in a Dairy and Its Economic Impact

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Summary

An outbreak of vesicular stomatitis occurred in a dairy herd in southwestern Idaho. An epidemiologic investigation and an economic impact evaluation were made. The virus spread in the herd despite the lack of insect vectors, and the vesicular stomatitis virus was isolated from saliva-contaminated water sources. Approximately 320 of 500 cows were affected, and the total estimated loss to the dairyman was approximately $50,000.

Introduction

Vesicular stomatitis (VS) is a viral disease that was first recognized clinically in horses more than 150 years ago. It was later shown that cattle and swine are susceptible to vesicular stomatitis virus (VSV). Vesicular stomatitis is confined mainly to the western hemisphere with central and northern South America considered endemic areas. Sporadic epidemics of VS occur in Mexico and southeastern United States. The spread of VS to more temperate zones occurs even less frequently. The disease usually occurs in summer and fall months coincident with the insect season. The seasonal nature of the disease has provided much of the basis for describing the transmission of VSV by insect vectors.[1,2,3]

An epizootic of VS was observed in the Rocky Mountain states during the summer and fall of 1982.[4] The epizootic, however, continued into the winter months in many areas, after the generally accepted insect season was finished.

There have been previous case reports of the economic effects of VSV in dairy cattle. One report from a 105-cow dairy in Alabama documented a loss of $40,000 in 1964.[5]

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In this report, we describe an epizootic of VS in a commercial dairy herd and its economic effects.

Case Report

A commercial dairy consisting of approximately 400 lactating Holstein cows being milked three times per day, and associated dry cows and heifers up to 6 months of age, located in Canyon County, Idaho, was in the process of expansion in the fall of 1982. From September 27 through November 8, 129 new replacements were purchased. The majority of these animals were springing heifers. There were 7 sources for these replacements, including 2 auction sales and 3 livestock dealers.

After arrival to the dairy, the purchased replacements were put in the close-up pen, or out in the dry-pen, depending upon their expected calving date. After they calved, they were moved into pen 4, which is a warm up pen, for a short time, and then into pen 1 or 3 which were high production pens *(Figure 1).*

The manager of the dairy recalled noticing a blister (vesicle) on the teat of one of the last springing heifers purchased when she arrived at the farm. He could not recall, however, the day he noted it nor the source of the heifer. The first documented vesicular lesion was on the teat of a purchased heifer from pen 4 on November 18, 1982.

The first lesion to be examined by the attending veterinarian was presented on November 23, 1982 *(Figure 2).* At that time, pseudo cow pox, herpes mammallitis, or frost bite/trauma were suspected as possible etiologies. Vesicular stomatitis was not considered because of the cold ambient temperatures occuring at the time of the outbreak and the absence of insect vectors *(Figure 3).* Vesicular fluid was harvested from an intact vesicle and sent on ice to the Washington Animal Disease Diagnostic Laboratory for virus identification.

An iodophor teat dip was in use at the time the first case was observed. The managers, however, decided to change the dipping routine, so on November 23, the teats were dipped twice per day with mineral oil, and once per day with the iodophor teat dip. This treatment was discontinued on the recommendation of the attending veterinarian on November 26, at which time the use of a chlorohexidine teat

Figure 1: Schematic map of dairy

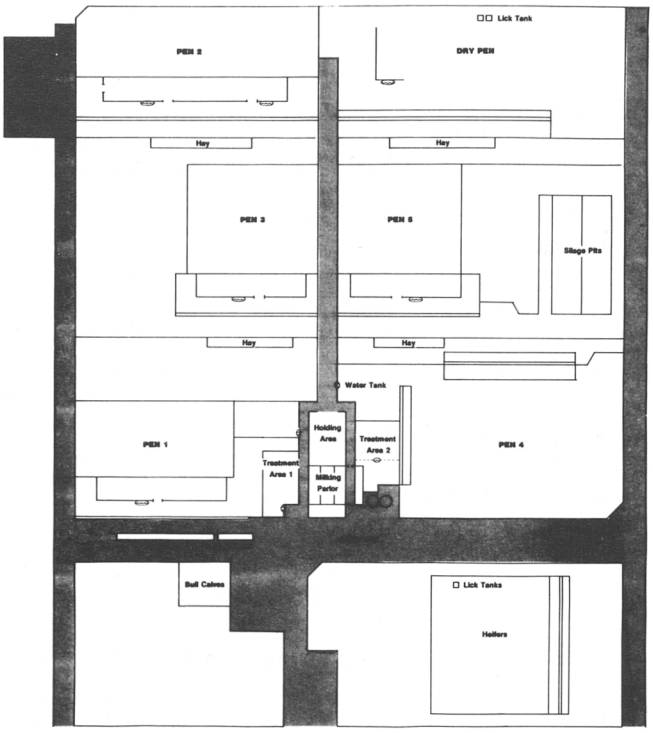


Figure 2: Teat lesions caused by VSV infection

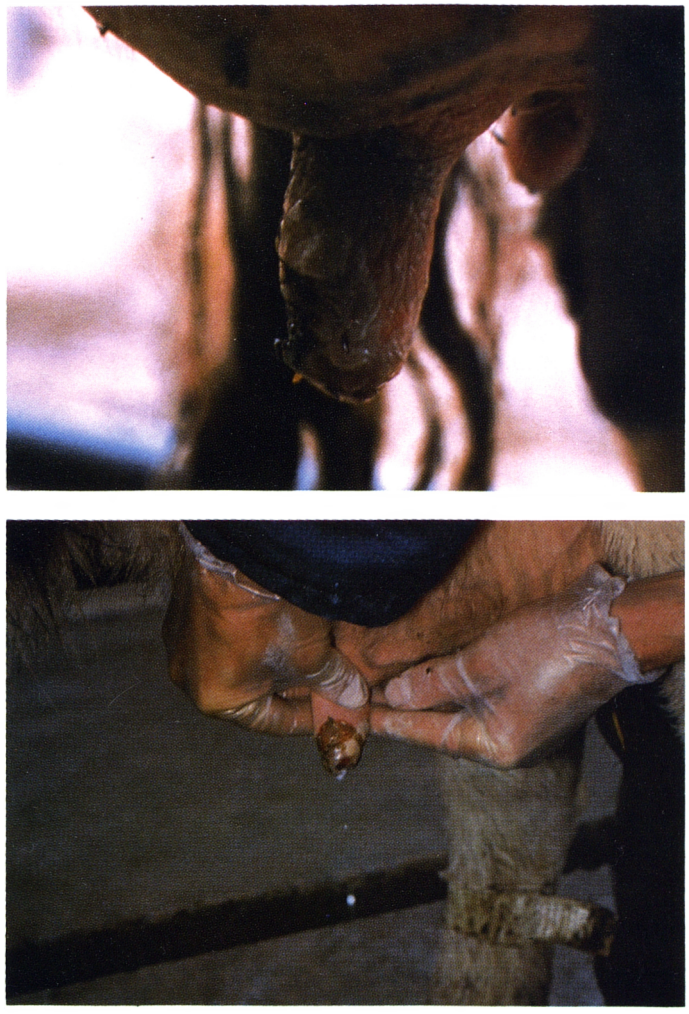
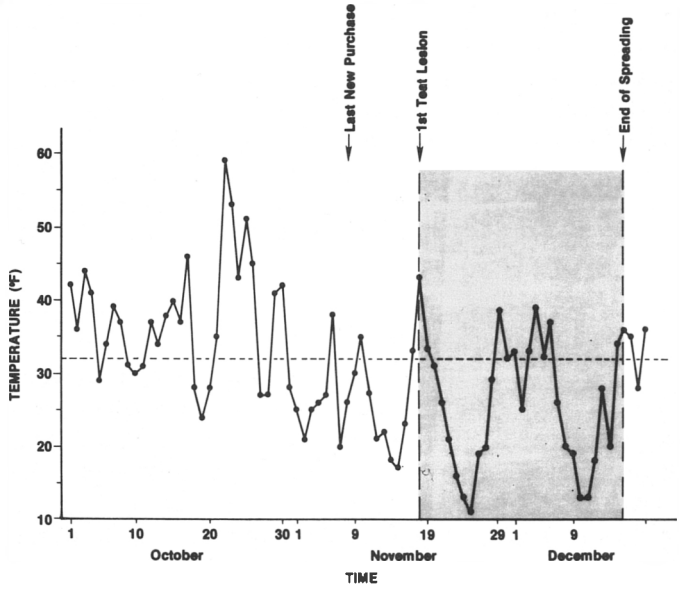


Figure 3: Ambient temperature data from National Weather Service monitoring station located five miles from dairy. Shaded area defines time of VS outbreak on dairy.



dip was instituted at each milking. This teat dip was used through February 1, 1983.

In the short time that the oil teat dip was used, it seemed that the rate of spread of teat lesions increased dramatically. Initially, all the teat lesions occurred in pen 4 *(Figure 1)*, but eventually cattle in other pens were affected. The spread of teat lesions showed considerably after the use of the oil teat dip was discontinued.

On November 27, 9 days after the first teat lesions, the first oral lesion was noted *(Figure 4)*. On this date VS was highly suspected. The first lesions on feet were noted on November 30, 1982 *(Figure 5)*.

A diagnosis of VS was made on December 1, 1982. This was based on virus isolation and electron microscopic examination of submitted vesicular fluid sent to the National Veterinary Services Laboratory, Ames, Iowa, for serotyping. The viral isolates were reported to the New Jersey strain of VSV as determined by the virus neutralization test.[4]

In an attempt to determine the incidence of clinical VS, 332 cows were examined for the presence of VS lesions on December 6, 7, and 9. About one-third of the cows were examined completely, while the remaining cows were given oral examinations. With information obtained from the examinations plus culling and treatment information for both mastitis and foot problems, an estimation of the incidence of VS lesions was made *(Table 1)*.

Figure 4: Oral lesions caused by VSV infection. Eroded lesions involving tongue and commissure of lips.

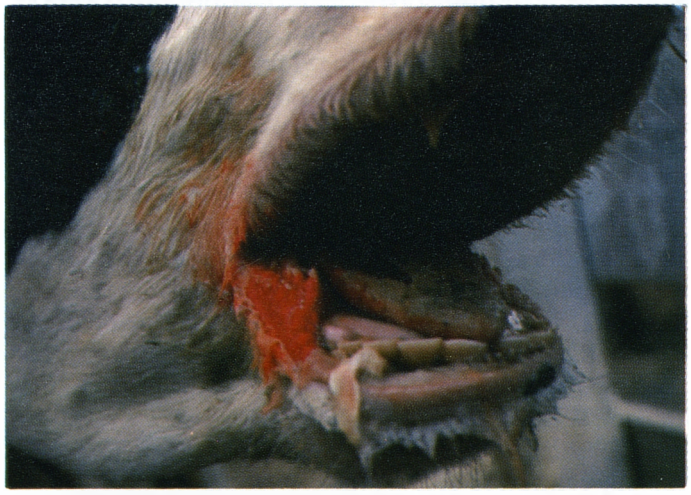


Figure 5: Vesicular lesion dorsal to coronary band and in interdigital area.

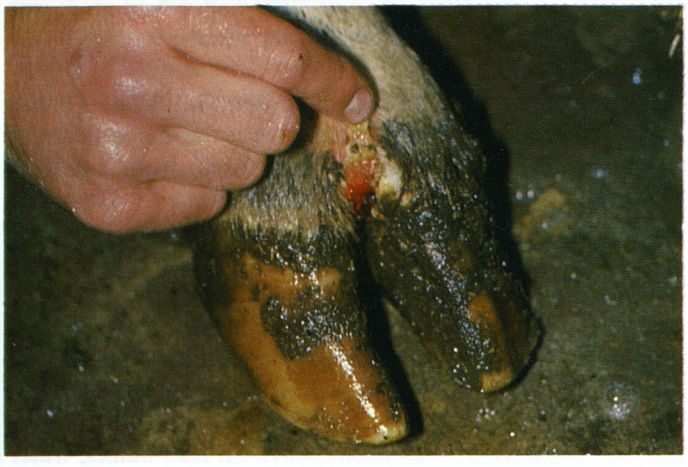


TABLE 1. Estimated incidence of VS lesions in 500 dry and lactating cows.

	% Affected
No lesions	36%
At least 1 lesion	64% *
Oral lesions	57%
Teat lesions	10.6%
Feet lesions	4%

* The summation of oral, teat, and feet lesions is greater than 64% because some cows had multiple lesion site involvement.

The spread of lesions through the mature cows herd was widespread, with all pens equally affected by December 9, 21 days after the outbreak began. None of the young heifers were affected. The 20 foot wide driveways *(Figure 1)* that separated them from the mature cows seemed to limit the spread of VS on this dairy.

The outbreak occurred in late November well after the insect season. The cattle were examined for lice, and none were found. Information obtained from a National Weather Service monitoring station within 5 miles of the dairy showed that there were 24 frosts (daily low temperature less than 32° F) prior to November 18, 1982. VSV spread for 4 weeks in the herd from November 18 through December 16, despite the fact that 18 of 28 days (64%) the temperature dropped below freezing *(Figure 3)*. The lowest temperature occurred on November 25 when it dropped to 11° F, while teat lesions were spreading and just prior to the appearance of oral lesions.

Other modes of transmission were suspected because of the distribution of clinically affected animals and the lack of evidence of insect vector transmission. We considered direct contact and fomites as the most important significant means of spread of VSV. Nearly all the heated waterers were contaminated with excessive amounts of saliva. Water samples were obtained from the waterers and VSV was isolated from one of the samples from pen 2 *(Figure 1)*. Fecal samples and other environmental samples, including teat dip oil, were also examined for VSV, but no isolations were made.

Economic Impact

The economic losses on this dairy due to VSV were substantial. In order to estimate the losses caused by the VSV outbreak, production and health records were analyzed. There were 4 major areas of loss caused by VSV on this dairy. They included 1) involuntary culling, 2) secondary bacterial infections and deaths, 3) lost milk production during the outbreak, and 4) losses due to early dry-offs.

When a cow is culled for reasons other than low production, she represents an economic loss. Thirty-eight cows in this herd were involuntarily culled due to the effects of VS. Of 53 cows affected with teat lesions, 30 were eventually culled. Culled cows were those that had extensive lesions which made milking impossible and/or developed severe mastitis secondary to erosions of teat ends. Eight additional cows were culled because of marked decreases in milk production.

The average salvage value of the 38 culled cows was $460.03. The cost to replace them, based on the average price the dairyman paid for the expansion heifers, was $1,038.80. The loss that the dairyman incurred is the difference between the two prices; $21,993.26.

Teat lesions caused by VS reduced the cows' defense mechanism against bacterial mastitis in many cases. The foot lesions caused by VSV were also susceptible to bacterial invasion. Many cows were treated with appropriate antibacterial drugs for mastitis and cellulitis of the pastern and fetlock region. The drug costs and associated milk withholdings were costs that were directly attributable to VS.

According to the treatment records there were 37 cases which were treated with antibacterial drugs. The cost of the

drugs to treat these infections was $401.28. The milk lost was valued at $1,849.37 according to daily production records and the price of milk ($12.97/cwt) at the time.

Two cows died due to VSV during the outbreak. Both of these cows were mature milking cows in the prime of their production careers. They were valued at $1,200 each. The total losses due to secondary infections and deaths was $4,650.65.

The daily production per lactating cow was monitored as part of the regular herd health program. Production was rising in early November because they were milking many fresh heifers. Milk production started dropping the week of November 16 and continued to drop until December 6, at which time it started to recover. It did not return to expected levels until the week of January 18 *(Figure 6)*. The area between the projected milk production and the actual represents losses in production directly attributable to VS. The total estimated losses due to decreased milk production were $13,944.14.

Many cows were no longer profitable to milk after clinical VS. These cows were either culled or dried off early depending upon their reproductive status. Economic losses due to shortened lactations and increased feed costs were estimated to be $8,741.31.

The total estimated losses for this dairy were $49,269.36 *(Table 2)*.

The aforementioned total does not include the costs of extra manpower needed in treating cows, ration changes which were employed to make the feed more palatable, or the weight loss the cows experienced during the infection. Therefore, the estimates are conservative. The losses when considered on an affected cow basis was approximately $150.

Figure 6: Milk production data. Expected production (dashed line) and actual production (solid line). The area between the lines represent the lost milk production.

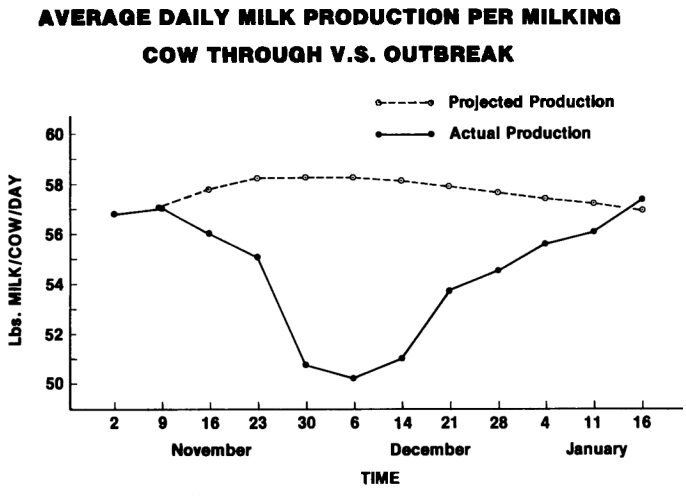


TABLE 2. Economic Losses Attributable to VS.

Involuntary culling	$21,933.26
Bacterial infection/death	4,650.65
Direct product losses	13,944.14
Early dry-off loss	8,741.31
	$49,269.36

Discussion

Vesicular diseases of cattle have been of concern due to the number of viruses that can cause similar lesions of which the most important is foot and mouth disease virus. With this outbreak, two problems emerged. The first involved the differential diagnosis of the vesicular diseases and the second pertained to the spread of the virus within the herd and how to control it.

Herpes mammillitis virus and pseudo cow pox were initially considered because the first lesions were confined to the teats.[2] VSV was not considered because of the low environmental temperatures experienced prior to the occurrence of lesions and reports from regulatory veterinarians that the spread of VSV had subsided. It was not until lesions were observed in the mouth that VSV was considered.

The seasonality and geographic distribution of VS has suggested that arthropod vectors serve as the major means of virus spread.[2,3] VSV has been isolated from sandflies *(phlebotomus)* and mosquitoes *(Culex, Aedes)*[1,3]. However, experimentally induced viremia has been insufficient to infect biting arthropods.[1]

Other modes of virus spread which may be equally as important as insects include horizontal transmission from one infected animal to a susceptible animal. This form of virus spread generally occurs within a herd by mechanical means such as milking machines,[2] as well as by susceptible animals contacting virus-laden saliva on feed bunks, water troughs or other surfaces.

Included in the animal transmission of VSV are wildlife and rodents. Serologic studies have shown that VSV infection occurs in deer, elk, antelope, bobcats, coyotes, raccoons, and birds.[3] Subclinical infection with VSV has recently been documented to persist in hamsters for more than 8 months.[6]

These data strongly suggest that there are at least two cycles for VSV. The first involves small rodents and wildlife with insects as virus carriers in nature. The second cycle involves livestock. In this cycle, the virus is potentially spread mechanically by insect vectors, but is enhanced by animal to animal contact.

Since VS is a self-limiting disease, there is a tendency to let the infection "run its course" through a herd once a definitive diagnosis is made.[2] The treatment of VS cases is symptomatic and is based on reducing secondary bacterial and mycotic infections of the oral cavity, teats, and feet. The observations from this outbreak indicated that both the mouth and foot lesions healed well with minimal

complications. However, the teat lesions did predispose to gland to mastitis. This was emphasized by the fact that 30 to 53 (57%) cows with teat lesions were culled due to inability to milk or severe mastitis. In a previous report 43 of 91 cows (47%) with teat lesions were culled.[5] Evidence presented from these two outbreaks suggest that approximately one-half of cows who develop teat lesions will be culled eventually.

Several management changes were made during this outbreak in an attempt to control the spread of VS. By discontinuing the use of the oil teat dip and the adoption of the chlorohexidine teat dip, the spread of teat lesions appeared to be greatly reduced. This points out the problems associated with the use of non-approved teat dips.

Other management changes to control the spread of VS included discontinuing parlour feeding and restricting movement between pens. The spread of VS, particularly the oral lesions, continued despite attempts to control it. The excessive environmental contamination with saliva from clinically affected animals probably was the reason for this.

The VSV was most likely introduced into this herd by the purchase of infected animals. The use of an isolation area could have saved this dairyman a substantial amount of money. This case points out the importance of such a facility and the care that should be taken when purchasing new animals.

VS is an economically important disease in its own right. It is not just a confounding problem in the diagnosis of foot and mouth disease. Presently there are no control programs to limit the spread of VS. Effective VS control programs are needed to prevent the type of economic loss reported in this case. It is hoped that research on VS vaccines and epidemiology of VS will aid in future control of the disease.

References

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