The Unexplained Abortion

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The title of this presentation was assigned me by the program committee. It probably represents wishful thinking on their part as much as any real expectation. On the other hand, they may have been trying to give me as wide a range for discussion as possible and since currently the great majority of bovine abortion are unexplained we have considerable scope.

Even ignoring the fact that in some of the “explained” abortions the explanation may well be inadequate, most laboratories diagnose less than 20% of the cases of abortion that are submitted to them. Costs of these examinations vary with laboratories; but in laboratories close to the 20% figure, they probably average $100 to $150 (1), which means that each “explained” abortion in a sense costs $500 to $750. Since this costly diagnosis usually only tells you what you already know, that is, that IBR, lepto, fungi, and a few other organisms can cause abortion in cattle, I have grave doubts whether these very expensive diagnoses are worth their costs. I checked with several practitioners and clinicians at the school to see if these diagnoses make a difference. They responded with various levels of indignation. It was as if I had put the knock on motherhood. They convinced me I had asked a socially unacceptable question, but they didn’t convince me that a cost-benefit analysis would show that the service is fiscally sound as it is currently performed.

This might be the subject for another editorial. But before we leave it one point should be made. With 80% of our bovine abortions unexplained we may well have abortogenic diseases, intoxications and hormonal problems we have not even considered. It is critically important to be sure, with the limited personnel and funds available, that any diagnostic effort have a research component and that our labs are not sunk in a sea of autolysing fetuses. It is very easy to confuse culturing rotten fetuses with effective investigation.

Having indicated that there may be major causes of abortion we don’t recognize, I’d like to spend most of my time discussing why diseases causing abortion represent such a diagnostic challenge and why we are unable to recognize even some of our old friends.

Pregnancies are maintained in essentially all mammalian species by progesterone. In the cow there is a shifting responsibility for the production of the hormone between the corpus luteum and the placenta. This changing source of progesterone production profoundly effects the manifestation of fetal death. Death of the conceptus early in pregnancy will not immediately cause a drop in progesterone production, so at least in a hormonal sense, the cow remains pregnant and fetus undergoes maceration. Abortion occurring at this period have been notoriously difficult to diagnose. The
Abortions following fulminating fetal infection. The fetuses seem to make. We have been speaking of abortion progesterone produced the feto-placenta component and this immune competence following fetal death and the associated drop in the antibody production but does not interrupt the pregnancy, and calves which suffered intrauterine infection late in gestation appear normal at birth and thrive. Therefore, the presence of antibody in a fetus may only indicate previous non-lethal infection. However, fetuses are apt to be killed or develop brain, eye, lung, or skin anomalies if infected by BVD before the 150th day of gestation. It is therefore extremely important in the investigation of congenital anomalies that blood be obtained for serologic examination before the calf gets colostrum. Studies of this type opened the door to our fuller understanding of BVD as a cause of abortion, congenital anomalies or inapparent fetal infections. Other infections such as IBR, although occurring late in gestation when the fetus is immunologically competent, kill the fetus before it can make antibodies.

There is at least one confusing experiment that seems to show abortion occurred during the "convalescent" period in PI3 infection. The fetuses appeared to "recover" from the acute disease and then died. Serologic fetal testing is more important in chronic infection such as Brucella and EBA.

This brings up another important point I’d like to make. We have been speaking of abortion following fetal death and the associated drop in the progesterone produced the feto-placenta component. This is typical of early abortion and abortions following fulminating fetal infection such as IBR. This produces the fetal stereotype of autolysis.

Normal fetuses secure their delivery by their own ACTH and glucocorticoid production at term. Chronically sick fetuses terminate their gestations in a similar way, the "stress" of the disease triggering ACTH and cortisol release. This adds some additional confusion in that the same disease may produce different appearing fetuses at different stages of gestation. That is, as the fetus ages, his defense improves and acute disease becomes more chronic and as the endocrine system matures premature delivery becomes a possibility.

Before I conclude, it is only fair that I say a few words about EBA, Epizootic Bovine Abortion. Because, very likely, I owe my invitation to speak here to it. Unlike most laboratories which provide diagnostic service, we often diagnose the cause of abortion in the majority of fetuses that are brought to us and best of all we do it without even trying hard, EBA fetuses being recognizable at 20 feet. EBA deserves mention here also because I believe it is an "unexplained" abortion. I don’t believe, as I once did, that it is caused by the psittacoid (chlamydial) agent that has been incriminated.

Since time is limited I will mention only those points which I find most convincing. There does exist in the foothill areas of California a very real disease which causes abortion in our cattle called, unfortunately, epizootic bovine abortion (EBA). This disease is seasonal and endemic. Susceptible animals exposed to hazardous ranges will often sustain abortion rates of 60-90%. There is approximately a three to four month period between exposure and abortion. The gross and histologic changes in the aborted fetuses are constant and unique.

In 1959 and 1960, Drs. Storz and McRkercher recovered chlamydial organisms and some of these fetuses and produced abortion by inoculating these agents into pregnant cows. It was presumed on this basis that the endemic abortion of cattle in California, EBA, was caused by this agent.

Concern about the validity of this assumption developed when vaccines failed to protect, although the natural disease appears to confer solid protection. There had been reason to question the relationship of these organisms to the natural disease even earlier for several reasons: the rate of recovery of the organisms from the natural disease was very low and inconstant, recoveries were made from abortions which did not fit the epidemiologic or pathologic patterns of EBA, and chlamydia infections, although regularly producing abortion,
do not reproduce the distinctive features of the natural disease. The experimental disease, like the chlamydial infection in sheep, enzootic abortion of ewe (E.A.E.), produces placentitis and usually only minor nonspecific fetal changes.

Cows experimentally inoculated with chlamydial agents respond with increase in CF titers and more specifically the experimentally aborted fetuses also have specific CF fixing antibody. This contrasts with the naturally occurring EBA abortions.

It seems likely from this evidence that the disease recognized as EBA in California is not caused by chlamydial organisms. If chlamydia produce a specific, naturally-occurring disease in cattle resulting in abortion, it has not been characterized or proved.

EBA is reported to occur outside of California. I am not sure if it does or not but I would be very interested to hear of any outbreak and would appreciate the opportunity to study tissues.

I am not sure I have shed much light on the “unexplained” abortion. I hope I have explained why the explanations are hard to come by and why, in spite of the fact that our last study appeared to move us back 10 years. I think more investigation is needed.

References


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**Weak Calf Syndrome**

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Weak Calf Syndrome is a specific disease of the bovine species characterized by abortions, stillbirths, neo-natal death and weak calves with the impairment of body functions of some animals visible throughout their natural life.

**History**

Since 1963 this disease entity has been recognized in beef and dairy herds in this author’s practice area, causing tremendous economic losses to individual ranchers and in certain years (1969) reaching epidemic proportions. Since 1969 the disease entity has been recognized by other veterinarians and ranchers in neighboring areas and states (Chart IV).

Due to failure to make a diagnosis of any known diseases, it has been tagged E.B.A. (variant), Bitter Root Crud, “Ward’s Disease” and Polyarthritis.

**Herd History**

A herd that is infected endemically will be plagued by a few scattered abortions during the last trimester of pregnancy involving approximately 1.5 to 2% of the total cow herd.

The second observation is a 6-8% death loss of calves shortly after birth, plus the same number of calves (6-8%) showing some form of weakness, diarrhea, stiffness and failure to do well as will be described.

The disease can be very sporadic or a very constant problem in a given herd during calving. Calf losses may be heavy at the beginning of calving, then stop; or, it may not become evident until one third or one half of the cows have calved. These factors depend on introduction of replacement animals, concentration, trucking, trailing and climatic conditions during the last 60 days of pregnancy.

The losses due to this syndrome will vary greatly depending on the above mentioned factors and natural immunity received from previous years of exposure.

**Etiology**

It is my belief that this disease is caused by a...