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Abortion Dilemma

Bruce Teachout
Iowa State University

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- Thesis, Iowa State University, Ames, Iowa, 1971.
3. Berthelson, J. D. The MMA Problem in the Sow, American Association of Swine Practitioners, March 21, 1972, 5-9.
 4. Cockerill, V. L., The MMA Complex, Hog Farm Management, May, 1970, 26-29.
 5. Gamhbiani, M. L., MMA syndrome in swine, VM-SAC, 6, March, 1969, 692-696.
 6. *Hog Information Please*. MMA, National Hog Farmer, Volume II, 1971, 115-119.
 7. Kopf, P. W., The Relationship of *E. coli* to Mastitis-Metritis-Agalactia Syndrome of Swine, M. S. Thesis, Iowa State University, Ames, Iowa, 1967.
 8. Kunesch, J., MMA—personal notes from Medicine, 1973.
 9. Lewis, C. J., The use of *chlorpromazine* in swine agalactia, Vet. Rec., 88, April, 1970, 380.
 10. Martin, C. E., Hopper, B. E., Armstrong, C. H., Armstrong, H. E. A clinical and pathological study of the mastitis-metritis-agalactia syndrome of sows, JAVMA, 151, December 1967, 1629-1634.
 11. Martin, C. E., Threfall, N. R., Non-infectious Aspects of MMA, Symposium: Effect of Disease and Stress on Reproductive Efficiency in Swine, Extension Service of University of Nebraska, 1970, 109-113.
 12. Martin, C. E. Lactation failure in the sow, Hog Farm Management, July, 1972, 22-23.
 13. Martin, C. E., Threfall, W. R., MMA Swine Health-Extension Service, University of Illinois, 1970.
 14. Martin, C. E., Threfall, W. R. Clinical evaluation of hormone therapy in the agalactia syndrome of sows, Vet. Rec., 87, 768-771.
 15. Modern Veterinary Practice. One panel reports on MMA syndrome, MVP, March, 1970, 46-49.
 16. Nachreiner, K. F., Ginther, O. J. Current studies on the mastitis-metritis-agalactia complex of swine in Wisconsin, JAVMA, 155, (Dec. 1969), 1860-1865.
 17. Nachreiner, R. F., Ginther, O. J., Ribelin, W. E., Carlson, I. H. Pathologic and endocrinologic changes associated with porcine agalactia, Am. Journal of Vet. Research, July 1971, 1065-1075.
 18. National Hog Farmer, Poll says Disease (MMA) at top No. 1, National Hog Farmer, October, 1968.
 19. Ringnarp, N. Clinical and experimental investigations into a postparturient syndrome with agalactia in sows. *Acta Agricultural Scandinavica*, Supp. 7, 1960.
 20. Roberts, S. J. *Veterinary Obstetrics and Genital Diseases*, 2nd ed., 1971, Edwards Bros, Ann Arbor, Michigan.
 21. Ross, R. F., Christian, L. L., Spear, M. L. Role of certain bacteria in mastitis-metritis-agalactia of sows, JAVMA, 155, Dec., 1969, 1844-1852.
 22. Ross, R. F. MMA—personal notes from Infectious Diseases, 1973.
 23. Smith, H. C. Mastitis, metritis, and diarrhea in swine, JAVMA, 147, 626-631.
 24. Swarbrick, O., The Porcine agalactia syndrome, Vet. Rec., March 2, 1968, 241-252.
 25. Tharp, V. L., Metritis, Mastitis, and Agalactia. *Diseases of Swine*, 3rd Ed., Edited by H. W. Dunne, University Press, Ames, Iowa (1970) 869-877.
 26. The Agalactia-Mastitis-Metritis Syndrome, Veterinary Medicine Professional Topics, University of Illinois, February 1966.
 27. Thurman, J. C., Simon, S. A field study of twelve sows affected with MMA syndrome, VM-SAC, 65 (March, 1970):263-272.
 28. Trapp, A. L., Keahey, K. K., Whitenack, D. L., Whitehair, C. K. Vitamin E-selenium deficiency in swine: differential diagnosis and nature of field problems, JAVMA, 157, 1970, 289-300.
 29. Zeitler, J. MMA—A Clinical Review, Senior Seminar 1971, Iowa State University, Ames, Iowa.

Abortion Dilemma

by
Bruce Teachout *

Abortion according to S. J. Roberts, DVM,MS, is the expulsion of a living fetus before it reaches a viable age, or more commonly the expulsion of a dead fetus of a recognizable size at any stage of gestation.¹ This unproductive termination of pregnancy is costly to the cattle producer and presents an enormous diagnostic task for the veterinarian in the field. A complete understanding of each cause of abortion and assistance from a diagnostic laboratory will enhance the efforts of the diagnostician in the field.

This article will deal mainly with the herd type problem of bovine abortion which means that over two percent of the dams in an individual herd aborts. Infectious cases of abortions will be the primary concern even though other causes of abortion such as nutrition, trauma, hormonal imbalances, genetic causes, and toxicities should be kept in mind if the diagnostic work fails to lead to a specific

pathological microbe. It should be stated that only about 43-47% of abortions are specifically diagnosed.

The agent most commonly diagnosed as causing abortion in the midwest is the IBR-IPV virus. Work done at the Iowa State Veterinary Diagnostic Laboratory and the South Dakota State Diagnostic Laboratory shows that approximately 86% and 25% respectively of bovine abortion diagnoses made in recent years were of IBR virus etiology.²³ This syndrome would present a history of contact with new stock and abortion primarily occurring after the sixth month of gestation, although abortion can be seen at any stage of the gestation period. The fetus will show some degree of autolysis and be expelled dead. Autolysis can vary from a slight degree of subcutaneous edema to complete autolysis. The fetal body cavities are filled with fluid while petechial hemorrhages are widespread. The placenta undergoes a nondescript autolysis and the uterus is generally free of disease. The laboratory

* Mr. Teachout is a fourth year student in the College of Veterinary Medicine, Iowa State University.

diagnosis is made by microscopic and fluorescent antibody examination of the fetal kidney and liver along with attempted isolation of the organism. Serology checks for changes in titers can also be of help.

Increased study in the field of mycology has shown that many abortions are caused specifically by fungi. Work done at the laboratory at South Dakota State has shown that 17.5% of the abortions diagnosed there between July 1, 1972 and June 30, 1973 were mycotic abortions.³ A field case will present a finding similar to the following: Abortion will occur sometime from the fourth month of pregnancy to term with possible expulsion of a slightly autolyzed fetus. This fetus will usually be in a fresh condition. The dams contract the organism via ingestion or inhalation; thus moldy feed sources might be found or fed prior to the abortion. The fetus may be dehydrated and have loose skin with mycotic lesions. The placenta will be thickened with a leathery texture. The placentomes will be edematous and necrotic with a pronounced cupping shape. Adventitious placentation may also be observed. The laboratory diagnosis is made by using both microscopic and gross lesions. Histopathology will show hyphae in the placenta and possibly in the lung concurrently with a fetal bronchopneumonia.

Bacteria are also many times diagnosed as the pathogenic etiology of bovine abortion in the midwest. Of these, *Corynebacterium pyogenes* is the most common etiologic agent. The laboratory at South Dakota State University in the period from July 1, 1972 to June 30, 1973 diagnosed 9.55% of the total determined cases as *C. pyogenes* abortion. (In a five year study at the same institution 7.2% of the total cases diagnosed were *C. pyogenes* abortion.)³ The initial infection can occur with a general bacteremia from a wound or infection can occur during coitus with an infected bull. The abortions are sporadic and can occur anytime in gestation. The gross and microscopic lesions seen with *C. pyogenes* abortion are very non-specific. There is a diffuse inflammatory placentitis and a bronchopneumonia in the fetus. The

cow may have a yellow discharge along with a vaginitis, cervicitis, and metritis which causes a retained placenta. Diagnosis is made by isolation of a pure colony of the organism from the placenta or fetus and microscopic lesions showing inflammatory placentitis and bronchopneumonia in the fetus.

Listeria monocytogenes isolation from the uterus is frequent but the occurrence of the clinical disease is less common. The laboratory at Iowa State University reported one case of *Listeria* abortion in the period from July 1, 1971 to July 1, 1972;² whereas the laboratory at South Dakota State reported forty cases or 7.95% of the total abortions diagnosed in the period from July 1, 1972 to June 30, 1973. The organism is transmitted by ingestion and the syndrome usually follows a history of feeding silage from a recently opened silo or introduction of new stock into the herd. Abortions occur during the seventh to ninth month of gestation. There is little autolysis of the fetus. One characteristic is that herd abortions usually occur in a short span of time. The dam may have shown an earlier period of malaise with rumen stasis with a septicemic type syndrome.

Vibro fetus venerealis abortion was diagnosed in 6.7% and 7.75% of the total diagnosed cases at the laboratories of Iowa and South Dakota respectively in the years previously mentioned. The disease is spread at the time of coitus or at the time of artificial insemination. The history often includes the addition of a new bull to the herd. The primary observation is herd infertility with an increased number of services necessary for conception. When abortion does occur, it is seen primarily from month four to month seven. The placenta is thickened and edematous while the cotyledons may be grayish and contain a white cheesy exudate. The fetal lesions observed resemble those of mycotic abortion. The fetus looks dehydrated with a wrinkled skin. There may be slight autolytic changes of the skin along with subcutaneous edema. The stomach is thickened and filled with a turbid material. There is a diffuse pleuritis, pericarditis,

and peritonitis with a blood tinged fluid filling the body cavities.

A tentative diagnosis of vibrio abortion is made by looking first at the herd records of breeding and conception rates. Confirmation is made by isolation and identification of the organism. A dark field microscopic examination of the abomasal contents will reveal the organisms if present. Stomach contents can also be cultured. Histopathologic examination will show placentitis and possible fetal bronchopneumonia. Vaginal, cervical or uterine swabs can also be cultured for vibrio isolation. Vaginal mucus can be collected and sent to labs for an agglutination test which will also confirm vibrio infection.

Leptospirosis is another common bacterial etiology of abortion. South Dakota State reported 6.17% of the diagnosed cases as leptospirosis and Iowa State reported 10% of the diagnosed cases as leptospirosis for the years stated previously. The primary organism found to cause abortion is *L. pomona* but *L. hardjo* has been isolated also. The organisms are shed in the urine and are transmitted via ingestion or invasion of lacerations on mucous membranes or skin. The cows may undergo a febrile period with anorexia, decreased milk production, and thickened milk. Abortion occurs during the last half of gestation but all susceptible cows in this stage of pregnancy may not abort.

The lesions are typical of a non-specific septicemia. The placental and fetal changes can vary from no autolysis to severe autolysis. Diagnosis is based mainly on serologic tests and lack of isolation of other organisms. Two serum samples taken two weeks apart are needed for the serological plate agglutination test. For best results, samples of serum should be taken at the time of acute illness and shortly after abortion. The laboratory at South Dakota State considers a rise in leptospiral titer from 1/1000 to 1/10,000 as being diagnostically significant for a positive diagnosis.³

The program of Brucellosis eradication and vaccination in the midwest has virtually eliminated abortions caused by *Brucella abortus* although five cases of

abortion were reported at the South Dakota State Diagnostic Lab for the period previously mentioned. Infection occurs after ingestion of infected genital discharges from aborting cattle but transmission can occur through mucous membranes of the eye and uterus (using artificial insemination). Manthei reported there is little evidence to show Brucellosis is spread via natural service from an infected bull.⁴ Abortions occur from the sixth to ninth month of gestation. Necropsy is of little value because the fetus shows signs of a generalized bacteremia with a fibrinous pleuritis, peritonitis, lymphadenitis, and pericarditis. The placenta is thickened with possible adventitious placentation. There is a purulent exudate on the surface of the cotyledon.

A positive diagnosis is made primarily by isolation of the organism from the fetal abomasum or placenta. Serology is also used for diagnosis. A plate agglutination titer of 1/50 is a suspect in a nonvaccinated cow and a titer of 1/100 is positive. In a vaccinated cow 1/100 is a suspect and 1/200 is positive. The serum should be drawn at the time of abortion and a sample taken 30 days later to allow a comparison of titers.

The last major etiologic agent of abortion reported at the South Dakota State Laboratory is *Chlamydia* which causes Enzootic Bovine Abortion (EBA). Transmission of the organism is poorly understood. The onset of the disease is sudden and abortion occurs primarily from the seventh to ninth month of gestation. The disease is sporadic, (occurring mainly in given areas and given herds). The fetus is fresh and has a pot belly appearance due to severe anascarca and ascites. Necropsy usually reveals a yellow nodular enlarged liver. Petechial and ecchymotic hemorrhages are seen throughout the organs and tissues. There is an enlarged heart along with fibrinous peritonitis. The placenta is edematous but of little help in the diagnosis.

Diagnosis is difficult because the organism is difficult to isolate. A complement fixation test using paired serum samples taken at the time of abortion and two

weeks later may aid in the diagnosis. A rise in titer should be observed.

The diagnosis of bovine abortion is made by the combination of many factors as seen above. After the field observation has been made the laboratory can be called upon for specific assistance. The laboratory should be aware of all field observations thus an explicit history should be sent along with the specimens. This history should include the size of the herd, when additions were made, number and rate of abortions, vaccination history, previous herd problems, and results of earlier laboratory tests.

An ideal specimen would include an uncontaminated placenta which has the cotyledons intact. The entire fetus should be submitted but if not possible the fetal lung, liver, spleen, kidney, and abomasal contents should be sent to the laboratory.

These specimens should be fresh and refrigerated. (There is too much tissue decomposition when the tissues are frozen and histological exam doesn't reveal good results). Serum samples from representative cows should also be sent for serological tests. These procedures could lead to the diagnosis of the abortion—but remember—"the diagnosis is only as good as the specimen presented."

Bibliography

1. Roberts, Steven J., (1971) *Veterinary Obstetrics and Genital Diseases* 2nd Ed. Roberts, Ithaca, New York.
2. Brown, Lowell N., Personal Communication, Iowa State University Veterinary Diagnostic Lab, Ames, Iowa. May 1973.
3. Wohlgenuth, Kurt, and Kirkbride, C. A. Personal Communication, South Dakota State Veterinary Diagnostic Lab, Brookings, S. D. June 1973.
4. Manthei, C. A. (1968) Brucellosis as a Cause of Abortion Today, in *Abortion Diseases of Livestock*, edit. by L. C. Faulkner, C. C. Thomas Co., Springfield, Ill.
5. Blood, D. C., and Henderson, J. A. (1971) *Veterinary Medicine*, 3rd ed., Williams and Wilkins Co., Baltimore, Maryland.
6. Hull, Bruce (July 1973) Personal Communication, Iowa State Univ. College of Veterinary Medicine. Ames, Iowa.

What's Your Radiographic Diagnosis?

by
Russell Mitten, B.V.Sc., M.R.C.V.S., D.V.R.*

An eleven-year-old quarter horse gelding was examined for a lameness of several weeks' duration in the left foreleg. Four months previously an imbedded nail had been removed from the hoof. Examination revealed that the hoof was very sensitive to hoof testers at the toe area, but no sinus tract swelling could be seen in the sole. Radiographs of the foot were taken—what is your diagnosis from examination of this radiograph? (figure #1)

Diagnosis—Keratoma of the hoof wall.

Keratoma is an excessive growth of horn on the inner wall of the hoof, nearly always appearing at the toe, and usually growing down from the coronary band. The radiograph shows a sharp-edged erosion of the pedal bone at the toe. There is no sclerosis in the surrounding bone, which would be seen in the case of an abscess. The keratoma causes pressure necrosis of the pedal bone as it grows downward, and occasionally an external bulge

is seen in the hoof wall. The etiology of keratoma is obscure in most cases, but some follow an injury to the hoof. There is no infection in most cases. Paring away the hoof will reveal the horny growth after it has been present for eight months or more. Radiographs of this case several weeks later showed that the eroded area of the pedal bone had enlarged (figure #2).

Differentiation between abscessation and a keratoma is based on finding necrosis of the pedal bone without any evidence of sclerosis, and the inability to demonstrate infection in the toe despite repeated attempts at paring and application of poultices. The recommended treatment for keratoma is surgical excision, but the prognosis should be guarded, due to the erosion in the pedal bone, and the tendency of the growth to recur after excision.

References

1. Creswell, H. and Smythe, R. H. in *Equine Medicine and Surgery*, page 433. First edition, 1963, A.V.P.
2. Douglas, S. W. and Williamson, H. D.: *Veterinary Radiological Interpretation*, page 132. First edition, 1970, Lea & Febiger.

* Dr. Mitten is an Assistant Professor of Veterinary Radiology at Iowa State University.