MMA—A Clinical Review

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Introduction

MMA (mastitis, metritis, agalactia) has in recent years become one of the most perplexing problems facing the pork producer. In a recent polling of members of the National Pork Producers Association (October, 1968), MMA was listed as the number one swine health problem.

Simply speaking, MMA refers to mastitis (inflammation of the mammary glands), metritis (inflammation of the uterus), and agalactia (lack of milk flow).

Economically, the disease ranks first or second in losses incurred by the swine producer. Losses are due to baby pig deaths and unthriftiness of survivors.

Occurrence

MMA has occurred nationwide and seems to have no predilection as far as age of the animal is concerned (first, second litter, etc.). Most cases are seen in confinement facilities, although it can occur with pasture farrowing facilities. The severity and morbidity of MMA varies from farm to farm and is seen on both well and poorly managed farms. The death loss in affected sows is low, 1-2%, but the losses in the young pigs due to starvation, hypoglycemia, and overlaying by the sows varies from 20-80%. The occurrence of MMA has no relation to herd size and is often preceded by a prolonged farrowing time.

Literature Review

Some of the names in the literature used to describe MMA have been post-parturient fever, sow agalactia, toxemic agalactia, mastitis-metritis syndrome, milk fever, and lactation failure. MMA therefore, has been used to cover a wide variety of postparturient disorders of the sow which include mastitis, metritis, and agalactia.

Etiology

The precise etiology of MMA has not been elucidated, but many infectious and non-infectious causes have been incriminated.

Some of the management procedures which have been stated as being possible causes are: feeding alfalfa meal, Vitamin E or Selenium deficiency, Vitamin B12 deficiency, overfeeding of concentrates prior to parturition, lack of bulk in the ration, Ca:PO₄ imbalance in the ration, and feeding excess protein.

Infectious agents which have been incriminated include: E. coli, Klebsiella sp., Proteus, Pseudomonas, Enterobacter, Citrobacter, Staphylococcus, Beta hemolytic Streptococci and Mycoplasma.

Toxic factors incriminated include ergotoxin—from feeding moldy rye, aflatoxin and estrogens (Fusarium)—from feeding moldy corn.

Bacterial endotoxins have been incriminated as a possible cause due to the constipation often seen with MMA.

Some workers have noticed a correlation between large litters and incidence of MMA.

Stress has also been incriminated as a possible cause. Stresses associated with farrowing might be emotional (onset of parturition), thermal (temperatures different from normal), confinement (being
enclosed in a tight stall), or environmental changes (i.e., odors in a confinement farrowing house).

One worker has postulated that agalactia in sows is a sign of pituitary disturbance and a disease of domestication, brought about by continuous selection of breeding animals with certain endocrine disturbances or characteristics, which bring about rapid growth characteristics.

Endocrine dysfunction has also been reported as a possible etiological agent because of reported changes in ovarian size, structural changes in the adrenal gland, and response to oxytocin, prednisolone, and dexamethasone. The reasoning behind this being that mammogenesis and lactation are a complicated interaction of the ovaries, pituitary gland, and thyroid gland.

Simple bacterial mastitis by itself could interfere with the milk letdown reflex and result in agalactia.

At the present time, most workers feel that the primary etiology is that of a hormonal imbalance with an infectious organism playing a secondary role in the causation of mastitis, metritis, and agalactia.

Transmission

MMA does not appear to be contagious. Because more cases are seen in confinement farrowing units, it might be related to bacterial buildup in the farrowing house.

Clinical Signs

The sow usually appears to be healthy, eats, drinks, and nurses the pigs for the first 12-24 hours (occasionally 36-48 hrs.) postfarrowing. Affected sows after this rapidly lose in milk production. Appetite is usually completely lacking and signs of tiredness and sluggishness are manifest.

Sows lie down on their abdomen to protect their udder. The sow seems insensitive (loss of maternal instinct) to squealing, hungry, rooting pigs which are trying to nurse. This is probably an attempt to cool the udder and prevent the pigs from nursing.

Generally, the sows will run a temperature ranging from normal 102.5° F to several degrees above normal (105°-107° F). Height of temperature rise is of questionable value in assessing the degree of agalactia.

The mammary glands are hot, swollen, firm, discolored, and painful to the touch. There are no strong changes in the visual appearance of the milk. The pH of the milk in MMA affected sows is usually more alkaline (7.0-7.2) than the normal (6.4-6.5).

There may be a discharge from the vulva, but this is variable. A normal sow’s lochia is sparse, muco-purulent, and often not observed. In MMA the lochial discharges are watery to thick muco-purulent and in general in greater quantity and less homogenous than normal lochial discharges. These discharges normally cease 3-6 days after farrowing.

Often a change in demeanor of the sow occurs ranging from semicomatose to aggressive and irritable. The sow may also have a hoarse characteristic voice.

The sow may be constipated with little or no feces being voided in the last 24 hours prior to onset of symptoms.

The agalactia (which is the only constant sign) or hypogalactia is a result of the sow refusing to let her milk down or milk production so scanty that the pigs starve.

Often baby pigs show signs of hypoglycemia and starvation. Comatose pigs, subnormal temperatures, and varying degrees of enteritis are frequently observed.

Differential Diagnosis

All sows exhibiting hypogalactia or complete agalactia do not show the typical MMA syndrome. In many of these cases, the causes of the lack of milk secretion can be determined and corrective or preventive measures taken. It is essential therefore, to make an accurate differential diagnosis.

Causes of agalactia:

1. Hypocalcemia or eclampsia
   (rare, can be treated with Ca, Mg products)
2. Chronic ergot hypoplasia
   (usually seen in gilts, insufficient

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prolactin release)

3. Udder and teat conditions
   a. Teat necrosis
   b. Actinomycosis of udder
   c. Traumatic agalactia
   d. Inverted nipples
4. Failure of milk let-down mechanism
   (responds to single injection of oxytocin)
5. Retained pig or placenta with accompanying infection
   (usually exhibits foul-smelling vaginal discharge)
6. Acute bacterial mastitis
7. Any septicemia
   (Erysipelas, influenza, hog cholera, TGE, etc.)
8. Baby pig diarrheas

Pathology

The hemograms of sows affected with MMA are essentially normal.
Lesions seen on post-mortem examination of affected sows include:
1. The musculature of the uterus, small and large intestines is atonic.
   A metritis is often present with varying degrees of purulent vaginal discharge.
2. Ovaries and thyroid glands are much smaller than normal.
3. The mammary tissue is hyperemic and congested with focal areas of bacterial mastitis.
4. Histologically, degenerative structural changes are seen in the adrenal glands. and mammary gland tissue development appears to be at a stage that would normally occur 4-5 days prior to lactation.

Prognosis

No symptoms are pathognomonic. The majority of sows recover spontaneously in 2-5 days, but usually cease lactating. Permanent udder changes may result, if untreated, because of induration of the mammary gland. Losses in sows are primarily due to degenerative changes in several organs, impaired circulation, septicemia, or hypoglycemia. If treatment is started early in affected sows, the prognosis is good.

Baby pigs often succumb to hypoglycemia or enteritis and remain as unthrifty slow gaining individuals for the rest of their lives.

MMA does not affect future fertility, nor does it impart immunity (some sows may develop the syndrome at every farrowing).

Treatment

Therapy is primarily symptomatic and geared to what works on individual farms and areas.

The following therapy regimes have been used:
1. Systemic antibiotics-penicillin, streptomycin, tetracyclines, tylosin, or any broad-spectrum antibiotic injected singly or in combination. Use is aimed primarily at combatting secondary bacterial infections.
2. Corticosteroids—used to improve clinical appearance, probably due to anti-allergic and anti-inflammatory properties.
3. Oxytocin—given to enhance milk let-down and to aid in expelling uterine contents.
4. Uterine therapy—furacin, sulfasulfadiazine, neomycin or other uterine boluses in the uterus if the cervix has already constricted, preparations such as antibiotics or antibiotic-hormone combinations may be deposited in the uterus with a bovine insemination tube.

Other products also used in therapy:
5. B-vitamins—help to stimulate sow’s appetite.
6. Antipyretics—used to decrease temperature.
7. Tranquilizers—quiet sow and potentiate the effects of oxytocin.
8. Laxatives—used to promote peristalsis and defecation.
9. Polyvalent coli antisera—used to combat the effects of secondary E. coli infections.

Sows will usually respond in 24 hours,
but in some cases retreatment may be necessary in 24-36 hours.

Treatment of affected pigs is also necessary. Pigs showing signs of hypoglycemia and diarrhea should be fed cow’s milk or milk replacer free choice or put on another sow for nursing. Pigs may also be treated with glucose injected intraperitoneally, antibiotics, vitamins, and iron.

**Control and Prevention**

Control of MMA involves proper sanitation, nutrition, environmental conditions, and perhaps a vaccination program. These may not decrease the incidence of the disease but affected sows may show decreased severity of clinical signs and respond more favorably to therapy.

The following management procedures have been used in MMA prevention.

1. Farrowing houses should be disinfected and fumigated.
2. Sows should be washed prior to entry in farrowing house.
3. Farrowing house should be well ventilated and proper comfort temperatures maintained inside.
4. Needle teeth of baby pigs should be clipped.
5. Dry bedding for pigs.
6. Sows should have a constant source of clean, fresh water.
7. Avoid any unnecessary stresses on sows by allowing them one to two days prior to farrowing to adapt to a farrowing stall.
8. During the gestation period sows should be given adequate exercise.
9. An effort should be made to see that all pigs receive colostrum.
10. Sows should be limit fed during the gestation period.
11. Bulky rations should be used prior to farrowing to prevent constipation.
12. Gilts should be selected for temperament (ability to adjust to confinement).
13. Use of bacterins—multiple injections of either mixed or autogenous bacterins during the gestation period.
14. Injection of polyvalent *E. coli* antiserum prior to farrowing.
15. Routine antibiotic and oxytocin injections after farrowing.
16. Some practitioners have recommended using high levels of antibiotics in the gestation ration. However research data indicates that this practice is of questionable value.

A ration that has been promoted as being some what effective in preventing MMA consists of: (4)

- 800 lb. Ground corn (medium grind)
- 400 lb. Wheat bran
- 200 lb. Alfalfa meal
- 200 lb. 40% Supplement
- 10 lb. Vitamin premix with:
  - 6,000,000 I.U. Vit. A
  - 1,200,000 I.U. Vit. D
  - 12,000 I.U. Vit. E
  - 5,000 mg Riboflavin
  - 45,000 mg Niacin
  - 16,000 mg Pantothenic Acid
  - 230,000 mg Choline
  - 30 mg Vit. B12
  - 500 gm Nitrofurazone

Full feed 10 days before and 10 days after farrowing.

**Summary**

MMA in sows is a disease complex associated with farrowing and characterized by partial or complete lactation failure. The primary losses from MMA are due to baby pig deaths and unthriftiness of remaining survivors. The exact etiology is not known and many different causes have been suggested. Treatment is primarily symptomatic. Prevention consists primarily of good management procedures. Until the exact etiology of MMA is elucidated we will have to rely on good management procedures and prompt early diagnosis and treatment of affected animals to minimize losses.

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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. 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 shows that approximately 86% of abortions are IBR virus etiology. 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 cavities are filled with fluid while petechial hemorrhages are widespread. The placenta undergoes a non-descript autolysis and the uterus is generally free of disease. The laboratory

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