Virus Pneumonia of Pigs

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VIRUS PNEUMONIA OF PIGS (VPP) is an inflammation of the lungs usually confined to the anterior-ventral portion and lower borders of the apical and cardiac lobes. It is usually a chronic disease that may develop into acute and subacute forms, particularly if "secondary break down" occurs due to secondary bacterial invasion. The disease is characterized by a chronic cough, consolidation of the anterior ventral portions of the lung, and secondary bacterial invasion. It is a distinct disease and is in no way related to the common swine influenza described by Shope in 1931.

Incidence of VPP

VPP is widespread throughout the major swine producing areas of the world. It is of particular importance in England and extensive research work has been done on the disease there. It is also very prevalent in the United States.

Although this disease is described as a relatively new disease in swine, just named as VPP by Betts in 1952, there is strong feeling that it has been present in swine for quite sometime. There is a report of a disease with similar characteristics in 1948 and Young reports on observing lung lesions similar to VPP in Eastern Nebraska in 1930. Just how long the disease has been present can be, at best, only pure speculation.

The lungs of over half of the pigs slaughtered in Britain are pneumonic and most of these belong to one distinct clinical and pathological entity, VPP. A virus can be isolated which is very different from the virus of swine influenza as described by Shope. Forty to seventy five percent of the pigs slaughtered in the United States show evidence of virus pneumonia. The lesions are microscopically indistinguishable from those of VPP as described in England. Studies have shown that occurrence of significant antibody titers against swine influenza is less than 15 per cent. It is worth pointing out that the incidence of lesions at slaughter does not give a true indication of the actual incidence of virus pneumonia, since experiments show that many pigs that have been injected will have recovered completely by the time marketable weight is reached.

Economic Importance

VPP does not have a high mortality rate. Rarely does a pig die from the disease unless complicated by secondary infection. The concern for VPP comes from its high incidence, conservatively estimated to effect 50 percent of the swine in the U.S. and its costliness because of slow growth and poor feed conversion. It has been shown that pigs infected with VPP require 17 per cent longer to get to market weight and require 22 per cent more feed to produce each 100 lbs. of pork. The animal loss in the United States due to slow growth and poor feed conversion has been conservatively estimated at $120,000,000.

Etiology

The etiologic agent is a virus which is capable of causing an extensive pneumonia by itself, but rarely does. In most natural infections both the secondary or-

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ganisms, P.P.L.O. and bacteria are found. The Pasturella sp. are possibly the most important bacteria involved, but other bacteria may also be involved such as *Corynebacterium pyogenes* and the streptococci. The virus is unusual in several respects; one of its most unusual features is its failure to produce any demonstrable immunity, thus precluding the possibility of using a vaccine as a control measure. Demonstration of antibodies against the virus in infected pigs has not been accomplished. Successful propagation of the virus in chick embryos has not been accomplished. Cytopathogenic effect has not been observed in tissue cultures. The only isolations made have been by passage through the natural host and this becomes difficult if the number of passages exceeds three or four.

Experimentally, the infection can be prevented by large doses of the tetracycline group of antibiotics. These antibiotics have no curative effect upon estabilshed lesions and thus are of no practical value in the treatment of the virus. The virus is not affected by chlormycetin, streptomycin, penicillin and sulfa drugs. All these drugs may cause a temporary clinical improvement by their action on secondary invading microrganisms.

The virus dies within a day outside the animal's body under farm conditions. In the lungs of the affected pigs it can persist for long periods of time and has been recovered from the lungs sixty-six weeks after infection.

The main differences between the viruses causing swine influenza and the VPP virus are the failure of any demonstratable immunity being produced by infection; failure of chick embryo propagation of the virus; and no cytopathogenic effect observed with VPP virus.

Symptoms

Pigs of all ages are susceptible to the disease, but it is most obvious in young pigs. Transmission is purely airborne and fomites play little or no part in the spread of infection. Pigs thus become infected from their dams or when diseased and healthy pigs are mixed together.

About the only obvious sign of the disease is a non-productive cough, which usually develops two or three weeks after contact. This cough may continue for the remainder of the pig's life or it may disappear after a few weeks.

The important thing about V.P.P. is that an affected pig does not thrive as well as a normal animal. Once a pig becomes infected there seems to be set up a delicate balance between the virus and secondary organisms on the one side, and the host on the other. Under conditions of poor husbandry and management the balance moves against the host, and the pig becomes more severly affected. If, however, the environment is good, the effect of the disease is minimized but is certainly not eliminated.

An interesting feature of the disease is that a number of pigs, which have contracted infection earlier in life, quite suddenly develop acute pneumonia at 19 to 26 weeks of age. This phenomenon has been termed "secondary breakdown" and is associated with the extension of a preexisting virus lesion and rapid multiplication of secondary organisms. At such times, pigs lose their appetites, become febrile and sometimes prostrate. Great difficulty in breathing is observed, and the pigs will "thump". The difficulty in breathing is a manifestation of the lesions present in the lungs. The pigs seem to eat well unless they are hopelessly sick. Although a few pigs die, the majority recover within a week or two. The malady is reflected in a sudden temporary reduction in growth rate or even an actual loss of weight. Actual weight loss and complete stunting of growth is more likely to occur under conditions of poor husbandry. Some animals are turned into hopeless runts due to permanent damage to the lungs. It is highly probable that many cases of so-called "pasteurellosis of swine" or "shipping fever of hogs" are, in fact, "secondary breakdown".

Quite apart from the spectacular effects on growth-rate caused by secondary breakdowns, the overall effect of VPP is a variable, but general depression of growth rate and efficiency of food utilization.

Lesions

The appearance of the lung in uncomplicated cases is quite typical. The apical, cardiac, and adjacent borders of the diaphragmatic lobes are the areas most commonly affected. The ventral portions of these lobes are usually consolidated, firm to the touch, and red or brownish in color. The affected lobe may have a mottled and uneven appearance due to the presence of alternate diseased and healthy lobules. A narrow streak of collapse and fibrosis may be observed along the course of the bronchial tree. The adjacent healthy lobules are usually emphysematous. If "secondary breakdown" has occurred, areas of suppuration will be found. The bronchial and mediastinal lymph nodes are enlarged and edematous and sometimes pleurisy and pericarditis accompany the pneumonia.

The characteristic microscopic changes are, lymphocytic infiltration and proliferative changes around the bronchioles with foci of interstitial pneumonia scattered throughout the lobule, often becoming confluent and more evident at the periphery. The alveoli may contain few or many cells. The alveolar lining cells usually predominate with many lymphocytes, plasma cells, a few histocytes, neutrophils, and occasionally giant cells are present. Marked hyperplasia of the lymph nodules with scattered foci of lymphocytic infiltration are constant features. Perivascular cuffing is frequently seen. Proliferative vascular lesions are also frequently seen. Varying degrees of fibrosis are present. The hyperplasia of the lymphoid tissue is, in many cases, remarkable, occurring in circumscribed masses which may cause pressure collapse of both bronchioles and alveoli.

Pattison has pointed out that histopathology seems to offer the more specific method of diagnosis of VPP. Lesions selected with the naked eye, as being typical of VPP, showed such variation, that when examined microscopically, additional causes of the lesions seemed probable. The principal histopathological finding of naturally and artifically infected animals was extensive lymphoid hyperplasia of the peribronchial, peribronchiolar, and perivascular tissues.

Pigs dying from "secondary breakdowns" frequently have in their lungs, necrotic areas which teem with bipolar organisms. Pleurisy is almost always presented and often pericarditis. The common occurrence of atelectatic lungs in swine may be due to VPP.

Differential Diagnosis

Virus pneumonia must be differentiated from other pneumonia in swine. In making a diagnosis of virus pig pneumonia, the primary disease that it must be distinguished from is swine influenza. Chronic and recurrent respiratory illnesses with "secondary breakdowns" should be diagnosed as virus pneumonia. Swine influenza may be differentiated by its acute nature, sudden onset, high temperature, anorexia and seasonal incidence. Animals suffering from "secondary breakdown" will exhibit an acute pneumonia, but the herd incidence of this acute pneumonia will be relatively low, whereas in swine influenza, which is also acute, the morbidity rate is high.

Transmission and Control

In view of the economic importance of virus pneumonia of pigs, a method of control is highly desirable. The fact that swine do not develop an appreciable immunity precludes the use of a vaccine. The antibiotics that are currently available are non-effective against the virus, although their use may give temporary improvement of clinical symptoms due to their action on secondary bacteria. The lack of a vaccine or an effective chemotherapeutic agent to control the disease places the veterinarian in a unique position as far as service to the client is concerned. Service will be rendered not through medication or vaccination, but through council and advice on how to maintain a herd of swine free of VPP and other diseases of similar nature. Such a venture will require much thoughtfulness and patience of both the veterinarian and the client.

Only control based on epidemiological consideration is possible. The following basic facts must be taken into consideration:

1. Virus pneumonia is a specific contagious disease and the only natural host is the pig.
2. The virus is not a common inhabitant of the respiratory tract of "normal pigs" with no pneumonia. If the virus is present, lesions of VPP will be present.
3. Almost indefinite persistence of the virus in the lungs, up to at least 66 weeks, presents a carrier problem by which the disease is passed from dam to offspring in succeeding generations.
4. The virus does not survive outside the natural host for more than a day under farm conditions.
5. The disease spreads solely by the airborne route; mechanical or intermediate vectors play no part in its transmission.

A control method based on epidemiological consideration has been developed and successfully applied under field conditions. It consists, essentially, of breaking the cycle of infection and is comprised of the following stages:

1. Farrowing sows in isolation to insure that any infection of the litter comes from the dam.
2. Determination of whether or not the the litter is infected by means of clinical examination supplemented by postmortem examination of one or more pigs per litter at weaning time. This method also determines the carrier sows.
3. Litters judged to be free from the disease may be grouped but retained in isolation.
4. Examination of lungs at slaughter from a considerable portion of the pigs when they reach market weight.
5. Replacement of original breeding stock with healthy progeny as soon as possible.

A more drastic method of control, but one which should be very effective is that described by Young and Underhal whereby "disease-free" pigs are obtained by hysterectomy.

Pasturella bacterin has been used on farms where VPP is a problem. Autogenous bacterin is believed to give the best results.

Brood spectrum antibotics are somewhat effective in preventing "secondary breakdown." Expectorants have been used to relieve chronic coughing but relief is usually only temporary.

Good management along with adequate nutrition will benefit pigs that are clinically sick. The elimination of dust accompanied by ascarid control is important. Dust may act as an irritant aggravating the condition. The migration of ascarid larvae through the lungs increases the severity of the lesions.

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