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PATHOLOGY OF THE MALIGNANT FORM OF FOOT AND MOUTH DISEASE IN SWINE

I. THE NATURAL DISEASE

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SUMMARY

Clinical and histological observations were made on pigs infected with Foot and Mouth Disease (FMD) on three separate pig farms in Colombia. Overall mortality rate was higher in young animals than adults. Approximately 159 fetuses were aborted.

Nineteen (19) spontaneously FMD infected pigs were necropsied. Lesions were found as follows : pedal lesions in 17 (89.5 per cent); myocardial necrosis in 16 (84.2 per cent); snout lesions in 14 (73.7 per cent); somatic muscle necrosis in 13 (68.4 per cent); liver lesions in 11 (57.9 per cent) and tongue lesions in 8 (42.1 per cent). Arteritis of the coronary vessels was observed in 4 out of eight pigs necropsied in the third outbreak. Lesions in the brain, pancreas and hairy skin were less consistent than others.

The suggestion is made that FMDV can be epitheliotropic, myotropic, or both, relative to epidemiological circumstances which will be discussed in a future paper of the present series.

Nota: Enviado a [illegible] Medicina [illegible]

INTRODUCTION

Since the isolation of the causative viral agent by Loefler and Frosh (1897). Foot and Mouth Disease (FMD) or "Fiebre Aftosa" has been well documented. The disease considered the most devastating malady of cloven footed livestock, occurs in most parts of the world with only few exceptions (Henderson, 1960).

Numerous clinical, virologic, immunologic and some histopathologic observations of the disease have been made on cattle and laboratory animals (Avellini., 1966; Gailiunas et. al., 1964; Geiger and Otte., 1958; Henderson et. al., 1948; Seibold., 1960; Skinner., 1951; Skinner et. al., 1952) but in spite of the importance of the disease in swine, information of this species is lacking.

In Colombia, attention has been directed primarily toward control of the disease in cattle. This study constitutes the first attempt to characterize some aspects of the malignant form of FMD in swine as it is seen in naturally occurring outbreaks. Major emphasis is directed toward histologic observations, with the aim of determining the most common pattern of lesions.

In the country as of June 1974, there exist two major types of FMDV, namely Vallee's A and O, and four subtypes comprising A_5, A_{18}, A_{27} and O_1.

MATERIALS AND METHODS

From October 6, 1970, through August 1973, a total of 19 swine necropsies were performed on three farms where FMD had been confirmed. Confirmation of diagnosis was based on

clinical signs, postmorten lesions and isol[illegible]n-characterization of the virus[a]. Table 1 shows the age and sex of the necropsied animals.

Tissues systematically collected for study in 10 per cent isotonic buffered formalin were : brain, spinal cord, submaxillary gland, tongue, esophagus, stomach (fundus), ileum, liver, pancreas, turbinates, trachea, lungs, hypophysis, thyroid, adrenal glands, tonsils, thymus, bronchial and mesenteric lymph nodes, spleen myocardium, aorta, kidney, urinary bladder, masseter, gastrocnemius and diaphragm muscles, pedal tissue, bone, eyes, and hairy skin.

All tissues were embedded in paraffin, cut at 5 to 6 μ thickness and routinely stained with haematoxylin and eosin. Special stains such as van Gieson, PAS, and Von Kossa were used when necessary.

First Outbreak- Vallee O, Subtype O_1

The pig farm was a breeder- fattening operation comprising 270 mixed and purebred Landrace pigs. Deaths from all causes in 1 month from the start of the outbreak numbered 82, fifty two sucklings and thirty fatteners. As the outbreak was 1 month old when reported, gross lesions varied widely in animals examined. Generally, pedal lesions were most severe with a tendency to involve all four limbs. Sick lactating sows had teat lesions and exhibited decubital ulcers. After the virus was identified, histologic studies were carried out on 6 animals.

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[a]Isolation- characterization was carried out by the Instituto Colombiano Agropecuario- Foot and Mouth Disease Diagnostic Center, located in Bogotá.

Second Outbreak- Vallee O, Subtype 0_1

The pig was a breeder- fattening operation comprising 200 mixed and purebred Landrace pigs. Death from all causes in two weeks from the start of the outbreak numbered 36, comprising 32 sucklings, two fatteners and two adults. Six pregnant sows aborted. One had fetuses approximately 20 days old and the other five had fetuses 2 to 3 month old.

Overall external lesions were extremely severe in adult animals. They were characterized by vesiculation of the snout, mammary gland of pregnant and lactating sows, and coronary bands of the feet. Sloughing of hooves was a common feature, the corium being exposed to trauma with excessive bleeding. Secondary bacterial contamination with swelling and ulceration of the skin of the metatarsal and metacarpal surfaces followed. Suckling pigs were cachectic, had muscular tremor, refused to move, and showed lesions in the snout, coronary bands, metacarpal and metatarsal surfaces. After viral identification, histological studies were carried out in 5 pigs.

Third Outbreak- Vallee A- Subtype A_{27}

The pig farm was a breeder- fattening operation comprising 1.093 mixed and purebred Landrace pigs. Deaths from all causes in 4 months from the start of the outbreak numbered 307 and 105 fetuses were aborted. Mortality was higher in fatteners (168), followed by sucklings (117), breeding females (18) and boars (4). The outbreak was extremely severe, the first 24 pigs (fatteners) dying in 48 hours. Due to the high mortality rate, the owner thought he was dealing with a hog cholera outbreak or some kind of massive intoxication. After viral identification, histologic studies were carried out in eight fattening pigs that have died in one of the pens.

RESULTS

In order of prevalence, tissues affected were : pedal epithelium, myocardium, snout, skeletal muscle, liver, and tongue (Table 2). Four of the animals that died acutely in the third outbreak had segmental necrotizing lesions involving the coronary artery (Fig. 1). This vascular damage related to the FMDV, could not be found described in the reviewed literature. Necrosis of the myocardium and somatic muscle was striking in most cases examined. The lesion varied in intensity from a rather mild mononuclear infiltration, loss of striation and homogenization of muscle fibers, to acute necrosis characterized by severe sarcolysis (Fig. 2). Dystrophic calcification was a feature in affected somatic muscle (Fig. 3). Lesions in the liver also varied in intensity. They consisted of lipid degeneration, vacuolization, with the presence of different sized hyaline bodies in hepatocytes, to frank centrilobular necrosis of the cardiac failure type. Coagulation necrosis (dry necrosis) in the tongue, was observed in only 3 of the 8 pigs that had oral lesions. This type of necrosis excluded the typical lifting of the upper most layers of the stratified squemous epithelium, that forms the full blown vesicle. Lesions in the pancreas in 4 animals were characterized by regression of acinar cells, loss of the cord like arrangement and hydropic degeneration of some islet cells. Noteworthy, was the appearance of typical FMD microscopic lesions in the ulcerated hairy skin of 4 of the affected animals (Fig. 4).

DISCUSSION

The morbidity and mortality rates were difficult to evaluate in the first two outbreaks because the disease was still in progress when the inspections were made. The third outbreak (Vallee A, Subtype A_{27}) could be followed from the start in August through November.

Overall total morbidity was 98.0 per cent. Mortality in August mounted to 21.7 per cent. In September it was 7.0 per cent approximately. In October, taking into account birth and sales, it went down to 0.0 per cent and in November it rose to 1.9 per cent. Aborted fetuses (105) were not taken into account.

Overall mortality in all three outbreaks seemed to be a higher in young animals, a factor that would indicate a greater susceptibility of piglets to FMDV. In piglets, however, in addition to myocardial and somatic muscle damage leading to cardiac failure and absorption into the circulatory system of shock producing substances from necrotic muscle, one should consider hypoglycemia as a possible cause of death. It has been demostrated experimentally, that hypoglycemic coma and death can easily be induced in baby pigs by starvation (Sampson et. al, 1942).

Blood glucose levels usually fall below 40 mg/100 ml. Animals in the early hypoglycemic stage seem to improve following dextrose therapy. This by itself, should prove an interesting study of the relationship between FMD, agalactia in lactating sows, and hypoglycemia, in baby pig mortality.

Prolonged anorexia per se, could account for the fatty changes seen in some of the livers examined histologically. The lack of availability of glucose, the increased output of growth hormone, and the heightened sympathetic nervous activity will cause the mobilization of free fatty acids from fat deposits to the liver. In favor of this stands the fact that this group of pigs (first outbreak) had been subject to a prolonged anorectic state.

The absence of similar changes in the liver of pigs necropsied in the second and third

outbreaks could be explained in the light acute deaths or early euthanasia following the onset of illness, that is, the animals were not subject to a prolonged anorectic state.

The atrophy seen in the pancreas and the hydropic degeneration of islet cells are of interest. The possibility of an association between pancreatic alterations, fatty metamorphosis of the liver, and diabetes mellitus might prove worthwhile to explore. Diabetes mellitus has been described as a sequel to FMD (Pedini et. al, 1962).

In the pig, diabetes mellitus has been described in relation to necrotic enteritis (Biester, 1925). The author shared the opinion of others, that several viral, bacterial and toxic diseases could account for the development of diabetes by bringing about anatomical, chemical or physiological alterations in the pancreas. Further association of the pancreas with the virus of FMD has been afforded by experimentally inducing acinar necrosis in mice and guinea pigs (Platt., 1956; Platt., 1958; Platt., 1959; Seibold., 1960).

Notable in the series of necropsies performed in the third outbreak was the absence of external lesions in two of the animals that died. This, in combination with a high mortality rate, could cause confusion when a rapid field diagnosis has to be made.

The present study suggests, that FMDV could behave only epitheliotropically, myotropically or both, the distribution of lesions being related to size and perhaps frequency of the challenging dose of virulent virus.

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Fig. 1 Segmental necrotizing lesion of ɔnary artery of FMD pig. H&E stain; x 60.

Fig. 2 Myocardium of FMD pig. Focus of acute necrosis characterized by severe sarcolysis (A). Intact and swollen myocytes are isolated in the center of the necrotic area (B). H&E stain; x 400.

Fig. 3 Gastrocnemius muscle of FMD pig showing severe necrosis with hyaline changes in the fibers. Some of the muscle fibers have undergone fragmentation and have piknotic nuclei (A). A small focus of distrophic calcification occurs at the right (B). H&E stain; x 400.

Fig. 4 Microscopic view of the prickle cell layer of the ulcerated hairy skin (A) showing typical FMD lacunae (B) filled with polymorphonuclear cells, H&E stain; x 400.

Table 1 The age and sex distribution of 19 pigs necropsied following infection with Foot and Mouth Disease Virus

	Sex		
Age	Male	Female	Total
0 to 56 days	1	8	9
57 to 120 days	4	1	5
121 to 180 days	5		5
Total	10	9	19

Table 2 Distribution of lesions associated with Foot and Mouth Disease in 19 pigs necropsied

Site	Number of pigs in which lesions were found	Prevalence of lesions %
Pedal Tissue	17	89.5
Myocardium	16	84.2
Snout	14	73.7
Gastrocnemius muscle	12	63.2
Liver	11	57.9
Tongue	8	42.1
Masseter	7	36.8
Pancreas	4	21.0
Hairy Skin	4	21.0
Brain	2	10.5