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CORONAVIRAL GASTROENTERITIS IN PIGS IN MALAYSIA

H.L. Too, K.L. Foo and A.R. Sheikh-Omar

Faculty of Veterinary Medicine and Animal Science,
Universiti Putra Malaysia, 43400 UPM Serdang, Selangor, Malaysia.

SUMMARY

Outbreaks of diarrhoea involving pigs of all age groups were reported by 24 farms in all the major pig rearing states of Peninsular Malaysia between the months of July and December 1994. Outbreaks of the disease were investigated in nine other farms. A diagnosis of coronaviral gastroenteritis, possibly porcine epidemic diarrhoea or transmissible gastroenteritis, was based on the presence of diarrhoea involving all pigs on affected farms, post mortem findings of severe intestinal villous atrophy and demonstration of coronavirus particles in intestinal contents of affected piglets. Based on piglet mortality, rapidity of spread and the extent of villous atrophy, porcine epidemic diarrhoea was considered the more likely suspect. The intestinal villi:crypt ratio of infected piglets ranged between 2.0:1 and 2.6:1. Experimental infection of sows showed that the incubation period was within 24 to 48 h. All outbreaks in geographically isolated farms were associated with new pig introductions while in densely populated pig areas, some outbreaks occurred without new introductions, suggesting that transmission by fomites may be less important in hot tropical environments. The nationwide outbreak also indicated a highly susceptible pig population and a deficiency in quarantine measures governing the importation of pigs.

Keywords: Porcine epidemic diarrhoea (PED), transmissible gastroenteritis (TGE).

INTRODUCTION

Epizootics of transmissible gastroenteritis (TGE) and porcine epidemic diarrhoea (PED) are almost clinically indistinguishable (Pensaert, 1992). Both are highly contagious and, in epizootic outbreaks, cause an enteric disease of pigs characterised by profuse diarrhoea in pigs of all ages, occasional vomiting and a high mortality in pigs under two weeks of age. Both are caused by coronaviruses that are antigenically distinct from each other.

The occurrence of TGE has been reported in many pig rearing countries including the United States, many European countries, Canada and some Asian countries like the Philippines, Korea, Taiwan and China (Saif and Wesley, 1992). The disease PED was first clinically recognised in the early seventies after outbreaks of acute diarrhoea with clinical features similar to TGE were reported in England and Belgium. Since then, the presence of PED was demonstrated by the presence of antibodies or by virus isolation in several countries in Europe, United Kingdom, Taiwan and China (DeBouck *et al.*, 1982, Pensaert, 1992).

Although there is little published information available about the occurrence of viral gastroenteritis in pigs in Malaysia, outbreaks of TGE involving many farms throughout the peninsula were thought to have occurred in the mid-1970s. The outbreak was self limiting and further outbreaks have not occurred possibly due to the warm tropical climate which may be too hostile for this heat labile virus (Too, 1993).

The aim of this paper is to report epidemiological and clinical features of a nationwide epidemic of coronaviral gastroenteritis in pigs in 1994.

MATERIALS AND METHODS

Postal survey

Two hundred and sixty questionnaires were sent in December 1994 to pig farms via the Federation of Livestock Farmers' Associations of Malaysia, State Livestock Associations (in Penang, Perak, Selangor, Negri Sembilan, Malacca and Johore) including farms registered for export. In each questionnaire, the syndrome of sudden onset of diarrhoea involving all age groups was described and the information solicited included farm location, whether there was a recent outbreak of diarrhoea that involved both sow and piglets, the month and duration of the outbreak, history of new introduction, source of the introduction and the estimated mortality rates of the different age groups within the farm.

Field investigations

The outbreak was further investigated in nine farms reporting outbreaks of diarrhoea involving all pigs between August and December 1994. The farms

were located in Selangor (3 farms), Negri Sembilan (3 farms), Malacca (1 farm) and Johore (2 farms). Post mortem examinations were conducted on dead and dying piglets in the affected farms. Small intestinal contents were collected in viral transport medium, kept chilled at 0°C and sent to Universiti Pertanian Malaysia (UPM) where virological and negative contrast electron microscopic examination were carried out.

In early to mid 1995, an outbreak of a similar enteric syndrome was also reported in Sarawak. Samples of pooled intestinal contents from two pig farms in Kuching, Sarawak were sent to UPM. The samples were also examined under negative contrast electron microscopy.

Epidemiological study of an outbreak

In a 1,000-sow pig farm in Negri Sembilan, an outbreak of diarrhoea was first detected on 14 November 1994.

Piglet mortality

The preweaning mortality of 112 litters involving 1138 piglets farrowed between the months of October and November 1994 was monitored daily to determine the relationship between the age of the piglets at infection and the mortality rate. All clinical observations were recorded and dead piglets were autopsied.

Experimental infection of sows

Forty two pregnant sows were fed fresh minced intestines derived from freshly killed piglets that had been clinically affected. The sows were individually housed in gestation stalls in a shed that contained a total of 120 sows. The 42 sows had not been observed to have had diarrhoea and were separated from each other by at least one sow that had not been so exposed. The latter sows were regarded as in-contact sows. The sows were observed daily and the onset and duration of diarrhoea were recorded.

Pathology of small intestine

One litter of 12 one-day-old piglets developed diarrhoea. One piglet daily from day 1 to day 5 of birth was sacrificed by means of intravenous pentobarbital sodium. The seven remaining piglets died. Two piglets from another affected litter which had diarrhoea for 6 and 7 days respectively were also available. One piglet from an unaffected litter served as a control.

Histopathologic sections of the small intestine measuring approximately 1.5 cm^2 from five different regions, namely the duodenum, proximal jejunum, mid jejunum, distal jejunum and terminal ileum were prepared by routine methods and examined by light microscopy. The villus length to crypt ratio of all intestinal sections was estimated using a method previously described (Moon *et al.*, 1975).

RESULTS

Postal survey

Of the 260 questionnaire forms mailed, 48 (18.5%) were returned. Of the latter, 24 farms (50%) reported the presence of an outbreak of diarrhoea involving pigs of all ages (Table 1). Except for Johore, the disease was reported in all the major pig rearing areas in Perak, Selangor, Negri Sembilan and Malacca. Nine out of twenty farms in Perak that responded to the survey reported outbreaks of the disease. The farms were located in Ipoh, Tronoh, Pantai Remis, Kampar and Bidor. In Selangor, the disease was also reported from farms in Sepang, Tanjung Sepat and Tanah Merah site "A". In Malacca, known affected areas were Kuala Sungai Baru and Mengkuang. Pig farmers from Perak and Selangor were the most cooperative with 28.6% and 32.5% of farms respectively responding to the survey while those from other states were not as forthcoming (Table 1).

Table 1. Number and distribution of pig farms responding to a postal survey of an outbreak of a TGE-like disease in Peninsular Malaysia

State	Questionnaires		No. farms reportedly affected
	No. sent	No. returned	
Penang	40	2	0
Perak	70	20	9
Selangor	40	13	10
N. Sembilan	40	3	2
Malacca	30	3	2
Johore	40	6	0
Unknown		1	1
Total	260	48	24

Twenty farms reported that the onset of the outbreaks was sudden and occurred between the months of July and December, 1994. Of these, four, six and eight farms reported that the onset was between July - August, August - September and September - October, 1994 respectively. At the time of the survey (i.e. December 1994), only two farms have reported that the outbreak was over and estimated that the duration of the outbreak was about three months. Fifteen respondents reported that the outbreak was not over (at the time of the survey). The earliest that the outbreak was reported was in July and the farms affected were from Perak and Selangor.

Eleven respondents admitted that there were new introductions to the farm prior to the outbreaks although the time between introduction and the observed onset of the disease was variable. Of these eleven respondents, nine bought their pigs from local sources while two farms imported pigs from overseas. One farm in Sepang reported that the outbreak occurred shortly after the introduction of a boar from overseas in the month of July, 1994.

The age group of pigs that were first observed to be affected in the outbreak varied from farm to farm. According to the respondents, the disease was first observed in the sows (3 farms), lactating sows and sucklers (1 farm), sucklers (3 farms), weaners (4 farms) or growers (10 farms). Most of the farms in Perak, reported that the disease was first noticed in either the sows or sucklers while those from Selangor (especially Tanjung Sepat) reported that the disease first occurred in grower pigs.

Only one respondent reported that one sow died; the rest reported that there was no sow mortality. Mortality rates among suckers, weaners and growers were estimated to be 25-50%, 10-20% and 0-10% respectively.

Field investigations

Outbreaks in the nine farms investigated occurred between the months of August and December. All except two farms had a history of recent introductions of new pigs within one week prior to the outbreak. All the introductions were from local sources. Of the two farms that had no history of new introductions prior to the outbreak, one was located in Sepang and the other in Tanjung Sepat. Both farms were located beside other pig farms. Four of the farms purchased breeding gilts while three brought in recently weaned pigs. Except for one farm in Negri Sembilan which purchased a group of weaned pigs from Sepang, none of the other farms would reveal their source.

In all farms, common observations include a sudden onset of profuse watery diarrhoea which eventually spread to involve all age groups. While the onset was reported as sudden, the speed of spread to other pigs in the farm appeared variable, ranging from four to eight weeks. The spread among suckling pigs the spread in the weaner sheds. The spread within the dry sows and the grower groups were the slowest. In some farms, the disease in preweaned pigs lasted for about one month. The duration of the outbreak varied from three to five months depending upon the size of the farm. In two farms attended by the senior author from the beginning of the outbreak, the clinical disease was still detected in weaned pigs more than three months after the initial outbreak.

The nature and odour of the diarrhoeic faeces of the various age groups appeared to be influenced by the diet; the faeces of sucklers being more watery, containing curds of undigested milk (Figure 1) and the odour more offensive. Vomiting was more often observed in suckling pigs than in other age groups. The vomitus from preweaned pigs resemble undigested milk curds. No systemic illness was observed in other pigs except in lactating sows which exhibited anorexia lasting for about two to three days. There was an age related susceptibility where the younger the pig, the higher the mortality. While the mortality rate among affected pigs within the first two days of life was almost 100 per cent, the overall mortality in pigs below one week old was estimated to be less than 50%. Most of the pigs affected after two weeks of age survive with minimal lasting effects. Nearly all the farmers believed that the use of antibiotics together with electrolytes especially in piglets was more effective in reducing the severity of diarrhoea and mortality than the use of electrolytes alone. Clinical signs in adult pigs were limited to diarrhoea, inappetance and an occasional pig vomiting. Adults usually recovered within a week or so.

Figure 1. A litter of piglets affected with coronaviral gastroenteritis. Note diarrhoeic faeces resembling undigested milk on the floor beneath the farrowing crate.

On post mortem examination, all piglets that died spontaneously showed severe dehydration with sunken eyes (Figure 2). The small intestines grossly appeared thin walled and translucent with diarrhoeic faeces often visible from the serosal surface (Figure 3). In all cases,

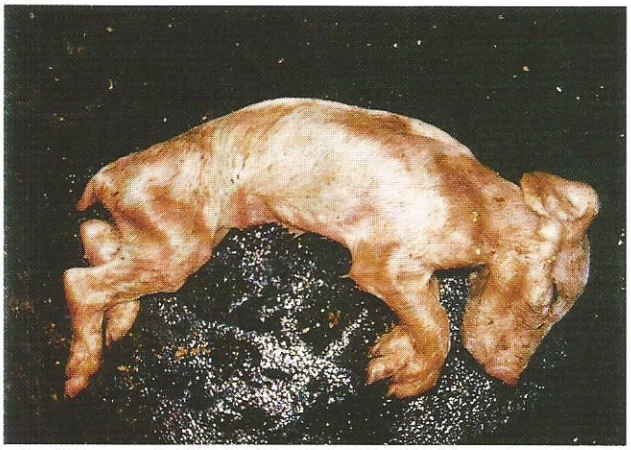

Figure 2. A grossly dehydrated two-day old piglet from a farm with an outbreak of coronaviral diarrhoea.

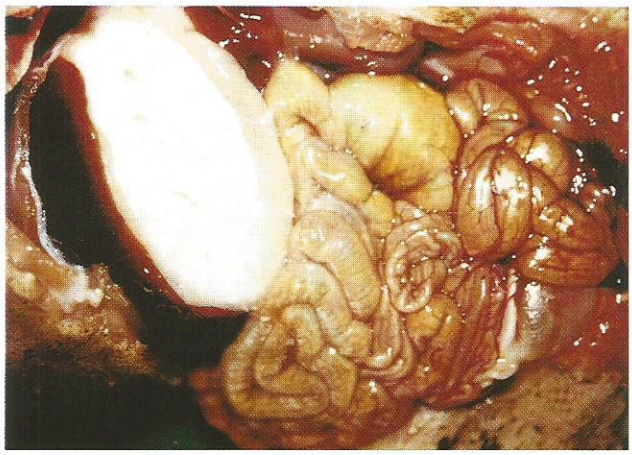

Figure 3. Undigested milk curd in the stomach and translucent small intestines in a piglet with viral gastroenteritis. Translucency indicated by the visibility of faeces from the serosal surface.

Coronavirus particles were detected in phase contrast electron microscopy from four samples of intestinal contents taken from piglets in the acute phase of the disease (Figure 4). The particles were pleomorphic and many of the particles appeared to have lost their spikes. The diameter of the virus ranged from 100 to 130 nm (excluding the spikes). Such virus particles was also similarly demonstrated in the samples from Sarawak. Attempts to isolate the virus were unsuccessful.

Epidemiological study of an outbreak

Piglet mortality

There was an increase in the preweaning mortality rate in the litters born between 25 October and 25 November 1994 with piglets born after 14 November experiencing a higher mortality (Table 2).

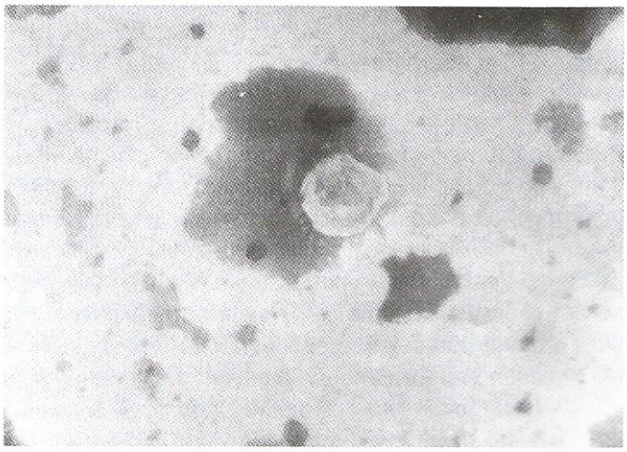

Figure 4. Electron micrograph showing one of the coronavirus particles from faeces of a piglet affected with diarrhoea. (X 100,000)

Experimental infection of sows

Of the 42 sows that were fed minced intestines from piglets that have died or sacrificed during the acute phase of diarrhoea, eight (19%) developed diarrhoea. Of these, six showed signs of the disease within 24 h while the remaining two developed diarrhoea after 24 h following experimental infection. About twenty four hours subsequent to this, five in-contact sows also showed signs of diarrhoea. Another 21 out of 120 sows in the same shed showed diarrhoea within the next 48 h. The duration of diarrhoea in the sows varied with 52%, 38% and 10% of the sows having had diarrhoea for one, two and three days, respectively.

Table 2. Preweaning mortality of pigs born in one month in 1994 in a piggery before and after the onset of an outbreak of viral gastroenteritis. (onset on 14 November).

No. farrowed	Date farrowed	Mortality rate (%)
190	25 Oct - 31 Oct	19.5
280	1 Nov - 7 Nov	25.0
352	8 Nov - 14 Nov	33.2
211	15 Nov - 21 Nov	35.1
105	22 Nov - 25 Nov	37.1

Pathology of small intestine

There was severe villous atrophy in the different regions of the small intestines of the piglets examined (Table 3). There appeared to be less severe intestinal villous atrophy in the pig that survived until day 7 of clinical disease.

Table 3. Mean villus length:crypt depth ratios in different sections of small intestines of piglets between day 1 and day 7 of birth. (Day 1 of birth = day 0 of diarrhoea)

Day of birth	villus length:crypt depth ratio					Mean
	duodenum	proximal jejunum	mid jejunum	distal jejunum	terminal ileum	
1	a	a	a	a	a	a
2	2.3:1	3.2:1	3.3:1	1.9:1	2.0:1	2.5:1
3	1.0:1	4.0:1	2.1:1	2.1:1	1.3:1	2.1:1
4	b	b	b	b	b	b
5	2.2:1	2.3:1	1.9:1	2.1:1	1.7:1	2.0:1
6	1.9:1	2.5:1	1.6:1	2.9:1	2.1:1	2.2:1
7	2.8:1	3.0:1	2.4:1	2.7:1	2.3:1	2.6:1
control	5.6:1	6.0:1	5.7:1	6.3:1	na	5.9:1

a = severe necrosis with complete loss of histological architecture
b = autolysed
na = not available due to poor section quality

DISCUSSION

The diagnosis of viral gastroenteritis due to coronavirus was based on the clinical syndrome of diarrhoea involving pigs of all ages, post mortem findings of severe intestinal villous atrophy and the demonstration of coronavirus particles in the intestinal contents of affected pigs during the acute phase of the disease. The two diagnostic possibilities were transmissible gastroenteritis and porcine epidemic diarrhoea, both of which are regarded as clinically indistinguishable (Pensaert, 1992). Other infectious causes of piglet diarrhoeas such as rotavirus and *Escherichia coli* can be excluded here due to the simultaneous presence of disease in adult pigs (Pensaert, 1992). However, the exact aetiological agent was not identified due to the failure to isolate the virus. The spread of the disease within individual farms in the present study was very variable (four to eight weeks). Pig farmers suggested that the present outbreak was different from outbreaks of TGE in the 1970s where piglet mortality rate was higher and the on-farm spread was faster than in the present outbreak. PED appears to be the more likely suspect because PED is known to take as long as one month to spread to all the age groups within a farm during an acute outbreak (Pensaert, 1992) while epizootic TGE infection usually spreads to involve most animals in the farm within a matter of days (Maes and Haelterman 1979).

In TGE, the intestinal villous atrophy is more severe than in PED. In TGE-infected piglets, the villi:crypt ratio was about 1:1 (Hooper and Haelterman 1969) and in PED-infected piglets, the ratio was about 3:1 (DeBouck *et al.*, 1981). The intestinal villi:crypt ratios of naturally infected piglets in the present study ranged between 2.0:1 and 2.6:1 and are therefore more consistent with PED.

Based on the replies to the questionnaire survey, the outbreak involved farms in nearly all the major pig rearing areas in Perak, Selangor, Negri Sembilan and Malacca. The senior author has also investigated outbreaks of the disease in two farms in Johore. Although only three out of 40 farmers surveyed in Negri Sembilan responded to the survey and only two farms admitted the presence of outbreaks, it is possible that many farmers have not reported the presence of the disease. However, the senior author's field investigations indicated that many farms in Negri Sembilan as well as some farms in Johore were similarly affected. Field reports from Sarawak also indicated the presence of a disease similar to that in Peninsular Malaysia. The detection of coronavirus particles in faecal samples from two pig farms in Kuching, Sarawak suggests that the outbreak spread to East Malaysia probably via the smuggling of pigs from Peninsular Malaysia. The occurrence of classical swine fever in Sarawak at the same time was also thought to be due to the smuggling of pigs from Peninsular Malaysia.

It is reasonable to assume that a nationwide outbreak of viral gastroenteritis must have resulted from the virus being imported from overseas since the disease is not known to be enzootic in Malaysia. Epizootics of this nature also indicate that the pig population in this country is highly susceptible. The present outbreak of the disease also indicates a deficiency in the quarantine measures governing the importation of pigs. There is no known carrier status for either of these two diseases. Pigs are not known to excrete the TGE virus for longer than two weeks (Pensaert *et. al.*, 1970). Experimentally, the PED viral antigen can be detected in faeces up to seven days after inoculation (Callebaut *et al.*, 1982). This means that if

shorter than two weeks. This outbreak also reveals a great deal about the nature of the trade in pigs in this country. Assuming that the disease entered the country through the importation of breeding pigs by a pig breeder farm, the rate of subsequent spread of the disease within the country is alarming in that at least 24 farms in different states are affected within a span of a few months. The outbreak is even more extensive if we were to assume that the number of farms affected based on the response to a postal survey is likely to be greatly underestimated. Since the most common method of spread is through the infected pig and a carrier status in pigs for either of these diseases has yet to be proven, the rapidity of spread would indicate that there is an extensive trade in live pigs between farms. Pig farmers in Malaysia can be said to be virtually exchanging pigs.

While many farmers admitted that there has been recent introductions of pigs into the farm prior to the outbreak, there are some cases in which there were no history of any recent introductions. However, in nearly all of the latter where the veracity of the history was not in doubt, the outbreak was first noticed in finisher groups of pigs and that these farms were situated in high pig density areas with other pig farms located in close proximity. It is possible that the virus could be spread passively by virus-contaminated clothing or shoes of pig catchers that go from farm to farm within these areas. In at least one farm in Selangor, an outbreak occurred among the pigs in the finisher pens a few days after market pigs were collected by pig catchers who have visited farms in the vicinity. However, in pig farms that are situated in isolated areas, outbreaks were associated with the introduction of new pigs despite the fact that some of these farms were also visited by the same pig catchers. This would appear to indicate that although the virus can be spread by fomites, the time interval between the farms visited may be an important factor. This is understandable since the TGE virus has been shown to be heat labile, losing its infectivity when held at 37°C for four days, (Harada *et al.*, 1968) as well as highly sensitive to sunlight (Haelterman, 1963) or ultraviolet light (Cartwright *et al.*, 1965). Although much less is known about the PED virus, the cell-adapted strain was shown to be slightly more heat stable, losing its infectivity when heated at 60°C for 30 min (Hoffmann and Wyler, 1989). This could probably explain why the spread by fomites is more likely to occur with pig catchers visiting farms located within the same densely populated area since the time interval between farms would be much shorter than would be the case with those farms that are located in isolated areas.

The incubation period of the disease in the present study is less than 24 h and this is consistent with earlier observations of viral gastroenteritis in pigs (Pensaert, 1992; Saif and Wesley, 1992). The short incubation period is deduced from the fact that the disease can be seen in day-old pigs and within 24 to 48 h following experimental infection of sows with minced intestines

It is interesting to note that only eight out of 42 sows that were so treated developed diarrhoea. It is possible that the minced intestines used did not contained sufficient viral particles to constitute an infective dose. It is also possible that these sows may have been naturally infected earlier but escaped detection. Consequently, these sows could have been already immune at the time of experimental infection. Although diarrhoea is the predominant clinical sign, it was observed that some sows became inappetant for a day or so within 24 to 28 h following experimental infection and subsequently recovered without showing signs of diarrhoea. When these sows eventually farrowed, their piglets were apparently protected from clinical disease. This would appear to indicate that even sows that do not develop diarrhoea can become actively immune following infection.

The duration of diarrhoea in sows that developed diarrhoea spontaneously or following experimental infection is between one and three days. This is characteristic of coronaviral gastroenteritis (Pensaert, 1992; Saif and Wesley, 1992).

The increase in preweaning mortality in the farm since November 1994 (Table 2) indicates that litters that were born after mid-November had a higher mortality while those that were born earlier (i.e. older) had a lower mortality. This shows that the younger the piglet at the time of infection, the higher the mortality. Therefore, a retrospective examination of the data on preweaning mortality on a farm which has had a previous outbreak of coronaviral gastroenteritis should show a bell-shaped curve as the piglet mortality can be expected to drop as more actively immune sows start to farrow later in the course of the outbreak.

Microscopic examination showed villous atrophy in all five different regions of the small intestine studied in naturally infected piglets in the present study. In TGE-infected pigs, the villous atrophy appeared to be more severe in jejunum and to a lesser extent in the ileum and was often absent in the proximal duodenum (Hooper and Haelterman 1969). In experimental infection studies (DeBouck *et al.*, 1981), where 3-day-old piglets were infected with the PED virus, extensive villous atrophy was reached between 24 and 26 h post inoculation whereas those caused by the TGE virus occurred more rapidly reaching a maximum villous shortening by 18-24 h post inoculation (Pensaert 1992). In the present study, extensive intestinal villous atrophy was detected in day 3 and day 6 of birth (Table 3). Since diarrhoea was first observed at day 1 of birth, the progression of the lesion appeared to have greater similarity to those previously described for PED (DeBouck *et al.*, 1981).

In conclusion, while TGE and PED are almost clinically indistinguishable, the clinical and epidemiologic features of the outbreak in the present report appear to have a greater resemblance to those of PED. Since there is no cross immunity between these coronaviruses, future outbreaks are distinct possibilities

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RINGKASAN

GASTROENTERITIS KORONAVIRUS DALAM BABI DI MALAYSIA

Wabak cirit birit yang melibatkan semua kumpulan umur telah dilaporkan dalam 24 buah ladang di kesemua negeri menternak babi utama di Semenanjung Malaysia di antara bulan Julai dan Disember 1994. Diagnosis gastroenteritis koronavirus, mungkin cirit birit epidemik atau gastroenteritis boleh pindah, adalah berasaskan wujudnya cirit birit yang melibatkan kesemua babi dalam ladang yang terkesan, penemuan pos mortem iaitu atrofi vilus usus teruk dan penunjukkan zarah koronavirus dalam kandungan usus anak babi terkesan. Berasaskan kadar kematian anak babi, kecepatan sebaran dan kadar atrofi vilus, cirit birit epidemik porsin telah anggap kejadian paling mungkin. Nisbah villi:krip anak babi terjangkiti adalah di antara 2.0:1 dan 2.6:1. Jangkitan ujikaji terhadap babi betina dewasa menunjukkan yang tempoh penginkubatan adalah di antara 24 dan 48 j. Kesemua wabak dalam ladang terpencil secara geografi telah terkait dengan pengenalan babi baru sambil dalam kawasan babi berpopulasi tinggi, setengah wabak berlaku tanpa sebarang pengenalan baru, dan ini menyarankan yang pemindahan melalui fomit mungkin merupakan perkara yang kurang penting dalam persekitaraan tropika panas. Wabak di seluruh negara juga menunjukkan populasi babi rentang tinggi dan kekurangan dalam langkah kuarantin yang mengawal pengimpotan babi.