

An Abattoir Survey on the Pathology of Swine Livers

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ABSTRAK

Satu kajian patologi pada hati babi yang telah rosak dijalankan selama lima minggu di rumah sembelih Shah Alam. Satu ratus hati telah dikumpulkan, 75 daripadanya ialah babi 'porker' dan 25 babi betina tua. Lesi-lesi yang dilihat ialah 36 'milkspots', 16 perihepatitis, 12 abses, 11 kolangiohepatitis, 8 kolangitis, 5 hiperplasia nodular, 4 nekrosis, 3 'post necrotic scarring', 2 lipidosis dan satu 'cystic bile duct hyperplasia', 'massive necrosis' dan hepatokasinoma. 'Milkspots' akibat daripada perpindahan larva Ascaris suum adalah satu masalah besar terutama dalam babi porker (41%). Empat belas (39%) daripada hati 'milkspot' diberikan gred lesi 4+ di mana semua lobanya terlibat. Perihepatitis dilihat hanya dalam babi porker dan hiperplasia nodular hanya dilihat dalam babi betina tua. Escherichia coli dan Kliebsiella sp. adalah dua spesies bakteria yang kerap diasingkan daripada 12 abses. Penyakit-penyakit yang mungkin menyebabkan lesi-lesi yang dilihat dibincangkan.

ABSTRACT

A five-week study on the pathology of condemned livers of 100 pigs comprising 75 porkers and 25 sows at Shah Alam abattoir was conducted. The liver lesions consisted of 36 with milkspots, 16 with perihepatitis, 12 with abscessations, 11 with cholangiohepatitis, 8 with cholangitis, 5 with nodular hyperplasia, 4 with focal necrosis, 3 with post-necrotic scarring, 2 with lipidosis and one case each of cystic bile duct hyperplasia, massive necrosis and hepatocarcinoma. Milkspots due to migration of Ascaris suum larvae was a major problem in porkers (41%). Fourteen (39%) of the milkspot livers were severely affected having a 4+ lesion score with all the lobes affected. Perihepatitis was seen only in porkers while nodular hyperplasia only in sows. Escherichia coli and Kliebsiella sp. were the common bacteria isolated from the abscesses. The possible causes of the lesions are discussed.

INTRODUCTION

The liver is the principal organ of metabolism for many endogenous and exogenous substances and as a result is one of the most frequently damaged organs in the body. It has a large capacity to regenerate in response to injury and as a result livers of clinically healthy animals show a spectrum of disease conditions at slaughter. In a study of 8,558 swine from an abattoir in Malaysia, the causes of condemna-

tions of livers comprised 0.12% with milkspots, 0.06% with cirrhosis, 0.05% with fatty change, 0.02% with abscesses and 0.01% with other changes (Tham and Sheikh-Omar, 1981). In another study, the majority (71.4%) of livers of 5,466 swine was condemned due to milkspots (Yap *et al.*, 1983).

This paper reports a survey on the pathology of livers of pigs slaughtered at the Shah Alam abattoir near Kuala Lumpur.

MATERIALS AND METHODS

One hundred condemned livers of pigs slaughtered at Shah Alam abattoir were collected on several visits to the abattoir in May and June, 1984. Most of the pigs slaughtered had come from Selangor and Negeri Sembilan and occasionally from Province Wellesly and Perak.

Each liver was examined for gross abnormalities. From each liver tissue, samples were taken, one each from the four main lobes and from any area that showed pathological changes. The specimens were fixed in 10% neutral buffered formalin and later embedded in paraffin wax and 4 μ m sections were stained with haematoxylin and eosin and where necessary, selected sections were also stained by the Masson's trichrome method for connective tissue and the Gram's stain for gram positive and negative bacteria.

The surface of all abscesses present was lanced and the pus sampled aseptically with

cotton swabs and cultured on blood agar and MacConkey agar aerobically at 37°C for 24 hours and anaerobically in an anaerobic chamber at 37°C for 48 hours. The organisms isolated were identified based on Gram reaction, microscopic morphology, colonial characteristics as well as standard biochemical techniques (Cowen, 1977).

RESULTS

Of the 100 condemned livers examined, 75 were from porkers and 25 were from sows. A combination of lesions was present in most livers. The most predominant lesions seen in each liver are presented in Table 1. The common lesions were milkspots (36%), perihepatitis (16%), abscessations (12%) and cholangiohepatitis (11%). In porkers, the most common lesions were milkspots (41%) and perihepatitis (21%) while in sows, cholangiohepatitis (20%), nodular hyperplasia (20%) and milkspots (20%) were most common.

TABLE 1
The most prominent lesions observed in condemned livers*

Lesion	Number of livers		
	Porker	Sow	Total
Milkspots	31	5	36
Perihepatitis	16	—	16
Abscessation	9	3	12
Cholangiohepatitis	6	5	11
Cholangitis	7	1	8
Nodular hyperplasia	—	5	5
Focal hepatic necrosis	3	1	4
Post necrotic scarring	1	2	3
Hepatic lipidosis	2	—	2
Cystic bile duct hyperplasia	—	1	1
Massive necrosis	—	1	1
Hepatocarcinoma	—	1	1
Total	75	25	100

*a combination of lesions was observed in most livers.

Milkspots were the most common lesions seen. Grossly, they were small stellate white spots, 0.1 cm to 1.5 cm in diameter. In a few livers, the bigger spots tended to have a nodular cystic centre of 0.1 cm diameter. The right lateral lobe and left and right medial lobes were most commonly affected. The lesions scored according to the number of lobes affected are shown in Table 2 and most livers had a score of 4+ in which all the lobes were affected. Microscopically, there was mild to marked focal fibrosis, biliary hyperplasia and cellular infiltration with predominantly eosinophils and lymphocytes.

TABLE 2
The severity of milkspot lesions in 36 pigs

Lesion score*	Number of livers affected
4+	14
3+	8
2+	3
1+	11
Total	36

*4+ — all lobes affected
3+ — three lobes affected
2+ — two lobes affected
1+ — one lobe affected

Perihepatitis was seen in 16 animals (16%), all of which were porkers. The affected livers had thickened and cloudy capsule, sometimes with fibrin tags causing fusion of the lobes and adhesion to the diaphragm. In severe cases, the fusion was complete and the liver appeared as one large mass. One liver was severely affected and adhered to the diaphragm. Microscopically, there was capsular and subcapsular fibrosis.

Abscessation was seen in 12 animals comprising 9 porkers and 3 sows. The abscesses ranged from 0.1 cm to 1.5 cm in diameter appearing as one focus or multifoci and affecting all lobes. The abscesses contained caseated, creamy whitish to greenish yellow material which was sometimes dry and calcified. The species of

bacteria isolated in mixed cultures and their isolation frequency are shown in Table 3. The most common isolates were *Escherichia coli* and *Kliebsiella* sp. One abscess each was positive for *Salmonella* sp. and *Pseudomonas pseudomallei*.

TABLE 3
Bacteria isolated from 12 hepatic abscesses

Species	Frequency of isolation*
<i>Escherichia coli</i>	7
<i>Kliebsiella</i> sp.	5
<i>Staphylococcus aureus</i>	3
<i>Citrobacter freundii</i>	2
<i>Proteus</i> sp.	2
<i>Corynebacterium pyogenes</i>	1
<i>Citrobacter intermedius</i>	1
<i>Pseudomonas pseudomallei</i>	1
<i>Proteus rettgeri</i>	1
<i>Salmonella</i> sp.	1

*in mixed cultures

Cholangiohepatitis was diagnosed microscopically in 11 animals (11%) comprising 6 porkers and 5 sows. Histopathological findings included fibrous tissue replacement of liver lobules, biliary hyperplasia and periportal infiltration by lymphocytes and eosinophils. Eight livers had cholangitis, 7 from porkers and one from a sow. Histologically, mild to moderate fibrosis with marked eosinophil infiltration in the portal triads were present.

Nodular hyperplasia was observed in sows only. The affected livers had a few firm nodules which were reddish on the cut surface and separated from normal liver parenchyma by a thin capsule. Histologically, the nodules consisted of normal hepatocytes but without the normal lobular architecture and were surrounded by a thin layer of fibrous tissue.

Four livers had focal hepatic necrosis, microscopically seen as focal areas of hepatocytes undergoing pyknosis, karyorrhexis and karyoly-

sis. Post necrotic scarring was seen in 3 livers which had typically marked periportal and portal fibrosis as well as distortion of hepatic lobules.

One case each of cystic bile duct hyperplasia, massive necrosis and hepatocarcinoma were also observed in sows. The first had cystlike dilations of bile duct with hyperplastic cuboidal epithelium. Massive necrosis was seen in a brown and friable liver which histologically revealed necrosis of hepatocytes of entire lobules which were filled with erythrocytes. The liver with hepatocarcinoma was enlarged and had a single raised nodule which appeared homogeneously brown and multilobulated on the cut surface. Histologically, there was loss of normal liver architecture with neoplastic cells present as solid masses separated by connective tissue septa. The cells were large round with prominent round to ovoid hyperchromatic nuclei and had no resemblance to normal hepatocytes.

DISCUSSION

The high frequency of livers with milkspots in this study is consistent with the findings of other workers (Bottle *et al.*, 1975; Polley and Mostert, 1980). This means that infection with the causative agent *Ascaris suum* is a problem in porkers in Malaysia and is most likely due to improper deworming schedules and poor sanitary measures at the farms. However, work done overseas has shown that *A. suum* infection still approaches 100 percent despite widespread use of anthelmintics and confined housings (Froe II, 1982). Egg of *A. suum* are very resistant to harsh environmental conditions and have been found to remain viable in manure collection pits up to 14 months (Smith, 1979). Untreated, infected pigs act as an important source of infection to other pigs by shedding 'infective' eggs when they reach 8 weeks of age thus contaminating the stalls (Froe II, 1982).

The pigs studied were clinically healthy. They were infected with *A. suum* probably at a grower phase and were able to acquire immunity against the parasite. Had the infection developed during the prestarter and starter phases, high

mortalities and growth retardation would have been prominent (Froe II, 1982). Segments of ascaris larvae and aggregations of lymphoid cells were not observed in this study because the lesions had been going on for some time. Such segments have been detected 21 days post infection with ascaris (Copeman and Gaafar, 1972).

It was interesting to find perihepatitis in this study but the cause has remained unestablished. Copeman and Gaafar (1972) described the possibility of ascarid larval migration causing clouding of the capsule 24 hours after infection and progressing to opacity by the 12th day. *Mycoplasma hyorhinis* and *Haemophilus parasuis* are the other possible causes (Leman *et al.*, 1981). In this study, all the affected livers had marked eosinophil infiltration suggestive of parasitic involvement.

The isolation of bacteria from the liver abscesses in mixed cultures mainly consisting of *Escherichia coli*, *Klebsiella* sp. and *Staphylococcus aureus* are similar to the findings of other workers (McCracken and McCaughey, 1973; Engvall and Schwan, 1983). The one liver abscess due to *Pseudomonas pseudomallei* had typical microscopic lesions of melioidosis (Omar, 1963; Thomas *et al.*, 1981).

Nodular hyperplasia which was seen in sows was consistent with the findings of Hayashi *et al.* (1983) who reported an incidence of 40 per million pigs and suggested hepatocarcinogens as a possible cause.

Hepatocarcinoma was the only neoplasm seen. Its occurrence is low in pigs, occurring at the rate of one per 5.5 million pigs (Moulton, 1978).

Hepatic lipidosis as seen in this study has been reported by Yap *et al.* (1983) at a higher frequency (11.9%). This problem usually arises when the fat is mobilised too rapidly from various fat depots when the animal is undernourished (Jubb and Kennedy, 1970). It is also seen in pigs suffering from severe protein malnutrition causing great reduction in hepatic phospholipid and disturbance in lipid metabolism (Gupta, 1973).

The source of the pigs studied could not be traced due to complete lack of history and hence epidemiological inferences could not be made. However the data presented do provide further information on the spectrum of disease conditions occurring in livers of slaughtered pigs.

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