Polychlorinated Biphenyl Poisoning in Chickens

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About two million chickens, including broilers and layers, were affected by an unknown acute disease showing high fatality in the western part of Japan in 1968. At the beginning, some feed poisoning was suspected in the field but the real cause of this disease was not immediately ascertained.

Meanwhile, a human disease characterized by the acne-like exanthema occurred almost simultaneously with the outbreak of the chicken disease. Because it was confirmed that the diseased persons had eaten the particular edible rice-bran oil manufactured by "K" company, the disease was named "Yusho" meaning oil disease. Then the Yusho Research Group was organized.

As a result of the investigations[7),8)] carried out by this research group, the causal agent of "Yusho" was clarified to be a kind of polychlorinated biphenyl (PCB), "Kanechlor 400". This substance had been used at "K" company as a heating medium for deodorizing rice-bran oil and was mixed in the oil accidentally in the manufacturing process.

"K" company had also produced "dark-oil" as a by-product of edible rice-bran oil. This by-product was mingled with the commercial assorted ration for chickens. Subsequently, it was proved that the "dark-oil" contained a lot of "Kanechlor 400" which might be easily transferred from the polluted rice-bran oil. This evidence was beneficial in verifying that the unknown chicken disease was PCB poisoning as well as "Yusho" in humans.

The authors[1),2),5)] had a chance to make clinical and pathological observations on field cases of this poisoning. Furthermore, the following experiments were attempted; (1) experimental reproduction of poisoning by administration of the assorted ration by which the poisoning in the field occurred, (2) experimental reproduction of poisoning by administration of "dark-oil" manufactured in "K" company and suspected to be poisonous, (3) the experiment inquiring about the relationship between the concentration of "Kanechlor 400" administered and the severity of poisoning, and (4) chronological observations on the poisoning due to "Kanechlor 400" administration. Through these experiments, the field cases were proved to be a poisoning caused by a kind of PCB, "Kanechlor 400".

Clinical signs

Diseased birds in broiler farms manifested droopiness, reduced appetite, gasping and abdominal distension. Symptoms began to appear in the flocks from the 4th day of feeding of the rations in question. Practically all diseased birds died within the period from the 3rd to 15th day after the beginning of symptoms.

About the same symptoms as those of the field cases were observed in the experimental cases administered toxic "dark-oil" or "Kanechlor 400" in the concentration between 300 and 100 ppm. On the other hand, when the broiler chicks were administered "Kanechlor 400" in the concentration of more than 600 ppm, all the cases died within 10 days showing no symp-

tom such as gasping or abdominal distension.

Pathology

Many birds manifested the anasarca mainly characterized by subcutaneous edema, hydropericardium, ascites, pulmonary edema, swelling and yellowish mottle formation of liver, thickening of proventricular wall, and thickening and hardening of keratinoid membrane of gizzard (Table 1). The same changes (Fig. 1) were also found in many cases of each experimental groups.

Table 1. Gross changes in field cases of PCB poisoning

Gross changes		Positive case number	(%)
Subcutaneous tissue	Edema	15	(40)
	Hemorrhage	3	(8)
Hydropericardium		21	(55)
Lung	Edema	9	(24)
	Dark reddish lesion	9	(24)
Air sacculitis		24	(63)
Ascites		11	(29)
Mottled appearance of liver		12	(32)
Swelling of proventricular wall		12	(32)
Hardening of keratinoid membrane of gizzard		22	(58)
Congestion of intestinal wall		19	(51)
Swelling of kidney		17	(46)

Fig. 1. Experimentally reproduced poisoning by administration of toxic "dark-oil" for 9 days. Slaughtered at 11 days of age. Remarkable hydropericardium is visible. Ascites and subcutaneous edema were also found

Subcutaneous edema was discovered in many cases at regions of the abdomen, inside of the thigh, thorax and back side of the neck. In some cases, subcutaneous edema was accompanied with ecchymosis progressing to intramuscular hemorrhage. Increased fluid in pericardial sac was generally straw-colored and clear, but the abdominal fluid was somewhat cloudy in some cases. In the experimental group fed on the ration including toxic "dark-oil" and slaughtered at 28 days of age, the severest case showed an increase of pericardial and abdominal fluid coming to 30 and 150 ml, respectively.

Such gross lesions as subcutaneous edema, hydropericardium, ascites, and swelling and yellowish mottle formation of the liver bore a close resemblance to those revealed in the chick edema disease occurred in the United States in 1957[3),4),6)]. A lot of studies have been conducted on the chick edema disease but the causal substance has not yet been reliably identified.

Except the lesions mentioned above, fading and swelling of the kidney and atrophy of bursa of Fabricius were recognized in some cases. Depression of weight gain was also observed obviously by the administration of "Kanechlor 400".

Histopathological changes of the field cases are represented in Table 2. Corresponding to the gross changes, anasarca was also found histopathologically almost all over the body; that is to say the skin, subcutaneous tissue to skeletal muscle, epicardium, interstitium of lung and serous membrane of the alimentary tract. A few of the field cases manifested a severe cell infiltration at the edematous lesion. Close by the edematous lesion, edematous change of blood vessel (Fig. 2) or fibrin thrombosis was often recognized.

As to the liver, vacuolar degeneration or zonal necrosis was revealed centrilobularly in several cases (Fig. 3). Hepatic necrosis was

Table 2. Histopathological changes in field cases of PCB poisoning

Histopathological Changes		Case number		(Positive percentage)
		Positive	Examined	
Skin	Serous dermatitis	3	21	(14)
	Edema	11	〃	(52)
	Hemorrhage	1	〃	(5)
Skeletal muscle	Serous myositis	1	20	(5)
	Edema	2	〃	(10)
	Hemorrhage	2	〃	(10)
Heart	Fibrinoid degeneration	5	36	(14)
	Granulomatous epicarditis	16	〃	(44)
	Epicardial edema	15	〃	(42)
	Heterophilic infiltration	12	〃	(33)
Lung	Catarrhal bronchopneumonia	3	38	(8)
	Interstitial serous pneumonia	9	〃	(24)
	Interstitial edema	31	〃	(82)
Liver	Necrosis	10	38	(26)
	Heterophilic infiltration	4	〃	(11)
	Hemorrhage	5	〃	(13)
	Activation of RES	8	〃	(21)
Alimentary tract	Serous serositis	3	37	(8)
	Serosal edema	9	〃	(24)
	Glandular epithel-desquamation of proventriculus	4	〃	(11)
Bursa of Fabricus	Degeneration of lymphoid cells	4	4	(100)
	Swelling of reticular cells	2	〃	(50)
Thymus	Degeneration of lymphoid cells	15	19	(79)
	Swelling of reticular cells	12	〃	(63)
Spleen	Atrophy of lymph follicles	12	28	(43)
	Activation of RES	14	〃	(50)
Kidney	Dilatation of renal tubules	26	37	(70)
	Cast formation	27	〃	(73)

often accompanied with hemorrhage. The reticuloendothelial cells were sometimes activated within or close by the necrotic foci. In some cases, bursa of Fabricius revealed the degeneration and disappearance of lymphoid cells accompanied with the hypertrophy and hyperplasia of reticular cells. Thymus revealed the same changes as in bursa. Such atrophic changes in these lymphoid tissues were also recognized in the spleen. In the kidney, slight cystic dilatation was produced in the superficial renal tubules in many cases.

Diagnosis

The above-mentioned clinical signs and pathological changes are thought to be quite characteristic for the PCB poisoning in chickens. But in order to make the exact diagnosis of PCB poisoning, the causal agent should be demonstrated from the diseased birds. Gas chromatography is usually used for this purpose. Many reports concerning the demonstration of PCB from wild birds or salt-water fish have been published recently. It is said that almost the whole world is polluted by PCB today.

Referring to the experiment results, it was concluded that the field cases in this report were caused by the take of rather high concentration of "Kanechlor 400", such as 300 to

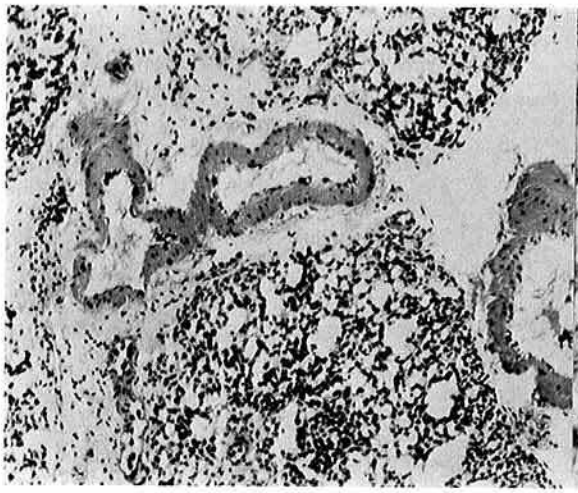

Fig. 2. Lung of a case administered the toxic feed for 21 days. Slaughtered at 52 days of age. The wall of arteries in the pulmonary interstitium undergoes edematous change. Hematoxylin and eosin (HB) stain, ×130

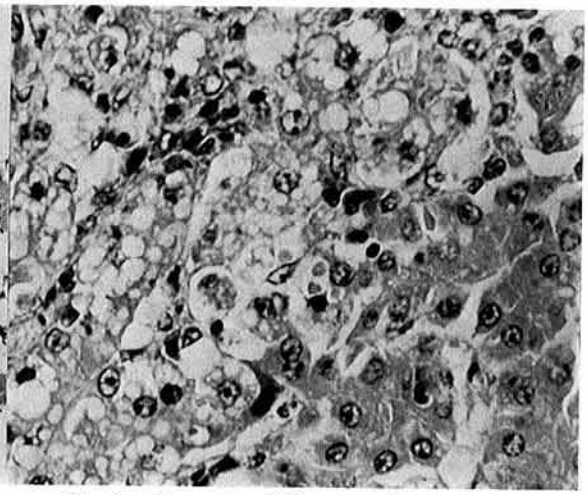

Fig. 3. Liver of a field case. Slaughtered at 38 days of age. Vacuolar degeneration of liver cells is remarkable in the centrilobular area. HE stain, ×500

100 ppm. There were no such obvious clinical signs or pathological changes when "Kanechlor 400" was administered experimentally in the concentration of less than 50 ppm during 22 days. The chronic poisoning of PCB produced by the lower-level and longer-term take should be investigated.

References

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