

## Peripheral Neuropathy of Dietary Riboflavin Deficiency in Racing Pigeons

Yoshihiro WADA, Hiroshi KONDO, and Chitoshi ITAKURA<sup>1)</sup>

Kamikawa Livestock Hygiene Service Center, 4-15 Higashitakaku, Asahikawa 071 and <sup>1)</sup>Laboratory of Comparative Pathology, Graduate School of Veterinary Medicine, Hokkaido University, Sapporo 060, Japan

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**ABSTRACT.** An occurrence of peripheral neuropathy in nine 14- to 55-day-old racing pigeons was documented. The predominant clinical signs were diarrhea, and leg and wing paralysis. Grossly, there was discoloration and swelling of all the peripheral nerve trunks. Microscopic lesions comprising swelling, fragmentation and demyelination of myelin sheaths, and proliferation of Schwann cells, were seen in the peripheral nerves of all birds examined. These changes were associated with moderate to severe swelling, fragmentation, atrophy and loss of axons. The peripheral nerve lesions in these cases were similar to those of dietary riboflavin deficiency in chickens. An analysis of the diet given to the pigeons indicated that the riboflavin concentration was only 0.9 mg/kg feed. — **KEY WORDS:** peripheral neuropathy, pigeon, riboflavin.

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Peripheral neuropathy due to dietary riboflavin (vitamin B<sub>2</sub>) deficiency has occasionally occurred in chickens [7, 8, 11], but has not been reported in pigeons. Since riboflavin was generally added to a poultry feed, the deficiency in chickens has been relatively uncommon. This note describes an occurrence of peripheral neuropathy in racing pigeons that had been fed a riboflavin-deficient diet.

A total of 119 pigeons (92 adults and 27 young) had been reared as a hobby by a farmer in Hokkaido, Japan. Out of them, 24 (8 adults and 16 young) showed clinical signs of diarrhea, and leg and wing paralysis from May 24 to June 20, 1991. These birds had been fed a diet formulated by the owner from hatching. An analysis of this feed indicated that the riboflavin concentration was only 0.9 mg/kg feed.

Seven 14- to 55-day-old pigeons showing severe leg paralysis and two 17- and 20-day-old dead ones were examined pathologically (Table 1). At necropsy, all the peripheral nerve trunks including the lumbosacral and brachial plexuses in all the birds examined were discolored and swollen approximately 2 or 3 times thicker than normal.

Histologically, variable degrees of peripheral neuropathy were found in all the nerves examined, including the sciatic and brachial nerves. On hematoxylin and eosin-stained sections, the lesions were mainly confined to the myelin sheaths of these nerves and consisted of swelling, fragmentation or presence of myelin globules, and demyelinations (Fig. 1). These changes were associated with swelling and proliferation of Schwann cells. Fragments of the myelin sheaths were stained positively with Luxol fast blue and Sudan III methods. On Bodian-stained sections of the peripheral nerves with degenerated myelin sheaths, axonal changes included mild to severe swelling, fragmentation, atrophy and loss of axons. There was considerable individual variation in the severity of the changes in both myelin sheaths and axons with age of the bird (Table 1).

Formalin-fixed sciatic nerves were embedded in Epon, from which 1  $\mu$ m thick sections were prepared and stained with toluidine blue. These sections showed degenerative changes consisting of the presence of myelin globules (Fig.

2). Teased, osmicated myelinated sciatic nerve fibers from selected cases showed granular breakdown and demyelination of the myelin sheaths (Fig. 3).

Concurrently, lesions of Newcastle disease were seen in 4 birds (Table 1), 2 of which were dead ones and had lesions associated with chronic bronchopneumonia and airsacculitis. The lesions of Newcastle disease consisted mainly of focal necrosis with astrocytic reaction in the cerebrum, scattered microglial nodules in the brain and spinal cord, and increased macrophages in the spleen. Newcastle disease virus was isolated from 2 of 5 cases examined.

Significant lesions in riboflavin-deficient birds are found mainly in the peripheral nervous system and are characterized by myelin degeneration accompanied with axonal alteration [7]. The peripheral nerve lesions in the present cases were similar to those in chicks suffering from riboflavin deficiency [7, 8, 11]. Jortner *et al.* [7] studied the sciatic nerves of chicks fed a riboflavin-deficient diet chronologically from 1 to 40 days of age. As a result, peripheral neuropathy was initially detected at 10 days of age, became more profound between 14 and 21 days, and then recovered with remyelination. Experimental riboflavin deficiency in chicks was induced by giving diets containing 1.8 to 2.5 mg riboflavin/kg feed [7, 8]. According to the standard nutrient requirements for chickens, the riboflavin concentrations should be 3.6 mg/kg from 0 to 6 weeks of age and 1.8 mg/kg from 6 to 14 weeks of age [10]. Although the feeding standard for pigeons is unknown, we have little doubt that the diet given to the present pigeons was deficient in riboflavin.

One characteristic clinical sign of riboflavin deficiency in chicks is "curled toe" paralysis [3, 5, 8, 11]. However, it does not always develop because the chicks die before it appears [12]. This sign was not observed in the present cases.

It should be noted that peripheral nerves are not involved in chickens [1, 6] and pigeons [9] infected with Newcastle disease virus. Focal necrosis in the cerebrum of the present cases resembled that in Newcastle disease in chickens [2] and was different from that in pigeons with thiamin deficiency [4].



Fig. 1. Sciatic nerve of case No. 4. Swelling, fragmentation, and demyelination of myelin sheaths with an increase in number of swollen Schwann cells. Hematoxylin and eosin stain.  $\times 500$ .

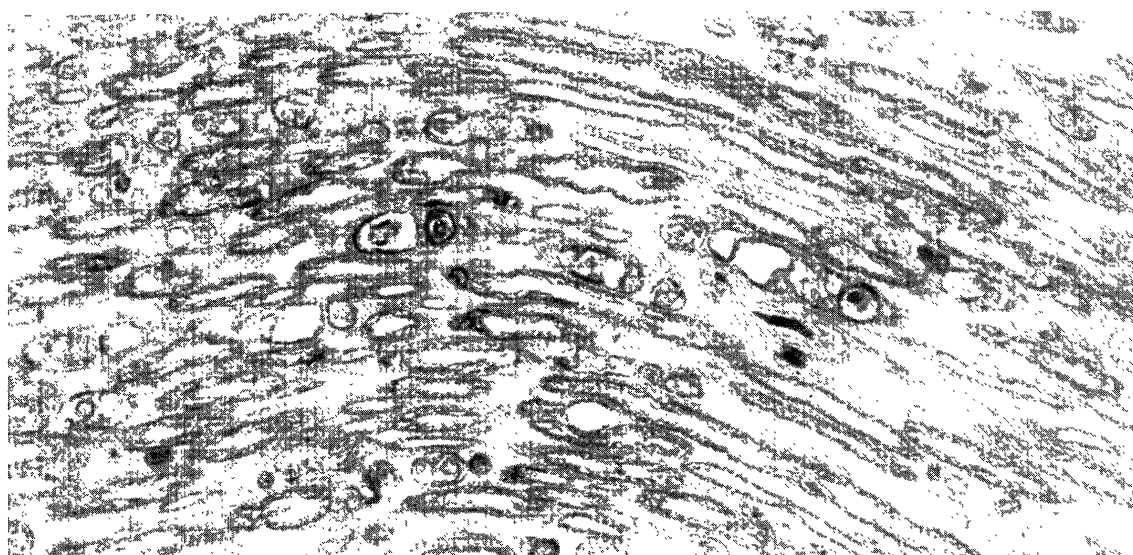


Fig. 2. Sciatic nerve of case No. 4. Many myelin globules in degenerative nerve fibers. Toluidine blue stain.  $\times 500$ .

Table 1. Main histopathological lesions in racing pigeons examined

Case No.	Age (day)	Termination <sup>a)</sup>	Neuropathy of sciatic nerve <sup>b)</sup>	Newcastle disease lesion in CNS
1	14	K	+	Yes
2	17	D	+	Yes
3	20	D	++	Yes
4	23	K	+++	Yes
5	25	K	+++	Yes
6	35	K	++	No
7	42	K	+	No
8	49	K	+	No
9	55	K	++	No

a) K; killed. D; dead.

b) Severity of lesion: +; mild. ++; moderate. +++; severe.

From the findings obtained here, it was concluded that the peripheral neuropathy in the present cases was caused by a riboflavin-deficient diet. Generally, pigeons are reared on a diet formulated by the owner. In such instances, riboflavin must be considered as an ingredient of nutrient supplements.

#### REFERENCES

- Alexander, D.J. 1991. pp. 496-519. *In: Diseases of Poultry*, 9th ed., Ames, Iowa State Univ. Press.
- Bhaiyat, M.I., Ochiai, K., Itakura, C., Islam, M.A., and Kida H. 1994. *Avian Pathol.* 23: 693-708.
- Chou, S. T., Sell, J. R., and Kondra, P. A. 1971. *Br. J. Nutr.* 26: 323-333.

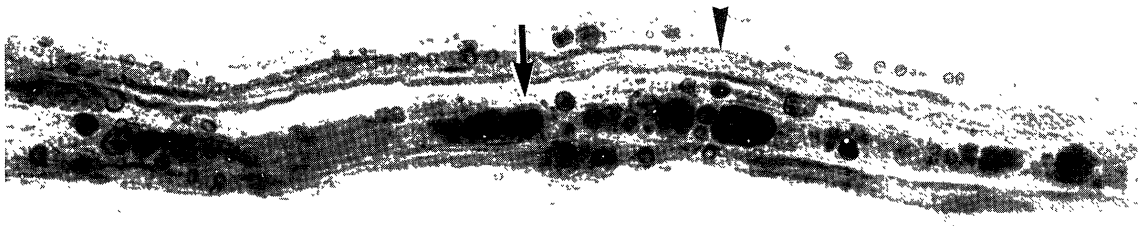


Fig. 3. Teased osmicated myelinated sciatic nerve of case No. 4. Marked myelin breakdown in a nerve fiber (arrow). An arrowhead shows an almost normal myelinated nerve fiber.  $\times 500$ .

4. Dreyfus, P. M. and Victor, M. 1961. *Am. J. Clin. Nutr.* 9: 414-425.
5. Gries, C.L. and Scott, M.L. 1972. *J. Nutr.* 102: 1286-1296.
6. Itakura, C., Yamagiwa, S., and Ono, T. 1971. *Jpn. J. Vet. Sci.* 33: 277-290.
7. Jortner, B. S., Cherry, J., Lidsky, T. I., Manetto, C., and Shell, L. 1987. *J. Neuropathol. Exp. Neurol.* 46: 544-555.
8. Jylling, B. 1971. *Nord. Vet. Med.* 23: 253-259.
9. Maeda, M., Koizumi, S., Yachi, M., Iwasaki, M., and Okubo, T. 1987. *Jpn. J. Vet. Sci.* 49: 217-223.
10. National Research Council. 1984. *Nutrient Requirements of Poultry*, 8th ed., Washington, National Academy of Sciences.
11. Phillips, P.H. and Engel, R.W. 1938. *J. Nutr.* 16: 451-463.
12. Wyatt, R. D., Tung, H. T., Donaldson, W. E., and Hamilton, P. B. 1973. *Poult. Sci.* 52: 237-244.