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AVIAN ENCEPHALOMYELITIS

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INTRODUCTION

VIAN ENCEPHALOMYELITIS (AE) is a viral disease characterized by leg weakness, incoordination, ataxia and tremors, especially of head and neck, generally affecting chicks of I to 5 weeks old. Besides chickens, pheasants are also susceptible. The disease can be experimentally produced in ducklings, young turkeys, pigeons and guineafowls (Van Rockel, 1965). The disease was first described by Jones (1932) in the U.S.A. followed by its occurrence in various parts of the globe. Rao (1965) and Mathur and Mukerji (1967) mentioned the occurrence of AE in India and suspected the presence of infection on clinical manifestation.

The present communication deals with the patho-anatomy of AE in natural and experimental chicks.

MATERIALS AND EXPERIMENTAL PROCEDURE

The source of infected materials was from day-old White Leghorn chicks received by a private farm. These chicks exhibited signs of illness and mortality, starting from second day of their arrival. Twenty chicks (a few of them were dead) were submitted to the pathology division for diagnosis. More birds were received from the same farm during the first week, second week and third week of April 1970. For experimental transmission, day-old chicks from a separate farm without any history of

this disease were used. A 10% saline brain suspension was made from infected chicks and intracerebrally inoculated into day-old and one-week-old chicks. Tissues from different regions were fixed in 10% formal-saline and processed for routine hematoxylin and eosin staining (H & E).

RESULTS

Clinical signs.—The main symptoms comprised of incoordination, bilateral ataxia with or without association of tremors in naturally infected cases (Fig. 1). Experimentally, clinical signs were produced after an average incubation period of 10 days (9-19 days) and comprised of incoordination, leg weakness and bilateral ataxia. Tremors of head and neck were occasionally associated with incoordination and ataxia but usually followed incoordination and ataxia. In a few instances the tremore were well marked and gradually decreased after 70 days post-inoculation (DPI). Mortality rate was low varying from about 10% to 21% of inoculated birds and usually occurs during 6 to 10th DPI. Number of chicks (dayold) showing clinical signs of the disease increased with serial passages (Table I).

TABLE I

Experimental transmission trials of AE

Passage No.	No. of chicks inoculated	Age of chicks in days	No. of chicks with clinical signs	Incubation period (DPI)
	32	1	7	20
ĨI	22	$\bar{1}$	11	10-18
III	30	7	9	13-17
īV	25	1	17	9-30
\mathbf{v}	10	1	7	9-19
VI	38	1	31	10-25
VII	2 l	1	21	8-23

Patho-anatomic changes.—No gross abnormalities were encountered in natural as well as in experimentally infected cases. Histopathological examinations of visceral organs of chicks from natural cases died during the first week of their life, revealed mild lymphocytic infiltration in a few birds. Microscopic examination of different regions of the brain and spinal cord showed presence of acute neuronal necrosis, especially marked in the region of pons-medulla and lumbar segments of the spinal cord (Fig. 2). Occasional areas of perivascular cuffings (Fig. 3) with lymphocytes and focal areas of gliosis were evident in a few cases.

Chicks examined after first week of their life with signs of pronounced leg weakness and ataxia exhibited moderate to marked infiltration of lymphocytic cells in the proventriculi and gizzard musculature, pancreas, heart, liver and kidneys. Lesions in the central nervous system (CNS) consisted of acute neuronal necrosis, areas of gliosis and perivascular cuffing. Perivascular cuffings were more pronounced in the brain whereas acute neuronal necrosis was prominent in the ventral horn of the spinal cord, especially in the region of lumbar spinal cord, just anterior to rhomboidal sinus.

Histopathological examinations of experimentally infected chicks revealed typical lesions in the CNS (Fig. 4) and visceral organs characteristics of AE. Lesions in the CNS (Figs. 5-6) were more pronounced than those seen in visceral organs. Inoculated chicks without clinical signs showed CNS lesions but mild visceral lesions when destroyed at variable periods between 15 and 25 DPI. Initially lesions of AE were evident in brain as early as 5 DPI, i.e., prior to the development of clinical signs.

A few birds destroyed after prolonged illness (54 and 113 DPI) revealed an occasional glial nodule in brain or spinal cord. Lymphoid foci were seen only in gizzard musculature, pancreas and in heart musculature.

DISCUSSION

The patho-anatomical changes in the CNS and visceral organs were indistinguishable from those described by various workers (Springer and Schmittle, 1968; Mohanty and West, 1968; Butterfield et al., 1969) for natural and experimental cases of AE. Tremors seen in experimentally inoculated cases usually developed after the onset of leg weakness, which indicates the late involvement of cerebellum. Histopathological changes in the cerebellum were suggestive of late involvement of this Mohanty and West (1968) observed region. similar changes in the cerebellum of experimentally infected chicks. Appearance of clinical signs within first week of the life of chicks in natural cases suggest egg transmission of the infection (Taylor et al., 1955), whereas variable incubation period in experimental cases corroborates our present findings in natural and experimental cases (Mohanty and West, Histopathological lesions in visceral 1968). organs in natural cases were more pronounced than experimental cases, while lesions in the CNS were more prominent in experimental cases. Butterfield et al. (1969) and Mohanty

and West (1968) recorded similar findings in natural and experimental cases of AE.

SUMMARY

An outbreak of Avian Encephalomyelitis (AE) was recorded in a private farm affect-

ing young chicks. Subsequent laboratory studies confirmed the disease on clinical, pathological and virological studies. The manifestations of the disease do not differ from those described in other countries.

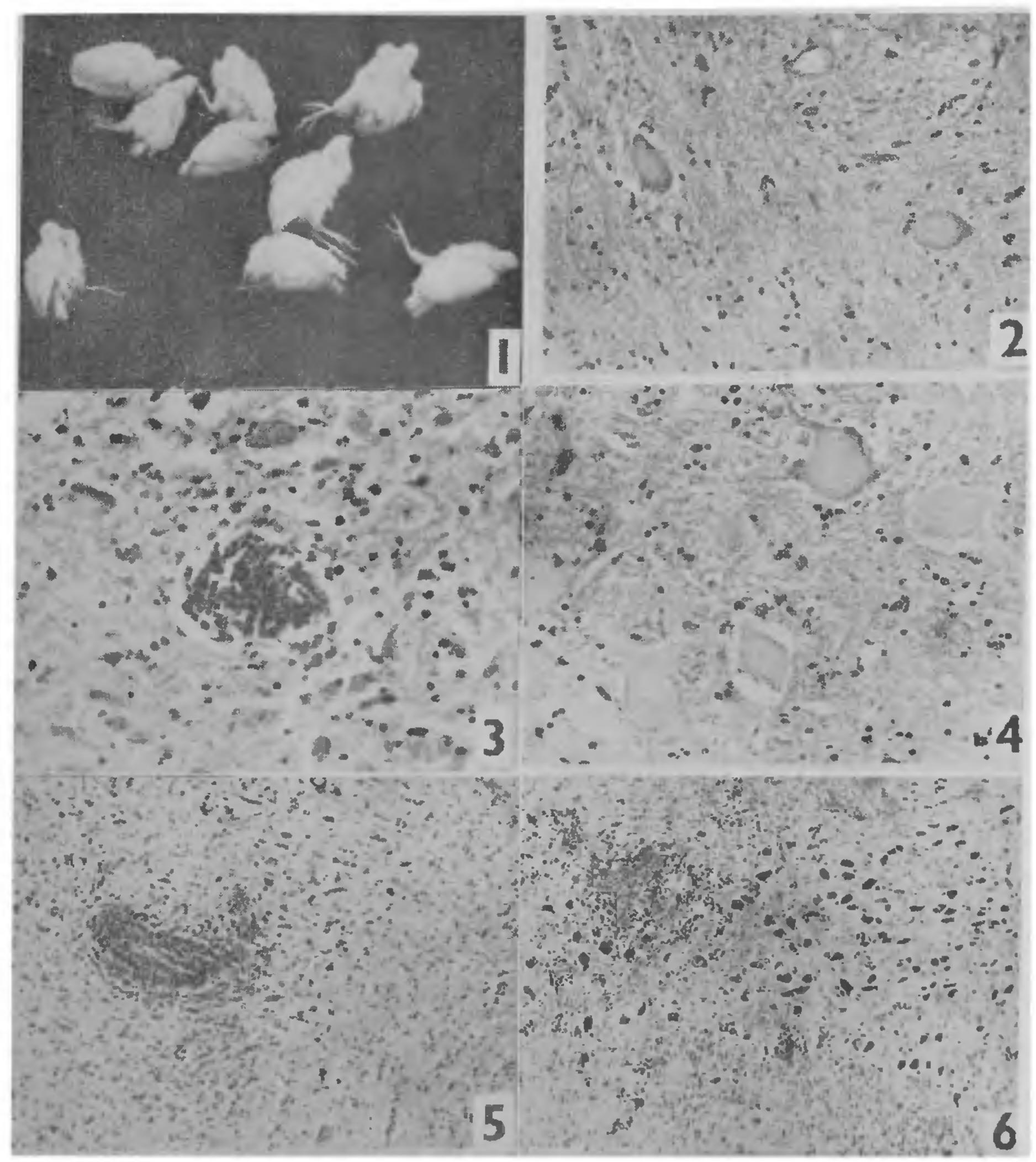


Fig. 1-6. Fig. 1. Avian Encephalomyelitis (AE) affected chicks (natural cases) showing bilateral ataxia. Fig. 2. Brain, Pons region of a natural case showing acute neuronal necrosis. If & E, × 260. Fig. 3. Lumbar region of spinal cord from a natural case of AE chick showing an area of perivascular cutting with lymphocytes. H & E, × 260. Fig. 4. Lumbar region of spinal cord from an experimentally inoculated chick with acute neuronal necrosis (13 days post-inoculation). If & E, × 310. Fig. 5. Lumbar segment of spinal cord from an experimentally inoculated chick showing an area of perivascular cutting with lymphocytes (20 days post-inoculation). If & E, × 120. Fig. 6. Brain, Pons region of an experimentally inoculated chick showing areas of perivascular cutting and gliosis (17 days post-inoculation). If & E, × 90.

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NUTRITIONAL QUALITY OF SOME POPULAR RICE VARIETIES

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India has increased from 21 million tonnes in 1951 to 39 million tonnes in 1965. The projected figure for 1971-72 is between 48-50 million tonnes. Only 17% of increase in the production has been attributed to an increase in area and the rest has been assigned to the increased production through better agronomic practices and the use of superior high-yielding photoinsensitive varieties.

Until very recently, Indian yields of rice per acre were among the lowest in the world. The first and foremost drawback was the plant-type itself. The subspecies indica, the traditional type of rice grown in Indian conditions, is characterised by tall growth habit, profuse number of narrow light-green drooping leaves, delayed flowering and late maturity. Though ideally suited to cloudy and rainy season, it succumbs to the modern high fertility farming. Additional fertilizer results in rapid vegetative growth and profuse leafing. These additional leaves mutually shade the plants and lead to the decreased photosynthetic efficiency in accumulating the carbohydrates. The planttype bottle-neck to higher yields has been broken through algeny2 and now we have a rice plant that is photoinsensitive, erect, short, has high capacity of tillering, matures early and possesses wide, dark and green leaves that remain photosynthetically active right upto the grain-filling stage. Twin characters of nonlodging and reduced mutual shading of the leaves have enabled it to stand very heavy doses of fertilizers to produce very high yields. It is in the background of this plant-type that

the nutritional aspects of the present and future rice varieties should be discussed. The composition of rice differs with plant-type, variety, soil conditions, manuring, location, soil moisture and weather conditions prevalent at the time of grain filling. A systematic investigation on these factors has just begun.³ Quantitative as well as qualitative characters are being considered in this context. From the studies, so far conducted, it could be concluded that among the above-mentioned factors, the genotype of the strains is most important.⁴

In the present study, an attempt was made to compare six popular rice varieties for their protein, carbohydrate and amino-acid composition. The length/breadth ratio and swelling number was also obtained to assess the cooking quality of these varieties. The data are presented in Tables I and II. In Table II, the reference aminogram for rice has been taken from Eggum (1968) for comparison.⁵ It is clear from the figures presented that with reference to protein content, there is significant difference between two distinct sets of varieties. Varieties Basmati-370, Sabarmati and Hamsa are having roughly 2% higher protein than Padma, Jaya and IR-8. These differences have been clearly discriminated by D.B.C., as well as microkjeldahl estimation. The starch properties as reflected by the amylose content are not widely different. However, if cooking quality is judged on the basis of swelling number and L/B ratio, it is evident that Sabarmati and Hamsa are similar to Basmati-370, therefore highly acceptable. Varieties Padma, Jaya and IR-8 seem to be inferior on the basis of these two criteria.